

Review

The Top 10 oomycete pathogens in molecular plant pathology

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SUMMARY

Oomycetes form a deep lineage of eukaryotic organisms that includes a large number of plant pathogens which threaten natural and managed ecosystems. We undertook a survey to query the community for their ranking of plant-pathogenic oomycete

species based on scientific and economic importance. In total, we received 263 votes from 62 scientists in 15 countries for a total of 33 species. The Top 10 species and their ranking are: (1) *Phytophthora infestans*; (2, tied) *Hyaloperonospora arabidopsidis*; (2, tied) *Phytophthora ramorum*; (4) *Phytophthora sojae*; (5) *Phytophthora capsici*; (6) *Plasmopara viticola*; (7) *Phytophthora cinnamomi*; (8, tied) *Phytophthora parasitica*; (8, tied) *Pythium ultimum*; and (10) *Albugo candida*. This article provides an

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introduction to these 10 taxa and a snapshot of current research. We hope that the list will serve as a benchmark for future trends in oomycete research.

Keywords: oomycetes plant pathology, microbiology, diversity, genomics.

INTRODUCTION

Oomycetes are eukaryotic organisms that superficially resemble filamentous fungi, but are phylogenetically related to diatoms and brown algae in the stramenopiles (Gunderson *et al.*, 1987; Jiang and Tyler, 2012; Lamour and Kamoun, 2009; Thines, 2014; Thines and Kamoun, 2010). Fossil evidence indicates that a number of oomycetes emerged as endophytes of land plants at least by the Carboniferous period, approximately 300–350 million years ago (Krings *et al.*, 2011). One species, *Combresomyces williamsonii*, described from ~320-million-year-old petrified stem cortex and rootlets of a seed fern, may have even been parasitic (Strullu-Derrien *et al.*, 2011). Phylogenetic analyses of modern taxa have revealed that plant parasitism has evolved independently in three lineages of oomycetes (Thines and Kamoun, 2010). Well-known plant pathogens, namely downy mildews, *Phytophthora* and *Pythium*, appear to have radiated from a common plant-parasitic ancestor (Thines and Kamoun, 2010). The impact of oomycetes on humankind is well documented as both a persistent threat to subsistence and commercial farming and as destructive pathogens of native plants (Agrios, 2005; Erwin and Ribeiro, 1996; Lamour and Kamoun, 2009). As a result, news related to plant diseases caused by oomycetes tends to capture the interest of the general public and is frequently featured in the media.

In the last two decades, increased awareness of the distinctive phylogeny and biology of oomycetes has driven the emergence of a specialist research community that is currently organized under

the umbrella of the 'Oomycete Molecular Genetics Network'. This community has moved the field beyond the gloomy view of the 1980s that oomycetes are a 'fungal geneticist's nightmare' (Shaw, 1983; discussed in Schornack *et al.*, 2009). It has produced novel paradigms in understanding host–microbe interactions, effector biology and genome evolution (Bozkurt *et al.*, 2012; Govers and Gijzen, 2006; Jiang and Tyler, 2012; Schornack *et al.*, 2009; Vleeshouwers *et al.*, 2011). The oomycete community was one of the first in plant pathology to initiate coordinated transcriptome and genome sequencing projects, and subsequently to exploit the resulting resources to drive conceptual advances (Govers and Gijzen, 2006; Pais *et al.*, 2013; Schornack *et al.*, 2009). These days, with the genomes serving as unique resources for basic and applied research, oomycetes are best portrayed as a 'genomicist's dream' (Govers and Gijzen, 2006; Jiang and Tyler, 2012; Pais *et al.*, 2013; Schornack *et al.*, 2009).

It is therefore particularly fitting that the oomycetes are at last covered by the Top 10 review series of *Molecular Plant Pathology*. The process to generate the list and the aim of this article are similar to those of previous contributions on plant-pathogenic viruses (Scholthof *et al.*, 2011), fungi (Dean *et al.*, 2012), bacteria (Mansfield *et al.*, 2012) and nematodes (Jones *et al.*, 2013). We undertook a survey to query the community for their ranking of plant-pathogenic oomycete taxa based on scientific and economic importance. In total, we received 263 votes from 62 scientists in 15 countries that yielded the Top 10 species (Table 1). To some degree, the results reflect the sizes of the subcommunities. Six of the ten species belong to the genus *Phytophthora*, which is more commonly studied than any other oomycete genus. Obligate parasites are also well represented with three species (*Hyaloperonospora arabidopsidis*, *Plasmopara viticola* and *Albugo candida*, Table 1). Another 23 species received votes but ranked outside the Top 10 (Table 2). The fish parasite *Saprolegnia parasitica* received enough votes to place it in the Top 10, but was removed, given that the list focuses on plant pathogens (Table 2).

Table 1 Top 10 oomycetes in molecular plant pathology.

Rank	Species	Common disease name(s)	Number of papers (2005–2014)	Number of votes
1	<i>Phytophthora infestans</i>	Late blight	1230	51
= 2	<i>Hyaloperonospora arabidopsidis</i>	Downy mildew	137	25
= 2	<i>Phytophthora ramorum</i>	Sudden oak death; Ramorum disease	378	25
4	<i>Phytophthora sojae</i>	Stem and root rot	276	22
5	<i>Phytophthora capsici</i>	Blight; stem and fruit rot; various others	541	17
6	<i>Plasmopara viticola</i>	Downy mildew	326	15
7	<i>Phytophthora cinnamomi</i>	Root rot; dieback	315	13
= 8	<i>Phytophthora parasitica</i>	Root and stem rot; various others	142	10
= 8	<i>Pythium ultimum</i>	Damping off; root rot	319	10
10	<i>Albugo candida</i>	White rust	65	9

The '=' sign before the ranking indicates that the species tied for that position. The number of papers published in 2005–2014 is based on searches of the Scopus database (<http://www.scopus.com>) using the species names as a query. For *H. arabidopsidis*, a search for the alternative name '*Peronospora parasitica*' was also performed and the combined number is shown. Searches with the terms 'oomycete*' and '*Phytophthora*' yielded 2068 and 4059 articles, respectively.

Table 2 Other oomycete species that received votes.

Rank	Species
11	<i>Aphanomyces euteiches</i>
12	<i>Albugo laibachii</i>
13	<i>Bremia lactucae</i>
14	<i>Phytophthora palmivora</i>
15	<i>Pseudoperonospora cubensis</i>
16	<i>Plasmopara halstedii</i>
17	<i>Peronophythora litchi</i>
18	<i>Peronosclerospora sorghi</i>
19	<i>Peronospora belbahrii</i>
20	<i>Phytophthora alni</i>
21	<i>Phytophthora brassicae</i>
22	<i>Phytophthora cactorum</i>
23	<i>Phytophthora meadii</i>
24	<i>Phytophthora phaseoli</i>
25	<i>Phytophthora plurivora</i> (formerly <i>P. citricola</i>)
26	<i>Plasmopara obducens</i>
27	<i>Pythium aphanidermatum</i>
28	<i>Pythium oligandrum</i>
29	<i>Sclerophthora rayssiae</i>
30	<i>Hyaloperonospora brassicae</i>
NR	<i>Saprolegnia parasitica</i> (fish parasite)
NR	<i>Lagenidium giganteum</i> (mosquito parasite)
NR	<i>Pythium insidiosum</i> (mammalian parasite)

NR, not ranked because the species is not associated with plants.

This article is based on contributions from the voters to provide an introduction to the 10 taxa and a snapshot of current research. Each section starts with a brief overview of the selected species, e.g. its importance, pathology, host range and life cycle. This is followed by a review of current research themes, particularly the unique findings that have emerged from the study of particular species, and an outlook on future research. We hope that this article will serve as a reference and resource for novice and experienced readers alike, as well as provide a benchmark for future trends in oomycete research.

1. PHYTOPHTHORA INFESTANS

Phytophthora infestans causes potato late blight (Fig. 1), a disease with major historical impact. Providing twice the calories of rye and wheat per hectare, potato was central to European agriculture between 1750 and 1850. In 1844, the arrival of *P. infestans* changed this situation. In addition to the well-documented Irish famine (Fig. 2), crop failures in 1845 and 1846 contributed to an estimated 750 000 hunger-associated deaths in continental Europe (Zadoks, 2008). The discovery that late blight was caused by a microbial pathogen, 15 years before Pasteur's formal confirmation of Germ Theory, places the 19th century migration of *P. infestans* as a significant milestone in the foundation of plant pathology as a scientific discipline. Today, late blight remains a major constraint to the production of potato, the world's third largest staple crop, and is thus a constant threat to food security (Fisher *et al.*, 2012; Haverkort *et al.*, 2008).

In the 1840s, trade in potatoes probably facilitated the long-distance migration of the pathogen. Whole-genome sequencing of European herbarium samples has revealed that populations belonging to the HERB-1 genotype were different from US-1, the genotype dominating the population globally in the mid- to late 20th century (Goodwin *et al.*, 1994; Martin *et al.*, 2013, 2014; Yoshida *et al.*, 2013, 2014). Waves of migration and genotype displacements in the 20th century have been well documented. The introduction of the A2 mating type to Europe in the 1970s (Drenth *et al.*, 1994)



Fig. 1 Potato plants with typical late blight lesions. Infection starts when a spore lands on the leaf and germinates. The germ tube forms an appressorium and an emerging penetration peg pushes into an epidermal cell. Then the inner cell layers are colonized. During the biotrophic phase, hyphae grow in the intercellular space, whereas haustoria enter plant cell cavities and invaginate host cell plasma membrane. Later, *Phytophthora infestans* switches to necrotrophic growth, resulting in the death of plant cells and the appearance of necrotic lesions on the infected tissues. In this phase, hyphae escape through the stomata and produce numerous asexual spores, named sporangia, that easily detach and disperse by wind or water. A sporangium that finds a new host can either germinate directly and initiate a new cycle or, at lower temperatures, undergo cleavage resulting in a zoosporangium from which six to eight flagellated spores are released. These zoospores can swim for several hours but, once they touch a solid surface, they encyst and germinate to initiate new infections. Under favourable conditions, the pathogen can complete the cycle from infection to sporulation in 4 days. In the field, this cycle is repeated multiple times during one growing season, resulting in billions of spores and a continuous increase in disease pressure. In addition to leaves, stems and tubers are also infected and *P. infestans* can continue to flourish on the decaying plant material. If not managed properly, infected seed potatoes or waste on refuse piles are often the sources of inoculum for new infections in the spring. An alternative route for surviving the winter is via oospores, sexual spores that can survive in the soil for many years. *Phytophthora infestans* is heterothallic; isolates are either A1 or A2 mating type, and sex organs only develop when isolates of opposite mating type sense the sex hormone produced by the mate.

and, more recently, the emergence of new, more aggressive lineages, such as 13_A2 (Blue 13) in Europe (Cooke *et al.*, 2012), US-22 in the eastern USA (Fry *et al.*, 2013; Hu *et al.*, 2012) and US-23/US-24 in Canada (Peters *et al.*, 2014), impose constant challenges to disease resistance breeding.

