Review

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Life histories of hosts and pathogens predict patterns in tropical fungal plant diseases

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Key words: biotrophic pathogens, endophytes, fungal disease, Janzen–Connell hypothesis, light-demanding species, necrotrophic pathogens, plant defence, shade-tolerant species.

Summary

Plant pathogens affect the fitness of their hosts and maintain biodiversity. However, we lack theories to predict the type and intensity of infections in wild plants. Here we demonstrate using fungal pathogens of tropical plants that an examination of the life histories of hosts and pathogens can reveal general patterns in their interactions. Fungal infections were more commonly reported for light-demanding than for shade-tolerant species and for evergreen rather than for deciduous hosts. Both patterns are consistent with classical defence theory, which predicts lower resistance in fast-growing species and suggests that the deciduous habit can reduce enemy populations. In our literature survey, necrotrophs were found mainly to infect shade-tolerant woody species whereas biotrophs dominated in light-demanding herbaceous hosts. Far-red signalling and its inhibitory effects on jasmonic acid signalling are likely to explain this phenomenon. Multiple changes between the necrotrophic and the symptomless endophytic lifestyle at the ecological and evolutionary scale indicate that endophytes should be considered when trying to understand large-scale patterns in the fungal infections of plants. Combining knowledge about the molecular mechanisms of pathogen resistance with classical defence theory enables the formulation of testable predictions concerning general patterns in the infections of wild plants by fungal pathogens.

I. Plant pathogens in tropical ecosystems

Microbial pathogens represent an unseen major component of natural biodiversity. Most groups of parasitic microfungi exhibit a higher degree of diversity in tropical than temperate regions (Piepenbring *et al.*, 2011). Furthermore, microbial pathogens, and fungal diseases in particular (Fig. 1), affect the survival of their plant hosts and, thus, the size and the genetic structure of plant populations. In consequence, plant pathogenic fungi are considered a major driving force that maintains plant diversity due to their 'Janzen–Connell' effect, which means that they disfavour abundant host species and favour the mixing of species in tropical ecosystems by enhancing the mortality rates of the offspring in close proximity to the mother tree (Connell, 1971; Janzen, 1971; for recent reports on the role of fungi in this context see Bagchi *et al.*, 2010; Hersh *et al.*, 2012; Liu *et al.*, 2012a,b; Terborgh, 2012, 2013). Furthermore, endophytes and pathogens of tropical plants might represent an as yet underestimated source of future crop diseases (Desprez-Loustau *et al.*, 2011; Fisher *et al.*, 2012). The proportion of plant-infecting fungi in the reports on emerging infectious diseases has increased > 10-fold in the last 15 yr (Fisher *et al.*, 2012). Climatic changes together with the resulting shifts in the species ranges of tropical plants, ongoing changes in ecosystem use and increased infection rates in disturbed areas (Pautasso *et al.*, 2012; Santos & Benitez-Malvido, 2012; Thompson *et al.*, 2013) affect the probability that pathogens will shift from noncultivated species to crops (Alexander, 2010; Fisher *et al.*, 2012), because the higher mobility of pathogens and hosts increases the possibilities of hybridization amongst pathogens that previously were geographically isolated (Chakraborty, 2013; Santini *et al.*, 2013) and exposes hosts to new pathogens. Roy (2001) investigated the association of flower-infecting rusts with the Brassicaceae and found that geographic proximity of potential hosts was a stronger predictor of host shifts than phylogenetic distances.

In this review we focus on fungal pathogens of tropical plants, because they are important drivers of biodiversity (see paragraph above) and because tropical ecosystems appear particularly prone to fungal infections. Their enhanced temperatures and humidity values together with longer annual vegetation periods are conditions which facilitate the infection of plants and increase the numbers of pathogen generations per year (Alexander, 2010) and thereby favour the evolution of new races of pathogens (Chakraborty, 2013). Besides fungal pathogens, we also include endophytes in our considerations. Endophytes are microorganisms that inhabit a living host without causing visible symptoms of disease, whereas pathogens either develop in living host tissue (biotrophs; see Fig. 1a) or kill the host cells (necrotrophs; see Fig. 1d) (Horbach *et al.*, 2011), but invariably cause disease symptoms (Fig. 2). Many plant pathogens (including very common and diverse genera such as *Colletotrichum*, and species such as *Septoria tritici* and *Magnaporthe grisea*) first pass a biotrophic phase but switch at a determined stage of their infection cycle to a necrotrophic phase and, therefore, are called hemi-biotrophs (Horbach *et al.*, 2011). Moreover, fungi frequently switch at the ecological and evolutionary timescale between an endophytic and a necrotrophic lifestyle, and a fungus that behaves as an endophyte in a given host and under a specific set of environmental conditions can act as a necrotroph in another host or in a changed environment (Delaye *et al.*, 2013). In

