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Comparative epidemiology of zoosporic plant pathogens

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Abstract Loss of zoospores has happened independently several times in different phylogenic lines and has, it is claimed, no major phylogenetic significance. But whether or not, how, and under which conditions plant pathogens retain the ability to produce motile asexual spores has fundamental importance from an ecological and epidemiological perspective. Recent molecular investigations of the early evolution of fungi and oomycetes are shedding light on the issue of zoospore loss in organisms able to cause plant diseases. Zoospore loss may have accompanied the development of new forms of dispersal adapted to the terrestrial environment, or the simplification processes which often follow the shift to parasitic or biotrophic life-forms. In this review we consider hybridisation events between *Phytophthora* species, long distance dispersal of oomycetes, sporangia and zoospore survival, direct and indirect infection processes and newly observed sporulating structures. These aspects are all relevant features for an understanding of the epidemiology of zoosporic plant pathogens. Disease management should not be based on the presumption that the zoosporic stage is a weak link in the life cycle. Oomycete plant pathogens show remarkable flexibility in their life cycles and ability to adapt to changing environmental circumstances.

Keywords Fungal phylogeny · Landscape pathology · Pathogen evolution · Plant epidemiology · Zoosporangia

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Introduction

Plant diseases are the outcome of the interaction of plants with a variety of pathogenic organisms in a disease-conducing environment. Many important plant pathogens are zoosporic, i.e. with motile asexual spores. Zoosporic plant pathogens cause significant crop losses worldwide and are the object of a substantial amount of epidemiological research. In our use of the term 'zoosporic plant pathogens' we include both zoosporic fungi and oomycetes. Although we wish to avoid becoming entangled in a systematics debate, modern molecular phylogenetic studies must be at the very heart of any attempt to discuss the comparative epidemiology (Kranz 1980, 2003) of plant pathogens in relation to the evolutionary loss of zoospores, a feature present in both true fungi and oomycetes. Zoospores, which are single-nucleated, formed in sporangia, and motile in aqueous environments, are however a key feature in the life cycle of many plant pathogens. They have been thought to be a weak link, as zoospores have no cell walls, which makes them particularly vulnerable and

transient (Lange and Olson 1983; Stanghellini 1997). Here, we aim at a selective review of relevant literature, focusing on a limited number of case studies that we believe provide insights in the issue of zoospore function and loss in plant pathogens. We then move on to discuss the epidemiological and ecological implications for sustainable plant disease management. We emphasize plant pathogens within the Straminipila[1], mostly oomycetes, but refer also to the true fungi with zoospores when appropriate. Zoospores are common in oomycetes and less common in the true fungi (Hardham et al. 1994; Lebeda and Schwinn 1994; Judelson and Blanco 2005).

Phylogenetic and epidemiological significance of zoospore loss

According to Dick (2002), "loss of the zoospore and therefore flagellation is a feature of both the Peronosporales and Sclerosporales and has minor phylogenetic significance." If the term 'fungus' is considered to be an essentially physiological concept and not a taxonomic one, then several independent phylogenetic lines of fungi have evolved (and lost) flagella (Dick 1997). We would not dismiss such a lack of phylogenetic significance for zoospore loss, but argue here that loss of flagellation and motility must have considerable significance from an epidemiological point of view. This follows from the differences in dispersal potential, infection processes and survival between pathogens with or without zoospores.

In spite of this, the ability to produce zoospores varies amongst different groups of oosporic plant pathogens. For example, it is usual in *Albugo* (e.g. Whipps and Cooke 1978), variable in *Plasmopara* (e.g. Kast and Stark-Urnau 1999), environmentally-dependent in *Phytophthora* (e.g. Judelson and Blanco 2005), and lost in *Hyaloperonospora* (e.g. Slusarenko and Schlaich 2003) and *Peronosclerospora* (e.g. Jeger et al. 1998). Whenever organisms have evolved to occupy niches in which their pre-existing complexity might have been superfluous, there has been the potential for features not contributing to the fitness of the species to be lost abruptly or over a period of time.

Whether zoosporic loss happened during punctuated events or over longer periods of time can only be the subject of speculation; given the paucity of the fossil record for fungi and oomycetes alike, the important point is that antagonistic interactions may inherently lead towards simplification—once one organism becomes dependent on another for its sustenance it may discard features previously required as a free-living organism. Parasitism, for instance, is often accompanied by morphological simplification involving, in the system we are interested here, the evolution of sporangia originally water-dependent and producing zoospores into sporangiophores producing directly germinating conidia (Brasier and Hansen 1992).

There may be here a conceptual connection with the argument that pathogens with higher genetic diversity and thus evolutionary potential pose a greater risk to plant populations, other things being equal, as these pathogens will be more likely than those with less genetic diversity to overcome the defence apparatus of their host(s) (McDonald and Linde 2002). Host specialization may on the one hand lead to genetic impoverishment, as the pathogen no longer needs the ability to infect various hosts, and can thus discard the machinery upon which it relied to successfully infect that host diversity; on the other hand, host specialization may also lead to the creation of new pathogen genetic diversity due to species-specific evolutionary arms races between host and pathogen (Clay and Kover 1996).