In the 1990s, *P. infestans* research led to the development of molecular approaches to study oomycete pathology and biology. The first DNA transformation system (Judelson *et al.*, 1991) and the first transcriptional profiling during infection (Pieterse *et al.*, 1991) paved the way to detailed molecular investigations of sexual and asexual development and pathogenicity (reviewed in Judelson, 1997). DNA fingerprinting revolutionized population studies (Goodwin *et al.*, 1992) and was a prelude to the first genome-wide genetic map of a *Phytophthora* species (van der Lee *et al.*, 1997). Observations that gene silencing occurred in *P. infestans* transformants (e.g. Judelson and Whittaker, 1995) sparked important studies of the mechanisms underlying transcriptional silencing (van West *et al.*, 1999). The first large-scale transcript sequencing studies (Kamoun *et al.*, 1999) ushered in the genomics era of *Phytophthora* research.



Fig. 2 One of the many Great Famine memorials around the world. These sculptures on Customs House Quays in Dublin, by artist Rowan Gillespie, stand as if walking towards the emigration ships on the Dublin Quayside (courtesy of Michael Seidl).

In the last 15 years, advanced genomic and functional approaches have placed *P. infestans* at the forefront of research to understand oomycete development (Judelson and Blanco, 2005) and pathogenicity (Kamoun, 2006). An elegant combination of bioinformatics, proteomics and functional genomics has revealed secreted proteins as potential factors influencing host–*P. infestans* interactions (Torto *et al.*, 2003). Apoplastic effectors that inhibit host secreted proteases were first described in *P. infestans* (Tian *et al.*, 2004), and the apoplastic phytotoxin-like SCR74 (Liu *et al.*, 2005) provided support for the use of diversifying selection as a criterion for the identification of candidate effectors. The apoplastic effectors EPIC1 and EPIC2B were found to converge on the host protease RCR3, which is also targeted by an independently evolved effector from a fungal plant pathogen (Song *et al.*, 2009). Recently, diversifying selection of the EPIC1 protease inhibitor was implicated in functional adaptation to the equivalent protease target after ‘jumps’ to a new host (Dong *et al.*, 2014).

The first reported *P. infestans* avirulence effector was AVR3a, the counterpart of resistance protein R3a (Armstrong *et al.*, 2005). Comparison with avirulence effectors from *Phytophthora sojae* and *H. arabidopsidis* identified conserved amino acid motifs RXLR and EER (Rehmany *et al.*, 2005), which act as signals for translocation into host cells (Whisson *et al.*, 2007). The genome sequence of *P. infestans* revealed hundreds of genes predicted to encode RXLR effectors, plus a second class of candidate effectors called Crinklers (CRNs) (Haas *et al.*, 2009), which may also be delivered inside plant cells (Schornack *et al.*, 2010). These effector genes are rapidly evolving and are typically variable between different *P. infestans* isolates (Cooke *et al.*, 2012). RXLR and CRN reside in gene-sparse, repeat-rich regions, potentially subject to rearrangement and rapid mutation, raising the concept of a ‘two-speed’ genome in evolutionary terms (Haas *et al.*, 2009; Raffaele *et al.*, 2010). Recent years have witnessed intense efforts to determine the biochemical function of RXLR effectors, notably by determining their protein targets in the host. The first target identified was ubiquitin E3 ligase CMPG1,

which AVR3a stabilizes to prevent programmed cell death in response to elicitors such as INF1 (Bos *et al.*, 2010).

Breeding for late blight resistance was initiated by James Torbitt of Belfast in the 1870s, inspired by the theories of Charles Darwin, with whom he shared considerable correspondence (DeArce, 2008). More than 130 years later, breeding efforts have yet to provide a durable solution. Occasionally, local successes are reported; for example, variety C88, widely cultivated in China for over a decade, retains its resistance (Li *et al.*, 2011). Many resistance (*R*) genes effective against races of *P. infestans* have been identified (Rodewald and Trognitz, 2013). Only with the recent identification of avirulence genes, all of which encode RXLR effectors, can breeders incorporate knowledge on how *P. infestans* evades detection into their breeding strategies (Vleeshouwers *et al.*, 2011). Such information is critical for the prediction of the durability of *R* genes. The use of effectors as tools to rapidly identify *R* genes is a powerful step towards rational selection and combination of lasting resistance (Vleeshouwers *et al.*, 2008).

We still know relatively little about how *P. infestans* effectors manipulate host plants to establish disease. How do they translocate into host cells? Do they define host range? How do they work in concert to modulate complex networks of regulatory processes occurring in the host? Only recently have researchers started to adopt structural biology to fully investigate functional relationships between interacting pathogen and plant proteins (Wirthmueller *et al.*, 2013). Could such research catalyse the modification of molecular interactions in favour of the plant immune system through synthetic biology approaches?

The basic knowledge gained on *P. infestans* in the last decade is starting to have an impact on the management of late blight disease. For the control of late blight, farmers largely rely on agrochemicals, many with unknown modes of action. The emergence of insensitive strains is possibly a result of high mutation rates in the pathogen (Randall *et al.*, 2014). The phenylamide metalaxyl was one of the first chemicals to exhibit specificity to oomycetes. Soon after its introduction and widespread use, fully insensitive strains emerged, but only 35 years later was the molecular basis of this insensitivity uncovered (Randall *et al.*, 2014). Mining of the *P. infestans* genome has revealed many novel proteins with domain combinations that are unique to oomycetes (Seidl *et al.*, 2011). Several probably function in signalling and may be promising fungicide targets. Mode-of-action studies are more accessible thanks to the available genomics resources, transformation tools and marker strains with tags to visualize the cytoskeleton and various subcellular compartments (Ah Fong and Judelson, 2003; Meijer *et al.*, 2014). In resistance breeding, the efficacy of stacking *R* genes into favoured cultivars, by either introgression or *trans*- or *cis*-genesis, is being investigated (Tan *et al.*, 2010). However, for success, the *R* genes to be combined should be selected carefully on the basis of our knowledge on the variation in the pathogen population, and the attendant vulnerability to resistance being overcome (Vleeshouwers *et al.*, 2011). Future efforts should consider the introduction of multiple barriers, perhaps combining *R* proteins with, for instance, membrane-localized pattern recognition receptors or components governing non-host resistance to *P. infestans*. Even then, it is unlikely that plant resistance can fully control late blight. Agrochemicals should not be abandoned. On the contrary, for more rational fungicide design, novel targets should be identified, an endeavour that will be enhanced by an even more profound insight into the biology of *P. infestans*.

2. *HYALOPERONOSPORA ARABIDOPSIS*

Hyaloperonospora arabidopsis (formerly *H. parasitica* and *Peronospora parasitica*) is one of 700 downy mildew species within the Peronosporaceae (Thines, 2014). Downy mildew pathogens cause harmful diseases on many important crops, notably *Peronospora* and *Hyaloperonospora* spp. on brassica crops, *Plasmopara viticola* on grape, *Peronosclerospora* spp. on maize and sorghum, *Pseudoperonospora cubensis* on cucurbits and *Bremia lactucae* on lettuce (Lucas *et al.*, 1995).

Most downy mildews have narrow host ranges and are completely dependent on their host for growth and reproduction. They can survive in the soil as quiescent oospores that initiate infection through roots. Spread of the pathogen mostly occurs through airborne sporangiospores that are formed on the lower leaf surface, giving downy patches (Fig. 3). These spores germinate on the plant surface and penetrate by forming appressoria (Koch and Slusarenko, 1990). Once past the epidermis, hyphae grow intercellularly and, similar to *Phytophthora* spp. and other downy mildews, develop haustoria, specialized structures that may function in feeding and the suppression of host defence by targeted secretion of effectors (Fig. 4, Whisson *et al.*, 2007).

Hyaloperonospora arabidopsis is a prominent pathogen in natural populations of *Arabidopsis thaliana* (Coates and Beynon, 2010; Holub, 2008). As such, it was adopted in the 1980s as one of two pathogens of *Arabidopsis*, together with the bacterium *Pseudomonas syringae* (Koch and Slusarenko, 1990). The Top 10 ranking of *H. arabidopsis* reflects the subsequent success of the *Arabidopsis*–*H. arabidopsis* pathosystem. *H. arabidopsis* was initially utilized as a 'physiological probe' of the *Arabidopsis* immune system (Holub *et al.*, 1994). This research led to the cloning of the first plant disease *R* genes against an oomycete, better understanding of the evolutionary dynamics of *R* genes, the definition of broadly important immune system regulators, the identification of downy mildew-resistant mutants and genetic definition of the complexity of the plant immune signalling network (reviewed in Coates and Beynon, 2010; Lapin and Van den Ackerveken, 2013; Slusarenko and Schlaich, 2003). On the pathogen side, research is hampered by the lack of protocols for culture and genetic transformation, established techniques with other oomycetes such as *P. infestans*. However, work in the early 2000s led to the development of genetic maps and DNA libraries that enabled the discovery of the first avirulence effector (Allen *et al.*, 2004), and later to the RXLR effector family (Rehmany *et al.*, 2005).

Genome sequencing of *H. arabidopsis* isolate Emoy2, completed in 2010, unveiled 134 predicted RXLR effectors and other components of the *H. arabidopsis* secretome (Baxter *et al.*, 2010). Notably, this report also revealed important genomic signatures of obligate biotrophy that have evolved convergently in other obligate oomycete and fungal lineages (reviewed in McDowell, 2011). Protein interaction assays have shown that *H. arabidopsis* effectors target a highly interconnected host machinery, helping to define a representative plant–pathogen interaction network (Mukhtar *et al.*, 2011). In addition, several high-throughput functional studies have investigated effector subcellular localizations, suppression of immune responses, molecular targets and cognate immune receptors (Cabral *et al.*, 2011, 2012; Caillaud *et al.*, 2011; Fabro *et al.*, 2011).