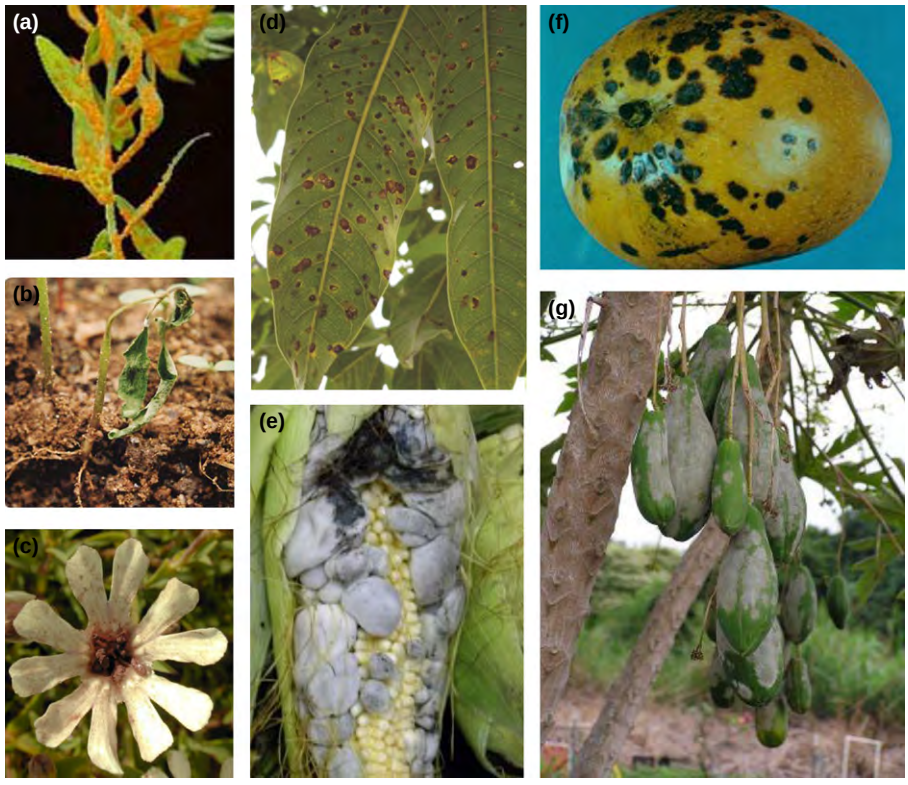

Fig. 1 Examples of common fungal diseases of plants. (a) Rust on flax (*Linum usitatissimum*) (© CSIRO); (b) damping-off disease (http://gardenofeaden.blogspot.mx/); (c) *Microbotryum violaceum*-infected Caryophyllaceae (© Malcolm Storey); (d) anthracnose of mango (*Mangifera indica*) leaves caused by *Colletotrichum gloeosporioides* (© Nigel Cattlin); (e) corn smut on a corn (*Zea mays*) ear caused by *Ustilago maydis* (http://plantali.blogspot.mx/); (f) anthracnose of mango fruit (© Wayne Nishijima); (g) powdery mildew of papaya (*Carica papaya*) caused by *Oidium caricae* (© Scot C. Nelson).

