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Review

The Top 10 fungal pathogens in molecular plant pathology

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SUMMARY

The aim of this review was to survey all fungal pathologists with an association with the journal *Molecular Plant Pathology* and ask them to nominate which fungal pathogens they would place in a 'Top 10' based on scientific/economic importance. The survey generated 495 votes from the international community, and resulted in the generation of a Top 10 fungal plant pathogen list for *Molecular Plant Pathology*. The Top 10 list includes, in rank order, (1) *Magnaporthe oryzae*; (2) *Botrytis cinerea*; (3) *Puccinia* spp.; (4) *Fusarium graminearum*; (5) *Fusarium oxysporum*; (6) *Blumeria graminis*; (7) *Mycosphaerella graminicola*; (8) *Colletotrichum* spp.; (9) *Ustilago maydis*; (10) *Melampsora lini*, with honourable mentions for fungi just missing out on the Top 10, including *Phakopsora pachyrhizi* and *Rhizoctonia solani*. This article presents a short resumé of each fungus in the Top 10 list and its importance, with the intent of initiating discussion and debate amongst the plant mycology community, as well as laying down a bench-mark. It will be interesting to see in future years how perceptions change and what fungi will comprise any future Top 10.

INTRODUCTION

Recently, the journal *Molecular Plant Pathology* considered which viruses would appear in a 'Top 10' of plant viruses on the basis of their perceived importance, scientifically or economically, in terms of the views of the contributors to the journal (Scholthof *et al*., 2011). This was carried out as many papers, reviews and grant applications claim that a particular plant virus is of huge importance, and this is probably rightly so.

As a result of the interest generated in this exercise for plant viruses, a similar survey was instigated for plant-pathogenic fungi, and with a similar mechanism to the virus review. All authors, reviewers, editorial board members and senior editors of the journal *Molecular Plant Pathology*, with an interest in fungi, were contacted and asked to nominate three plant-pathogenic fungi they would expect to see in a list of the most scientifically/economically important fungal pathogens. The review, by its very nature, is similar in format and layout to the Top 10 virus review (Scholthof *et al*., 2011).

The survey generated 495 votes from the international community, and allowed the generation of a Top 10 fungal plant pathogen list for the journal *Molecular Plant Pathology* (see Table 1).

The fungus making the strongest appearance in the voting was *Magnaporthe oryzae*, in first place with almost double the number of votes of *Botrytis cinerea*, in second place. All voters highlighted the economic importance of *M. oryzae*; with over one-half of the world's population relying on rice as the main source of calories, this pathogen can have devastating effects; however, many also highlighted how this pathogen has developed into a model system for the study of plant–pathogen interactions.

Botrytis cinerea, in second place, clearly has an impact in many areas because of the broad host range, causing severe damage, both pre- and post-harvest. It may be one of the few entries in the Top 10 that can claim a potential beneficial use, through its role in some aspects of wine production, with some benefits also claimed for the ninth entry, *Ustilago maydis*, in infected cobs.

Puccinia spp., with many voters grouping the three wheat-infesting rust diseases together, are grouped together in third position. These fungal pathogens were already a serious disease

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Table 1 Top 10 fungal plant pathogens.

Rank	Fungal pathogen	Author of fungal description
1	*Magnaporthe oryzae*	Ralph Dean
2	*Botrytis cinerea*	Jan A. L. van Kan
3	*Puccinia* spp.	Zacharias A. Pretorius
4	*Fusarium graminearum*	Kim Hammond-Kosack
5	*Fusarium oxysporum*	Antonio Di Pietro
6	*Blumeria graminis*	Pietro Spanu
7	*Mycosphaerella graminicola*	Jason J. Rudd
8	*Colletotrichum* spp.	Marty Dickman
9	*Ustilago maydis*	Regine Kahmann
10	*Melampsora lini*	Jeff Ellis

The table represents the ranked list of fungi as voted for by plant mycologists associated with the journal *Molecular Plant Pathology*.

threat, but have increased in impact with the emergence of race Ug99, now posing a serious challenge to wheat production.

In fourth and fifth places are two *Fusarium* species, but with contrasting host ranges, with *F. graminearum* causing significant damage predominantly to cereals and a few noncereal species, and *F. oxysporum* having a wide host range, with severe losses in crops as diverse as tomato, cotton and banana. However, pathogens of cereals continue to have a significant presence in the Top 10 with *Blumeria graminis* in sixth position and *Mycosphaerella graminicola* in seventh.

Colletotrichum spp., in eighth position, are members of an important genus of plant pathogens, for which the taxonomy may be described as being in a state of flux. The proposed number of species within the genus ranges from 29 to over 700 depending on taxonomic interpretation. *Colletotrichum* spp. have long served as a model system for hemibiotrophic pathogens, with a short biotrophic stage, followed by a switch to tissue ramification and necrotrophic development.

It is interesting that *Ustilago maydis* and *Melampsora lini* make up ninth and tenth positions, respectively, with many voters indicating strong scientific rather than economic reasons for their inclusion. Both are now developed as model systems, which are readily tractable, and provide vital insights into the molecular basis of plant immunity and infection processes.

Although the aim of this review article was to identify the Top 10 most important plant-pathogenic fungi according to the contributors to *Molecular Plant Pathology*, we are very much aware that importance and priorities can vary locally across continents and disciplines. We are also aware that not all fungi can make it into any Top 10, because of obvious numerical limits, although such fungi can still be regarded as hugely important. We therefore felt it appropriate to cite honourable mentions for fungi just missing out on the Top 10 list, including *Phakopsora pachyrhizi* (Goellner *et al*., 2010) and *Rhizoctonia solani* (Gonzalez *et al*., 2011; Vilgalys and Cubeta, 1994), both clearly important. In future years, when another survey of the Top 10 is carried out, it will be interesting to see whether fungi such as *Phakopsora pachyrhizi*, the causal agent of Asian soybean rust, makes an appearance, as it is clearly a disease that has only recently appeared in certain parts of the world and will potentially increase in importance (Goellner *et al*., 2010).

This review contains single-page descriptions of the Top 10 plant-pathogenic fungi to introduce the reader to each of them, with illustrative figures and key references for further reading. The intention of this review is to trigger discussion and debate amongst the plant mycology community, as well as to lay down a bench-mark, as it will be interesting to see how perceptions change in future years and which fungi enter and leave the list.

1. *MAGNAPORTHE ORYZAE*

Magnaporthe oryzae, a filamentous ascomycete fungus, is the causal agent of rice blast disease, the most destructive disease of rice worldwide (Ou, 1980). Its importance is underscored by the fact that approximately one-half of the world's population relies on rice for its primary caloric intake (Khush, 2005). All foliar tissues are subject to infection; however, infection of the panicle can lead to complete loss of grain. Losses of 10%–30% are typical, although regional epidemics can be devastating. In addition, the fungus is highly amenable to genetic and molecular genetic manipulation (Jeon *et al*., 2007; Talbot, 2003; Valent and Chumley, 1991). Consequently, as a result of its agronomic significance and tractability, *M. oryzae* has emerged as a seminal model in the study of host–fungal pathogen interactions (Dean *et al*., 2005). Although host resistance is the most economically viable and environmentally sound practice to manage this disease, the fungus overcomes blast resistance quickly, and cultivars typically become ineffective within 2–3 years (Ou, 1980; Zeigler *et al*., 1994). *Magnaporthe oryzae* is part of a species complex that can cause disease on a variety of grasses and related species, including crops such as barley, wheat and millet (Couch *et al*., 2005). New wheat strains have emerged in South America, elevating concerns that the fungus poses a serious threat to global wheat production (Urashima *et al*., 1993). Knowledge of the biology, genetic diversity and adaptability of this pathogen is key for the development of novel and durable strategies to manage this and related devastating fungal diseases (Fig. 1).

The discovery of Guy11, a fertile Mat1-2 strain from French Guiana, more than 20 years ago stimulated a surge in scientific interest in the rice blast pathogen (Leung *et al*., 1988; Silué and Notteghem, 1992). The ability to perform genetic analyses led to the cloning and characterization of several genes for host and cultivar specificity (Bryan *et al*., 2000; Jia *et al*., 2000; Orbach *et al*., 2000; Valent *et al*., 1991). Today, much effort is focused on cloning and characterizing the many avirulence and corresponding rice resistance genes that have been genetically identified (Chao and Ellingboe, 1991; Skamnioti and Gurr, 2009). The development of genetic markers, such as MAGGY, MGR583 and MGR586, from repetitive elements has provided the means to not only create valuable genetic maps for *M. oryzae*, but also the molecular tools to assess population diversity and the evolution of lineages, valuable knowledge for the breeding and timely release of resistant cultivars (Farman *et al*., 1996; Hamer *et al*., 1989; Nitta *et al*., 1997; Skamnioti and Gurr, 2009).