Future studies with the *H. arabidopsis* experimental system will include: (i) direct or *Agrobacterium*-mediated transformation for genetic manipulation required for the molecular analysis of downy mildew pathogenicity; (ii) the establishment of the temporal hierarchy of effectors during penetration, colonization and sporulation, which may serve as a blueprint for a better understanding of the molecular basis of biotrophy; (iii) the role of genetic recombination and epigenetics on the emergence of new effectors; (iv) the development of tools to understand how plant-originated molecules regulate pathogen response; and (v) the relevance of interspecies transfer of small RNAs. These investigations on *H. arabidopsis* will continue to provide new insights into the molecular mechanisms of downy mildew pathogenicity, and contribute to comparative and functional analysis of (obligate) biotrophic oomycete and fungal pathogens.



Fig. 3 *Hyaloperonospora arabidopsis* disease symptoms on a 2-week-old *Arabidopsis* seedling. Mature sporangiospores are visible as white structures on the right side of the leaf.

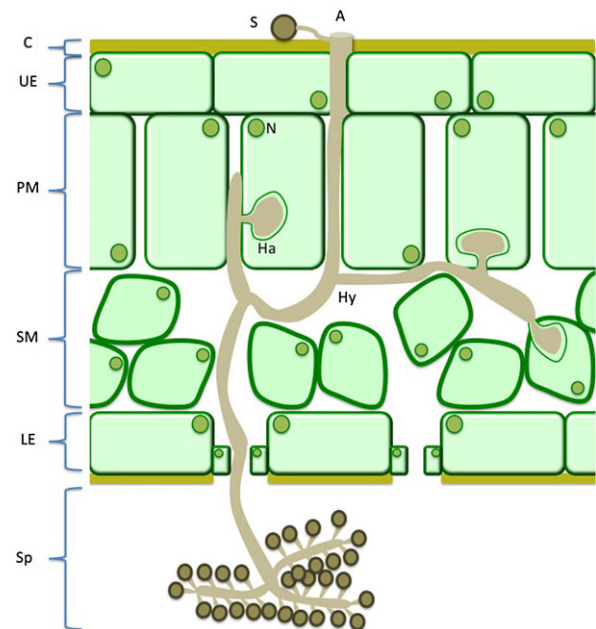


Fig. 4 Diagram depicting a compatible interaction between *Hyaloperonospora arabidopsis* and *Arabidopsis* initiated by a sporangiospore landing on the leaf surface. A, appressorium; C, cuticle; Ha, haustorium; Hy, hyphae; LE, lower epidermis; N, nucleus; PM, palisade mesophyll cells; S, sporangiospore; SM, spongy mesophyll cells; Sp, mature sporangiospore; UE, upper epidermis. Note: sporangiospores are not drawn to scale.

3. PHYTOPHTHORA RAMORUM

Phytophthora ramorum is the most destructive disease of oaks worldwide and the cause of sudden oak death, sudden larch death and ramorum blight (Brasier and Webber, 2010; Grünwald *et al.*, 2008; Rizzo *et al.*, 2005; Werres *et al.*, 2001) (Fig. 5). The host range of *P. ramorum* is one of the widest of any *Phytophthora* spp., and includes many species of hardwood trees and ornamentals. To date, four distinct clonal lineages have been recognized by various molecular markers and two mating types have been found (Grünwald *et al.*, 2009; Van Poucke *et al.*, 2012). These lineages appear to be anciently diverged, yet have emerged repeatedly in both North America and Europe (Goss *et al.*, 2009a; Grünwald *et al.*, 2012) (Fig. 6). Lineages NA1, NA2 and EU1 are currently found in Canada and the USA, whereas EU1 and EU2 exist in Europe (Goss *et al.*, 2009b, 2011; Grünwald *et al.*, 2012; Van Poucke *et al.*, 2012). These lineages differ in aggressiveness in controlled assays, but field experiments have not been conducted given quarantine restrictions (Elliott *et al.*, 2011; Grünwald *et al.*, 2008; Hüberli *et al.*, 2012; Kasuga *et al.*, 2012; Van Poucke *et al.*, 2012). Unique ecological attributes, such as a wide host range and survival in dry, hot Mediterranean summers, combined with the ability to reproduce from chlamydospores, are thought to provide the basis for sudden oak death in California (Garbelotto and Hayden, 2012).

The availability of the genome sequence just a few years after the identification of the pathogen provided rapid and novel insights into the biology of this pathogen (Tyler *et al.*, 2006). Like other *Phytophthora* spp., *P. ramorum* has a large number of candidate effectors interacting with the plant hosts, including RXLR, CRNs and the necrosis and ethylene-inducing peptide 1 (Nep1)-like protein (NLP) gene families (Goss *et al.*, 2013; Tyler *et al.*, 2006). RXLR effectors diversify rapidly, despite the clonality of this organism, using mechanisms such as loss or gain of repeated domains, recombination or gene conversion among paralogues and selection on point mutations (Goss *et al.*, 2013). Recent work has focused on the discovery of endogenous small RNAs and description of the silencing machinery (Fahlgren *et al.*, 2013). *Phytophthora ramorum* and two other *Phytophthora* spp. examined produce two primary, distinct 21- and 25-nucleotide small RNA classes, including a novel microRNA family. Two argonaute classes and two dicer-like proteins appear to be involved in each pathway (Fig. 7), but this remains to be formally tested. Epigenetic mechanisms have been implicated recently in phenotypic diversification, namely colony morphology, colony senescence and virulence on coast live oak and California bay laurel (Kasuga *et al.*, 2012).

Phytophthora ramorum provides a unique opportunity for the study of the evolution of a genome that has two mating types, yet appears to lack sexual reproduction in the known field populations. The epigenome, transcriptome and proteome of *P. ramorum* remain a mystery. The apparent documented phenotypic variation driven by host association might be epigenetic in nature and invites further study. Several basic questions remain unanswered. Why does *P. ramorum* have such a large number of RXLR effectors, given that the genome is adapted to a wide host range? What are the genes conferring a wide host range compared with *P. infestans* and *P. sojae*? Is there a centre of origin? This centre might be located in Asia where the closest relative, *P. lateralis*, has been found in an old growth *Chamaecyparis* forest (Brasier *et al.*, 2010). The discovery of a centre of origin might also reveal a host with which *P. ramorum* might have co-evolved, providing a mechanism of *R* gene discovery, a critical tool needed for the management of sudden oak death. Comparative genomics of related species, such as *P. lateralis*, *P. syringae* and *P. hibernalis*, amongst others, will provide novel insights into core effectors and genes under purifying selection in the clade, but diverged among clades.



Fig. 5 Impact of sudden oak death in California. Tanoak mortality evidenced by defoliated or wilted canopies on the Bolinas Ridge at Mt. Tamalpais, Marin County, CA, USA. Photograph courtesy of Janet Klein (Marin Municipal Open Space District).

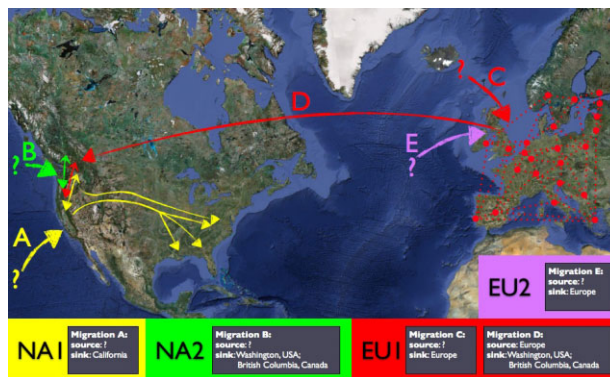


Fig. 6 Inferred pattern of migration of the four clonal lineages of *Phytophthora ramorum*. Modified from Grünwald *et al.* (2012).

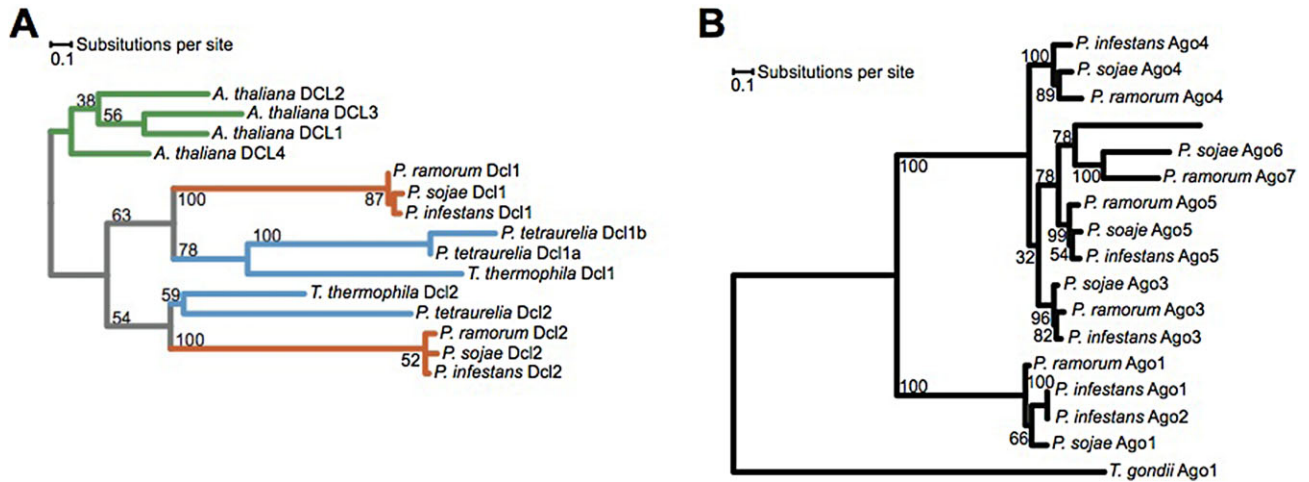


Fig. 7 Silencing machinery in *Phytophthora*. Phylogenetic placement of dicer-like (DCL) (A) and argonaute (Ago) (B) proteins in the genus *Phytophthora*. For more details, see Fahlgren *et al.* (2013). Species correspond to: *Arabidopsis thaliana*, *Paramecium tetraurelia*, *Phytophthora infestans*, *Phytophthora ramorum*, *Phytophthora sojae*, *Tetrahymena thermophila* and *Toxoplasma gondii*.