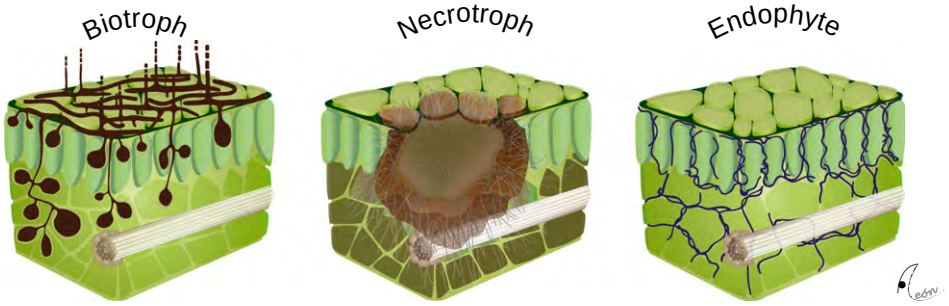

Fig. 2 Lifestyles of plant-infecting fungi. The fungi discussed in this review are biotrophs (which develop in living host tissue and cause symptoms of disease), necrotrophs (which actively kill host cells and live in dead tissue) and symptomless endophytes (which develop in living host tissue without causing any symptoms of disease). See Section IV Necrotrophic and biotrophic pathogens vs symptomless endophytes for major characteristics of these lifestyles.

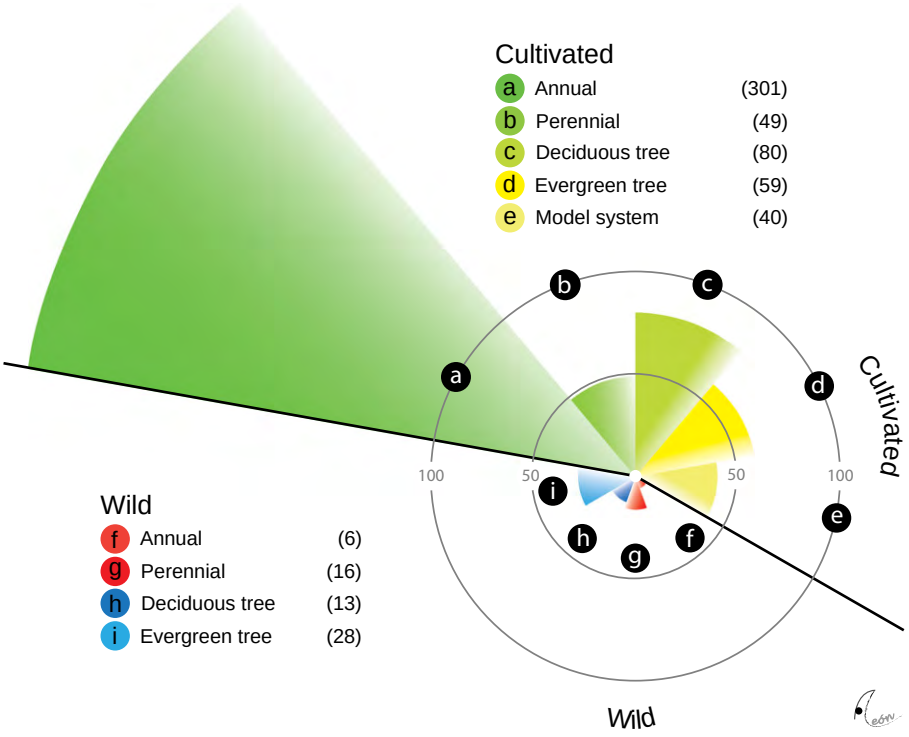

Fig. 3 Number of research reports on diseases of plants. The chart indicates the number of reports found in the literature search on compatible infections for each of the different plant lifestyle groups, which each have different demands concerning light conditions. The length of each element symbolises the number of cases detected (which is also indicated after each host lifestyle), grey concentric rings indicate 50 and 100 cases, respectively. Our results illustrate that the majority of reports are focussed on diseases of annual crops. The literature search of the ISI® Web of Science was performed between 20 May and 6 June 2013 by searching for 'plant disease', 'plant AND pathogen' and 'plant AND infection' in literature published between 1 November 2011 and 6 June 2013. Figure produced with help by Carlos Mazza, taking concepts as seen in Ballaré *et al.* (2012).

summary, the terms 'endophyte', 'biotroph' and 'necrotroph' as we use them here should be considered as evolutionarily and ecologically flexible outcomes of the infection of a certain plant by a specific fungus. These terms represent the endpoints of a continuum of possible outcomes, rather than genetically fixed lifestyles.