The early stages of infection have been studied in great depth in *M. oryzae*. Like many fungal plant pathogens, *M. oryzae* elaborates an appressorium, a specialized infection cell (Howard and Valent, 1996). Appropriate development of this cell is required for infection; indeed, several effective fungicides, such as tricyclazole, which inhibit melanization of the appressorium, block host penetration (Woloshuk *et al*., 1983). The ability to induce appressoria *ex planta* and to perform functional analyses of genes by targeted gene knockout has provided powerful access to many of the underlying molecular processes (Leung *et al*., 1990; Oh *et al*., 2008). Considerable knowledge has been acquired regarding the perception of environmental cues (Lee and Dean, 1994), starvation responses (Donofrio *et al*., 2006), cell signalling pathways (Lee and Dean, 1993; Nguyen *et al*., 2008; Xu and Hamer, 1996), turgor pressure generation (deJong *et al*., 1997), recycling cellular contents (autophagy) (Veneault-Fourrey *et al*., 2006) and cell cycle checkpoints (Saunders *et al*., 2010) which regulate and orchestrate the development of this specialized cell (Fig. 2).

Over the last 6 years, following the publication of the genome sequence for strain 7-15 (a derivative of Guy11) (Dean *et al*., 2005), genomic resources for *M. oryzae* and related species have grown dramatically and presently include more than 30 genomes from strains around the globe, as well as various transcriptome datasets [expressed sequence tags (ESTs) and small RNAs] (Gowda *et al*., 2006; Nunes *et al*., 2011; Oh *et al*., 2008) and emerging proteome datasets. Early examination of these data revealed dramatic differences in gene content and organization of repetitive elements. Other studies have revealed fundamental insights into host defence and gene silencing mechanisms (Ikeda *et al*., 2002; Nguyen *et al*., 2008). With the emergence of new sequencing technologies, population biology studies and phylogenomic analyses of *M. oryzae* are poised to grow significantly in the very near future, providing deep insight into the evolution of phytopathogenesis.

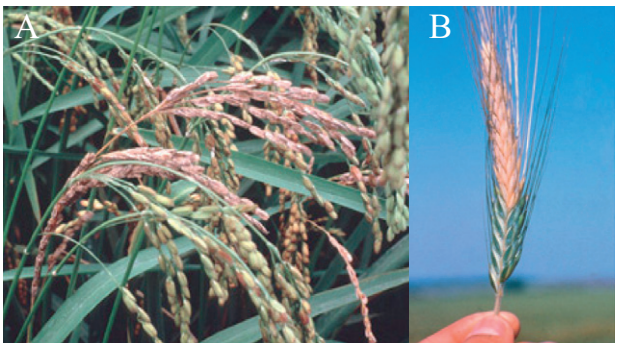

Fig. 1 Disease resulting from the infection of rice and wheat with *Magnaporthe oryzae*. (A) Classical symptoms of panicle blast on rice, although the fungus can cause disease on all foliar tissues. (B) Head blast on wheat. The symptoms on wheat are typically restricted to the head and can be mistaken for wheat scab caused by *Fusarium graminearum*.

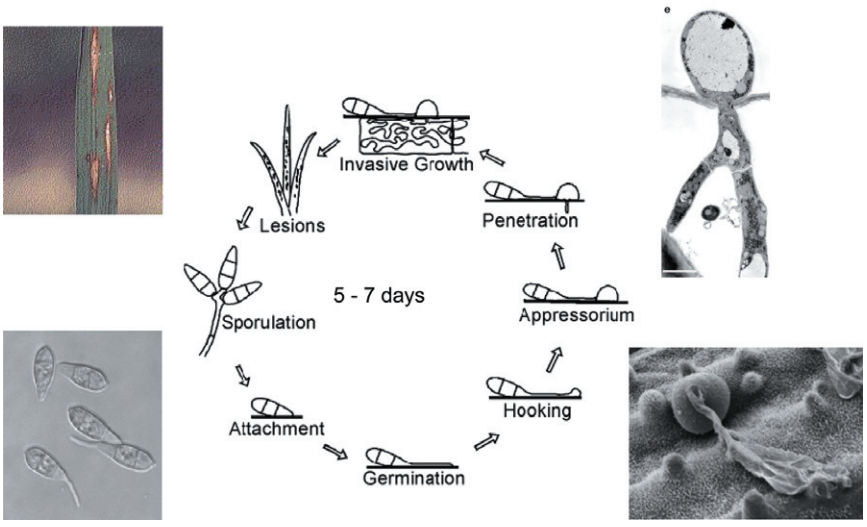

Fig. 2 Infection cycle of the rice blast fungus. Conidia produced from lesions are splashed onto new plants where they attach firmly and germinate within a few hours. Subsequently, the germ tube ceases polar growth, the tip swells and, by 12 h, forms a highly melanized dome-shaped structure, the appressorium. Typically within 24 h, turgor pressure increases within the appressorium, forcing a penetration peg into the underlying tissues. Eye-shaped necrotic lesions do not appear until several days after infection, from which, under appropriate conditions, conidiophores emerge bearing conidia to re-initiate the infection cycle. (Scanning electron microscope image in bottom right courtesy of Nicholas Talbot and Michael Kershaw, University of Exeter, UK). [Correction added on 5 July 2012, after online publication: The bottom right image has been amended and acknowledgement has been included in the legend].

2. *BOTRYTIS CINEREA*

Botrytis cinerea Persoon: Fries [teleomorph *Botryotinia fuckeliana* (de Bary) Whetzel; Fig. 3], known as grey mould, can infect more than 200 plant species. The fungus is considered as a typical necrotroph, which co-opts programmed cell death pathways in the host to achieve infection (van Baarlen *et al*., 2007). The genome sequences of two *B. cinerea* strains failed to identify 'silver bullets', unique features which distinguish it from other pathogenic and nonpathogenic fungi (Amselem *et al*., 2011). The availability of the genome sequence and a variety of molecular tools [ease of transformation to obtain knockout mutants (van Kan *et al*., 1997) or to achieve gene silencing (Patel *et al*., 2008, 2010)], together with its economic relevance, have contributed to *B. cinerea* being the most extensively studied necrotrophic fungal pathogen. These studies have considerably advanced our understanding of the infection strategies of *B. cinerea*, yet only very few absolutely essential virulence determinants have been identified by candidate gene approaches (Tudzynski and Kokkelink, 2009).

Botrytis cinerea is most destructive on mature or senescent tissues of dicotyledonous hosts. This fungus sometimes remains quiescent for a considerable time before rotting tissues when the host physiology changes and the environment is conducive (Williamson *et al*., 2007). Infestation can occur all the way from the seedling stage until product ripening. Serious damage can occur following the harvest of seemingly healthy crops. Harvested commodities can be spoiled in the retail chain, during storage, transport to distant markets or during display at the retailer (Fig. 4).

The costs of *Botrytis* damage are very difficult to estimate because of the broad host range. Costs are diffuse as *Botrytis* damage occurs in different stages of the production and retail chain. No reliable figures are available for *Botrytis* damage during crop cultivation, yet it must be huge. In spite of the increasingly effective application of biocontrol in some crops (Moser *et al*., 2008), fungicide application remains the common method to control *Botrytis*. The average cost for chemical *Botrytis* control (all crops, all countries) is about €40/ha (Steiger, 2007). Fungicides specifically targeted against *Botrytis* ('botryticides') cost €540 million (2001), representing 10% of the world fungicide market (UIPP, 2002). The wine and table grapes segment represents 50% of the value of the total market for botryticides, with solanaceous vegetables, cucurbits, strawberries and ornamentals each making up 5%–9% (Steiger, 2007). The costs of broad-spectrum fungicides also effective against *B. cinerea* are unknown. Fungicide resistance is an increasingly problematic issue, with significant proportions of the fungal population being resistant to fungicides (Leroch *et al*., 2011). Multidrug resistance has been reported and is mostly caused by the increased expression of ABC transporter genes (Kretschmer *et al*., 2009).

In wine and table grapes, expenses for the control of *Botrytis* bunch rot are a major cause of reduction in profit in Australia (AUS $52 million/year; Scholefield and Morison, 2010), Chile (US$ 22.4 million/year; Esterio *et al*., 2009) and South Africa (SA Rand 25 million/year). Estimates for other countries could not be obtained. These expenses predominantly include the chemicals, but do not cover the losses experienced by the lack of treatment (organic farming, low-input farming), failed treatment (fungicide resistance) or quality loss (fungicide levels exceeding export requirements; grape quality insufficient for high-quality wines).

