Taken from *Slayers, Saviors, Servants, and Sex: An Exposé of Kingdom Fungi* by David Moore; published by *Springer-Verlag, New York: 2001*, ISBN 0387950982.

Chapter 2 Blights, rusts, bunts and mycoses. Tales of fungal diseases

There's a notion gaining ground that food is the driving force of human history. Finding enough to eat today so that you stay alive into tomorrow is the mark of individual success. Finding so much today that you can afford to devote tomorrow to thinking is the start of civilization. Then the argument would run that tools were devised to improve some aspect of food gathering, so the origins of technology are also to be found in the prime directive 'Feed me!'

The main concern of early humans was clearly food gathering and in that exercise fungi most probably featured just like any other bounty of nature. Mushrooms are likely to have been collected for food alongside any other fruits and berries. I'm also very sure that the ability of yeasts on fruit left to one side to ferment the stuff into an alcoholic soup was a very early discovery indeed! But an early philosopher of even genius proportions would never have been able to recognize the influence of fungi in this miraculous event. However, when those early humans gave up their nomadic hunter-gatherer existence and turned to agriculture to solve the food problem they would rapidly have been challenged by the fungi. Early farmers must have learned very quickly that crops are very uncertain resources, prone to variations in weather, fire, floods, weeds, insect pests, and those upsets which came to be referred to collectively as 'blights' which were various sorts of plant disease.

Great crop losses can be suffered, caused by any of these factors, but by bringing the crops together into fields in the first place, the early agriculturalist created ideal conditions for the spread of plant disease. And the more selective his farming, the closer his crops came to being true monocultures, the greater the extent of agricultural losses due to any single agency like a particular plant disease.

Standing out among the examples of how damaging a crop disease can be is the Irish famine of 1845/46, which was caused by the failure of the potato crop in Europe because of just one plant disease, the Potato Late Blight. This is an astonishing story of how a crop disease affected the structure of our civilization and our understanding of Nature - while causing the deaths of one in eight of the Irish population. It is a story which goes far beyond statistics of number of deaths due to starvation, number of people emigrating, or crop losses and reduction in agricultural yield, and I will tell you that story in some detail. But it is a piece of our history which we must read about in the knowledge that even today world agriculture suffers significant losses due to plant disease, despite all our scientific advances of the past 150 years. Hopefully, in that time we have learned enough at least to avoid massive calamities like the Irish famine, and today's losses can be reported in terms of monetary losses. But behind each such statistic there must be personal tragedies in which the lives of individuals and families are changed dramatically.

The greatest losses suffered by agricultural crops today are caused by insect damage and plant diseases, with plant disease being the most damaging. Of course, there are more than fungi out there to cause disease. There are bacteria, viruses, and even nematode worms (eel worms), as well as fungi. There are serious plant diseases caused by all these other pathogens, but fungi probably cause the most severe losses around the world. For one thing there are more plant pathogenic fungi than there are plant pathogenic bacteria or viruses. One survey made several years ago in the State of Ohio came up with the estimate that the State had one thousand diseases of plants caused by fungi, one hundred caused by viruses and only fifty due to bacteria.

Agricultural survey statistics make it clear that crop losses directly attributable to fungi are very considerable. Of course, it's changing all the time because at least in part it depends on the weather, but it appears that world agriculture sustains average losses (in terms of monetary value) of around thirteen percent annually as a result of plant diseases. This overall average conceals instances of good news - with disease loss in the one to two percent range as well as bad news of a season of

unusually heavy disease incidence which might involve losses in the thirty to forty percent region. This is '21st century Agriculture' we're talking about, not some primitive agriculture of the distant past. Today, at this very moment, *on average* one in every eight crop plants will fail to yield because of fungus disease. That's a terrible tax on our activities to take into the shiny new millennium!

We lose more than mere money, too. A disease of the native American chestnut, Chestnut Blight (caused by an introduced parasite), effectively eliminated a stately and valuable timber and nut-crop tree from the United States. A similar loss, equally difficult to quantify because it is a loss of amenity as much as monetary value, happened in England when large elm trees were killed by Dutch Elm Disease (also caused by an introduced parasite, though this time the introduction was *from* the US and *into* Europe).