For species of the genus *Phytophthora*, both specialization to a single host and general aggressiveness towards a wide range of hosts are observed (Brasier and Hansen 1992; Hardham 2007). For example, *P. cinnamomi* affects several tree, shrub and herbaceous species in the Jarrah forest of South-Western Australia (e.g. Shearer et al. 2007). A similar wide range of potential and actual hosts is found with *P. ramorum* (e.g. Rizzo et al. 2005). Conversely, *P. sojae* (e.g. Tyler 2007), *P. ilicis* (Coyier 1981) and *P. porri* (Smilde et al. 1995) are all examples of Phytophthoras which are specialized to a single host or to a taxonomically related group of hosts. This host specialization implies a distinct co-evolution of attack

[1] Alternatively, Stramenopila: spelling of the taxon and of its various derivatives urgently needs standardization. Analysis of 32 publications since 2000 breaks down into 20 using the spelling above and 12 using the alternative. In some cases both taxon spellings are given as keywords (Money et al. 2004; Honda et al. 2007).

and defence in these pathosystems. Zoospore loss seems not to be dependent on whether or not a certain *Phytophthora* has undergone host specialization, but rather on environmental conditions.

Increasing numbers of molecular studies are elucidating the early evolution of various groups of plant pathogens, including the true fungi (James et al. 2006a) and oomycetes (Göker et al. 2004; Tyler et al. 2006). Assembling the fungal tree of life (Bruns 2006) also provides insights on the issue of zoospore loss in organisms able to cause plant diseases. The ancestors of fungi are believed to have been simple aquatic forms with flagellated spores (James et al. 2006a). Also the earliest fungi were aquatic and lacked aerial spore dispersal. The traditional view is then that a monophyletic core developed producing zoospores (phylum Chytridiomycota, with the exception of *Hyaloraphidium curvatum*, where the presence of flagella has never been reported; Ustinova et al. 2000). As opposed to that, loss of zoospores was generally thought to have happened in the Zygomycota, with the exception of the single-flagellated *Olpidium* (Lange and Olson 1976), which has now been reclassified (James et al. 2006b). However, recent molecular work based on a six-gene phylogeny suggests that the Chytridiomycota are not monophyletic, and that at least four independent events of zoospore loss can be traced back in the kingdom Fungi (James et al. 2006a).

This surge of molecular activity is not just relevant for the production of a more accurate phylogeny (Tyler et al. 2006; Göker et al. 2007), but also for applied epidemiology, as zoosporic fungi can act as vectors of plant viruses (e.g. Teakle 1983; Adams 1991; Campbell 1996; Rochon et al. 2004), although suspicions that oomycetes may be implicated in virus transmission, e.g. *Lagena radicicola* and flame chlorosis of cereals (Haber et al. 1991), have not been confirmed. But before dealing with the ecological and epidemiological implications of zoospore loss in oomycetes, we briefly discuss potential explanations for such an evolutionary development and some case studies.

Explanations for the loss of zoospores

Loss of flagellated spores is believed to have been concurrent with the development of new mechanisms of spore production and dispersal (James et al. 2006a). When fungi moved on to the terrestrial environment, some of them shed their 'ancient baggage' which had made them successful in water, and focused on new means of dispersal, more adapted to the new life in periodically water-poor environments. For example, in the Peronosporales, *Hyaloperonospora parasitica* has no zoosporic stage in its life cycle, and this has been related to its independence from the aqueous environment (Slusarenko and Schlaich 2003).

Alternatively, zoospore loss may have accompanied the development of parasitism and biotrophy. An example is *Peronospora*, which is thought to derive from a *Phytophthora* that lost the ability to produce zoospores and became an obligate biotroph (Cooke et al. 2000). There is a wide spectrum of angiosperm hosts that is parasitised by the morphologically 'advanced' (i.e. lacking zoospores) genus *Peronospora*. For species-specific parasitic interactions, it has been claimed that suppression and inhibition are likely to be less important than attraction and growth stimulation (Dick 2002).

There are many examples of zoosporic loss of plant pathogens in relation to the presence or absence of humidity in their typical environment. Prime case studies are tropical graminaceous downy mildews of sorghum and pearl millet (Jeger et al. 1998; Fig. 1). On the one hand, *Sclerospora graminicola* produces zoospores and affects pearl millet, which is generally found in regions with higher temperatures and lower rainfall than sorghum. Sorghum is affected by *Peronosclerospora sorghi*, which does not produce zoospores in spite of sorghum growing in regions of higher humidity than those where pearl millet is cultivated. Given that flagellated zoospores are propagules for dispersal in the presence of humidity, it is perhaps counter-intuitive that *S. graminicola* should have kept zoospores whilst *P. sorghi* should have lost them. Conversely, it can be argued that zoospores are even more important in an arid environment where water is available only rarely and needs to be used efficiently.

There are recent examples where plant pathogens have made a rapid transition to a new environment. Turf grass rapid blight disease has recently emerged as a terrestrial plant pathogen (Olsen 2007). It was first observed in California in 1995 and was subsequently associated with high salinity irrigation in