In 2002, about 15%–20% of rose and gerbera bunches traded through Dutch flower auctions contained detectable *Botrytis* infection, leading to vase life reduction of such bunches by 3 days. The loss in revenue for rose growers alone was estimated at €1.3 million (Vrind, 2005). Such figures do not include flowers that were infected before harvest and did not make it to the auction, or flowers that were spoiled during transport to retailers and became unsellable. Reduction of the shelf life (fruit) or vase life (flowers) is a serious quality issue. Consumers experience economic loss when berries rot before they are eaten, or when roses do not last for a satisfactory period. From an economic perspective, the loss experienced by consumers is often disregarded because producers and retailers have obtained income. However, dissatisfied customers may refrain from purchasing the product again for several weeks, an effect difficult to incorporate into estimates of economic losses. Official figures of the costs of grey mould in soft fruit and ornamentals must therefore be considered as underestimates.

Botrytis cinerea is an exceptional pathogen, as it may occasionally be beneficial! Under specific climatic conditions, *B. cinerea* may cause noble rot in grape berries, which are used to produce sweet wines (Sauternes, Tokaj). The most prestigious *Botrytis* wines are sold for prices up to €500/bottle. Nevertheless, the overall impact of *B. cinerea* is negative, even in the wine industry.

Altogether, global expenses of *Botrytis* control (cultural measures, botryticides, broad-spectrum fungicides, biocontrol) easily surmount €1 billion/annum. The impacts of product loss occurring despite disease control, and the quality loss during the retail chain, are likely to be far higher.

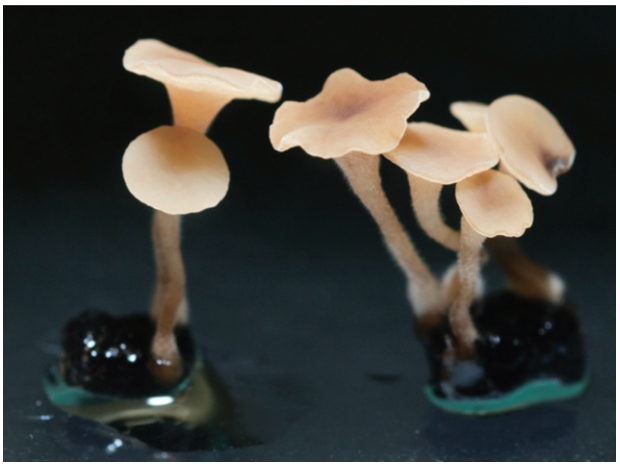

Fig. 3 Sexual fruiting bodies (apothecia) of the teleomorph *Botryotinia fuckeliana* (de Bary) Whetzel.

Fig. 4 *Botrytis cinerea* on raspberry fruits (from Williamson *et al*., 2007).

3. *PUCCINIA* SPP.

Three rust diseases occur on wheat, namely stem (black) rust (caused by *Puccinia graminis* f. sp. *tritici*) (*Pgt*) (Fig. 5), stripe (yellow) rust (*P. striiformis* f. sp. *tritici*) (*Pst*) (Fig. 6) and leaf (brown) rust (*P. triticina*) (*Pt*). Prolific sporulation, efficient dissemination, pathogenic variability and the widespread cultivation of wheat, often in conducive environments, all contribute to the destructive potential of these rusts. Historically, stem rust has been most notorious for damaging wheat crops. The disease was feared in ancient Rome where rituals ('Robigalia') were performed to save crops from rust (Zadoks, 1985).

Pgt, *Pst* and *Pt* are obligate, biotrophic basidiomycete fungi with macrocyclic, heteroecious life cycles (Bolton *et al*., 2008; Jin *et al*., 2010; Leonard and Szabo, 2005). Obligate biotrophs differentiate specialized infection structures, efficiently suppress host defence responses and obtain nutrients through the formation of specialized feeding structures, called haustoria, which are situated inside plant cells (Voegele and Mendgen, 2011).

Stem rust is frequently perpetuated by repeating uredinial stages on common and durum wheat, barley and triticale. However, basidiospores can infect alternative hosts, such as *Berberis vulgaris*, furnishing primary inoculum for wheat and new virulence combinations as a result of sexual re-assortment of genes (Jin, 2011). The barberry eradication programme in the USA between 1918 and 1980 (Roelfs, 1982) and in the UK, which also started in 1918 and continues today, must be viewed as one of the major achievements in plant pathology, in both scope and disease management.

Significant and repeated crop failures caused by rusts occurred in North America between 1904 and 1962 (Hodson, 2011; Roelfs, 1985). Serious epidemics also occurred in Europe and China (Leonard and Szabo, 2005), with less frequent outbreaks in Eastern Europe, India, Australia, Mexico, Chile, Ethiopia and South Africa (Hodson, 2011; Pretorius *et al*., 2007). In many instances, exotic incursions of wind-borne spores have occurred, establishing new race lineages (Park, 2007; Singh *et al*., 2011). Recently, severe and widespread stripe rust epidemics have been ascribed to new and more aggressive races adapted to warmer environments (Hovmøller *et al*., 2011). The elucidation of the complete *Pst* life cycle (Jin *et al*., 2010) and the genome sequence of this pathogen (Cantu *et al*., 2011) are significant steps towards a better understanding of virulence, and thus breeding for durable resistance.

Early stem rust epidemics initiated studies on pathogenic variability, epidemiology and host–pathogen genetics in *Pgt* (reviewed by Loegering, 1984 and Roelfs, 1985). The specialization of *Pgt* in different races has impacted strongly on wheat breeding and production. Numerous cultivars protected by single genes have become susceptible to stem rust, often with devastating 'boom-and-bust' effects (e.g. Jin, 2011; Kolmer *et al*., 2007; Martens and Dyck, 1989; Park, 2007; Pretorius *et al*., 2007).

The occurrence of race Ug99 of *Pgt* in East Africa with virulence for *Sr31* (Pretorius *et al*., 2000), a commonly used resistance gene, has renewed stem rust research (Singh *et al*., 2011). Seven variants within the Ug99 lineage have been reported, varying in virulence for *Sr21*, *Sr24*, *Sr31* and *Sr36* (Singh *et al*., 2011). As a result of its adaptive capacity, fitness and virulence attributes (90% of the world's wheat is susceptible), the Ug99 race group has been recognized as a major threat to food security (Flood, 2010; McIntosh and Pretorius, 2011; Singh *et al*., 2011; Vurro *et al*., 2010).

Together with progress in the detection, genetic mapping and management of genes and quantitative trait loci (QTL) conferring resistance to Ug99 (Durable Rust Resistance in Wheat Project, http://www.globalrust.org), significant advances are being made in understanding the molecular basis of pathogenicity in *Pgt* (Duplessis *et al*., 2011). Continued research on surveillance and race analysis, coupled with pathogen genomics, will enable the discovery, characterization and utilization of sustainable management strategies for resistance. The release and adoption of widely adapted resistant cultivars are essential for future and effective rust control globally (Lowe *et al*., 2011; McIntosh and Pretorius, 2011).

Fig. 5 Stem rust-infected wheat showing uredinial and telial spore stages.

Fig. 6 Wheat flag leaf infected with stripe rust caused by *Puccinia striiformis* f. sp. *tritici*.

4. *FUSARIUM GRAMINEARUM*

The ascomycete *Fusarium graminearum* (teleomorph *Gibberella zeae*), which resides in the order Hypocreales, is a highly destructive pathogen of all cereal species (http://www.scabusa.org). Locally, *F. graminearum* co-exists and co-infects with other *Fusarium* species. The greatest economic losses occur when the floral tissues become infected (Fig. 7). This disease mainly reduces grain quality, rather than lowering grain yield, and results in mycotoxin-contaminated grain. Worldwide, all the major cereal-growing regions have reported a re-emergence of *Fusarium* epidemics (Leonard and Bushnell, 2003). During the post-harvest period, if infected cereal grain is stored or transported at too high a moisture content, post-harvest growth of the fungus occurs and mycotoxin levels increase (Magan *et al*., 2010).

Mycotoxin-contaminated grain is often unsafe for human consumption, animal feed or malting purposes. In Europe, the USA and other regions, strict upper limits on specific mycotoxin levels in grain and foods have been imposed [Commission Regulation (EC) 1881/2006; http://www.scabusa.org]. *Fusarium graminearum* produces several trichothecene mycotoxins, the most important of which are deoxynivalenol (DON), acetylated DON derivatives, nivalenol and the phytoestrogen zearalenone. DON binds to the peptidyl transferase protein in the ribosome and inhibits protein translation. Different natural isolates (termed chemotypes) produce different mycotoxin types (Alexander *et al*., 2011).