All groups of fungi, from the most primitive to the most advanced can cause serious plant diseases. For example, Late Blight of Potatoes and Downy Mildew of Grapes are diseases caused by the most primitive of fungi (so primitive, that many experts would not include them as *true* fungi at all), whereas Rusts and Smuts are diseases caused by members of the group of fungi which is the most advanced in evolutionary terms. Chestnut Blight, Peach Leaf Curl, Dutch Elm Disease, Net Blotch of Barley, Beet Leaf Spot, Apple Blotch, Maple Leaf Spot and thousands of other diseases are caused by all those fungi in between these extremes.

Talking about extremes brings us back to the Potato Murrain. The word 'murrain' is not in common usage these days, in fact my dictionary describes it as being obsolete. It's the word that was used at the time and the word stayed with the story as it passed into history. A 'murrain' is a great pestilence, a disease so widespread that it becomes a plague. The story I'm going to tell you deals with just such a terrible disease of the potato crop, and it should come as no great surprise, considering what the rest of this book deals with, that it's a disease caused by a fungus. Before developing the story, though, it's essential to put it into historical *scientific* context. The events mostly took place in 1845 and 1846. This was around twenty-five years *before* the great French microbiologist Louis Pasteur proposed his theory that diseases (of humans and animals) were caused by 'microbes' or 'germs'. The mid-nineteenth century had little concept of disease beyond its being an affliction. Your individual beliefs and prejudices might then determine your convictions about the cause of the affliction - a judgement from God, maybe, or the work of the Devil, or maybe that witch down the lane, or even him next door, you know, the one with the evil eye. Nobody at that time would immediately associate dying plants with an infection. If such thoughts are not part of your every-day 'world view', whether you are the King, Scientist or Pauper, then just what *can* you do when all the plants in your fields die?

The first report of what was to develop into such a calamity, can be found in a letter from Dr Bell Salter in the Isle of Wight to *The Gardeners' Chronicle and Agricultural Gazette.* In the edition dated August 16th, 1845 Dr Salter reported the arrival in England of a new malady of the potato. Matters progressed so rapidly that the distinguished editor of that journal, Dr John Lindley published in his editorial the next week, August 23rd, that: 'A fatal malady has broken out amongst the potato crop. On all sides we hear of the destruction. In Belgium the fields are said to have been completely desolated. There is hardly a sound sample in Covent Garden Market.'

The potato crop had been affected before - just like other crops. Damaged by too much rain in wet seasons, or by too little rain when there was a drought. Sometimes the tubers were scabby and inferior quality, sometimes the leaves curled up and the crop was reduced. But nothing as destructive as this new murrain had ever been seen before. Growing plants (remember this was in August - the summer season) looked like they had been badly affected by frost. Lindley's editorial

told the story: ‘The first obvious sign is the appearance on the edge of the leaf of a black spot which gradually spreads; the gangrene then attacks the haulms (stems), and in a few days the latter are decayed, emitting a peculiar and rather offensive odour. When the attack is severe the tubers also decay.’ Lindley recognized that if this ‘gangrene’ continued to spread, an important part of the country’s food supplies for the coming winter would be lost. He offered little hope for treatment, though, saying: ‘As to cure for this distemper there is none. One of our correspondents is already angry with us for not telling the public how to stop it; he ought to consider that Man has no power to arrest the dispensations of Providence. We are visited by a great calamity which we must bear.’