4. PHYTOPHTHORA SOJAE

Root rot disease of soybean caused by *P. sojae* was first recognized in North America in the 1950s (Hildebrand, 1959; Kaufmann and Gerdemann, 1958). The alternative names *P. megasperma* var. *sojae* and *P. megasperma* f.sp. *glycinea* were commonly used for this species in the past (Erwin and Ribeiro, 1996). Infection of soybean by *P. sojae*, which is a hemibiotroph, typically initiates below ground and eventually produces spreading cankers that destroy root tissues and travel up the stem (Fig. 8). The pathogen thrives in wet conditions and in compacted or heavy clay soils. Motile, water-borne zoospores are released from sporangia and are attracted to soybean root exudates (Morris and Ward, 1992). *Phytophthora sojae* is homothallic and creates abundant, thick-walled sexual oospores that are long lived and provide a soil-borne inoculum. *Phytophthora sojae* has a narrow host range and its economic damage is limited to soybean. It has been suggested that *P. sojae* originated in North America as a pathogen of lupins, as some 26 species of the genus *Lupinus* are susceptible to infection (Erwin and Ribeiro, 1996). In laboratory tests, *P. sojae* can also be made to infect lima bean (*Phaseolus lunatus*), string bean (*Phaseolus vulgaris*) and cranesbill (*Geranium carolinianum*) (Erwin and Ribeiro, 1996; Hildebrand, 1959). *Phytophthora sojae* is one of the most damaging disease problems confronting soybean growers (Wrather and Koenning, 2006). The management of *P. sojae* in soybean has primarily relied on breeding for resistance.

Research on *P. sojae* has focused on the mechanisms of pathogen virulence, host resistance and the molecular basis of recognition between *P. sojae* and soybean (Dorrance and Grünwald, 2009; Gijzen and Qutob, 2009; Tyler, 2007). *Phytophthora sojae*, together with *P. ramorum*, was the first oomycete to have its genome sequenced (Tyler *et al.*, 2006). Genomic information, including gene expression data and multiple genome sequences, have driven rapid progress (Qutob *et al.*, 2000; Torto-Alalibo *et al.*, 2007; Wang *et al.*, 2011). The genome of *P. sojae*, and later other oomycetes (Haas *et al.*, 2009), was found to be partitioned into stable regions rich in housekeeping genes displaying extensive synteny with other oomycete genomes, interspersed with dynamic, transposon-rich regions that contained rapidly evolving genes implicated in virulence (Jiang & Tyler, 2012; Tyler *et al.*, 2006) (Fig. 9). Multiple large, rapidly diversifying families of genes with functions in virulence were discovered (Jiang and Tyler, 2012; Tyler *et al.*, 2006), including hydrolases, hydrolase inhibitor proteins,

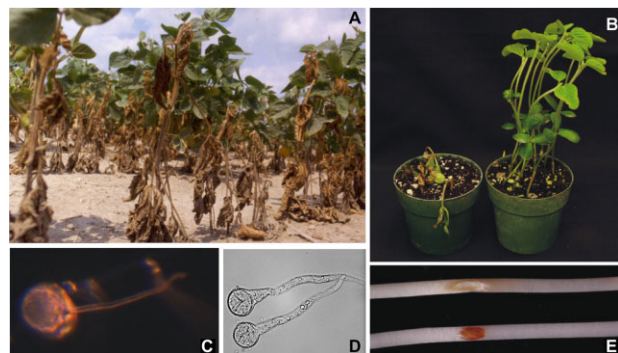


Fig. 8 *Phytophthora sojae*. (A) Diseased soybean plants in the field, infected with *P. sojae*. Plant height is 20–30 cm. (B) Susceptible (left) and resistant (right) soybean plants inoculated in the stem with *P. sojae*, 7 days after infection, illustrating *R*-gene-mediated resistance. Pots are 10 cm in diameter. (C) Germinating oospore of *P. sojae* growing on water agar. Oospore is 35 μ m in diameter. (D) Germinating *P. sojae* cysts growing on water agar. Cysts are 15 μ m in diameter. (E) Etiolated soybean hypocotyls inoculated with a 10- μ L water droplet containing 10^3 zoospores from a virulent (top) and avirulent (bottom) strain of *P. sojae*, 48 h after infection, illustrating strain-specific variation in avirulence effectors and the hypersensitive response. Soybean hypocotyls are 5 mm in diameter.

toxin-like proteins, such as the NLP family (Qutob *et al.*, 2006), and two huge, diverse classes of effector proteins (RXLR and CRN effectors) (Haas *et al.*, 2009; Jiang *et al.*, 2008; Tyler *et al.*, 2006) that can cross into the cytoplasm of host plant cells (Dou *et al.*, 2008; Kale *et al.*, 2010) (Fig. 9). All 11 avirulence genes cloned from *P. sojae* proved to encode RXLR effectors detected by host R proteins (Jiang and Tyler, 2012) (Fig. 9). Genetic inheritance studies in *P. sojae* using molecular markers led to discoveries of high-frequency gene conversion (also referred to as loss of heterozygosity) (Chamnanpant *et al.*, 2001) and epigenetic silencing (Qutob *et al.*, 2013) as important mechanisms underlying pathogen variation.

Many areas of future research into *P. sojae* will be shared with those of other oomycete and fungal pathogens. One area will be to define in detail how apoplastic and host cell-entering effectors of *P. sojae* manipulate soybean physiology to enable infection, including the soybean molecules targeted by the effectors (Fig. 9) and the mechanisms by which RXLR and CRN effectors gain entry (Fig. 9). The identification of the *P. sojae* effectors most important for infection (e.g. using new tools for genetic manipulation, such as tailored nucleases) should lead to new targets for disease control. The cell biology of *P. sojae* infection (Enkerli *et al.*, 1997) must be defined in much more detail, incorporating the spatial distributions of pathogen and host transcripts, proteins and metabolites. Understanding fully the role of epigenetic mechanisms in the generation of variation in pathogen populations will also be important for the design of effective strategies for disease control. Sequencing the genomes and transcriptomes of numerous isolates of *P. sojae* and its nearest sister species will shed light on the genetic adaptability of the pathogen. In practical terms, a large translational research effort will be required to convert the rapidly accumulating knowledge about the basic biology and pathology of *P. sojae* and other oomycetes into effective disease control solutions.

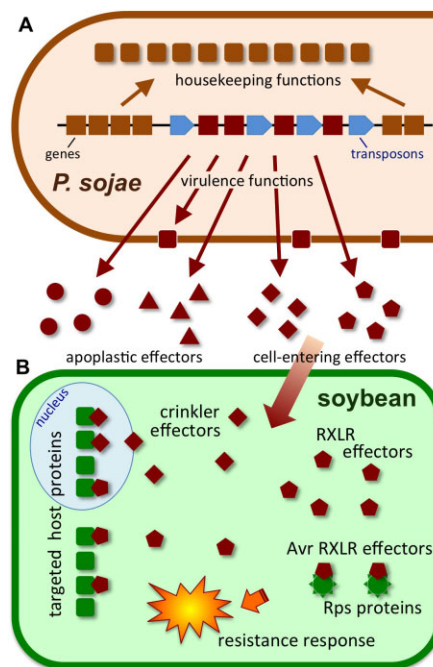


Fig. 9 Large diverse families of virulence proteins encoded by the *Phytophthora sojae* genome. (A) The *Phytophthora sojae* genome contains clusters of conserved housekeeping genes (brown) that have conserved orders among *Phytophthora* species, separated by dynamic transposon-rich regions that contain genes (red) encoding virulence proteins, many of which are secreted. (B) Secreted virulence proteins (effectors) may act in the apoplast, or be transported inside the cell. Cell-entering effectors may have targets in the nucleus or cytoplasm, and may be detected by resistance proteins (Rps; resistance against *P. sojae*).

5. PHYTOPHTHORA CAPSICI

Phytophthora capsici is a highly destructive invasive pathogen that attacks solanaceous (pepper, tomato), legume (lima and snap beans) and most cucurbit hosts (Hausbeck and Lamour, 2004; Leonian, 1922). Disease is favoured by warm (25–28 °C) and wet conditions, and the asexual epidemiology is often explosive (Granke *et al.*, 2009). Initial infection is biotrophic, followed by transitions within 24–48 h to necrotrophy and the production of deciduous sporangia on the surface of infected tissues (Fig. 10). The importance of the sexual stage differs by region. In Peru and Argentina and across much of China, long-lived and widely dispersed clonal lineages dominate (Gobena *et al.*, 2012; Hu *et al.*, 2013; Hurtado-Gonzales *et al.*, 2008; Sun *et al.*, 2008). This is in contrast with the USA, South Africa and the northern provinces of China, where populations have high levels of genotypic diversity and outcrossing is frequent (Dunn *et al.*, 2010; Gobena *et al.*, 2012; Meitz *et al.*, 2010). Once introduced to a field site, *P. capsici* is difficult to control and often impossible to eradicate. *Phytophthora capsici* grows rapidly and sporulates heavily on simple media, and isolates from sexual field populations are often fecund (Gobena *et al.*, 2012; Lamour and Hausbeck, 2000). *Phytophthora capsici* is one of the most genetically diverse eukaryotic organisms yet described, and there is significant genetic variation in the form of single nucleotide polymorphisms within individual genomes and across world populations (Lamour *et al.*, 2012).

Current research includes the investigation of resistance (host and nonhost) in commercial vegetable and experimental plants, the discovery and characterization of genes and proteins driving pathogenicity and virulence, and studies to measure the evolution of natural and laboratory populations. Under controlled conditions, *P. capsici* infects at least 26 plant families (Granke *et al.*, 2012). Natural resistance to *P. capsici* in pepper and cucurbits appears to be rare (Mallard *et al.*, 2013) but, in tomato, it may be extensive (Quesada-Ocampo and Hausbeck, 2010). Recent molecular studies of the CRN class of effectors have indicated that they are often localized to the host nucleus and play an important role in the infection process (Chen *et al.*, 2013; Stam *et al.*, 2013a, 2013b). Transcriptome studies using RNAseq and microarrays have indicated dramatic changes from pre-infective spores through the early biotrophic and later necrotrophic stages of infection (Chen *et al.*, 2013; Jupe *et al.*, 2013). Studies of individual effectors have suggested that some manipulate the host to allow infection, whereas others trigger plant cell death (Chen *et al.*, 2013; Feng and Li, 2013; Feng *et al.*, 2010; Sun *et al.*, 2009).

A high-quality reference genome and a high-density single nucleotide polymorphism-based genetic linkage map have been completed recently (Lamour *et al.*, 2012). These important resources illuminated an important source of asexual genetic variation, known as loss of heterozygosity (Lamour *et al.*, 2012). Loss of heterozygosity occurs when variable length tracts (300 bp to >1 Mbp) of the diploid genome switch to one of the two possible haplotypes. Loss of heterozygosity has been described in laboratory-produced sexual progeny, field isolates maintained on agar medium and field isolates genotyped directly from naturally infected tissue (Gobena *et al.*, 2010; Hu *et al.*, 2013; Hulvey *et al.*, 2010). Loss of heterozygosity is associated with spontaneous switches from the A2 to the A1 mating type, loss of pathogenicity and reduced virulence (Hu *et al.*, 2013; Lamour *et al.*, 2012).