In spite of their omnipresence and their importance, fungal pathogens of tropical noncultivated plants have received little scientific attention: this is likely to be because plant pathologists traditionally focus on crops and the model plant *Arabidopsis* rather than wild species. In consequence, the vast majority of studies on plant–pathogen interactions deal with fast-growing annuals that grow in monocultures under high-light conditions (Fig. 3). Much less is known about pathogen infections in perennial plants and in diverse and highly structured natural ecosystems (García-Guzmán & Morales, 2007; Alexander, 2010). The only generally accepted pattern appears to be that pathogens show a high level of 'host conservatism', meaning that the probability that a pathogen which shows a compatible interaction with a given host species can infect another one decreases with the phylogenetic distance among the hosts (Gilbert & Webb, 2007; Schulze-Lefert & Panstruga, 2011; but see Roy, 2001). Risk assessments concerning novel pests and pathogens are based on this phylogenetic signal in the host range of pathogens (Gilbert *et al.*, 2012), and the same considerations apply when one tries to predict the potential influence of a specific pathogen as a 'Janzen–Connell-agent' in the maintenance of biodiversity. However, considering the phylogenetic conservatism of a pathogen is not enough to reliably predict the probability at which a specific pathogen will shift to a certain future host (Roy, 2001; Barrett & Heil, 2012; Juroszek & von Tiedemann, 2012; Swinfield *et al.*, 2012; Chakraborty, 2013). Here, we discuss why plant life-history not only determines the genetic structure and evolution of parasite populations (Barrett *et al.*, 2008; Giraud *et al.*, 2010), but can also affect disease incidence and the relative

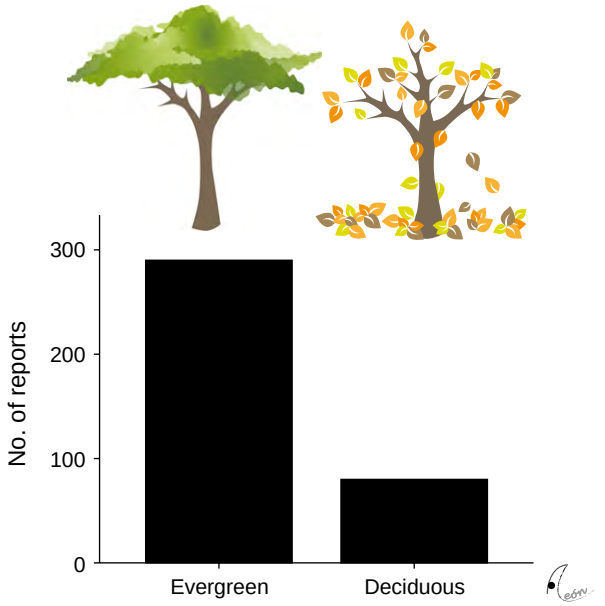

Fig. 4 Number of reports of compatible (symptom-causing) fungal infections in evergreen vs deciduous tropical woody plant hosts. In keyword searches in Web of Knowledge® (for 'endophyte', 'biotroph', 'necrotroph', 'fungal pathogen' and 'fung*' AND 'plant' AND 'life history', time period 1990–2012 and also considering cited work) and in Google Scholar, evergreen species were significantly more affected by disease than the deciduous species ($\chi^2 = 45$, d.f. = 1, $P < 0.001$).