But who would bear the greatest calamity? As August became September reports of the spread of the murrain came from Poland, Germany, Belgium, France, and from all over England. Lindley’s fears were not exaggerated. Every strain of potato in cultivation was attacked. When they were dug from the field the potatoes were blotched with the dark patches of rotting flesh that were symptomatic of the disease. Patches coloured like bruised and battered human flesh. Patches that smelled of pestilence. And they spread. This was not something which ended when the crop was lifted. Leave potatoes on the floor of a barn for a few days and they became worse than when they were lifted. This was the unique character of the potato murrain. It spread amongst potatoes in the ground and *also* amongst those in store. The crop you might have thought safely harvested and stored could rot away in a few days; every tuber, no matter how slightly affected, would be lost. There is a record of a shipment of potatoes sent, routinely, by ship from the East coast seaport of Kingston-upon-Hull to be marketed in Belgium. Two thousand tons of potatoes left Hull in sound, palatable condition, but the entire cargo was rotten before the ship reached its destination just a few days later.

Then, on September 13th Lindley made the most dramatic announcement in *The Gardeners’ Chronicle*: ‘We stop the Press, with very great regret, to announce that the Potato Murrain has unequivocally declared itself in Ireland. The crops about Dublin are suddenly perishing ... where will Ireland be, in the event of a universal potato rot?’

The potato murrain affected the whole of Europe, but right from the very start, it was clear that the Irish peasants would suffer most. Loss of the potato crop in England would bring distress, of course, but the poorest laborers in England lived on oat gruel and bread as well as potatoes. The cereal crops were intact and so a major proportion of their normal diet was still available (no doubt at higher price - competitive pricing strategies, you know). In Ireland, on the other hand, the poorest members of the population lived almost exclusively on potatoes. On average four to seven kilograms of potatoes per day, per person, day in, day out, for ten months of the year. Over one hundred kilograms per family per week. The only break in the cycle occurred in July and August, the gap between the old and the new potato crops, when they had to subsist on wheat meal and anything else they could find. The population of Ireland had grown from four million in 1800 to over eight million in 1845, largely because of the ability of the potato plant to provide large crops of easily-stored tubers from small holdings on even relatively poor land. One acre of average land could produce six tons of potatoes each year. The tubers were easy to store over winter, so the potato had brought reliability of food supply to the poorest peasant farmers. It was clear to everybody that if the Potato Murrain spread through the small-holdings of Ireland there would be millions of men, women, and children, who would starve to death. And spread it did.

The crops of 1845 and 1846 were lost. In 1847 there was good weather and though there was a very much reduced acreage under potatoes in Ireland, the murrain was localized and relatively unimportant. With brilliant sunshine from July to September that year over the whole of Europe, harvests of grain and potatoes were good. The two worst years of famine were past but the misery went on in the shattered Irish countryside. Two years of famine were devastating. In the years from

1845 to 1860, one million people died in Ireland as a direct consequence of the famine, and over two million emigrated. Those that survived changed the world. Many headed for North America and the 'Paddies' became the labour force that built the foundation of the USA. They brought the Catholic Church to a position of prominence in a nation founded by Protestants. And now twenty percent of the US population claim Irish ancestry. They changed Ireland, too, creating a pattern of Ireland being an exporter of people. There's an estimate that in the hundred years between 1830 and 1930 fifty percent of those born in Ireland left the country to make their permanent home elsewhere. Ireland, now becoming a European 'Tiger economy' within the European Union, reported its first net gain in population since the famine in 1996.

The social upheaval caused by the Irish potato famine changed the demographic and political structure of the whole world and made an enormous contribution to the structure of the socio-economic civilization we enjoy today. It was caused by the potato murrain, so just exactly what *was* it?