The diversity and plasticity of *P. capsici* present challenges and unique opportunities for research (Fig. 10). Future research questions include the following. What is the significance of a highly diverse effector arsenal? Are these effectors important for host-specific interactions and the broad host range? How important is loss of heterozygosity in laboratory and field populations? The genomic plasticity of *P. capsici* and the ease of laboratory manipulation provide a unique opportunity to measure genome stability and adaptive evolution at a fine scale, and may provide useful insights into this and other oomycete pathogens (Lamour and Hu, 2013).

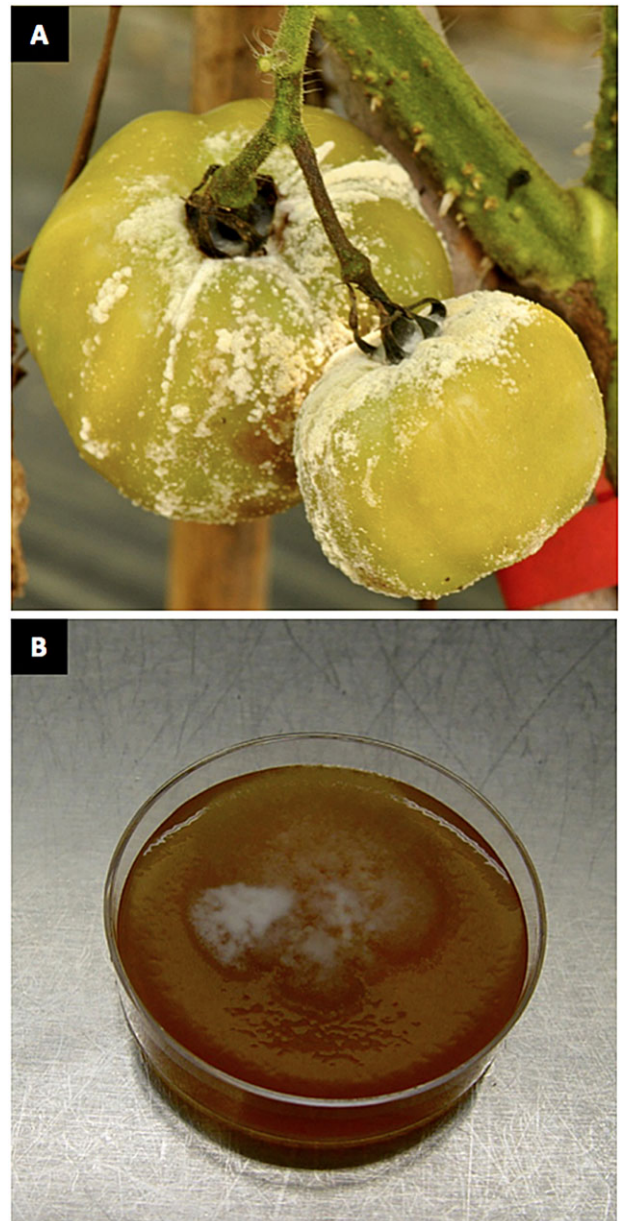


Fig. 10 Heavy sporulation and spontaneous morphological variation in the vegetable pathogen *Phytophthora capsici*. (A) Naturally infected tomato fruit with sporangium production on the surface of the fruit. (B) A single zoospore-derived field isolate of *P. capsici* sectoring on V8 agar medium following long-term storage.

6. *PLASMOPARA VITICOLA*

The obligate biotrophic oomycete *Pl. viticola*, the causal agent of grape downy mildew, is native to North America and was inadvertently introduced into Europe at the end of the 19th century (Millardet, 1881; Viennot-Bourgin, 1949), causing severe damage to *Vitis vinifera*, which had evolved in the absence of this pathogen (Galet, 1977; Gessler *et al.*, 2011). *Plasmopara viticola* belongs to the family Peronosporaceae; its life cycle includes an asexual multiplication phase occurring during the plant vegetative period and a sexual phase that ensures pathogen overwinter survival (Wong *et al.*, 2001). The pathogen overwinters as oospores in dead leaf lesions or as mycelium in infected twigs. In spring, and in particular during rainy periods, oospores germinate to produce sporangia, which are transported by wind or water to the wet leaves near the ground, which they infect through stomata on the lower surface. The mycelium then spreads into the intercellular spaces of the leaf from which sporangiophores arise and emerge through the stoma, ready to start new infection cycles (Gobbin *et al.*, 2005). Downy mildew affects leaves, fruits and shoots, with young tissues being particularly susceptible to infection (Fig. 11) (Kennelly *et al.*, 2007). A disease cycle may take from 5 to 18 days, depending on the temperature, humidity and varietal susceptibility (Agrios, 2005; Gessler *et al.*, 2011).

Downy mildew is still most destructive in Europe and in the eastern half of the USA, where it may cause severe epidemics year after year (Agrios, 2005; Madden *et al.*, 1995). When the weather is favourable and no protection against the disease is provided, downy mildew can easily destroy up to 75% of production in a single season (Madden *et al.*, 2000; Rossi and Caffi, 2012). Occasionally, *Pl. viticola* can be destructive in other humid parts of the world in which *V. vinifera* is cultivated.

In the last 50 years, research on grapevine downy mildew has focused on the pathogen life cycle and epidemiology (reviewed in Gessler *et al.*, 2011), and on the genetic identification of host resistance loci for the establishment of molecular markers (revised in Töpfer *et al.*, 2011). In the last decade, studies on *Pl. viticola* have highlighted its enormous potential in the development of fungicide resistance (Blum *et al.*, 2010; Chen *et al.*, 2007) and in breaking down plant resistance of interspecific hybrids, such as 'Bianca' (Casagrande *et al.*, 2011; Peressotti *et al.*, 2010) and 'Regent' (Delmotte *et al.*, 2013). Population genetic studies have been carried out (Fontaine *et al.*, 2013 and references therein) to assess pathogen dynamics and diversity in Europe. Moreover, it has been proposed that grapevine downy mildew is not caused by a single species, but instead by a complex of cryptic species that have diverged on Vitaceae (Rouxel *et al.*, 2013). Trade-offs between the size and number of sporangia produced have been reported, leading to ecological advantages for this pathogen (Delmotte *et al.*, 2013). For these reasons, the *Vitis-Pl. viticola* pathosystem was elected as a prime candidate for the study of host specialization of biotrophic plant pathogens (Rouxel *et al.*, 2013) and pathogen adaptation to partial host resistance (Delmotte *et al.*, 2013).

Control methods against *Pl. viticola* are essentially based on the use of chemical fungicides and the application of disease models that serve as decision support systems (Lalancette *et al.*, 1987; Rossi *et al.*, 2009, 2013; Vercesi *et al.*, 2010). As a result of the growing public concern on the use of chemical pesticides, and the need to adhere to the European Directive 2009/128/EC encompassing a framework 'to achieve a sustainable use of pesticides' by promoting Integrated Pest Management (IPM), the search for alternative methods for the control of *Pl. viticola* on grapevine appears to be urgent. Several alternative approaches have been proposed in the last 20 years, but none has been transferred to practical use (Gessler *et al.*, 2011). Moreover, the lack of precise characterization of *Pl. viticola* isolates is dramatically limiting the reliable control of the pathogen. Although molecular studies have previously confirmed a high diversity in the pathogen population, there is a surprising lack of phenotypic characterization of pathotype strains or races which could be used to study the mechanisms of interaction with host genotypes with different levels of resistance (Gómez-Zeledón *et al.*, 2013). In conclusion, interdisciplinary studies and consistent resources should be invested for the study of the *Pl. viticola-V. vinifera* pathosystem in order to find new, effective alternatives for the IPM of this highly adaptive and destructive pathogen.



Fig. 11 Downy mildew symptoms with well-evident sporangiophores on the lower side of a grape leaf (A) and a young cluster (B).

7. PHYTOPHTHORA CINNAMOMI

Some call it the 'biological bulldozer' for its capacity to destroy natural plant communities across the globe, and disease caused by *P. cinnamomi* has broad and economically important impacts in forestry and horticulture, and in the nursery industry. Like other *Phytophthora* spp., *P. cinnamomi* has a number of strategies for survival, propagation and dissemination. The motile zoospore is recognized as the main infective propagule following encystment and attachment to roots and stems (Fig. 12). Recent work has suggested that, in addition to chlamydospores, *P. cinnamomi* is also associated with lignituber formation which enables survival under harsh conditions (Jung *et al.*, 2013). Both A1 and A2 mating types are pathogenic, but wide differences in the distribution and frequency of occurrence suggest that the A2 mating type is more invasive and is generally recognized as being the more aggressive of the two mating types. Where introduced, this pathogen has had enormous impacts on natural systems, including those of Australia, southern Europe and the USA. In Australia, for example, it has been estimated that >3000 largely endemic plant species from numerous plant families are under threat, and a new National Threat Abatement Plan has been urgently implemented (Australian Government Department of Environment, 2014) (Fig. 13). The control of disease, especially across the large areas of native vegetation affected, is still a great problem, but some success has been achieved by the use of phosphite in both natural systems and in agriculture (Akinsanmi and Drenth, 2013; Crane and Shearer, 2014), by the use of calcium amendments to soil (Serrano *et al.*, 2013) and by containment and eradication of spot infections (Dunstan *et al.*, 2010). Climate change is predicted to have a significant influence on the intensity and distribution of disease (Thompson *et al.*, 2014).

Phytophthora cinnamomi is a challenging organism to work with. Sequencing of its genome has recently been completed (JGI; <http://genome.jgi-psf.org/>) and has opened up a variety of opportunities to investigate the critical features that enable this pathogen to be so wide-ranging in its hosts. A real change in emphasis in research on *P. cinnamomi* has occurred in recent times, with an increasing number of groups using genomic, transcriptomic and proteomic approaches to understand the pathogen and/or its interaction with the host (for example, Reeksting *et al.*, 2014). Experimental plants, including *Arabidopsis*, *Zea mays* and *Eucalyptus*, for which comprehensive genomic information is available, are increasingly being used as host-pathogen platforms (for example, Allardye *et al.*, 2013; Dempsey *et al.*, 2012; Rookes *et al.*, 2008). Fundamental studies on *P. cinnamomi* plant cell wall-degrading enzymes and the genes that encode them are being undertaken (Hee *et al.*, 2013; Adrienne Hardham, Australian National University, Canberra, ACT, Australia, personal communication), as well as examination, at the molecular level, of defence pathways and their control (Eshraghi *et al.*, 2014; Gunning *et al.*, 2013).