Control of *Fusarium* floral infections remains problematic. In most cereal species, the resistance sources identified are only partially effective and are major QTL based (Buerstmayr *et al*., 2009). Some azole fungicides are moderately effective, but spray coverage and the timing of applications remain difficult. Minimizing consecutive cereal crops and ploughing under any infected residues remain the best methods to reduce disease pressure locally. *Fusarium graminearum* infection of noncereal species in the crop rotation is increasingly being reported, e.g. in soybean and sugar beet.

Excellent genetic, biochemical, molecular genetic, genomic, transcriptomic and isolate collection resources now exist for *F. graminearum* (http://www.scabusa.org, http://www.plexdb.org, http://www.fgsc.net). The genome lacks repetitive sequences and contains a low level of duplicated genes, but a high level of polymorphism exists between strains. When aligned to a genetic map, a 'two-speed' genome is recognized, with discrete regions of high recombination and high single nucleotide polymorphisms (SNPs) located near telomeres and in the middle of the four large chromosomes. Comparisons with the sequenced genomes of *F. verticillioides* and *F. oxysporum* f. sp. *lycopersici* suggest that the high-diversity sites in the *F. graminearum* genome were the result of ancestral chromosome fusion events (Cuomo *et al*., 2007; Ma *et al*., 2010).

Direct replacement of a gene of interest with a selectable marker via homologous recombination is now relatively straightforward. The production of DON mycotoxin contributes to disease formation on wheat floral tissue (Proctor *et al*., 1995). In the absence of DON, strong host defence responses are activated in the rachis and hyphal colonization is restricted to the florets (Jansen *et al*., 2005). DON synthesis is tightly regulated by at least three transcription factors: TRI6, TRI10 and TRI15. An additional 160 pathogenicity/virulence factors are now recognized to contribute to cereal floral infection, with the majority functioning post-penetration (http://www.phi-base.org; Urban and Hammond-Kosack, 2012).

A recent re-investigation of the biology of the wheat floral infection process has revealed that a considerable phase of symptomless infection exists in which hyphae advance extracellularly between the living host cells. High *TRI* gene expression is detected at the advancing front and diminishes thereafter (Brown *et al*., 2010, 2011). Host cells only die on intracellular hyphal invasion, and extensive degradation of plant cell walls is a relatively late process. These findings indicate that *F. graminearum* uses a stealth approach to facilitate successful floral infections (Fig. 8).

Fig. 7 The floral tissues of hexaploid wheat severely infected with *Fusarium graminearum*. This disease is frequently referred to as *Fusarium* head blight (FHB), *Fusarium* ear blight (FEB) or head scab.

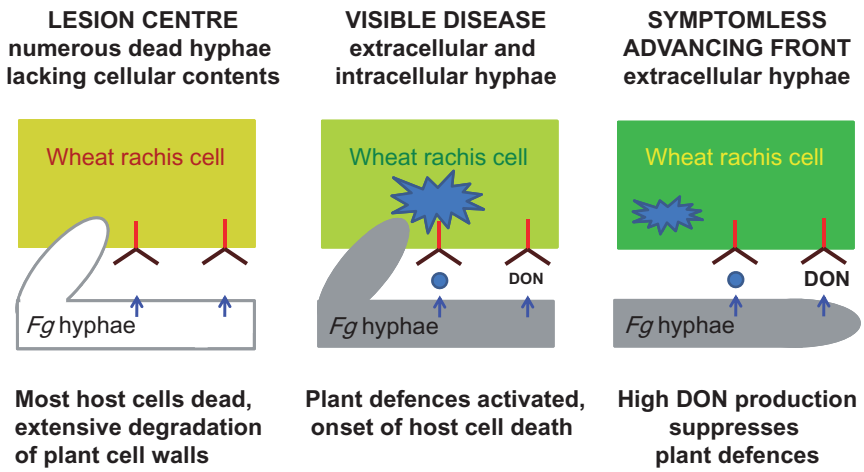

Fig. 8 A simplified three-phase model of the *Fusarium graminearum* (*Fg*) infection process through wheat rachis tissue. At the advancing hyphal front, deoxynivalenol (DON) mycotoxin inhibits protein translation, which greatly suppresses the burst of plant defence responses (depicted in blue). Once hyphae enter the plant cells, the presence of the released proteins and sugars and the high density of fungal hyphae lead to a strong activation of plant defence responses. Later, within the lesion centre (>10 days), the cellular contents of fungal cells residing deep within the dead cortical tissue are relocated to the hyphae just below the rachis epidermis and asexual sporulation then occurs.

5. *FUSARIUM OXYSPORUM*

Fusarium oxysporum Schlecht. is a ubiquitous soil-borne pathogen that causes vascular wilt on a wide range of plants. Characteristic disease symptoms include vascular browning, leaf epinasty, stunting, progressive wilting, defoliation and plant death (Agrios, 2005). The *F. oxysporum* species complex comprises different formae speciales (f. sp.), which collectively infect more than 100 different hosts, provoking severe losses in crops such as melon, tomato, cotton and banana, among others (Michielse and Rep, 2009). *Fusarium oxysporum* is also an emerging pathogen of humans that can cause invasive infections in immunocompromised patients (Nucci and Anaissie, 2007; O'Donnell *et al*., 2004).

In contrast with the remarkably broad host range at the species level, individual isolates of *F. oxysporum* cause disease only on one or a few plant species (Armstrong and Armstrong, 1981; Gordon and Martyn, 1997). This dichotomy has both fascinated and puzzled generations of plant pathologists. Adding to the intrigue, phylogenetic studies suggest that different isolates of a given forma specialis, infecting the same host plant, have originated independently during evolution (O'Donnell *et al*., 1998). Because *F. oxysporum* lacks a known sexual cycle, the mechanism through which these new pathogenic lineages emerged in otherwise distinct genetic backgrounds has long remained elusive. Recently, analysis of the complete genome sequence of the tomato pathogenic form *F. oxysporum* f. sp. *lycopersici* (*Fol*) has revealed the presence of lineage-specific (LS) genomic regions, including four entire chromosomes that are absent from other *Fusarium* species, such as the cereal pathogens *F. graminearum* and *F. verticillioides* (Ma *et al*., 2010). Transfer of two LS chromosomes from *Fol* to a nonpathogenic isolate enabled it to cause disease on tomato plants. This suggests that horizontal transfer of small chromosomes could account for the emergence of new pathogenic lineages (Ma *et al*., 2010). Genome sequences from additional *F. oxysporum* isolates will provide invaluable tools to further explore this hypothesis.

Dominant plant resistance (*R*) genes against different races of *F. oxysporum* have been identified in several crops (Simons *et al*., 1998). The interaction between tomato and *Fol* was used to study the molecular basis of disease resistance and susceptibility (Houterman *et al*., 2008, 2009; Rep *et al*., 2004). These studies led to the identification of a classical gene-for-gene system with at least three fungal avirulence genes, some of which can function as both elicitors and suppressors of *R* gene-based plant immunity (reviewed in Takken and Rep, 2010).

The unusual ability of a single *Fol* isolate to cause disease on both tomato plants and in immunodepressed mice provides a unique model to study trans-kingdom pathogenicity in fungi (Ortoneda *et al*., 2004). Genetic analysis using targeted mutants has revealed that signalling components which are essential for the infection of tomato plants, such as a mitogen-activated protein kinase (MAPK) or a small G-protein, are dispensable for virulence in mice (Di Pietro *et al*., 2001; Martinez-Rocha *et al*., 2008; Ortoneda *et al*., 2004). Others, such as the pH response transcription factor PacC, are required for virulence in the mouse model, but not in plants (Caracuel *et al*., 2003; Ortoneda *et al*., 2004). These results indicate that *F. oxysporum* employs fundamentally distinct infection strategies on plants and mammals. Further studies are addressing the presence of common virulence mechanisms that are required on both types of host.

Recent advances towards an understanding of the genetic basis of pathogenicity in *F. oxysporum* include genome-wide insertional mutagenesis screens (López-Berges *et al*., 2009; Michielse *et al*., 2009a) leading, among others, to the discovery of a master regulator of pathogenic development (Michielse *et al*., 2009b), the identification of a conserved nitrogen response pathway regulating invasive growth functions (López-Berges *et al*., 2010) and the characterization of a novel mucin-type transmembrane sensor (Pérez-Nadales and Di Pietro, 2011). Future studies in the *F. oxysporum* system will continue to provide new insights into the molecular mechanisms of fungal pathogenicity (Fig. 9).