The main intellects of the day were unable to come up with a cause, much less a cure. There was an official Commission of Enquiry which recommended some perfectly sensible methods of storage for *sound* potatoes. But the crop then being harvested was not sound and the tubers continued to rot in the stores. It's easy to be dismissive in retrospect but at the time it's difficult to see what other advice the government's scientists could offer. Probably, for the time, the best the Government of the day could do is what it did. Prime Minister Peel requested a survey of the state of the Irish potato crop. In response to this, on 16 September 1845 the Inspector General of Constabulary, a Mr D. Mc Gregor, issued a strictly confidential circular from the Constabulary Office in Dublin Castle which started: 'Information having reached the Government that the POTATO CROP of the present year has totally failed, from disease, in many districts of this Country, County and Sub-Inspectors of Constabulary are hereby directed to make full and immediate inquiries respecting the state of this Crop in their several Districts, and to report the result of such inquiries without loss of time ... These inquiries are not only to be regarded as confidential but they are to be so conducted as to prevent speculation on the possible motives for seeking the information required.' Over two hundred of these disease reports had been received by the end of September. The extent of the disease was quite clear. The impact was also evident. Sir William Wilde wrote in the *Dublin University Magazine* of 1854 that 'The late Bishop Brinkley, one of the most profound thinkers we have ever had in Ireland, who predicted the loss of the potato many years ago, and calculated mathematically the extent of ruin which was likely to follow, declared to his relative, the late Dr. Graves, that he was unable to sleep for an entire night, owing to the effect that it had upon him.' From conscientious gathering of information the government knew that about two million people were dependent entirely on the potato for food in Ireland and eight million pounds sterling were put into a relief effort in eighteen months between 1845 and 1847. That is about one pound sterling for every soul in Ireland at a time when the agricultural labourer earned about two pounds sterling per year. A colossal amount for the day, though the tragedy was so great that even this relief effort was overwhelmed.

As to the real cause, well, there were some hints. The peculiar changes of weather which had occurred during the summer of 1845 must have had much to do with the outbreak and spread of the potato murrain. The early part of the season that year was good; good for planting, good for early growth, so the crops looked really promising right up to July. In July the weather was hot and dry. The temperatures up to four degrees above the average for previous years. Somebody called Mr F. J. Graham recorded all this in an essay on the history of the murrain, which won a prize and was eventually published in the *Journal of the Royal Agricultural Society*. He went on: 'It then suddenly changed to the most extraordinary contrast that I ever witnessed in this fickle climate, the atmosphere being for upwards of three weeks one of continued gloom, the sun scarcely ever visible

during the time, with a succession of most chilling rains and some fog, and for six weeks the temperature was from 1.5 degrees to 7 degrees *below* the average for the past nineteen years.'

Dr Lindley theorized about the cause of the murrain, though his explanation was wrong. Lindley argued that it was the result of this cold and wet July weather causing the potato plants to become overladen with water. The man who got the explanation right, at the time, was the Reverend Miles Joseph Berkeley who was, in Lindley's words, 'a gentleman eminent above all other naturalists of the United Kingdom in his knowledge of the habits of fungi'. Berkeley associated the murrain with growth of a kind of mould in the affected tissues. He saw the damaged foliage himself in his parish, near King's Cliffe in Northamptonshire, and put forward the revolutionary theory that the mould was the cause and not the consequence of the potato murrain. That the murrain, in other words, was a disease.

Lindley argued that Berkeley was taking his interest in fungi too far, and that any mould that might be present was one of the '... myriads of creatures whose life could only be maintained by the decomposing bodies of their neighbours ...' The way in which moulds and mildews appeared, almost overnight, and how they reproduced was a great mystery at the time. Felice Fontana had examined Rust on wheat under a microscope in 1767, recognizing it as a minute vegetable with bodies resembling seeds. Indeed, in 1807 in France, Bénédict Prévost had actually seen the spores of the Bunt fungus on wheat germinating like seeds in water. Nevertheless, it was still commonly believed that small fungi could be produced in decaying matter by spontaneous generation. Robert Hooke published his *Micrographia* in 1667. He was the first person ever to use a compound microscope to describe the appearance of fungus growth. He examined a blue mould on some old leather and some rose leaves which had yellow spots on them. His opinion was that 'the blue and white and several kinds of hairy spots, which are observable on different kinds of putrify'd bodies are all of them nothing else but several kinds of small and variously-figured mushrooms, which from convenient materials in those putrefying bodies, are by the concurrent heat of the air, excited to a certain kind of vegetation'.