High-throughput, genome sequencing is emerging as an exciting way to examine the pathogen, its hosts and the soil environment, and is now being used to examine pathogen ecology and diversity within soils, and for the analysis of the response of host plants (Treena Burgess, Murdoch University, Perth, WA, Australia, personal communication). Much of our progress in understanding host-pathogen interactions will come from such approaches. We still lack knowledge on why plants are resistant to this pathogen and what is the basis for induced resistance, for example, following phosphite treatment. The search for host resistance to *P. cinnamomi* is an active area in which signalling pathways and their control are being elucidated. Mapping the disease over the large invaded areas is still not straightforward, although advances in remote sensing, high-resolution digital photography, hyperspectral imaging (David Guest, Sydney University, Sydney, NSW, Australia, personal communication) and 'drone' technology will probably see our ability in this area being greatly enhanced in the near future. The era of nanotechnology offers new opportunities for the delivery of molecules that can influence disease outcome, yet this area has not been explored to date, although preliminary research with various nanoparticle systems (Hussain *et al.*, 2013; Nadiminti *et al.*, 2013) has indicated that these may be useful in delivering molecules, including pathogen effectors, DNA and miRNA, which may modify host processes and their response to pathogen infection.

Future research will focus on the following questions. (i) What are the virulence factors that enable *P. cinnamomi* to infect and colonize susceptible species and therefore should be targeted for control? (ii) What constitutes host resistance and how can it be manipulated? Can we look to nanotechnology for some answers? (iii) Is phosphite the only chemical that we can use to control *P. cinnamomi*, and how does phosphite alter the host response?

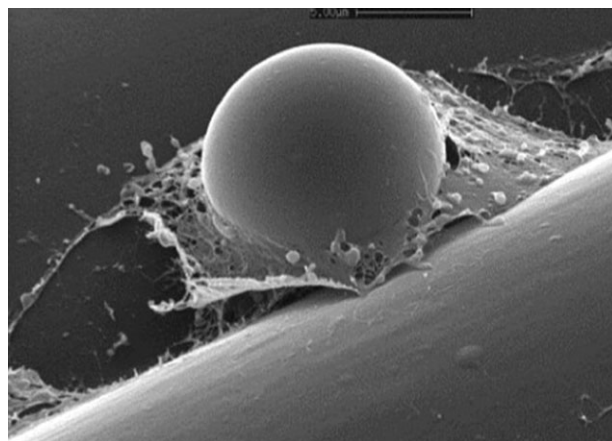


Fig. 12 Cryo-scanning electron micrograph of a *Phytophthora cinnamomi* cyst between two epidermal cells on a root of tobacco. Note the adhesive material that surrounds the cyst that has been expelled by zoospore peripheral vesicles. Photograph courtesy of Adrienne Hardham, Australian National University.

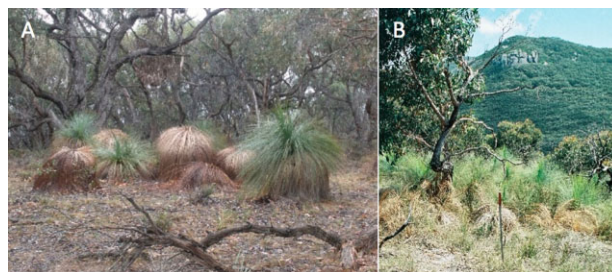


Fig. 13 (A) Individual plants of *Xanthorrhoea australis* (austral grass tree), a highly susceptible native Australian species, infected by *Phytophthora cinnamomi* within a dry sclerophyll eucalypt forest at Anglesea, Victoria, Australia. Note the dead and dying plants that have brown, collapsed leaves compared with the healthy green and erect leaves of plants which are yet to be killed. These individual plants range in age from approximately 20 years (the smallest in the centre) to around 70 years (green individual on the right of the image). (B) Advancing disease front caused by invasion by *P. cinnamomi* in *Xanthorrhoea australis*-dominated understorey in eucalyptus open forest at Wilsons Promontory, Victoria, Australia. The disease has moved from the foreground of the picture, where all susceptible vegetation including *X. australis* has been killed, and its progress can be seen as a line of dead and dying *X. australis* (brown collapsed plants) at the disease margin. Healthy green plants behind them will soon be killed. Loss of the major understorey components, as in the foreground, results in complete structural change and loss of all susceptible species.

8. PHYTOPHTHORA PARASITICA

Phytophthora parasitica (= *P. nicotianae*) is a worldwide distributed pathogen (Erwin and Ribeiro, 1996). Primarily known to cause tobacco black shank and citrus root rot and gummosis (Fig. 14), it is also responsible for severe foliar and fruit diseases, as well as root and crown rots on herbaceous and perennial plant species in more than 250 genera, including solanaceous crops (Fig. 15), and horticultural and fruit trees (Cline *et al.*, 2008). *Phytophthora parasitica* produces both asexual zoospores and thick-walled sexual oospores. Oospores constitute a potential source of genetic variation and, with resting chlamydozoospores, contribute to survival in unfavourable conditions in soil or within infected plant tissues.

Contrasting with the broad host range observed at the species level, most individual *P. parasitica* isolates display host preference (Erwin and Ribeiro, 1996). Frequent cases of differential virulence on a range of hosts have been revealed (Colas *et al.*, 1998; Matheron and Matejka, 1990), pointing out the

need to decipher the genetic structure of *P. parasitica* on a global scale. In agreement with pathogenicity tests, recent single nucleotide polymorphism analyses conducted with mitochondrial and nuclear genes have revealed a specific association between the host of origin and genetic grouping which was particularly evident for tobacco and citrus isolates (Mammella *et al.*, 2011, 2013). In contrast, no clear genetic structure was revealed for isolates from other hosts, especially potted ornamentals in nurseries. A significant geographical structuring was revealed for tobacco, but not for citrus, isolates (Bonnet *et al.*, 1994; Colas *et al.*, 2001; Mammella *et al.*, 2013). Further studies relying on whole-genome sequencing programmes (see below) are necessary to determine whether these molecular groups represent evidence of physiological races, pathotypes or even subspecies within *P. parasitica*.

As some isolates may also infect many hosts, including *Arabidopsis thaliana* (Attard *et al.*, 2008, 2010), *P. parasitica* has emerged as an ideal pathogen to develop specific studies with the aim to advance our knowledge on the mechanisms underlying general pathogenicity and those governing host specificity. Furthermore, *P. parasitica* is phylogenetically related to

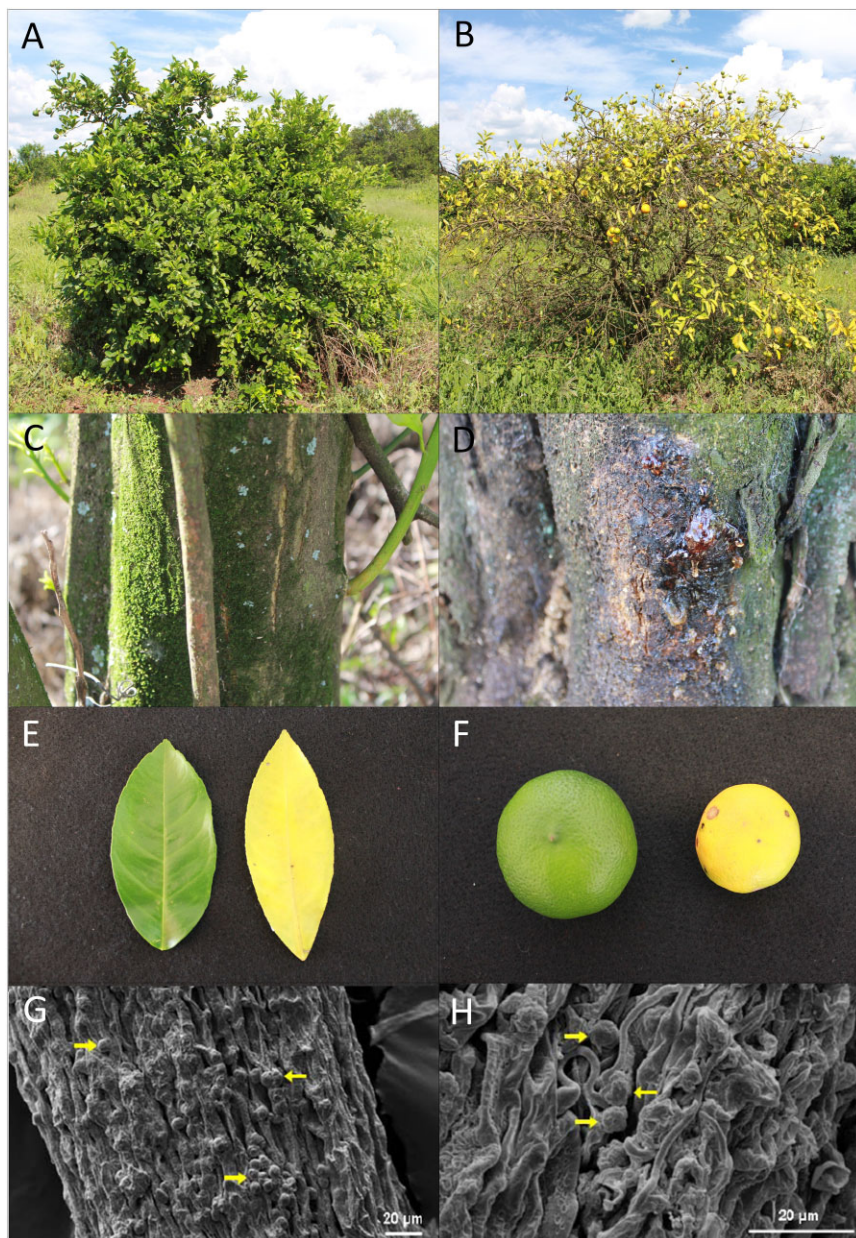


Fig. 14 Impact of *Phytophthora parasitica* infection on citrus plants. (A, B) Five-year-old citrus plants not infected and infected, respectively, with *Phytophthora parasitica*. (C, D) Symptoms of *P. parasitica* on stems: (C) not infected; (D) infected plant displaying gummosis symptoms. (E, F) Leaves and fruits of infected (left) and healthy (right) plants. (G, H) Scanning electron microscope images of citrus fine roots infected with *P. parasitica*. Yellow arrows show encysted zoospores and germ tube of *P. parasitica*. Bar represents 20 μ m. (A–F) Photographs courtesy of R. J. D. Dalio. (G, H) Photographs courtesy of M. E. Escanferla.

