**Fusarium: a ubiquitous fungus of global significance**

*Fusarium* is one of the most economically important genera of fungal plant pathogens, causing significant crop losses and contamination of grain by mycotoxins on a global basis. Some species also cause infections (mycoses) of humans and other animals. *Fusarium* includes many species, a significant number of which cause a wide range of plant diseases that affect many crops including major food and fibre crops such as wheat, barley, maize, bananas and cotton, often with devastating socio-economic impact. The diseases are often insidious and extremely difficult to control. Its success as a plant pathogen can be attributed to wide host ranges, endophytic infection, and varied modes of survival and dispersal. Representatives occur in virtually all bioclimatic regions of the world in agricultural and natural ecosystems. In this article we present a summary of the key aspects of the biology and morphology of *Fusarium* and then briefly discuss several plant diseases to illustrate the diverse nature and devastating effects of these fungi, their mycotoxins, the impact of no-till farming systems on disease incidence, and the poorly understood but key role of endophytic colonisation in the disease cycle. Inevitably, the coverage is selective but it indicates the potential global impact of this fungal genus on plant disease and food security.

**Biology and morphology**

*Fusarium* is a filamentous fungus producing thread-like hyphae that enable it to penetrate plant surfaces and ramify through (colonise) host tissues as primary or secondary invaders. Many species can colonise plants endophytically, an insidious process as it does not lead to symptom development, but contributes to a build-up in inoculum levels. However, stress may alter the relationship between a *Fusarium* endophyte and its plant host, leading to disease development, as discussed below. Many *Fusarium* species produce toxic secondary metabolites called mycotoxins that diffuse from the hyphae into the surrounding substrate such as grains or other infected tissues.

All species produce canoe-shaped spores called macroconidia formed in spore masses called sporodochia, from which they are splash-dispersed over short distances. Many species also form small spores called microconidia that are formed singly or in delicate chains. Microconidia of some species are splash-dispersed, whereas others are dry air-dispersed, presumably over long distances. Incredibly we know little about the dispersal of microconidia, or their role in the epidemiology of plant disease. Some species also form a sexual stage (structure) called a perithecium, from which ascospores are forcibly discharged into the atmosphere where they can be carried considerable distances. Some species also form thick-walled survival spores called chlamydospores, adapted to long-term persistence in soil.

Representatives of *Fusarium* are ubiquitous and are found in most bioclimatic regions of the world. In Australia, for example, we have isolated *Fusarium* species from soil and/or plants from tropical and temperate cropping areas, as well as savannah woodlands, hummock grasslands, and temperate and alpine grasslands. Natural ecosystems are reservoirs of genetic diversity of *Fusarium*, including newly recognised species, as well as known and potential pathogens. Indeed studies on *Fusarium* in natural ecosystems have contributed significantly to our understanding of the taxonomy, diversity, biology and ecopathology of *Fusarium* species.

*Fusarium* species have generally been referred to as soil-borne fungi based largely on studies of pathogenic species in traditional farming systems involving cultivation (tillage) and incorporation of crop residues. However, our more recent ecological studies in natural ecosystems, no-till farming ecosystems and tree crops in Australia and elsewhere indicate that many species normally infect and colonise above-ground plant parts especially stems or stalks, and are presumably dispersed as airborne spores, in seeds, other infected plant materials, and by insects. These species are not necessarily found in non-cultivated soils. The adoption of no-till farming practices, and particularly the retention of crop residues on the soil surface, has led to major changes in the ecology of many *Fusarium* species in agricultural ecosystems.
The remarkable diversity of plant diseases caused by *Fusarium* includes vascular wilt diseases, root, crown, stalk, head and cob roots of grain crops, growth distortion diseases, cankers, and storage rots of tubers, bulbs and corms (Table 1).

### Fusarium vascular wilt diseases

These diseases are caused by over 100 *formae specialae* of *F. oxysporum*. These pathogens persist in soil as chlamydospores, infect through the feeder rootlets and then colonise the vascular system, leading to severe wilting and death, depending on cultivar susceptibility. A *forma specialis* normally affects only one primary host species, but may endophytically colonise the roots of secondary hosts, so augmenting the population of chlamydospores in soil, an insidious process. Fusarium wilt of bananas, (Panama disease), caused by *F. oxysporum* *Esp. cubense*, illustrates the global importance of the vascular wilts. It devastated the export banana industries in Central America and the West Indies in the 1940s and 1950s before the resistant Cavendish type was introduced. More recently a new race, Tropical Race 4 (TR4), has wreaked havoc in South-east Asia and other areas, contributing, for example, to the collapse of the banana export industry in Indonesia. Fusarium wilt of cotton is another wilt of global importance. A new strain that appears to have evolved in Australia has had major socio-economic impacts in this country, causing serious losses and forcing many growers out of production.