The belief of the day, therefore, was that putrefaction caused the appearance of the lesser organisms. The Rev. M. J. Berkeley was going against this core belief by making his suggestion. But he was in frequent correspondence with other naturalists in Europe so from their observations he was able to confirm that the same mould fungus was associated with this potato disease across the Channel. The same mould was on the diseased potatoes themselves. Though Berkeley had never seen this particular species of mould before, it resembled one he had seen growing on onions and shallots. It also appeared to be related to the fungus which was associated with a very serious disease of silkworms in Europe. The potato murrain, the Rev. M. J. Berkeley insisted, was due to the growth of this particular fungus, and no other, as a parasite on the potato plants. Berkeley called it a species of *Botrytis infestans*, today we call it *Phytophthora infestans*, the causal organism of the Late Blight of Potato. The cool, wet and very humid weather at the end of July favoured the spread of this moisture-loving fungus and the disease became an epidemic.

There is great scientific significance in Berkeley's ideas as to the most probable cause of the potato murrain. His was a new conception of the nature of disease. By advancing the hypothesis that a living parasitic organism on the potato foliage was the cause and not the consequence of the potato murrain the Rev. Berkeley was doing no less than anticipating the germ theory of Pasteur by almost twenty-five years. Of course, Pasteur *proved* it, Berkeley only suggested it. But it's a measure of the intellect that was at work in King's Cliffe in Northamptonshire late in the summer of 1845.

Sadly, despite all the varied intellects which were devoted to the potato murrain no cure or treatment emerged. We will never know what twists human history might have taken if some person

had remained alive at some stage, or some other person had succumbed. We will never know what human genius was withered away before full bloom, caught in the tragedy of illness, famine or mortal accident. But we do know that in the last weeks of July 1845 there was set in train a series of events that were to change the whole course of human history. And the agent was a fungus.

A sad postscript to this story is that one person of the day *did* have an effective way of saving the potato crop. It was widely known that the murrain started on the foliage of the potato plant and then made its way to the tubers. A Dr Morren in Belgium, and within two weeks of the first appearance of the murrain in 1845, pointed out that if the upper parts of affected plants were removed and destroyed, the tubers remained healthy. Small, perhaps, but healthy. If Morren's measure had been adopted, much of the potato crop could have been saved in the famine years.

There are an enormous number of plant disease fungi, so many that we could continue to catalogue them until Hell freezes over. Indeed, I've heard that there is a monastery somewhere in the Himalayas where the monks are listing all the names of plant diseases. When they've entered the last one in their list, the Universe ends and we start all over again. But next time mushrooms rule, OK?

There is just one other plant disease which I will tell about in some detail because of the general points that it shows up. It is Dutch Elm Disease. This tree disease first really came to notice in Holland in 1918 and 1919. Elms died soon after first showing symptoms and mature trees were lost in large numbers. For most people the immediate aftermath of the First World War in Europe was a more immediate concern, rather than this disease of elm trees. But there were numerous ideas as to its cause, including its being a side effect of poison gas from the war front. Dead and dying elms were not confined to sites where these fanciful explanations might apply, however. It came to be called Dutch Elm Disease because its true cause was established at that time by Dutch scientists. They found it was caused by a fungus with two different types of spore. Today we know the fungus as *Ophiostoma ulmi.* It was actually brought to Europe from the Dutch East Indies in Southeast Asia during the late nineteenth century. Symptoms of Dutch elm disease are symptoms of lack of water and nutrients - wilting. First, leaves droop and turn yellow. In a few days to weeks, the leaves are brown and dead, then larger branches begin to die and most trees are completely dead within two years. The reason for this progression is that the fungal cells plug the channels which normally distribute water and nutrients around the tree. As more channels are plugged the diseased tree is starved of food and water. When the tree dies the fungus will grow on the wood that remains. But there is more than a fungus involved. Left to itself, the fungus has difficulty passing from tree to tree. In this case, what turns a disease into a raging epidemic is a relationship between the fungus and several species of elm bark beetles. Adult females of these insects lay eggs in recently dead elms. Their eggs hatch and young larvae tunnel into the inner bark and outer wood to feed on it. If the tree has been killed by Dutch elm disease, the fungus sporulates in the beetle tunnels so the adult beetles that emerge are covered with fungus spores. These are transported to the first tender, young, healthy elm twigs that the young beetles bite into. Elm bark beetles, therefore, are vectors of Dutch elm disease.