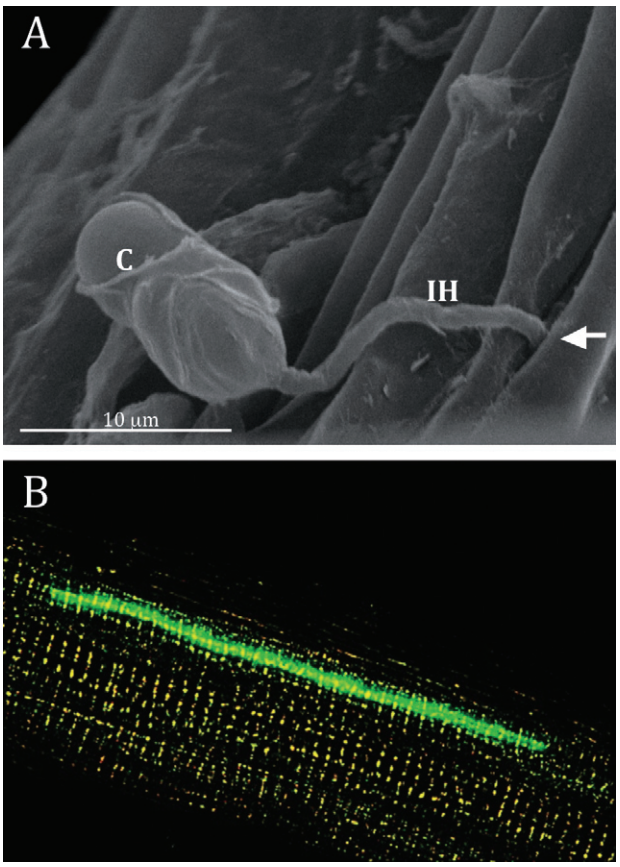

Fig. 9 (A) *Fusarium oxysporum* microconidium (C) germinating on the surface of a tomato root. Penetration occurs by directed growth of the infectious hypha (IH) towards a natural opening between epidermal root cells (penetration site indicated by an arrow). (B) *Fusarium oxysporum* hypha growing in a xylem vessel of a tomato root (from Di Pietro *et al*., 2001).

6. *BLUMERIA GRAMINIS*

Blumeria graminis is an ascomycete belonging to the Erysiphales (Takamatsu, 2004). It causes powdery mildews of grasses (Fig. 10), including wheat and barley, which are amongst the top agricultural crops world-wide (FaoStat, 2011). Mildew infections of cereals reduce grain yield and must be kept in check to ensure an economically viable product. Control of *Blumeria* is achieved by the deployment of fungicides and disease-resistant plant varieties. These control measures need to be continually revised, updated and developed because of the emergence of fungicide resistance, changing regulatory constraints and the evolution of mildew strains capable of overcoming host resistance.

The Erysiphales exhibit a wide range of host specificity, ranging from broad polyphagy observed in many of the dicot mildews (Ing, 1990) to the extremely narrow host ranges of *B. graminis*, where the 'formae speciales' *tritici* and *hordei* can infect only wheat or barley, respectively (Wyand and Brown, 2003). Other Erysiphales cause mildews which affect many other high-value fruit and vegetables, including tomato, cucumbers, grapes and strawberries. Their biology, aetiology and pathology are, overall, similar to those of the cereal mildews (Glawe, 2008). All powdery mildews are strictly obligate biotrophic pathogens: they are absolutely dependent on a live host plant.

Epidemics are caused by a rapid succession of asexual cycles which begin with the landing of airborne conidia on a host surface. Within a few minutes, the conidia germinate. *Blumeria*, unlike other mildews, produces first a short primary germ tube dedicated to surface sensing (Wright *et al*., 2000; Yamaoka *et al*., 2006). After a few hours, a secondary germ tube emerges from the conidium, septates and differentiates an elongated, hooked appressorium from which a peg penetrates through the host cuticle and epidermal cell wall. The peg develops a complex multidigitate haustorium surrounded by a perihaustorial matrix and a specialized host membrane (Fig. 11) (Hückelhoven and Panstruga, 2011). In common with other obligate biotrophic pathogens, the haustorium is devoted to the feeding and control of host immunity and metabolism (Panstruga and Dodds, 2009). Plant-derived nutrients are transported to hyphae growing on the plant surface. Within 3 days from inoculation, conidiophores develop from specialized foot cells and produce masses of conidia: the eponymous 'powder' of these mildews. At the end of the host growth season, compatible strains mate and produce chasmothecia (Braun *et al*., 2002), which also act as resting structures to survive adverse conditions.

The genomes of barley and other powdery mildews are >120 Mb, i.e. much larger than those of related Ascomycota (Spanu *et al*., 2010). This is caused by an extraordinary proliferation of retrotransposons. Genome expansion is accompanied, on the one hand, by a partial loss of genes involved in secondary metabolism, carbohydrate degradation and nitrate uptake and, on the other, by massive proliferation of genes predicted to encode protein effectors. Two classes of effector are currently recognized: the 'EKA' effectors paralogous to the Avrk1 and Avra10 genes closely associated with retrotransposons (Ridout *et al*., 2006; Sacristán *et al*., 2009); and the more conventional small secreted proteins which are further characterized as highly lineage specific (Spanu *et al*., 2010).

The importance of *B. graminis* is thus a result of its persistent and central role as the causal agent of disease of prominent cereal crops (Murray and Brennan, 2010), and because it is a model to study other mildews and other obligate biotrophic pathogens.

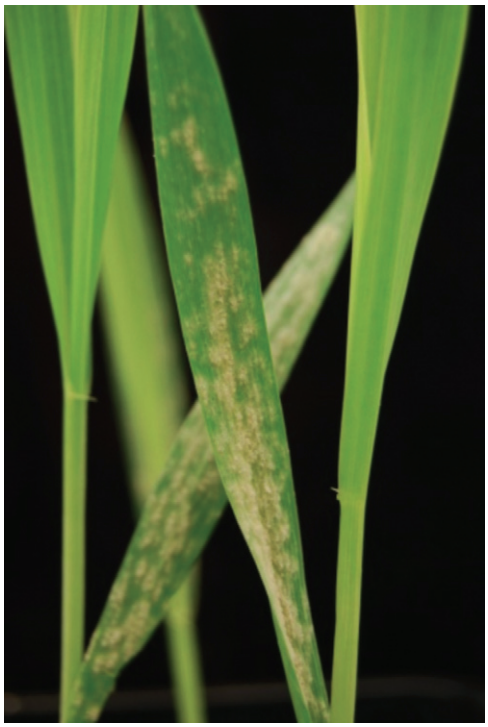

Fig. 10 Barley leaves infected with *Blumeria graminis* f. sp *hordei*. The typical powdery 'pustules' produced by the mildew colonies which grow on the outer surface of the host plant consist of hundreds of thousands of highly infectious asexual conidia blown by air currents to propagate the disease. The epiphytic colonies are fed by intracellular haustoria which develop inside the epidermal cells (see Fig. 11).

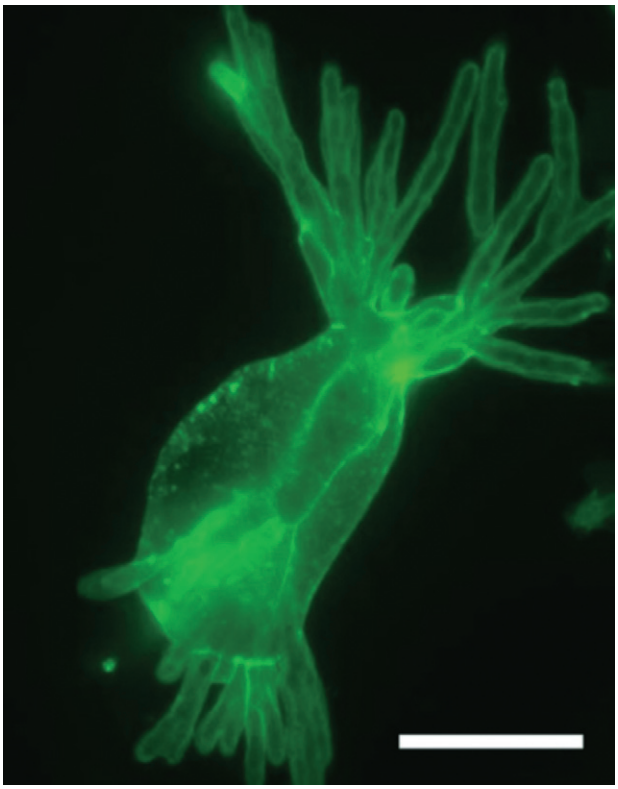

Fig. 11 *Blumeria graminis* f. sp. *hordei* haustorium. This haustorium was isolated by manually dissecting the epidermis of an infected barley leaf and digesting away the host cell wall with a protoplasting cocktail; it was stained with a lectin (wheatgerm agglutinin) bound to Alexa-288. The multidigitate structure is surrounded by the perihaustorial membrane of host origin. In common with other mildews, this specialized membrane is continuous with the host plasma membrane, but has very different biochemical properties (Micali *et al*., 2011). In mildews, the perihaustorial membrane is thought to be the gateway for nutrients and effectors. Bar, 10 μm.