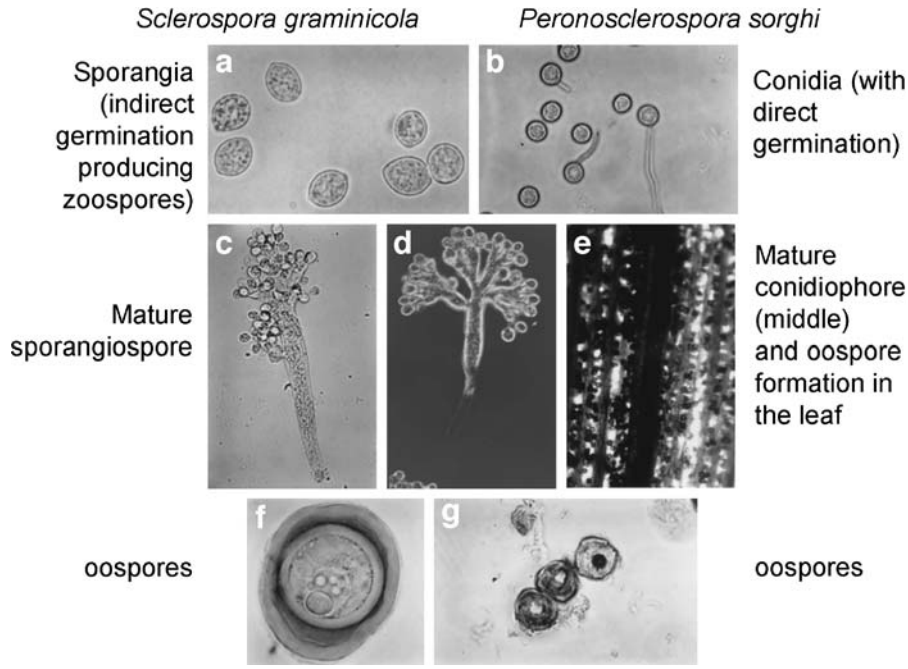

Fig. 1 Sexual and asexual phases of *Sclerospora graminicola* **a**, **c**, **f** and *Peronosclerospora sorghi* **b**, **d**, **e**, **g** (from Jeger et al. 1998, with kind permission of Blackwell)

water and golf courses. Preliminary diagnosis identified the pathogen as a species of the *Labyrinthula* genus, which is associated with the marine environment. For example, *L. zosterae* causes marine grass wasting disease (Olsen et al. 2003). The pathogen (Fig. 2) was then aptly named as *Labyrinthula terrestis* sp. nov. (Bigelow et al. 2005), as it is the first observation of this type of organism (a straminipile; Leander and Porter 2001) on land plants. It is considered to have originated from a single infected population and to share a recent common ancestor with other labyrinthulids (Craven et al. 2005). *Labyrinthula terrestris* builds digitate colonies in an extracellular network produced by specialized organelles called bothrosomes and uses these networks to move rapidly (Stowell et al. 2005). The disease has spread onto golf courses in Arizona and nine other US states; there has been a first report of a *Labyrinthula* sp. on amenity turf grass in the UK (Entwistle et al. 2006). In many Labyrinthulid species there is an absence of zoospore production, although biflagellated zoospores are clearly described (Amon and Perkins 1968; Perkins 1973; Amon 1978). Perhaps the formation of the extracellular network enables the local but rapid movement of somatic cells analogous to the swimming of zoospores.

Fig. 2 Vegetative cells of *Labyrinthula terrestris* illustrating longitudinal cell division (photo, D. Bigelow, with kind permission of American Phytopathological Society)

Pythium species are root-infecting oomycetes closely related to Phytophthoras (Brasier and Hansen 1992; Deacon and Donaldson 1993). They are characterized by flexibility in their life cycle. Oospores can either germinate directly or produce cysts via sporangia and zoospores. Zoospores can also be produced by sporangiophora on infected seedlings (van West et al. 2003). Some species, e.g. *P. glomeratum* from soil, are reported to produce no sporangia or zoospores (Paul 2003) but as a rule *Pythium* species do have the ability to undergo

zoosporogenesis (Walker and van West 2007). Other species, such as *P. helicoides*, are reported to produce only sporangia and zoospores in ebb-and-flow culture systems (Kageyama et al. 2003; Fig. 3). Some related oomycetes, e.g. *Saprolegnia* species, are able to release a new secondary zoospore after encystment of a primary zoospore. The secondary zoospore is the better swimming spore (Walker and van West 2007). Thus *Pythium* and related species such as *Aphanomyces* show remarkable flexibility in their life cycles and the ability to respond and adapt to changing environmental conditions.

Epidemiological and ecological implications

Zoospore loss has been reported widely in plant pathogens, but it is important to relate this knowledge to its potential epidemiological implications and to its relevance for disease management (Jeger 2004; Madden 2006). We discuss here hybridisation events for Phytophthoras, long distance dispersal for tobacco blue mold, the relation of sporangia and zoospore release with pathogen survival, infection processes (direct and indirect germination), sporulating structures in *Phytophthora ramorum,* integrating life cycles in *P. syringae*, and epidemic modelling in *P. infestans*.

Hybridisation events

The advent of molecular phylogenetics has revealed the potential for interspecific hybridisation of many plant pathogens (Schardl and Craven 2003). Hybrids may create devastating disease on both cultivated and wild plants (Olsen and Stenlid 2002) and have the potential to jump on new host species or to increase their virulence on traditionally infected hosts. For *Phytophthora*, the occurrence of multiple species in the rhizosphere of individual nursery plants can enhance the evolution and emergence of new tree diseases (Brasier and Jung 2003). Natural hybrids of *P. nicotianae* and *P. cactorum* have been observed in glasshouse hydroponic systems (Bonants et al. 2000). Similarly, there are reports of interspecific crosses between *Phytophthora sojae* and *P. vignae* (May et al. 2003) and of nuclear hybrids from protoplasts of *P. parasitica* and *P. capsici* followed by completion of the parasexual cycle (Gu and Ko 2000). In vitro fusion of zoospores of *P. nicotianae* and *P. capsici* has been achieved (Érsek et al. 1995; English et al. 1999).