Chinese and Siberian elms are highly resistant to the disease, but those native to North America are not. So the next step in the story is that Dutch elm disease was first found in North America in 1930 in Cincinnati, Ohio. The evidence indicates that it was introduced on elm logs from Europe that were landed at ports on the eastern seaboard. The American elm had become an important amenity tree throughout the continent, being planted in urban sites to such an extent that it's no exaggeration to describe the plantings as urban forests. But by 1950 the disease was spreading through seventeen states (and into south-eastern Canada). Today, Dutch Elm disease occurs wherever American elms grow in North America. Countless millions of trees have been killed, with a corresponding multi-

billion dollar cost of removing and disposing of them and replacing the trees that were lost with new plantings. But the story has yet another twist. In May 1963 a shipment of *American* elm logs was landed in the United Kingdom ... goodbye to twenty-five million British elm trees!

There is no cure so the key to control of Dutch elm disease is sanitation. Dead, dying, or weak elm wood must be destroyed to eradicate both fungus and beetle. It goes beyond the aerial parts of the tree, though. The roots of adjacent elms tend to fuse together over time, resulting in a shared root system between several trees. This type of root grafting may occur between elms within 50 feet of one another. When a single elm tree in such a group becomes infected, the fungus may move down the diseased tree into the roots and then into the next healthy tree through root grafts. The sanitation processes must include disruption of these root grafts by digging a trench two feet deep along a line around the beyond the longest branches of the diseased tree. Saving elms is a hard, costly job!

Fungi cause the majority of plant diseases, but they figure in only a minority of animal diseases. The fungus diseases of humans are called *mycoses* and the majority, perhaps all, are not caused by dedicated pathogens, but rather by fungi common in other situations taking advantage of a host weakened in some way or of a particularly advantageous set of environmental conditions. There are about one hundred different human fungal pathogens, together with several other species that cause allergic reactions. The fact that there are relatively few mycoses does not mean that they are rare. Far from it. What they lack in diversity, the human disease fungi make up for by being very widespread. It must surely be true to say that almost everybody suffers from Athlete's foot at some time in their life. This is caused by a tropical import called *Trichophyton rubrum*. You don't have to go to the tropics to collect your share because it so likes those nice warm and moist shoes we all wear that it is now distributed throughout the temperate climatic zones. Athlete's foot may be little more than a nuisance, which can be successfully treated with over-the-counter remedies.

Another remarkable statistic about mycotic disease is that it is now unusual for a woman to go through her reproductive years without at least one significant infection by the yeast *Candida albicans*. This is a normal inhabitant of the human mouth, throat, colon, and reproductive organs. Usually it causes no disease but lives in ecological balance with other micro-organisms of the digestive system. However, other factors such as diabetes, old age, pregnancy, but also hormonal changes, can cause *C. albicans* to grow in a manner that can't be controlled by the body's defence systems and candidiasis results, with symptoms ranging from irritating to life threatening.

We are prone to fungal invasion of the skin, nails and hair. Athlete's foot has already been mentioned, ringworm is another such infection, but is a family of mycoses because the cause is a fungus in one of two closely related genera, *Microsporum* or *Trichophyton.* Each fungus is very specific to a particular part of the body. Animals can also suffer ringworm diseases of skin and fur, and the fungi spread readily to humans.

We also breathe in a lot of fungal spores. Some of these may only challenge the immune system, but others find the lungs a good place to grow and can cause mycoses. *Cryptococcus neoformans* causes cryptococcosis; spores of the fungus are inhaled, begin to grow in the lungs and can enter the bloodstream and be carried throughout the body. It can cause cryptococcal meningitis which is usually lethal. The fungus grows and sporulates well on pigeon droppings. This bird is so common that most of us must go near a source of infection fairly often. Designers of buildings please note! Histoplasmosis is another human respiratory disease associated with exposure to bird droppings. It is caused by the fungus *Histoplasma capsulatum*. Inhaled spores colonize the lungs causing a disease similar to tuberculosis. Chicken farmers are the highest-risk group, but stay away from the roosts of large populations of starlings!