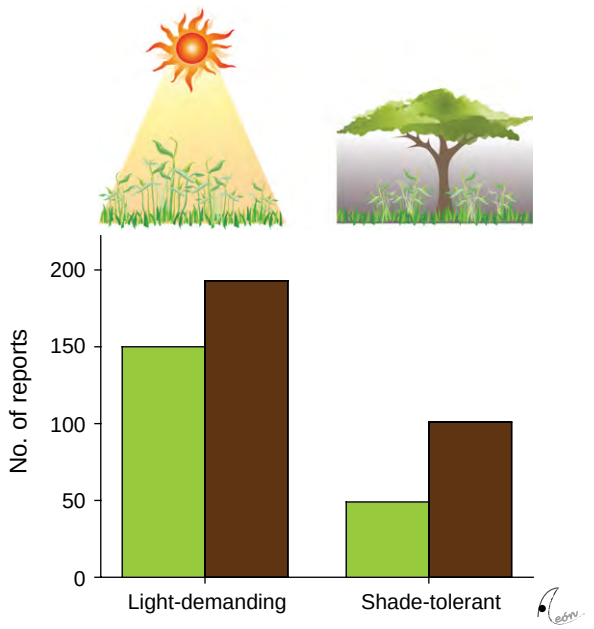

Fig. 5 Number of reports of compatible (symptom-causing) fungal infections in light-demanding vs shade-tolerant plant hosts. We conducted keyword searches in Web of Knowledge® (for 'endophyte', 'biotroph', 'necrotroph', 'fungal pathogen' and 'fung*' AND 'plant' AND 'life history', time period 1990–2012 and also considering cited work) and in Google Scholar for fungal infections in hosts with different light requirements. χ^2-tests indicate that there were no differences in the number of disease reports for light-demanding herbs (green bars) and woody (brown bars) species ($P > 0.05$); however, there were significantly more reports of shade-tolerant woody species than for herbaceous shade-tolerant species ($\chi^2 = 15.6$, d.f. = 1, $P < 0.001$).

frequency of compatible infections in plants by fungal pathogens with different life-history strategies.

II. Questions and patterns

Which type of pathogen is most likely to infect each type of plant? Which type of plant under which growing conditions is most prone to infection? Under which conditions will infections cause the most detrimental effects on host fitness? Which classes of pathogens function as the most efficient 'Janzen–Connell-agents'? Here we discuss how an understanding of the molecular mechanisms that underlie disease resistance and knowledge of the basic life-history traits of both host and pathogen can inform traditional theory regarding plant anti-herbivore resistance to yield testable predictions on fungal infections in plants. We use patterns in the numbers of reports on the compatible infection of tropical plants by fungi to discuss why: (1) fungal infections are more commonly reported from evergreen than deciduous hosts (Fig. 4); (2) fungal infections are more commonly reported from fast-growing and light-demanding species than from slow-growing and shade-tolerant species (Fig. 5); and (3) necrotrophic pathogens are more commonly reported to infect plants in the understorey whereas biotrophic pathogens are more commonly reported on light-demanding hosts (Fig. 6). These patterns are highly likely to be biased, most importantly due to unequal sampling efforts in different habitats and by the specific focuses of most researchers on selected groups of fungi. However, with this overview and the suggested explanations, we hope to guide future research aimed at corroborating these trends and experimentally testing the causal mechanisms that we suggest underlie these general patterns.

III. Pathogens as destructors and constructors

Fungal pathogens can reduce the fitness of their hosts or even kill them (Bell *et al.*, 2006; Beckman *et al.*, 2012; Fisher *et al.*, 2012; Hersh *et al.*, 2012; Liu *et al.*, 2012a). However, their effects on the fitness of the individual host are determined by the phase of the life cycle of the host at which infection occurs (Gilbert, 2002; Mordecai, 2011; Sánchez-Martín *et al.*, 2011). For example, germinating seeds and young seedlings are prone to attack by soil-borne pathogens that can cause a range of damping-off diseases (Fig. 1b) and result in the absolute loss of individual fitness (Bell *et al.*, 2006; Terborgh, 2013). Infections at the mature stage are usually less likely to be fatal, but they can still reduce the survival rates of plants. For example, the rust *Melampsora lini* can reduce the population size of its host *Linum marginale* by 60–80% during severe epidemics (Jarosz & Burdon, 1992; Nemri *et al.*, 2012). Other pathogens may affect the fecundity, growth and competitive potential of their host owing to the cumulative effects of multiple foliar lesions (Burdon, 1993; Allan *et al.*, 2010). Pathogens that directly attack the reproductive organs or the developing embryos can severely affect fecundity (Curran *et al.*, 2012). For example, *Microbotryum violaceum* completely sterilizes plants of many species in the Caryophyllaceae (Fig. 1c) by causing the abortion of female reproductive structures and replacing the anthers of infected plants with teliospores (Fujita *et al.*, 2012). Fungal