7. *MYCOSPHAERELLA GRAMINICOLA*

The ascomycete *Mycosphaerella graminicola* (anamorph *Septoria tritici*) is in the order Dothideales and causes *Septoria tritici* blotch (STB) disease of wheat (Fig. 12). This is a major economic constraint on wheat productivity, particularly in temperate growth regions (Orton *et al*., 2011). *Mycosphaerella graminicola* infection of wheat begins with hyphal extension on the leaf surface and penetration through stomata without differentiation of an appressorium (Fig. 13). A lengthy period of symptomless intercellular colonization (>7 days) then follows, which culminates in the formation of necrotic leaf lesions within which *My. graminicola* sporulates asexually (Kema *et al*., 1996; Keon *et al*., 2007). This latter phase involves a switch to necrotrophic nutrition (Fig. 13).

Mycosphaerella graminicola is a well-established model organism for the study of pathogen population dynamics and evolution. Long-term datasets (from 1843 to 2003) have demonstrated that STB is influenced by climatic changes, including those associated with the industrial revolution (Bearchell *et al*., 2005). It has also been reported that as much as 90% of the global genetic variation in this fungus can be present within a single infected wheat field (Zhan *et al*., 2003). This level of genetic diversity arises from the heterothallic sexual reproductive system and the formation of ascospores that initiate epidemics each season (Chen and McDonald, 1996; Linde *et al*., 2002; Waalwijk *et al*., 2002). Much of the economic cost incurred by *My. graminicola* arises indirectly from its rapid evolution in response to selective pressures, including disease-resistant wheat cultivars and the sustained use of fungicides. Examples of this include the speed with which *My. graminicola* field populations evolved and propagated a genetic mutation providing resistance to the strobilurin class of fungicides (Fraaije *et al*., 2005), and the rapid evolution of the CYP51 target protein in response to the widespread use of particular azole fungicides (Cools *et al*., 2011).

Mycosphaerella graminicola has also been the subject of intense genetics and genomics analyses. The genome sequences of a model isolate, together with relatives collected from wild grasses, have been published recently (Goodwin *et al*., 2011; Stukenbrock *et al*., 2011). These studies have highlighted the existence of 21 chromosomes, the eight smallest of which are all dispensable for plant infection and vary in number and structure between isolates (Goodwin *et al*., 2011; Stukenbrock *et al*., 2011; Wittenberg *et al*., 2009). This represents the largest number of dispensable chromosomes so far reported in fungi (Wittenberg *et al*., 2009). Functional genomics analysis of the plant infection process has identified a number of genes important for signalling transitions to hyphal growth and stomatal penetration (Orton *et al*., 2011). Subsequent post-penetration stealth pathogenesis is supported through a reduced gene set for cell wall-degrading enzymes (Goodwin *et al*., 2011) and high-level expression of secreted effector proteins which suppress the activation of plant defences (Marshall *et al*., 2011). This initial evasion of plant defences reflects a mechanism more typical of obligate biotrophs. However, what follows is an acute activation of plant defences involving host cell death as the fungus transitions to necrotrophic colonization (Keon *et al*., 2007; Rudd *et al*., 2008). This constitutes an intriguing mechanism for future analysis (Fig. 13), involving initial 'subterfuge', followed by subsequent 'hijack' of plant defence signalling (Deller *et al*., 2011), which may be shared by other members of the genus *Mycosphaerella*, which comprises the largest number of plant-pathogenic fungi.

Fig. 12 *Septoria tritici* blotch (STB) disease of wheat caused by *Mycosphaerella graminicola*.

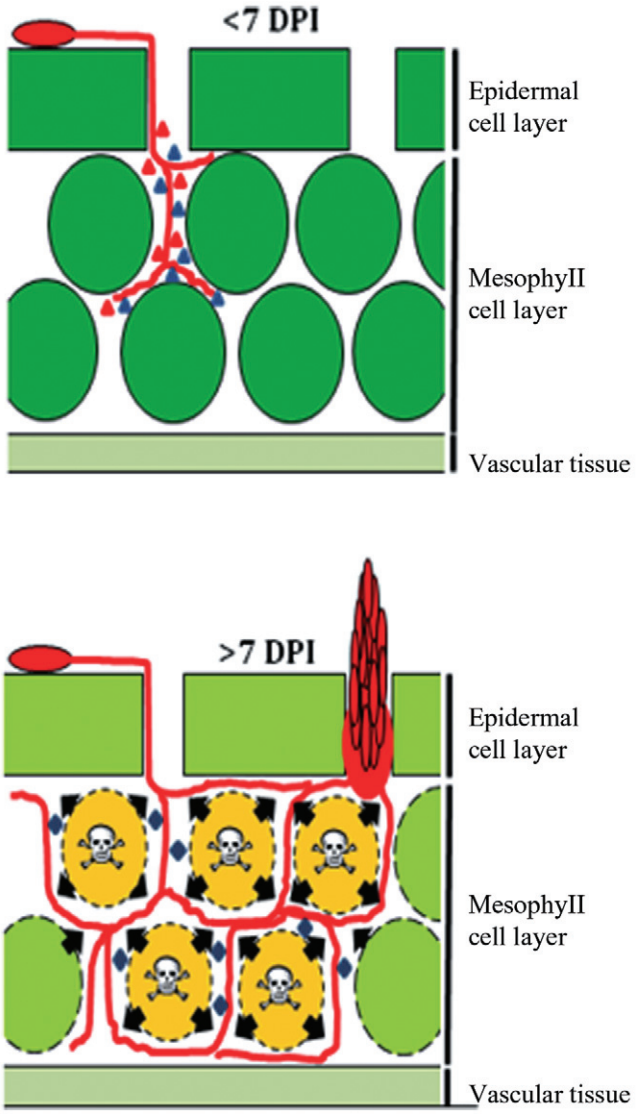

Fig. 13 A model for wheat leaf infection by *Mycosphaerella graminicola*. Early symptomless colonization (<7 days post-leaf inoculation, DPI) involves the release of plant defence-suppressing apoplastic effectors (red and blue triangles) from slow-growing intercellular hyphae. After 7 days, leaf cell death and the loss of membrane permeability occur, allowing nutrient release into the apoplast. Defence-suppressing effectors are 'switched off', although other potentially toxic effectors (blue diamonds) may be produced. Extensive hyphal growth and asexual sporulation (pycnidia and pycnidiospore formation) are now supported within leaf lesions.

8. *COLLETOTRICHUM* SPP.

Colletotrichum is one of the most common and important genera of plant-pathogenic fungi. Virtually every crop grown throughout the world is susceptible to one or more species of *Colletotrichum*. These fungi cause anthracnose spots and blights of aerial plant parts and post-harvest rots. Members of this genus cause major losses to economically important crops, especially fruits, vegetables and ornamentals. The damage caused by *Colletotrichum* spp. extends to important staple food crops, including bananas, cassava and sorghum, grown by subsistence farmers in developing countries throughout the tropics and subtropics. It is particularly successful as a post-harvest pathogen because latent infections, which are initiated before harvest, do not become active until after the fruit has been stored or appears on the market shelf. Up to 100% of the stored fruit can be lost as a result of *Colletotrichum* disease (Prusky, 1996).

Colletotrichum is an asexual genus, classified within the Fungi imperfecti. It belongs to the Coelomycetes, producing its conidia in acervuli. Despite significant developments, the taxonomy of *Colletotrichum* remains in a state of flux. Many uncertainties exist with regard to the systematics of fungal pathogens from this genus and, depending on criteria, the number of species can range from 29 to over 700 (von Arx, 1957; Sutton, 1992). One of the most confusing species is *C. gloeosporioides*. For example, 594 species of *Colletotrichum* were reclassified by von Arx as synonyms of *C. gloeosporioides* (Table 2).

As a result of its importance as a pathogen, its unique intracellular hemibiotrophic lifestyle and the ease with which it can be cultured and manipulated, *Colletotrichum* has a long and distinguished history as a model pathogen for fundamental, biochemical, physiological and genetic studies. For example, the phenomenon of pathogenic variation (race/cultivar specificity) was first recognized in *C. lindemuthianum* (Barrus, 1911). *Colletotrichum* on bean was the model for many of the early studies on phytoalexins (reviewed by Kuc, 1972). During the 1970s and through the 1990s, much of the work that established and characterized the phenomenon of systemic acquired/induced resistance (SAR) was performed using a *Colletotrichum*–cucumber model pathosystem (Durrant and Dong, 2004). Key genes of the cyclic adenosine monophosphate (cAMP), MAPK, and RAS/small G-protein and calcium-mediated signalling pathways have been cloned. The function of these genes, particularly during conidial germination and appressorium development, has been characterized in several *Colletotrichum* species (reviewed by Chen and Dickman, 2004; Chen *et al*., 2006; Dickman and Yarden, 1999; Dickman *et al*., 1995; Takano *et al*., 2000).