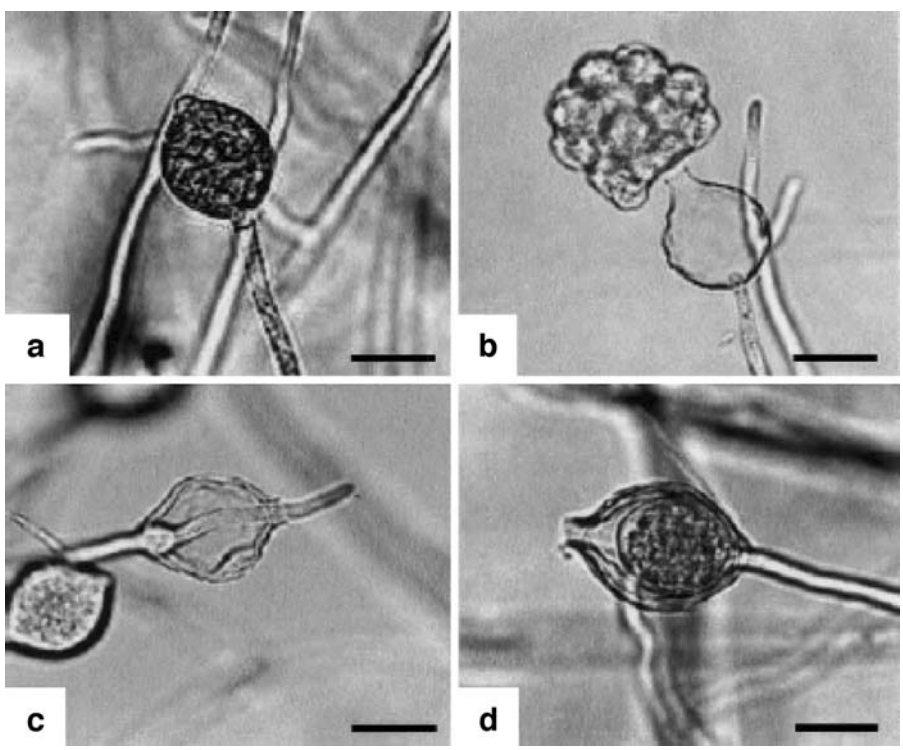

Fig. 3 Morphology and germination mode of group P of *Pythium* (scale bars = 20 μm). **a** Papillate sporangium, **b** zoospore formation in a vesicle originating from a sporangium, **c** hyphae proliferating from the base of the sporangium, **d** a sporangium proliferating from inside an old sporangial wall (from Kageyama et al. 2003, with kind permission of Blackwell)

There has been much less work done with downy mildews although genetic recombination through the parasexual cycle has been demonstrated in *Plasmopara halstedii* (Spring and Zipper 2006).

The emergence and spread of the hybrid alder *Phytophthora* is a good example of the potential of hybridization events to create new pathosystems (Brasier et al. 1995, 2004). Extensive field surveys of riparian and plantation alder in Bavaria (Germany) have revealed that symptoms were widespread on the majority of river courses and one third of plantation stands (Jung and Blaschke 2004; Fig. 4; see also Gibbs et al. 1999 for Britain, and Streito et al. 2002 and Thoirain et al. 2007 for France). The source of inoculum was traced back to young infected alder plantations at sites that drain into the river system. Rootstocks of alder plants might have been infected in nurseries, possibly due to the presence of disease propagules in irrigation water. The subsequent direct spread of zoospores from infected plantations (during seasonal flooding or waterlogged sites) to older and naturally regenerating trees, as well as to river catchments and riparian alders, can be seen as an example of disease spread at the landscape level along a physical network (Holdenrieder et al. 2004; Jeger et al. 2007).

Long-distance dispersal

Long-distance dispersal of plant pathogens is a fundamental process in the dynamic of plant epidemics, as it enables disease to jump from patch to patch of susceptible hosts, overcoming efforts at containing disease development with local control measures. Long-distance spread of pathogens is helped by man-made connectivity of previously separated continents creating what are known as 'small-world' networks, and is of concern given the lower disease threshold of epidemics in such networks compared with regular lattices (Pautasso and Jeger 2008). *Phytophthora infestans,* the cause of potato late blight, moves over long distances aerially by producing asexual sporangia which can infect plants by germinating directly or by releasing zoospores (e.g. Ristaino 2002). Natural long-distance spread of sporangia of *P. infestans* is limited by exposure to UVB radiation, the short infectious period of the pathogen, and rapid mortality of the host plants (Campbell 1999; Brown and Hovmøller 2002; Zwankhuizen and Zadoks 2002), but disease expression may be facilitated by current and future climate warming (Baker et al. 2005; Garrett et al. 2006; Hannukkala et al. 2007; Jeger and Pautasso 2008). Aylor (2003) assessed the critical gap width for dispersal to be approximately 35–50 km. However, *P. infestans* has been shown to spread rapidly and over long distances due to movement of infected tubers (Goodwin et al. 1998).