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Aspergillosis is a respiratory mycosis caused by about eight different species of the green mould, *Aspergillus*. Inhaled spores may cause asthmatic reactions but they may also grow within the lungs. The disease can be fatal if unchecked. *Aspergillus* species can produce enormous numbers of spores and they are common moulds (already mentioned in Chapter 1 as food contaminants that can produce toxins). The common name for aspergillosis, 'farmer's lung', suggests that the traditional victims are those who handle mouldy hay and grain (farmers and brewery workers) but faulty or dirty air conditioners can cause spore-laden environments in modern office buildings, so don't get too complacent!

And finally. The fungus *Coccidoides immitis* grows in desert areas throughout the Western Hemisphere and causes coccidiomycosis or 'Valley fever'. Inhalation of its spores infects the lungs. The young and healthy may suffer a slight cough, but it clears up. If the victim is not in the best of health, the infection can spread throughout the body and may prove fatal.

Health statistics indicate the sinister fact that mortality from fungal disease is on a steady increase against a background of steady decline in mortality caused by all other infectious agents. There are a number of reasons for this that are evident. There has been an increase in diagnosis of fungal disease (that is, not necessarily more disease, but more of what does occur is being diagnosed as of fungal origin). To some extent this reflects the introduction of techniques, especially molecular methods that can rapidly identify fungi. Secondly, increased availability of international travel has taken more people into the tropics, and tropical regions do seem to harbour more fungal pathogens. Thirdly, drug therapies used to manage the immune system in transplant and cancer patients have the unfortunate side effect of weakening the body's defences against fungal pathogens. A fourth reason is that AIDS patients have similarly weakened immune defences against fungi. Indeed, it is likely that most AIDS-related deaths are due to fungal diseases.

So what do we do about it? Across the board, whether plant or human diseases are concerned, the effort to counter the fungal menace must start with proper management: try to *avoid* giving the fungus any advantage. Effective management of plant diseases requires a good deal of information about fungal biology. Knowing the complete life cycle and infection conditions are important. Such knowledge may suggest control measures which target a vulnerable stage in the life cycle of the pathogen. The prime example of this is being the causal agent of Black Stem Rust of Wheat, which alternates between two hosts - wheat and barberry. Eradicating barberry plants growing in the vicinity of wheat breaks the cycle and protects the crop plant. Methods to control plant diseases aim at the pathogen itself, development of a host more resistant to disease, and attempts to use environmental features to reduce the effectiveness of the pathogen. The 'environmental features' do not have to be terribly grand. Procedures like crop rotation, improving ventilation by wide spacing of plants, arranging better drainage, can do a lot to provide conditions less favourable for the growth of the fungus. Measures aimed against the pathogen include quarantine and sanitation measures. The first aims to avoid introduction of disease to an area where it is not established; the second aims at eradicating diseased plant material from the vicinity of the crop to reduce the inoculum. Ultimately, of course, we have to resort to application of chemical agents to injure or kill the pathogen.

Control of fungal pathogens of humans is not so different in essence. Avoid infection, don't encourage the environmental conditions that fungi prefer, and fall back on a panel of effective fungicides. Chemical control of fungi in human disease is more difficult than control of bacterial diseases because the chemistry of fungal cells is so much more similar to that of human cells. A fungicide is toxic to the fungus, it may also be toxic to the host and do too much damage to the healthy cells of the patient. We need to understand the biological mechanisms which cause fungi to grow because this is fundamental to the development of treatments for fungal diseases in humans,