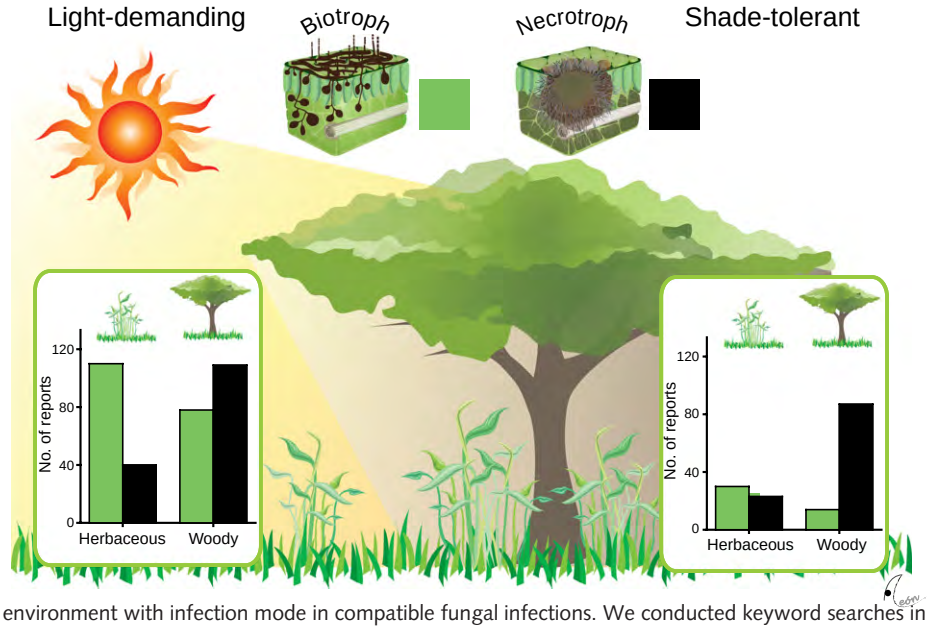

Fig. 6 Interaction of light environment with infection mode in compatible fungal infections. We conducted keyword searches in Web of Knowledge® (for 'endophyte', 'biotroph', 'necrotroph', 'fungal pathogen' and 'fung*' AND 'plant' AND 'life history', time period 1990–2012 and also considering cited work) and in Google Scholar, and illustrate here the number of reports relating to compatible infections by biotrophic vs necrotrophic fungal pathogens in dependence of plant lifestyle (herbaceous vs woody hosts) and light conditions (light-demanding vs shade-tolerant). For both woody and herbaceous host species, χ^2-tests indicate an interaction of light environment with fungal lifestyle: light-demanding herbaceous species were mostly affected by biotrophs ($\chi^2 = 32.7$, d.f. = 1, $P < 0.001$), whereas shade-tolerant woody species were mostly affected by necrotrophs ($\chi^2 = 33.5$, d.f. = 1, $P < 0.001$).

pathogens can also infect ripening fruits (Fig. 1e–g) and then cause significant pre-dispersal mortality of the developing seeds (Beckman & Muller-Landau, 2011).

Plant pathogens usually show some degree of host specificity and require a vector (such as insects, water or wind), which always shows at least some degree of distance dependency. In consequence, the probability at which a certain plant becomes infected is positively correlated with its distance to the closest conspecific plant that can function as a source of pathogens. Thus, pathogens decrease the survival rate of plants – and particularly of seedlings – in a density-dependent manner, favour rare and disfavour common species, favour the mixing of species, and thereby help to maintain plant diversity (Freckleton & Lewis, 2006; Lewis, 2010).

After its original formulation (Connell, 1971; Janzen, 1971), the 'Janzen–Connell hypothesis' has received empirical support from multiple studies. For example, the natural succession of plant species in coastal dune vegetation has been attributed to the action of plant-specific soil-borne diseases (van der Putten & Peters, 1997; van der Putten *et al.*, 2013). Pathogens can also kill the seedlings of several species of tropical trees in a density-dependent manner (Gallery *et al.*, 2010; Lewis, 2010; Liu *et al.*, 2012a,b; Swinfield *et al.*, 2012; Schweizer *et al.*, 2013). For obvious reasons, host-specific pathogens are likely to represent the most effective 'Janzen–Connell agents' and, indeed, most authors have reported that density-dependent mortality rates in tropical seedlings are caused by fungal host specialists (Bagchi *et al.*, 2010; Konno *et al.*, 2011; Liu *et al.*, 2012a,b; Terborgh, 2012). In summary, besides their role as destructive agents, pathogens also play an important role as constructive agents that help to maintain tropical biodiversity (Mordecai, 2011).