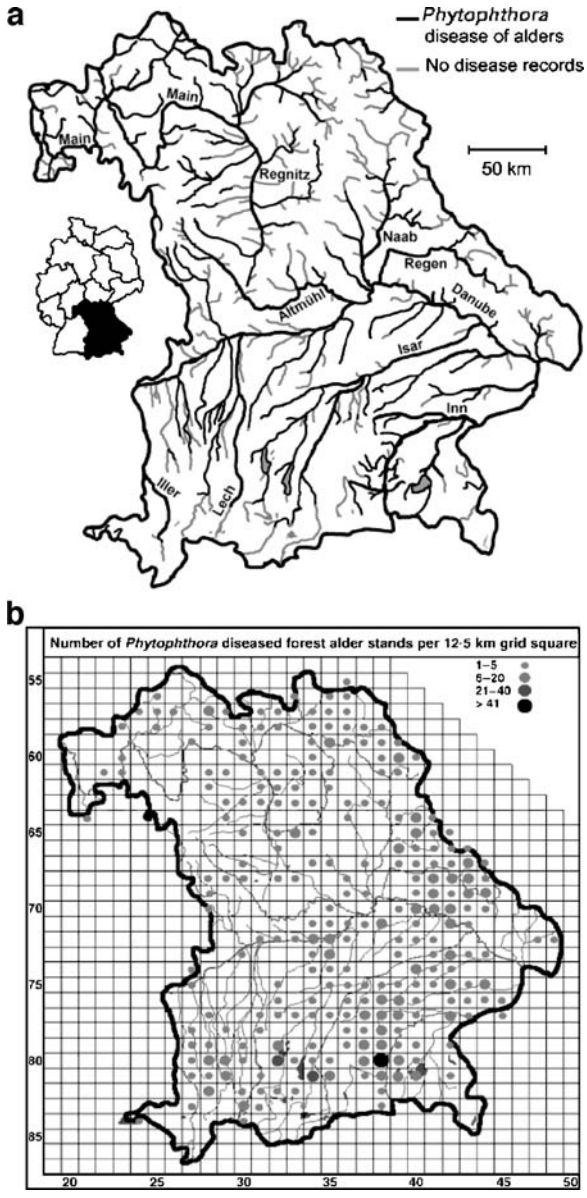

Fig. 4 Distribution in Bavaria of *Phytophthora* root and collar rot of alders **a** along main rivers and streams and **b** in forest alder stands (from Jung and Blaschke 2004, with kind permission of Blackwell)

Long-distance dispersal of tobacco blue mold (*Peronospora tabacina*) is another example of the potential for plant pathogens to spread and act over vast regions. Each year, blue mold advances in a wave from the southern-most tobacco-growing regions to the northern-most ones in the eastern USA (Aylor 1999). This is consistent with the observed low rates of genetic diversity in this pathogen throughout the USA (Sukno et al. 2002). Calculated rates of advance range from 9 to 18 km per day. Aylor (2003) estimated the critical gap width for disease spread to be 10^2 km for dispersal under full sun and 10^3 km under cloud cover, depending on spore density. The effects on disease spread of the mode of dispersal of inoculum, with particular attention to Phytophthoras, was summarized by Ristaino and Gumpertz (2000). In general, although flagellated spores have epidemiological relevance, the presence of absence of zoospores does not necessarily have an impact on dispersal, particularly for foliar pathogens.

Survival

The occurrence of full sun or cloud cover is an important variable in plant epidemics, as it can affect the survival of spores. Some chytrids have the ability to survive periodic drying and high summer temperatures typical of cropping soils (Gleason et al. 2004). There are many examples of the influence of environmental conditions on oomycetes, both above ground and below. Solar radiation is the dominant factor determining survival of sporangia of *Bremia lactucae* in California. Infection by sporangia that have survived a day is only likely on cloudy days or shaded leaves (Wu et al. 2000, 2005). However, there is a lower ability of zoospores of *P. infestans* to survive under the cool temperatures which favour their development. Sporangia that do not form zoospores under conditions favourable for formation may be specially adapted for survival in the absence of a host (Porter and Johnson 2004). Release of zoospores from sporangia of *Plasmopora viticola* occurred for at least seven days if free water was available (Kast and Stark-Urnau 1999). Many sporangia of *P. viticola* do not survive during clear daylight periods following their production. However, with overcast conditions for 12–24 h, 50% still released zoospores (Kennelly et al. 2007). The formation of sporangia in *P. viticola* has been shown to be photosensitive, with a prolonged period of dark as a necessary condition (Rumbolz et al. 2002). Assessment of survival abilities in soil, and hence the influence of edaphic factors, depends on the techniques used. Assays for detecting and quantifying surviving *P. capsici* in soil differed in efficacy according to propagule type: oospores, mycelial fragments, sporangia and zoospores. Zoospore inoculum was detected at 10 propagules per gram (ppg) of soil, whereas sporangia were detected at 1 ppg (Larkin et al. 1995).

Infection processes

Host targeting is a fundamental strategy for zoosporic plant pathogens to successfully infect their hosts (Tyler 2002). This is true both in aquatic and terrestrial environments. Zoospore chemotaxis was observed in mangrove strains of *Halophytophthora vesicula* (Leano et al. 1998). However, no evidence for this phenomenon was obtained for *Pythium porphyrae* parasitising the red alga *Porphyra yezoensis* (Uppalapati et al. 2001). For terrestrial pathosystems, it is known that host factors can influence the development of *Plasmopara viticola* by (1) accelerating the release of zoospores from mature sporangia, (2) coordinating the morphogenesis of the germ tube, and (3) directing zoospores to stomata (Kiefer et al. 2002). Similar evidence for host-mediation of zoospore development was reported for *Phytophthora infestans* infecting *Solanum phureja* (Oyarzun et al. 2004). However, *Pythium oligandrum* zoospores are not attracted to hyphae of their fungal host, but if encysted on hyphae show a significant germ-tube emergence towards the host (Madsen et al. 1995).

Direct germination of conidia may be an advantage in some cases. Conidia of *Peronospora rubi* germinate and infect most commonly through direct penetration or enter through stomata (Williamson et al. 1995). Conidia of *Peronospora parasitica* enter through the stigma, ovary wall and establish in the ovary enabling embryo infection and seed transmission (Jang and Safeeulla 1990). Direct germination exists in *Phytophthora drechsleri*, where sporangia are stimulated by microbial interaction in soil. With indirect germination zoospore infectivity may be suppressed (Hardy and Sivasithamparam 1991). A study on the effect of the biocontrol bacterium *Burkholderia cepacia* on *Pythium aphanidermatum*

indicated that although antibiosis was the main mechanism involved in suppression there was some contribution of competition for zoospore homing compounds (Heungens and Parke 2000). This effect was not apparent against *Aphanomyces euteiches* zoospores.