Most *Colletotrichum* species initially establish infection through a brief biotrophic phase, associated with large intracellular primary hyphae, although some species are described as subcuticular, e.g. *C. capsici*. The fungus later switches to a destructive, necrotrophic phase, associated with narrower secondary hyphae which ramify throughout the host tissue (Fig. 14). The molecular details that govern the hemibiotrophic lifestyle (also known to occur in other fungal species, e.g. *Magnaporthe*) have long been of interest to phytopathologists. In particular, factors regulating the transition from biotrophy to necrotrophy await identification. The recently completed *C. graminicola* sequence (Vaillancourt *et al*., Department of Plant Pathology, University of Kentucky, Lexington, Kentucky), together with several other *Colletotrichum* species in the pipeline, promises to increase our understanding of this important fungal phytopathogen.

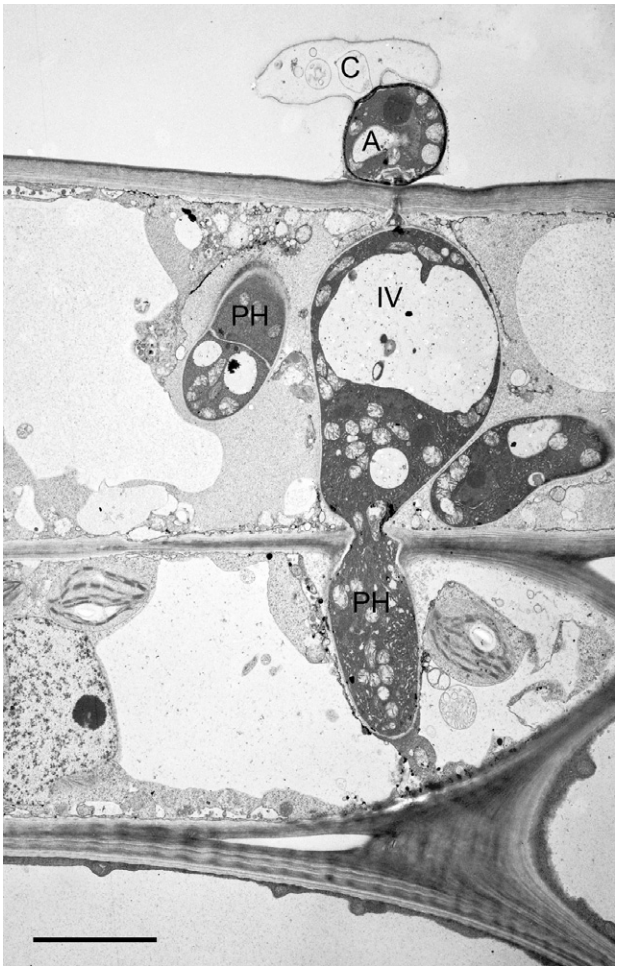

Fig. 14 Transmission electron micrograph showing hemiotrophic growth of *Colletotrichum destructivum* during cowpea infection. Note the thick biotrophic infection vesicle (IV) following appressorium penetration. The host cell is still alive and its plasma membrane can be seen surrounding the hypha. Subsequently, thinner necrophic penetrating hyphae (PH) degrade tissue whilst growing inside the cell. A, appressorium; C, conidium. Photograph courtesy of Dr Richard O'Connell (Department of Plant Microbe Interactions, Max Planck Institute for Plant Breeding Research, Cologne. With permission).

Table 2 The major species of *Colletotrichum*.

Colletotrichum species	Host	Lifestyle
*C. gloeosporioides**	Papaya/citrus/many other hosts	Hemibiotrophy
C. acutatum	Strawberry/others	Necrotrophy
C. coccodes	Tomato	Hemibiotrophy
C. graminicola	Corn	Hemibiotrophy
C. boninense	Broad host range	Hemibiotrophy
C. trifolii	Alfalfa	Hemibiotrophy
C. capsici	Pepper/other hosts	Necrotrophy
C. destructivum	Legumes/tobacco	Hemibiotrophy
C. caudatum	Several grasses	?
C. crassipes	Dryandra	Hemibiotrophy
C. dematium	Radish/other hosts	Hemibiotrophy
C. kahawae	Coffee	Hemibiotrophy
C. orbiculare	Melon/cucumber	Hemibiotrophy
C. sublineolum	Sorghum	Hemibiotrophy
C. truncatum	Legumes	Hemibiotrophy
C. musae	Banana	Hemibiotrophy
C. fragariae	Strawberry/other hosts	Hemibiotrophy
C. spinaceae	Spinach	Hemibiotrophy
C. lindemuthianum	Common bean	Hemibiotrophy

*Dependent on host and infected tissue.

9. *USTILAGO MAYDIS*

Corn smut is not an economically important or devastating disease. Therefore, *Ustilago maydis* has not made it into the Top 10 for this reason. Although its symptoms on corn can be quite dramatic (Fig. 15, centre), in most cases, infections remain local, do not spread and are hence not associated with severe losses in corn yield. Quite the contrary, farmers in Mexico infect corn artificially to harvest infected cobs for the preparation of Huitlacoche, a traditional dish popular in pre-Hispanic times.

What are the attractions of this fungus and why has it become a model for biotrophic, plant-pathogenic basidiomycetes? *Ustilago maydis* can be grown in culture in defined media, the fungus is haploid and grows by budding (Fig. 15A) and forms compact colonies on plates that can be replica plated. These were actually some of the reasons for choosing this fungus for seminal early studies on homologous recombination (Holliday, 2004). For host colonization and symptom development, corn seedlings can be infected. Symptoms can develop on all above-ground parts of maize plants in 5–6 days (Fig. 15D) and an infection cycle is completed in about 2 weeks. Typical for smut fungi is that pathogenic development equals sexual development, and mating and the formation of a dikaryotic filament are absolutely required. These steps can be reproduced in the laboratory (Fig. 15B) up to the formation of appressoria on appropriate hydrophobic surfaces (Fig. 15C). The proteins controlling these events are known: pheromones and pheromone receptors, as well as a heterodimeric transcription factor formed after cell fusion that triggers a regulatory cascade (Brefort *et al*., 2009; Heimel *et al*., 2010a, b). This knowledge has allowed the construction of haploid solopathogenic strains that cause disease without need for a mating partner (Bölker *et al*., 1995; Kämper *et al*., 2006). Such strains enable forward genetic screens and greatly reduce work in reverse genetics approaches. Homologous recombination is amazingly efficient (Kämper, 2004) and makes use of four dominant selectable markers (Fig. 15G; Basse and Steinberg, 2004). Tools include regulated promoters for the study of essential genes and a variety of staining methods to visualize the fungus during biotrophic growth (Fig. 15D), and fluorescent proteins for live cell imaging of gene expression and visualization of subcellular structures (Fig. 15F) are available (Böhmer *et al*., 2009; Mendoza-Mendoza *et al*., 2009; Steinberg and Perez-Martin, 2008). Since 2006, the 20.5-Mb manually annotated and curated genome sequence has been available (Kämper *et al*., 2006; http://mips.helmholtz-muenchen.de/genre/proj/ustilago). The genome is 'lean' and contains little repetitive DNA. Transcriptional profiles (Fig. 15E) have been established for the most important developmental fungal stages and plant responses (accessible through GeneExpressionOmnibus: http://www.ncbi.nlm.nih.gov/geo/). This has spurred reverse genetics approaches and has allowed the identification of clustered genes encoding secreted effectors that play crucial roles during host colonization, and the determination of which tissue can be infected (Doehlemann *et al*., 2009, 2011; Kämper *et al*., 2006; Skibbe *et al*., 2010). From genome analysis, it has also become apparent that *U. maydis* is more closely related to humans than to budding yeast, and numerous proteins are shared only by *U. maydis* and *Homo sapiens*. This includes proteins involved in basic principles of long-distance transport, mitosis, motor-based microtubule organization and homologous recombination. *Ustilago maydis* is therefore a perfect host for the study of such processes (Banuett *et al*., 2008; Holloman, 2011; Steinberg and Perez-Martin, 2008). Where is this system heading? It will allow the establishment of the hierarchy of effector action during plant colonization, provide insights into fungal nutrition during plant colonization (Eichhorn *et al*., 2006; Wahl *et al*., 2010) and serve as the blueprint for comparative approaches (Schirawski *et al*., 2010), which are likely to reveal insights into both genome evolution and host specialization.