other animals and crops. With better knowledge of the fungal growth mechanisms we can develop more specific, and less environmentally damaging, treatments for fungal diseases. Unfortunately, we are not doing the research that is necessary. People interested in fungal biology are being pensioned off from Universities, research institutes and commercial companies around the world at the very time when we most need their expertise. The world wide market for treatments of human fungal diseases is worth approximately four billion US-dollars. It is divided into the three main sectors of over-the-counter remedies (the largest market sector), topical preparations (ointments and similar things that are put onto a particular part of the body) and systemic drugs (whole-body treatments given by mouth or injection). In 1995 the systemic sector accounted for sales of one and a half billion US-dollars and had a growth rate of fifteen percent per year because of the accelerating use of these drugs to combat fungal infection resulting from immune-suppressing therapies used during cancer treatments and transplant management, and in HIV/AIDS patients. The most successful antifungal drug is Pfizer's Diflucan (fluconazole). It was first launched in 1988 and by 1997 sales had reached nine hundred million US-dollars.

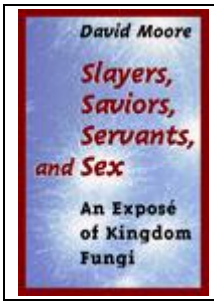
On the other hand, the world wide market for the use of fungicides in agriculture was estimated at almost six billion US-dollars in 1996, about fifty percent larger than the pharmaceutical market for antifungals. The two markets use similar chemical technology. Several products in both markets are derivatives of chemicals called azoles, which have two nitrogen atoms side-by-side. Azoles are relatively more toxic to the fungus rather than being non-toxic to the patient or host plant. Really, we do not have enough effective, broad spectrum antifungal drugs and there is growing concern about the appearance of resistance to those antifungals which are presently available commercially. In particular, we have not yet found a non-toxic antifungal or fungicide. Unfortunately, we are unlikely to find such a drug because of the lack of good scientific research in basic fungal biology.

Despite the colossal amounts of money invested in scientific research by industry, current corporate research strategies are aimed at corporate success not the advancement of fundamental science. This is very clear in the current concentration on screening programs. Most pharmaceutical and agrochemical companies are involved in these. They represent safe science and are wonderful things for managers and accountants to understand because they have aims ('find something that works in this test') and *measurable* objectives ('we must screen twenty thousand compounds this year because our competitor is screening fifteen thousand'). Science which is constrained by the profit motive is not 'good' *fundamental* science because it is aimed at doing little, if anything, to advance our knowledge or understanding. Recognize it for what it is: it is 'good' *commercial* science, aimed at providing sufficient marketable products to repay the megabucks that these programs absorb.

Even the much vaunted 'genetic engineering/cloning' (GM or genetic modification) programmes are not necessarily good science either. True, they are technically wonderfully competent and they provide tools that would enable us to unravel so many mysteries. But when GM programs are directed towards corporate success they have about as much to do with real science as the mechanic who repairs your PC has to do with designing the next generation of computer chips. Zilch. Nevertheless, that's where the research money is being spent because the manager/accountant can easily understand that a very good way to make lots of money is provided by the cycle (1) be sole supplier of pesticide, (2) use GM to introduce resistance gene into crop and be sole supplier of pesticide-resistant seeds, (3) make crop plant sterile somehow so that fresh seed is needed every season. Even politicians can understand that. So they invest large sums in the 'close to market research' to make it happen. It doesn't matter whether it is desirable or not (ethics is the responsibility of somebody else). It doesn't matter that it doesn't improve our overall understanding of biology (research scientists are being employed aren't they?). The driving criterion is that marketable products will be produced. And they *will*. Markets *will* be satisfied. Some problems *will*

Taken from *Slayers, Saviors, Servants, and Sex: An Exposé of Kingdom Fungi* by David Moore; published by *Springer-Verlag, New York: 2001*, ISBN 0387950982.

be solved. But if only a tiny fraction of the funding devoted to this sort of market-lead science was used to fund research on *organisms* rather than ever smaller bits of them, we could advance *understanding* sufficiently to find alternative ways, undreamed-of ways, of achieving the same ends. Yes, I know, I'm ranting again, aren't I?



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