Many studies have shown that temperature has an important effect on zoospore infection. For example, heat stress (40°C rather than 25 to 35°C) enhanced the severity of root rot caused by *Phytophthora cryptogea* on container-grown *Chrisanthemum* (MacDonald 1991). Also for *P. cryptogea* on *Lycopersicon esculentum*, enhanced temperature (above 25°C) was ineffective to counter established infection in summer-grown plants (Kennedy and Pegg 1990). Together with wetness duration, higher day temperature was found to be associated with increasing incidence and severity of *P. cactorum* on apple and pear fruits (Grove and Boal 1991). However, citrus root colonization by *P. citrophthora* and *P. parasitica* was shown to be restricted or limited above a certain temperature threshold (27 and 33°C, respectively). A similar result was obtained for early infection of *Vitis vinifera* by *Plasmopara viticola* in Western Australia (Williams et al. 2007). In general, the effect of temperature on disease severity caused by zoosporic plant pathogens will depend not only on the temperature preferences of the pathogens, but also on the temperature threshold at which they will tend to switch from zoospore to sporangial infection (Judelson and Blanco 2005), and will be confounded by other factors such as inoculum density and plant age (Raftoyannis and Dick 2002).

Sporulation structures

In *Phytophthora ramorum*, the causal agent of sudden oak death and ramorum dieback of many shrubby species (Rizzo et al. 2005), sporangia and zoospores are the elements driving the observed disease epidemic. Moralejo et al. (2006) observed structures termed sporangiomata on susceptible woody species. This is the first description of stromata produced by a *Phytophthora* species, and may be a significant environmental adaptation in *P. ramorum*. In particular, adaxial positioning suggests adaptation for rain-splash dispersal. Moreover, sub-epidermal positioning of the stroma may in part protect from desiccation or solar radiation and clustering of sporangia may contribute to moisture retention. Oversummering survival structures may provide a way to avoid the challenge posed by the Mediterranean climate in the current region of outbreak, as well as in other regions with potentially susceptible hosts (Moralejo et al. 2006).

Integrating life cycles and predictive models

Oospore germination and zoospore infection in *Phytophthora syringae* also pose a challenge to understanding disease epidemiology and management. *Phytophthora syringae* persists as oospores in fallen apple leaves. Oospores germinate by giving rise to one or two sporangia and, when free water is available, each sporangia produces 20 to 30 motile spores. Undehised sporangia may germinate to create a secondary sporangium which may produce zoospores or give rise to a tertiary sporangium, potentially an important adaptation providing flexibility in response to variable environmental conditions. One open question in this pathosystem is the long-term viability of ungerminated zoospores. Harris and Xu (2003) found that infection of fruit depended mainly on sufficient rain being available to keep soil moist for at least 2–3 days (oospore germination) and wetness periods of at least 4 h (zoospore infection; Fig. 5).

Typically mechanistic and/or forecasting models should take account of zoospore behaviour, because in many cases this factor seems to be essential in understanding and predicting epidemic development. Examples of various predictive models where zoospore activity could significantly improve forecasting involve outbreaks of *Phytophthora infestans* (Johnson et al. 1996; Aylor et al. 2001; Bourgeois et al. 2004; Andrade-Piedra et al. 2005; Powell et al. 2005).

Disease management

Other than resistance breeding and sanitation, disease management for zoosporic plant pathogens has relied heavily on chemical control, and the emergence of resistance has been observed repeatedly. Apparently, the cost of fungicides used against *Phytophthora infestans* on *Solanum tuberosum* accounts worldwide for approximately 25% of the total sum spent on fungicides on all crops (Erwin and Ribeiro 1996). In

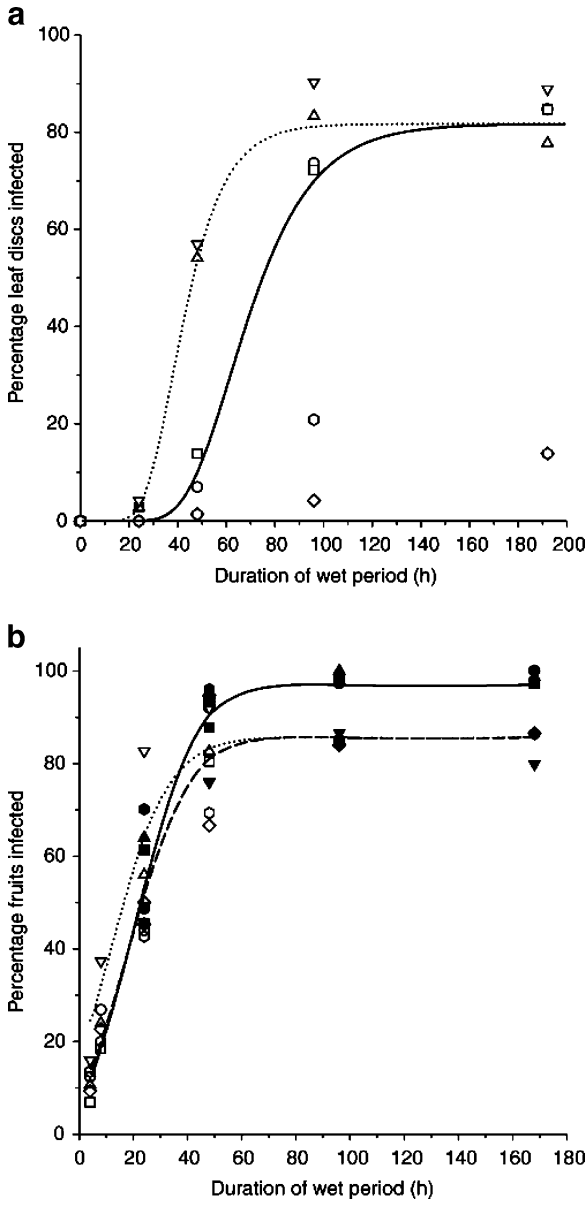

Fig. 5 Observed and predicted percentage of **a** *Phytophthora syringae* oospore activation, estimated as the percentage of infected leaf discs, and **b** apple fruits infected by zoospores of *P. syringae*, in relation to temperature and duration of wet period; *circle* 10°C, *square* 12°C, *triangle* 14°C, *inverted triangle* 16°C, *diamond* 18°C, *hexagon* 20°C; **a** *solid line* 10 and 12°C, *dashed line* 14 and 16°C; no models can be fitted to data at 18 and 20°C; and **b** *solid line* 10, 12 and 14°C, *dotted line* 16°C, *dashed line* 18 and 20°C (from Harris and Xu 2003, with kind permission of Blackwell)