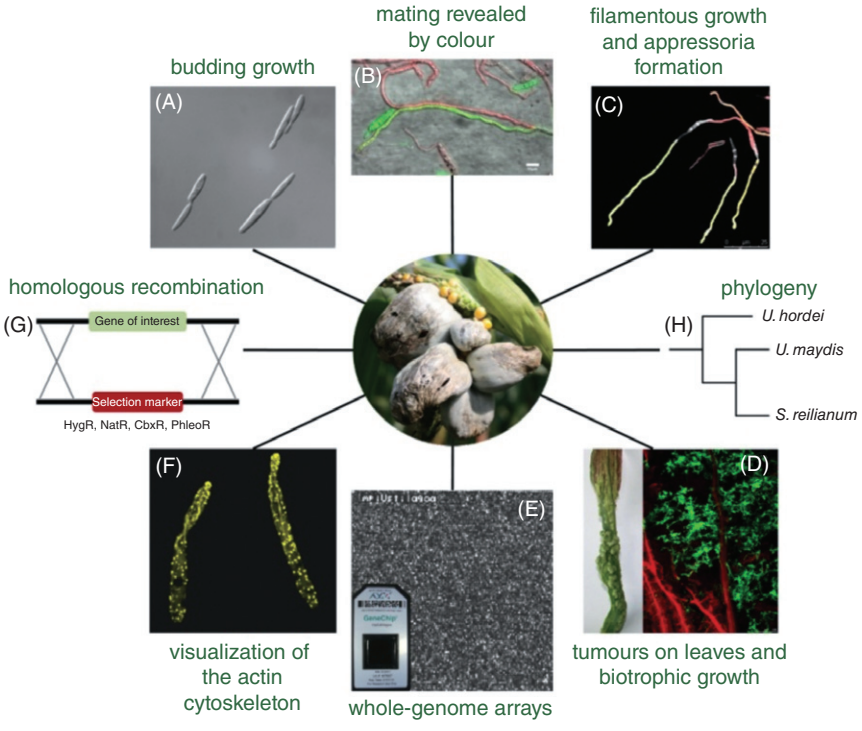

Fig. 15 Developmental stages of *Ustilago maydis*, tools and disease symptoms. (A) Haploid cells display budding growth. (B) Compatible haploid *U. maydis* strains expressing cytoplasmic red fluorescent protein (RFP) (red) and green fluorescent protein (GFP) (green) under a constitutive promoter mate and produce a filamentous dikaryon (yellow). (C) A solopathogenic strain expressing cytoplasmic RFP from a constitutive promoter and an appressorial marker gene fused to triple GFP efficiently forms appressoria on a hydrophobic surface, after stimulation with hydroxy-fatty acids. (D) Macroscopic *U. maydis* disease symptoms on a maize leaf 12 days post-infection (left) and fungal hyphae in tumour tissue 10 days post-infection (right) are visualized by confocal microscopy; fungal hyphae are stained with WGA-AF488 (green); plant cell walls are stained with propidium iodide (red). (E) Affymetix gene chips allow genome-wide expression studies. (F) Visualization of F-actin through LifeAct-YFP in budding cells. (G) Highly efficient homologous recombination is used for gene replacement employing hygromycin resistance (HygR), nourseothricine resistance (NatR), carboxin resistance (CbxR) or phleomycin resistance (PhleoR) as dominant selectable markers. (H) Phylogenetic tree of *U. maydis* and its closest relatives. Figures were kindly provided by Patrick Berndt and Rolf Rösser (*U. maydis* disease symptoms in infected maize cob, centre), both at the Max Planck Institute for Terrestrial Microbiology. Also, please note that their permission for using these Figures has been obtained.

10. *MELAMPSORA LINI*

In contrast with most of the other pathogens on this 'Top 10' list, flax rust is more famous than infamous. Although it was initially studied as a pathogen having an impact on the flax and linseed industries, its main impact on food and fibre production came (and comes) from its role as a model system, providing insights into the molecular basis of plant immunity.

Classical genetic studies (approximately 1926–1981)

Through pioneering genetic studies (Fig. 16) of the pathogenicity of different physiological races of the pathogen and the resistance and susceptibility to rust in flax, H.H. Flor observed that there was a specific relationship between each gene determining pathogenicity in rust [avirulence (*Avr*) genes] and corresponding genes [resistance (*R*) genes] in the host determining resistance (Flor, 1956; Lawrence, 1988; Lawrence *et al*., 2007). This became known as the 'gene-for-gene' relationship, which provided pathologists and plant breeders with a logical foundation to reconcile the process of breeding other crop species for resistance to pathogens. The observations also provided insights into breeders' experience that single resistance genes deployed over large areas frequently 'break down' as a result of the mutation of a single corresponding avirulence gene or genetic re-assortment during sexual reproduction of previously 'hidden' recessive virulence alleles to produce progeny homozygous for virulence. In practical terms, this classical genetics-based knowledge also underpinned the approach to more durable resistance through the pyramiding of multiple resistance genes in a single genotype of crop species. The gene-for-gene hypothesis also gave rise to the postulate that resistance genes encode receptor proteins that detect the presence of specific avirulence proteins in the pathogen, and a resulting recognition event that gives rise to host resistance, the foundation for the development of the current knowledge of the basis of plant immunity.

Molecular studies (1988 to present)

The flax rust system was among the first three systems to provide cloned resistance genes and the identification of a new class of immune receptor, cytoplasmic nucleotide-binding leucine-rich repeat (NB-LRR) proteins, specific members of which provided host plant resistance against fungi, oomycetes, bacteria, viruses, nematodes, sucking insects and parasitic plants (Ellis *et al*., 2007). The same protein group was subsequently identified in animals as part of the innate immune system.

The examination of alleles from the *L* resistance locus and chimeric genes between alleles expressed in transgenic flax provided the first indication that the specificity of gene-for-gene interactions was determined by sequence variation of the LRR domain of the receptor proteins, and that the likely origin of new resistance specificities in nature results from point mutations, re-assortment of the mutations, and duplication and deletion of LRR units by intragenic recombination (Ellis *et al*., 2007).

The intracellular nature of the flax resistance proteins implied that the Avr (effector) proteins were detected by R proteins within host cells. This was subsequently confirmed by the cloning of several *Avr* genes from flax rust which encode diverse proteins secreted from the rust and were taken into the plant cell through the mediation of uptake signals located near the N-terminus of the Avr proteins (Rafiqi *et al*., 2010). The molecular basis for gene-for-gene recognition was shown to rely on specific and direct interaction between flax R proteins and corresponding Avr proteins. Only those R and Avr proteins that interacted in yeast two-hybrid assays induced plant resistance responses when co-expressed in plants (Wang *et al*., 2007). The study of the flax rust fungus and its interaction with its host is now set for further advances following the development of an *Agrobacterium* transformation system for this obligate biotroph (Lawrence *et al*., 2010) (Fig. 16).

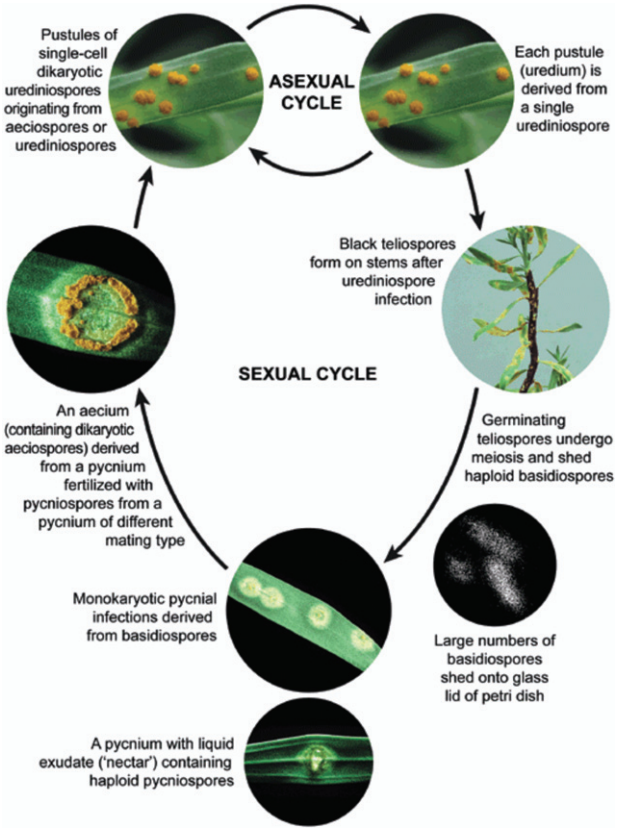

Fig. 16 Life cycle of flax rust, *Melampsora lini*. Genetic analysis in flax rust depends on the ability to replicate all stages of the life cycle under controlled conditions to produce selfed and outcrossed progeny from flax rust isolates, and is complicated by the rust being an obligate biotroph, requiring all manipulations to be undertaken on the living host plant.

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