IV. Necrotrophic and biotrophic pathogens vs symptomless endophytes

Plant-infecting fungi are a heterogeneous group of organisms that strongly differ in important life-history traits such as dispersal mechanisms, types of reproduction and modes of parasitism (Burdon, 1993; Jarosz & Davelos, 1995; Gilbert, 2002; García-Guzmán & Morales, 2007; Burdon & Thrall, 2009). Thus, the outcome of a given infection depends on multiple traits of both the fungus and its host (Burdon, 1993; Pan & Clay, 2002; Barrett *et al.*, 2008; Nemri *et al.*, 2012). Nonetheless, the importance of lifestyle in influencing the timing, severity and selective impacts of disease has received little attention in natural systems (Burdon & Thrall, 2009).

Fungal pathogens obtain resources from their hosts in two principally different ways: as biotrophs or as necrotrophs (Mengiste, 2012) (Fig. 2). Whereas many fungi invariably follow one of these strategies, recent research demonstrated for an increasing number of species that they keep the cells of their host alive during the early phase of their infection cycle and then shift to killing host cells (hemi-biotrophs). However, it remains unknown whether the early phase of these fungi, which in most cases originally were considered necrotrophs, can be directly compared to the lifestyle of obligate biotrophs (Horbach *et al.*, 2011). Whereas biotrophs extract their nutrients from living host tissue, necrotrophs actively kill the cells of their host and then obtain nutrients from the dead tissue. Plant resistance responses to pathogens in these two major groups are orchestrated by two independent but interacting signalling pathways: salicylic acid (SA)-mediated signalling mainly acts against biotrophs, whereas jasmonic acid (JA)-mediated signalling mainly

acts against necrotrophs (see section VI. 'Plant resistance to necrotrophs and biotrophs', and Santner & Estelle, 2009; Mengiste, 2012; Thaler *et al.*, 2012; Lyons *et al.*, 2013; Wasternack & Hause, 2013; Wen, 2013; Westfall *et al.*, 2013 for reviews). Therefore, we focus here on these two major types of parasitism.

Necrotrophic pathogens are notorious for their aggressive virulence strategies that allow them to feed on dead or dying cells. These fungi produce a variety of phytotoxins, cell-wall-degrading enzymes and reactive oxygen species, which induce cell necrosis to support penetration and favour the leakage of nutrients (Mengiste, 2012). Cell-wall degrading enzymes are over-represented in the genomes of necrotrophic fungi and might contribute to the provision of carbohydrates to the fungus (Horbach *et al.*, 2011). Necrotrophs can produce a variety of low-molecular-weight phytotoxins that are targeted either against specific hosts or a variety of plant species (van Kan, 2006). By contrast, biotrophic pathogens derive nutrients from living tissues and usually must establish long-term relationships with their host to complete their life cycle. Biotrophs generally lack toxin production and produce low quantities of cell wall-degrading enzymes (Mengiste, 2012); that is, they exhibit coevolved pathogenesis mechanisms that control host physiology to keep their host alive (Mendgen & Hahn, 2002). Obligate biotrophs indent the plasma-membrane of the host cells. In fact, the extra-haustorial space, which is formed by both the haustorium and the host cell, represents a key determinant of biotrophy (Horbach *et al.*, 2011). Interestingly, the genome of the biotroph *Blumeria graminis* f. sp. *hordei* showed multiple gene losses, most of which affect enzymes of primary and secondary metabolism, carbohydrate-active enzymes and transporters (Spanu *et al.*, 2010). Consequently, biotrophs lack the ability to synthesize important metabolites and are highly dependent on their hosts (Kemen & Jones, 2012).