many cases the effects of oomycete fungicides have been tracked through the various stages of zoospore formation, release, motility, cyst formation, germination, and infection (e.g. Mitani et al. 2001; Reuveni 2003) and similarly for plant extracts (Rohner et al. 2004), secondary metabolites (Shimai et al. 2002) and mineral supplementation (Xu and Morris 1998). In relatively few studies has the relative effect of control of sporangia/conidia and zoospores been directly compared.

In a comprehensive study the response of *Plasmopara halstedii* to anti-oomycete fungicides varied during ontogeny defined in terms of 13 developmental stages of the pathogen (Viranyi and Oros 1991). A principal component analysis of responses formed two main groupings with same separation of sporangial and zoosporic responses in one of the two groups. Famoxadone used against *P. infestans* and *Plasmopara halstedii* inhibited zoospore release and caused lysis of zoospores. Higher doses were required to inhibit direct germination (Andrieu et al. 2001). In *P. infestans* zoospore encystment and cyst germination were highly sensitive to dimethomorph; direct sporangial germination less so (Stein and Kirk 2003). Multi-drug resistant isolates of *P. infestans* significantly reduced sporulation and sporangial germination but not differentiation into zoospores (Ziogas et al. 2006). Tomato treated with PGPR, and BABA for induced systemic protection had reduced germination of *P. infestans* sporangia and zoospores with a marginally greater effect on sporangia (Yan et al. 2002; Fig. 6). Both direct and indirect germination of sporangia of *P. infestans* were suppressed by a range of calcium-modulating treatments, marginally greater for indirect germination (Hill et al. 1998; Fig. 7).

From a biological control point of view, a different line of work has built on the discovery that biosurfactants produced by the bacterium *Pseudomonas aeruginosa* were an effective way to protect hydroponic plant specimens inoculated with four species of *Pythium* and *Phytophthora parasitica* (Stanghellini and Miller 1997). In order to achieve long-term sustainability, strategies alternative to pesticides are needed for the management of zoosporic plant pathogens (Hoitink and Boehm 1999; Martin and Loper 1999; Paulitz and Belanger 2001; Hong and Moorman 2005). This research showed that rhamnolipids (Nitschke et al. 2005) produced by bacteria or directly applied to plants are able to lyse the membranes of zoospores (e.g. Kim et al. 2000; Maier and Soberon-Chavez 2000; see also Tomlinson and Faithfull 1979). Subsequent work showed that fluorescent pseudomonads colonizing the rhizosphere are

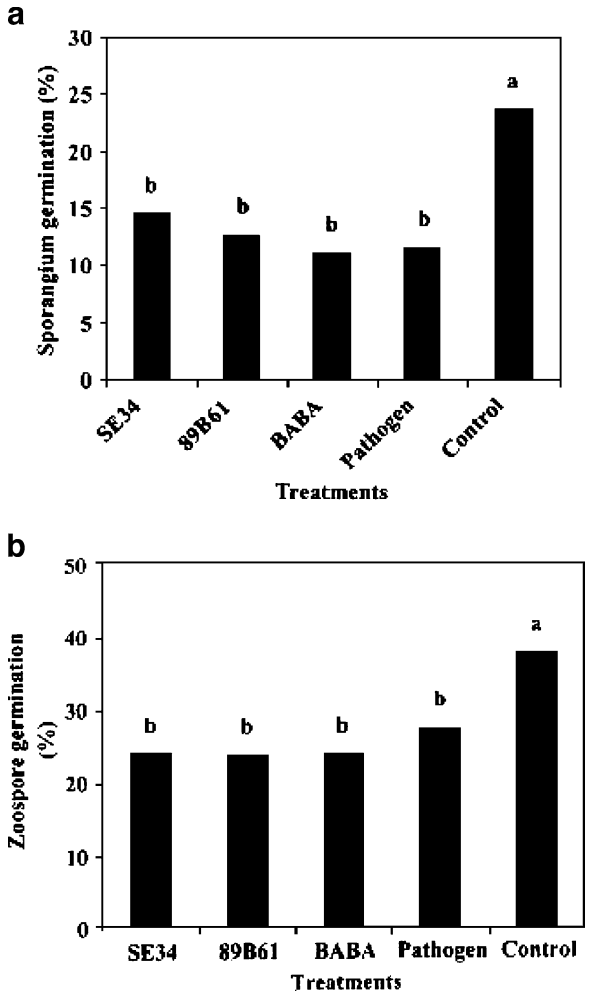

Fig. 6 Percent germination of **a** sporangia and **b** zoospores of *Phytophthora infestans* on tomato leaves induced with plant growth-promoting rhizobacteria (PGPR) strains SE34 and 89B61, β-amino butyric acid (BABA), and pathogen. Data are means of two experiments (from Yan et al. 2002, with kind permission of American Phytopathological Society)

able both to elicit systemic defence response in plants and to affect the pathogenicity of zoosporic plant pathogens (Haas and Defago 2005). The potential of the approach has been confirmed empirically in various pathosystems (e.g. *Phytophthora capsici* on *Capsicum annuum*; Ristaino and Johnston 1999; Nielsen et al. 2006; *Albugo occidentalis* on *Spinacia oleracea*; Irish et al. 2002; *Pythium aphanidermatum* on *Cucumis sativus*; Folman et al. 2004; *Phytophthora cryptogea* on *Cicorium intybus*; De Jonghe et al. 2005; *Phytophthora infestans* on *Solanum tuberosum*; Lozoya-Saldana et al. 2006; *Pythium aphanidermatum* or *Phytophthora* spp. on *Lycopersicon esculentum*; Calvo-Bado et al. 2006; Sharma et al. 2007). Widespread adoption is dependent on economic circumstances in different crop production systems.