P. infestans in *Phytophthora* clade 1, and overlaps its host range, which includes potato (Taylor *et al.*, 2008). Comparative analyses on these two species varying in genome size (83 and 240 Mb for *P. parasitica* and *P. infestans*, respectively) will facilitate the understanding of the evolution behind pathogenicity and host range among *Phytophthora* spp. Through the sequencing of the genome of isolates of diverse host range and geographical origins, the international '*Phytophthora parasitica* genome initiative' project will enable the characterization of genes that determine host range. In-depth genomic and transcriptomic analyses of 14 sequenced genomes are currently being performed. Special efforts are being devoted to characterize the repertoire of effector proteins. As a broad host range pathogen, *P. parasitica* provides a unique opportunity for intra- and interspecific comparative analyses, looking for the presence of various effector families, their organization, their role in plant recognition and infection, and their evolution among strains and species that display broad or restricted host ranges. The identification of conserved effector groups, as well as of other pathogenicity genes, will allow the evaluation of the evolutionary pressures of exposure to different host defence responses to the diversification of effectors and their role in adaptation to host plants.

Phytophthora parasitica has not been as extensively studied as its economic importance should warrant. However, it is likely to gain importance in the foreseeable future for the following reasons. First, it is prominent in nurseries of potted ornamentals and fruit tree species, the trade of which seems to represent one of the most efficient dissemination pathways of *Phytophthora* (Moralejo *et al.*, 2009; Olson and Benson, 2011). This makes *P. parasitica* an ideal species to study diffusion pathways of *Phytophthora* and other soil-borne pathogens on a global scale. For aesthetic reasons, the ornamental industry requires the extensive use of anti-oomycete chemicals (Olson *et al.*, 2013). The global nursery trade thus increases the risk of the rapid spread of resistant *P. parasitica* strains to new areas following the inappropriate use of fungicides, and constitutes a growing threat to local agriculture and natural ecosystems (Brasier, 2008). In addition, *P. parasitica* will probably benefit from the warming climate. Its host range generally includes those of other species of prime economic importance (*P. infestans*, *P. capsici*, *P. citrophthora*), but generally requires warmer conditions than these potential competitors (Erwin and Ribeiro, 1996). Consequently, the foreseen global warming is likely to provide a catalyst for the geographical expansion of this species, as has already been proposed in Mediterranean climates (Andres *et al.*, 2003; Saadoun and Allagui, 2008) and in eastern India (Guha Roy *et al.*, 2009).



Fig. 15 Severe infection of brinjal fruit with *Phytophthora parasitica*. Typical symptoms are brown, soft, water-soaked patches which rapidly cover the whole fruit. Brinjal is also known as aubergine or eggplant (*Solanum melongena*). Photograph courtesy of S. Guha Roy.

9. *PYTHIUM ULTIMUM*

Pythium resides in the peronosporalean lineage of oomycetes, with *Phytophthora* and downy mildews (Dick, 2001). One of its most significant members is *Py. ultimum*, which causes damping off and root rot on >300 diverse hosts, including corn, soybean, wheat, Douglas fir and ornamentals (Farr and Rossman, 2014). *Pythium ultimum* is a common inhabitant of fields, ponds and streams, and of decomposing vegetation worldwide. Contributing to the ubiquity of the species is its ability to grow saprotrophically in soil and plant residue, a trait shared by most *Pythium* spp., but not other peronosporaleans, which must colonize living hosts. *Pythium ultimum* is also homothallic, producing oospores capable of long-term survival (Martin and Loper, 1999). Mycelia and oospores in soil can thus initiate infections of seeds or roots, leading to wilting, reduced yield and mortality (Fig. 16A). Disease management is difficult, but focuses on sanitation, fungicides and biological control, particularly in glasshouses. Quantitative resistance has been reported in several hosts, but has a limited effect (Lucas and Griffiths, 2004; Wang and Davis, 1997).

The role of asexual spores depends on the strain. *Pythium ultimum* is a species complex that includes *Py. ultimum* var. *ultimum* and var. *sporangiferum*, which have indistinguishable internal transcribed spacer (ITS) sequences (Schroeder *et al.*, 2013). Sporangia and zoospores are produced rarely by the former, but abundantly by the latter. *Pythium ultimum* var. *ultimum* forms sporangia-like hyphal swellings which germinate to yield infective hyphae (Fig. 16B; Stanghellini and Hancock, 1971). Similar to other oomycetes, it also produces sexual oospores (Fig. 16C).

Genome sequencing of several *Pythium* spp., including *Py. ultimum* var. *ultimum* and var. *sporangiferum*, has provided insight into their biology (Adhikari *et al.*, 2013; Levesque *et al.*, 2010). Annotated in the c. 43-Mb genomes of two subspecies are 15 290 and 14 086 genes, respectively, which is less than the smallest *Phytophthora*, but more than downy mildews. Whether this difference in gene number is significant requires more analysis, as the two assemblies vary in quality and only single isolates were sequenced. Large size variation between isolates was reported on the basis of electrophoretic karyotyping (Martin, 1995).

Nevertheless, it is evident from genome analysis that there are many differences between *Pythium* and *Phytophthora*, including a 25% reduction in *Pythium* of the fraction of proteins that are secreted. In part, this reflects the absence of RXLR effectors, which may not be needed for necrotrophic lifestyles. *Pythium* also lacks haustoria, which appear to be used by biotrophic and hemibiotrophic oomycetes to deliver RXLRs to host cells (Whisson *et al.*, 2007). Another likely sign of the necrotrophy of *Pythium* is its greater abundance of lipases and proteases. However, *Pythium* lacks cutinases and pectin esterases, but this is probably because it infects primarily nonsuberized tissue, whereas *Phytophthora* (which encodes these enzymes) can penetrate plant cuticles. *Pythium* encodes sufficient enzymes for the maceration of host cell walls, but cannot degrade some ubiquitous wall components, such as xylans, which suggests that soluble sugars may be a more important carbon source (Zerillo *et al.*, 2013).

Markers derived from *Py. ultimum* genomes should help reveal what distinguishes the subspecies and provide more resolution to population studies, which can help to identify the sources of outbreaks. Studies of *Py. ultimum* var. *ultimum* have indicated that populations are not clonal and some outcrossing may occur (Francis and St. Clair, 1997). A greater evolutionary question concerns the organization of the *Pythium* genus as a whole. Division of the >120 *Pythium* species over five new genera has been proposed on the basis of the sequence analysis of two loci (Uzhashi *et al.*, 2010). If accepted by the community, *Py. ultimum* would be renamed *Globisporangium ultimum*.

Of much interest is the interaction between *Py. ultimum* and biocontrol agents, such as *Trichoderma*, *Streptomyces*, *Pythium oligandrum* (a mycoparasitic member of the genus) and others (Gracia-Garza *et al.*, 2003; Martin and Loper, 1999). The extent to which these organisms function by altering rhizosphere microflora or attacking *Py. ultimum* directly is an open question (Naseby *et al.*, 2000; Vallance *et al.*, 2012); *Pythium ultimum* competes poorly with organisms having higher saprotrophic capabilities.

Pythium ultimum is just one of many destructive members of the genus, with *Py. aphanidermatum* and *Py. irregulare* also topping lists of important

pathogens (Martin and Loper, 1999). Opportunities for understanding their biology, ecology and evolution have been enhanced by genome sequencing. DNA-mediated transformation has been achieved for several species, but few studies of gene function have been reported (Grenville-Briggs *et al.*, 2013; Weiland, 2003). As core promoter structure appears to be conserved within the Peronosporales, vectors and technologies applied to species, such as *Phytophthora*, should be transferable to *Pythium* (Roy *et al.*, 2013). *Pythium* spp. are easily grown and have the potential to develop into valuable experimental systems for necrotrophic and saprotrophic oomycete lifestyles. Their growth on byproducts of the agro-food industry has also attracted interest as sources of polyunsaturated fatty acids for human consumption (Stredansky *et al.*, 2000).

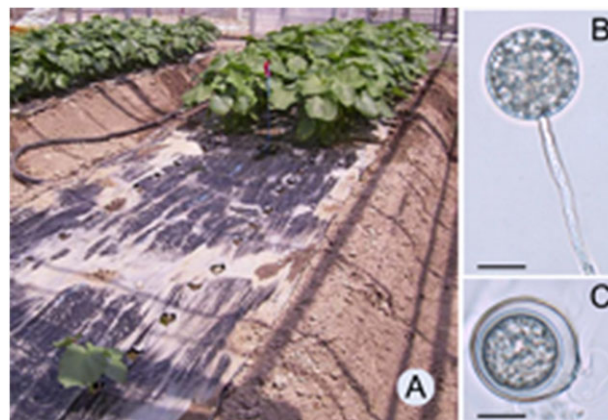


Fig. 16 *Pythium ultimum* var. *ultimum*. (A) Pre-emergence damping-off in okra, resulting in the death of most plants at the front of the row. At the back, disease was controlled using metalaxyl. (B) Terminal hyphal swellings; bar, 10 μ m. (C) Oospore within oogonium; bar, 10 μ m. Images adapted from Kida *et al.* (2006) with permission.

10. ALBUGO CANDIDA

White blister rust is a disease caused in many dicotyledonous plant species by obligate biotrophic parasites. For example, *A. candida* infection of *Brassica juncea* (Indian mustard) has resulted in significant crop losses in India (Awasthi *et al.*, 2012), Canada (Rimmer *et al.*, 2000) and Australia (Kaur *et al.*, 2008). The white rusts, order Albuginales, are phylogenetically distant from the Peronosporales, and probably represent an independent acquisition of biotrophy (Thines and Kamoun, 2010; Thines and Spring, 2005). All *Albugo* species infecting the Brassicaceae were thought to be races of *A. candida*, but molecular studies of isolates from various hosts and locations have led to the description of specialists, for example *Albugo laibachii* on *Arabidopsis thaliana* (Thines, 2014; Thines *et al.*, 2009). Specific *A. candida* races can grow on diverse plant hosts, including Brassicaceae, Cleomaceae and Capparaceae (Thines, 2014). *Albugo* spp. provide an interesting experimental system for the study of plant immune suppression, disease resistance and host–pathogen co-evolution.

Albugo spp. reproduce asexually via zoosporangia, which release flagellated motile zoospores on incubation in water. On the surface of a plant leaf, zoospores settle in stomata, and each extends a germ tube into the substomatal chamber (Holub *et al.*, 1994). Coenocytic hyphae then grow intercellularly through the plant. Small globose haustoria penetrate into plant cells (Soylu *et al.*, 2003). When an *Albugo* infection is mature, zoosporangia rupture the plant epidermis with force and enzymatic digestion (Heller and Thines, 2009). This results in characteristic ‘white blister’ pustules. *Albugo* also has a sexual cycle, producing tough oospores that can survive difficult environmental conditions (Petrie, 1975). During the systemic infection of Brassicaceae hosts, the inflorescences become misshapen, forming so-called ‘stagheads’ (Fig. 17A).