### Fusarium diseases of cereals – pathology and toxicology

*Fusarium graminearum* is a versatile pathogen and endophyte of global significance. It has a wide host range, but is best known as the cause of Fusarium head blight (FHB) of wheat (Figure 1, C) and barley, and stalk, and cob rot (Figure 2), of maize. Indeed it has long been known that regular rotations of maize and wheat or barley can lead to serious epidemics of FHB and stalk and cob rot. The persistence of perithecia on crop residues, especially maize residues, is a critical phase in the disease cycle. The perithecia produce ascospores that initiate infection of the following crop. Consequently the adoption of no-till farming systems involving the retention of crop residues has led to a dramatic re-emergence of FHB in many regions even in the absence of maize. For example, FHB has had a devastating socio-economic impact on cereal growing communities in the USA, causing losses estimated at US$1 billion in 1993 alone. FHB reduces grain quality and yield, and may lead to contamination of the grain with high levels of the mycotoxin deoxynivalenol (DON) making it unacceptable for consumption by humans and farm animals. Furthermore, brewers will not accept barley with detectable DON, as it impairs fermentation. In epidemic years in China FHB may affect over 70 million ha, and continues to cause cob rot in maize (Figure 2). In contrast to wheat, *F. graminearum* produces two mycotoxins in infected maize kernels, DON, and zearalenone an oestrogenic-like compound that can cause infertility in animals. These examples illustrate the effect of substrate on mycotoxin production and the ability of some species to produce a number of different mycotoxins. Climatic conditions, especially temperature and available moisture are also important determinants of the nature and extent of mycotoxin contamination of infected grain prior to harvest and in storage. In Australia, the adoption of no-tillage practices in the eastern grain belt has also led to the emergence of FHB albeit at a low level in most areas. However, it has caused serious losses in localised areas of the Liverpool Plains region where wheat is grown in rotation with maize under centre pivot irrigation, or in the vicinity. The centre pivots have acted as foci or hotspots of infected residues, from where the fungus can be dispersed to more distant dryland crops by ascospores. In 1999 there were significant losses from FHB in such hotspots, and intermittent outbreaks since then. The occurrence of FHB was accompanied by grain contamination with DON, leading to rejection of grain at the silos. It is fascinating that *F. graminearum* can endophytically

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<td><em>F. circinatum</em></td>
<td>Pitch canker complex of <em>Pinus</em> species</td>
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<td><em>F. fujikuroi</em></td>
<td>Bakanae (foolish seedling) disease of rice</td>
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<td><em>F. sacchari</em></td>
<td>Pokkah boeng (crazy top) disease of sugarcane</td>
<td>Rugosity in leaves, internal stem necrosis, top necrosis</td>
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n.a. not applicable
infect and colonise the modern maize hybrids grown under near optimal conditions in these hotspots without causing any symptoms (Philip Davies, personal communication). However, abundant perithecia are produced on the infested maize residues within three months of harvest (Figure 1, B), providing a critical link in the disease cycle, as illustrated in (Figure 1). This is an example of the insidious nature of *Fusarium* pathogens.

*Fusarium verticillioides* also infects and colonises maize stalks endophytically but most modern hybrids are resistant to stalk rot unless subjected to severe moisture stress late in the season. However, the fungus continues to cause white cob rot in many parts of the world, especially in hot dry conditions. In Australia *F. verticillioides* has caused sporadic outbreaks of cob rot in maize in the eastern grain belt, invariably in crops subjected to moisture stress and/or other stressors late in the season. Some of these outbreaks have resulted in contamination of grain by the fumonisins group of mycotoxins, leading to toxicoses in horses, and pulmonary oedema in pigs. Indeed it is difficult to find maize anywhere in the world that does not contain at least traces of fumonisins that have also been linked to an increased incidence of oesophageal cancer and neural tube defects in humans in developing countries, especially in China and South Africa.

*Fusarium pseudograminearum*, a stubble-borne pathogen, causes crown rot of wheat and barley. This disease was first reported in Australia in 1966, but has since been recorded in many other countries where dryland wheat is grown under warm, semi-arid conditions. It is another insidious disease as the fungus can infect and endophytically colonise the crown (stem base) region of the plant without causing obvious symptoms under optimal conditions. However, hot dry conditions during anthesis and grain fill can lead to severe crown rot, which disrupts the uptake of water causing whitehead formation (Figure 3), heads containing shrivelled or no grain, the conspicuous symptoms of the disease. As this fungus persists in stubble (stem) residues it has emerged as a major problem in eastern Australia following the adoption of no-tillage practices and stubble retention. Crown rot has, for example, been estimated to cost the grains industry in Victoria alone A$11–16m per year, based on surveys over 1997–2009. The disease is particularly important in marginal areas where options for rotation are limited. However, in more reliable areas it can be virtually eliminated by regular rotation to crops such as sorghum, pulse crops and canola.

**Endophytic colonisation** is shown to have a role in the disease cycle of all of the diseases discussed above. The ability to infect and colonise a plant endophytically has significant ecological
advantages for a fungus as it maximises its substrate, and subsequently the level of infested residues, facilitating infection of the next crop, a remarkable adaptation. Furthermore, it is the basis for their insidious nature, a feature that has complicated their understanding by plant pathologists, agronomists and farmers alike!

**Global impact of Fusarium**

Globally, *Fusarium* has significant socio-economic and international trade implications for food security through its ability to devastate crop yields and contaminate plant products with mycotoxins. The impact will vary from country to country, a reflection of the major crops, agronomic practices and climatic conditions, factors that dictate the fungi that are present in a farming system and their activity. Furthermore, the level of food contamination coupled with local socio-economic factors and food cultural practices determine human exposure to mycotoxins. Some predictions indicate that *Fusarium* diseases and mycotoxin contamination of grain will increase with global warming. \(^{14,15}\) Furthermore new populations of *Fusarium* pathogens will continue to emerge through micro-evolution, and through incursion of exotic pathogens. Thus it is imperative that we maintain stringent quarantine measures to minimise the impact of such developments on the production of food, fibre and other natural plant products.

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**References**


**Biographies**

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