One often overlooked management strategy is the effect of spatial and temporal mixtures of resistant and susceptible species or varieties on diseases. Devoting different fields to different crops and rotating crops

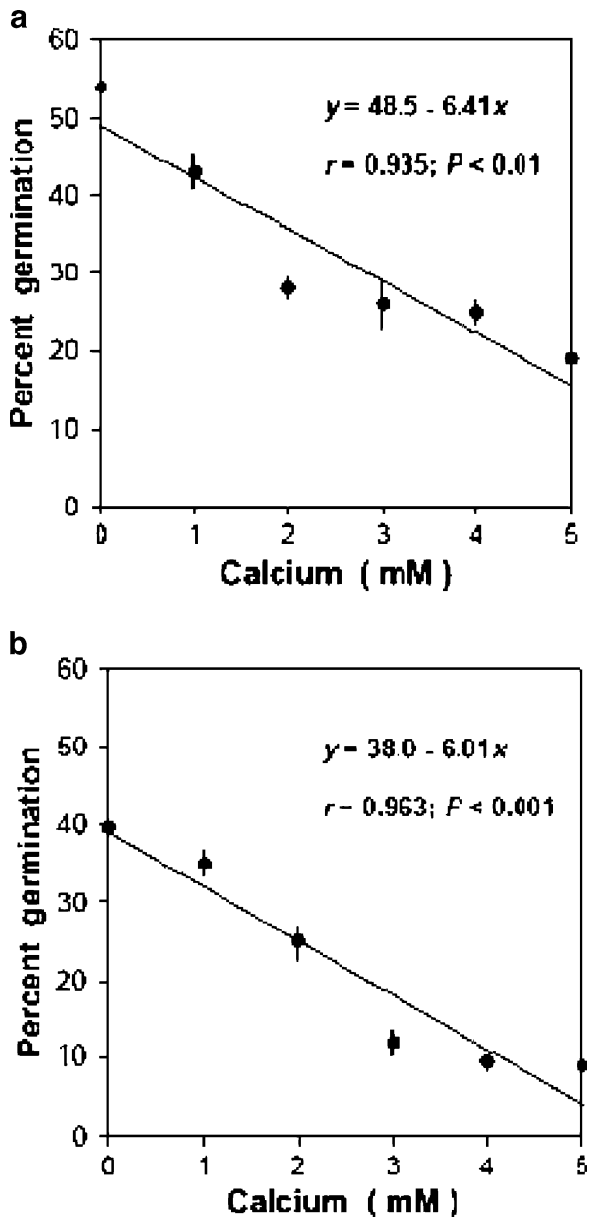

Fig. 7 Effect of $[Ca^{2+}]$ on sporangial germination by **a** hyphal outgrowth (20°C) and **b** zoospore release (12°C). Data points are means ± SE of three replicates, based on counts of 100 sporangia in each replicate (from Hill et al. 1998, with kind permission of Kluwer)

from year to year is a traditional agricultural practice which makes sense also as a control strategy for zoosporic plant pathogens. Indeed, monocultures grown year after year in the same soil are often remarkably susceptible to disease, as exemplified by potato late blight. A study of the effect of mixtures of *Solanum tuberosum* varieties with differing levels of susceptibility to *P. infestans* showed that mixtures of an immune or near immune variety substantially reduced disease on susceptible ones (Phillips et al. 2005). That host diversity can reduce potato blight severity has been now shown repeatedly, although with varying degrees (Garrett and Mundt 2000; Garrett et al. 2001; Andrivon et al. 2003; Pilet et al. 2006). It is likely that the mechanisms underlying these findings involve sporangial dispersal, as immune plants constitute a physical barrier and reduce the overall density of susceptible individuals in a field (Burdon and Chilvers 1982; Keesing et al. 2006; see also Jactel and Brockerhoff 2007). At a landscape level, a similar protective mechanism could be implemented for sudden oak death. In this emerging pathosystem, connectivity of woodland patches is playing a key role in the spread of *Phytophthora ramorum* and forests could be managed so as to decrease connectivity of susceptible hosts (such as bay laurel) by increasing the diversity of resistant understory species (Condeso and Meentemeyer 2007). In tropical forests, *Phytophthora* and *Pythium* species have been suggested as contributing to the high tree diversity by producing density-dependent mortality of seedlings close to parent trees (e.g. Packer and Clay 2000; Hood et al. 2004; Pautasso et al. 2005; Bell et al. 2006; Augspurger and Wilkinson 2007).

Conclusions

Loss of flagellated cells, zoospores, has occurred independently in different phylogenetic lineages. No single explanation is apparent for these evolutionary losses. The case studies discussed in this review suggest that it would be an oversimplification to view lack of zoospores as progressing from free-living aquatic to parasitic terrestrial organisms. Indeed, oomycetes show remarkable flexibility (and redundancy) in 'spore' structure and function in relation to their environment. Zoospores have perhaps mistakenly been seen as the weak link in pathogen life cycles. Evidence from disease management studies on the best targets for control interventions is inconclusive and needs further comparative analysis.

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