Albugo infection has long been associated with ‘green islands’, where infected tissue appears to be healthy and senescence is delayed. Infection by *Albugo* also greatly enhances susceptibility to co-infections with downy mildews (Bains and Jhoo, 1985; Crute *et al.*, 1994). Cooper *et al.* (2008) investigated the ability of *A. laibachii* and *A. candida* to suppress host immunity. They showed that *A. laibachii* can suppress the ‘runaway cell death’ of *Arabidopsis lsd1* mutants after inoculation with avirulent *H. arabidopsidis*. Furthermore, when pre-infected with virulent *A. laibachii*, resistant *Arabidopsis* accessions were no longer resistant to avirulent *H. arabidopsidis* isolates (Fig. 17B), lettuce downy mildew or powdery mildew. Suppression was also observed on *B. juncea* with *A. candida* and Brassica downy mildew (Cooper *et al.*, 2008). These results suggest that *Albugo* is effective at broad suppression of plant immunity, including effector-triggered immunity.

The first step in understanding how *Albugo* spp. impose such susceptibility is to examine their genomes. Links *et al.* (2011) and Kemen *et al.* (2011) sequenced *A. candida* and *A. laibachii* genomes, respectively. The genomes are around 40 Mb and compact; about 50% of the assemblies consist of coding sequences (see Fig. 18). Both genomes show adaptations to obligate biotrophy; they are missing sulfite oxidases, nitrate and nitrite reductases and, in the case of *A. laibachii*, the whole molybdopterin biosynthesis pathway. The *A. candida* secretome consists of 929 proteins (without transmembrane domains), compared with 672 in *A. laibachii*, perhaps reflecting its wider host range. Within the secretomes, there is no enrichment of putative RXLR effectors. Kemen *et al.* (2011) discovered the CHXC (cysteine, histidine, any amino acid, cysteine) motif at the N-terminus of a class of candidate effectors. The CHXC-containing N-terminus is sufficient to translocate the C-terminus of *P. infestans* AVR3a (an RXLR effector) into host cells (Kemen *et al.*, 2011).

Several *A. candida* races can infect some, but not all, *A. thaliana* accessions and, from crosses between resistant and susceptible accessions, an *R* gene against four *A. candida* races, *WRR4* [encoding a toll interleukin 1 receptor-nucleotide-binding-leucine-rich repeat (TIR-NB-LRR) R protein], was identified (Borhan *et al.*, 2008). *WRR4* can also provide resistance to *A. candida* when transformed into susceptible cultivars of *B. napus* and *B. juncea* (Borhan *et al.*, 2010). In *A. thaliana*, *RAC1* (also encoding a TIR-NB-LRR R protein) confers resistance to *A. laibachii* (Borhan *et al.*, 2004). The inheritance of avirulence of a *B. juncea* isolate (Ac2V) was studied through a cross between two *A. candida* isolates; this work predicted a

single avirulence gene for the incompatibility between Ac2V and *B. rapa* (Adhikari *et al.*, 2003).

There are open questions about *Albugo* from both fundamental and translational perspectives. Thines (2014) speculated that the broad host range of the *A. candida* meta-population is maintained through frequent genetic exchange where the host range of individual isolates overlap. Comparing the genomes of multiple isolates from different hosts would test this hypothesis and build up a clear picture of population variation. This would also aid the discovery of new effector candidates through the identification of secreted proteins under strong selective pressure. A more extensive analysis of *Albugo* effectors should be made. The presence of the CHXC effectors inside host cells needs to be confirmed and the translocation mechanism elucidated. It is unclear which *Albugo* effector proteins are recognized by the few known R proteins. *Arabidopsis thaliana* cannot be colonized by most *A. candida* isolates. The molecular basis for this resistance could be exploited to introduce durable resistance to Brassica crops.

Lastly, and perhaps most interestingly, is the question: how does the remarkable defence suppression by *Albugo* work? We need to define the extent of this suppression. What other pests or pathogens can grow on plants infected with *Albugo*? What changes occur in the microbiome of *Albugo*-infected leaves in the field? Recent data have suggested that *Albugo* could be widespread as an asymptomatic endophyte (Ploch and Thines, 2011). The implications of these infections as a reservoir for (i) *Albugo* and (ii) further *Albugo* suppression-enabled infections by other species remain to be discovered.

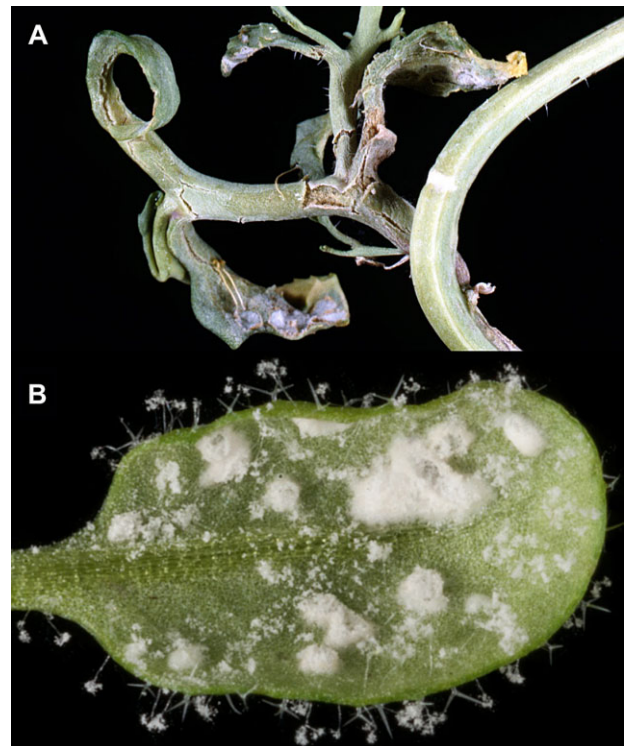


Fig. 17 Disease symptoms of *Albugo* infections. (A) Disease resulting from the infection of *Brassica juncea* with an unknown *Albugo* species. The misshapen inflorescence phenotype is known as a ‘staghead’. (B) An example of immunity suppression by *Albugo laibachii*: *A. thaliana* Col-0 is resistant to *Hyaloperonospora arabidopsidis* Emoy2 via RPP1 but, when pre-infected with *A. laibachii*, can support the growth of both pathogens.

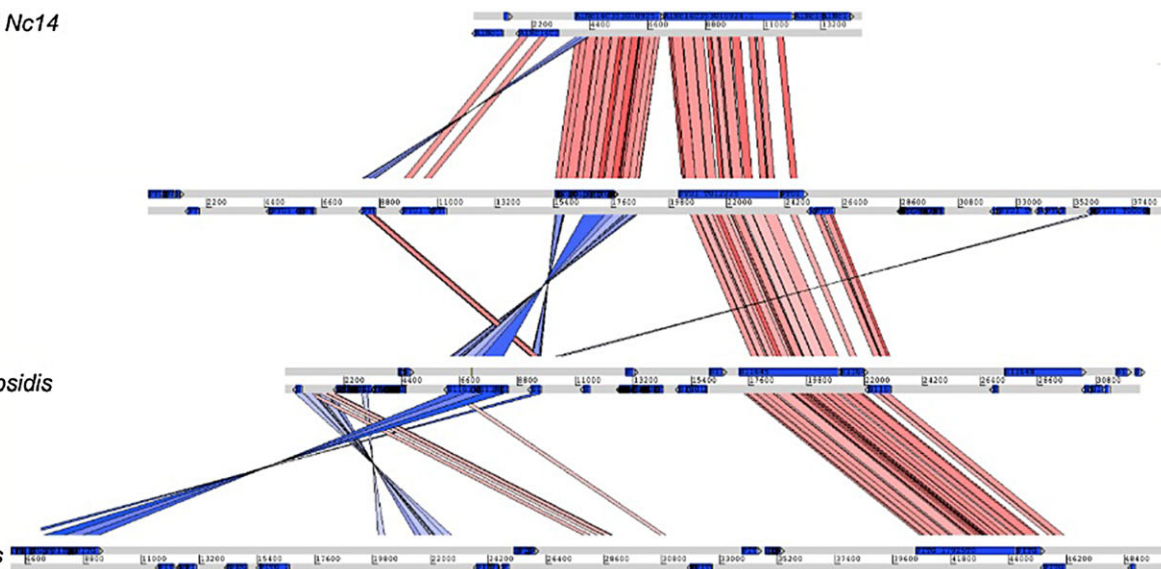
A. laibachii Nc14*P. ultimum**H. arabidopsidis**P. infestans*

Fig. 18 *Albugo* spp. have compact genomes. Synteny between *Albugo laibachii*, *Pythium ultimum*, *Hyaloperonospora arabidopsidis* and *Phytophthora infestans*. The region shown is an example of the relatively dense clustering of genes in *Albugo* species. With increasing genome size, the distance between both genes increases and re-organizations occur (red, synteny without inversion; blue, inverted regions). Reproduced from Kemen *et al.* (2011).

CONCLUSION

Although the study of oomycete plant pathogens has always been an important topic in plant pathology, it has recently taken an even more central role with the advent of genomics and the discovery of large effector repertoires in oomycete genomes. This article provides a benchmark for future trends. Many topics remain to be investigated in more depth. For example, the large number of pathogenic species and the wide diversity of host species and host ranges should provide a rich basis for the investigation of the genetic and physiological mechanisms of host adaptation and specialization. It will also be interesting to track how the Top 10 list will develop in the coming years. Many pathogenic oomycetes among the 33 listed (Table 2) have evolved unique adaptations in their parasitic lifestyle that will undoubtedly reveal fascinating processes and mechanisms. Thus, future research efforts should take into account a diverse spectrum of taxa beyond the widely studied species.

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AUTHOR CONTRIBUTIONS

All authors voted and contributed to the writing of the 10 sections. SK oversaw the voting and writing. Other authors are listed in reverse order based on the species ranking. The species coordinators are listed last in their section and are preceded by other contributors listed in alphabetical order. The species coordinators are: *Albugo candida* (OF and JDG), *Pythium ultimum* (HSJ), *Phytophthora parasitica* (FP), *Phytophthora cinnamomi* (DC), *Plasmopara viticola* (MR and AF), *Phytophthora capsici* (KL), *Phytophthora sojae* (MG and BMT), *Phytophthora ramorum* (NJG), *Hyaloperonospora arabidopsidis* (GvdA and JMcD) and *Phytophthora infestans* (PRJB and FG).

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