A third group of fungi that infect tropical plants is represented by endophytes (Fig. 2). Endophytes are very common and diverse (Arnold *et al.*, 2000, 2003; Saikkonen, 2007; Gazis & Chaverri, 2010), and it has been suggested that all plant species in nature are inhabited by them (Promputtha *et al.*, 2007; Hyde & Soytong, 2008; Rodríguez *et al.*, 2009; Aly *et al.*, 2011; Porras-Alfaro & Bayman, 2011). Some studies have considered fungal endophytes as mutualists because they can enhance plant resistance to pathogens (Arnold *et al.*, 2003; Herre *et al.*, 2007; Mejia *et al.*, 2008), herbivores (Hartley & Gange, 2009; Vega *et al.*, 2009; Estrada *et al.*, 2013) or abiotic stress (Márquez *et al.*, 2007). In spite of their ubiquity, surprisingly little is known about why some fungi live as asymptomatic endophytes, whereas others cause disease symptoms when they infect a plant and then behave as pathogens. A recent phylogenetic analysis (Delaye *et al.*, 2013) revealed that biotrophy usually represents a derived and evolutionarily stable trait, whereas fungi can easily switch between an endophytic and necrotrophic lifestyle (Fig. 7).

V. Endophytes as a reservoir of necrotrophic pathogens?

Fungal endophytes are likely to comprise > 1 million species (Arnold *et al.*, 2000) and represent an understudied component of tropical biodiversity. These fungi live entirely within plants and can contribute significantly to their phenotype (Gundel *et al.*, 2013; see Partida-Martínez & Heil, 2011 for a review), usually without causing any symptoms of disease (Rodríguez *et al.*, 2009; Purahong & Hyde, 2011). Factors that shape the community of endophytes and, for example, define their specificity towards specific host species, or organs, still remain to be discovered (Peršoh, 2013), although insect vectors play a central role in this context (Vega *et al.*, 2009; Cory & Ericsson, 2010; Pazoutova *et al.*, 2013). It also remains an open question how endophytes generally suppress (or escape from) their host's resistance mechanisms. However, several species can produce disease symptoms under particular conditions, for example, during leaf senescence (Stone, 1988; Kriel *et al.*, 2000) or in response to particular light conditions (Alvarez-Loayza *et al.*, 2011). As a result, several endophytic species have been considered as latent pathogens (Begoude *et al.*, 2011; Goodwin *et al.*, 2011; Andrew *et al.*, 2012; Sánchez-Márquez *et al.*, 2012). Among them, *Deightoniella torulosa*, a common endophyte in wild banana (*Musa acuminata*), causes leaf spots under certain environmental conditions (Photita *et al.*, 2004), and *Diplodia mutila*, an endophyte of the palm *Iriartea deltoidea*, can shift to pathogenic behaviour under elevated light conditions (Alvarez-Loayza *et al.*, 2011). Whether endophytism represents a true lifestyle or just a more or less prolonged state during the infection cycle of pathogens is still unclear. Nonetheless, this example suggests that climate conditions can trigger some fungal species to change roles from endophytes to pathogens.

Interestingly, fungal species that shift between the endophytic and the pathogenic lifestyle usually behave as necrotrophs when entering the pathogenic state. A recent phylogenetic analysis revealed that most endophytes cluster within taxa of necrotrophic pathogens (Fig. 7, and Delaye *et al.*, 2013). Furthermore, once biotrophy evolves there is no regression to one of the other two lifestyles (Delaye *et al.*, 2013). Thus, fungi can easily switch at the evolutionary or ecological timescale between symptomless endophytic growth in live host tissue and life as a necrotrophic pathogen, whereas biotrophy usually represents a derived and evolutionarily stable trait (Delaye *et al.*, 2013). It appears likely that the specific adaptations that are required by biotrophs strongly contribute to the evolutionary stability of the biotrophic lifestyle. These observations indicate that future studies aimed at understanding general patterns in plant disease should consider endophytes as a putative reservoir of necrotrophic fungal pathogens.

VI. Plant resistance to necrotrophs and biotrophs

Which factors determine the host range of a certain pathogen? Rather than being limited by primary metabolic demands, the capacity of fungi to infect a host is mainly determined by the interplay of host resistance traits and pathogen counter-adaptations (Schulze-Lefert & Panstruga, 2011; Barrett & Heil, 2012). Hosts have evolved sophisticated mechanisms to detect and resist invaders and perceive at least two major classes of molecules that indicate attack by a pathogen. Conserved microbial molecules such as chitin and flagellin serve as pathogen-associated molecular patterns (PAMPs), which hosts perceive via pattern recognition receptors

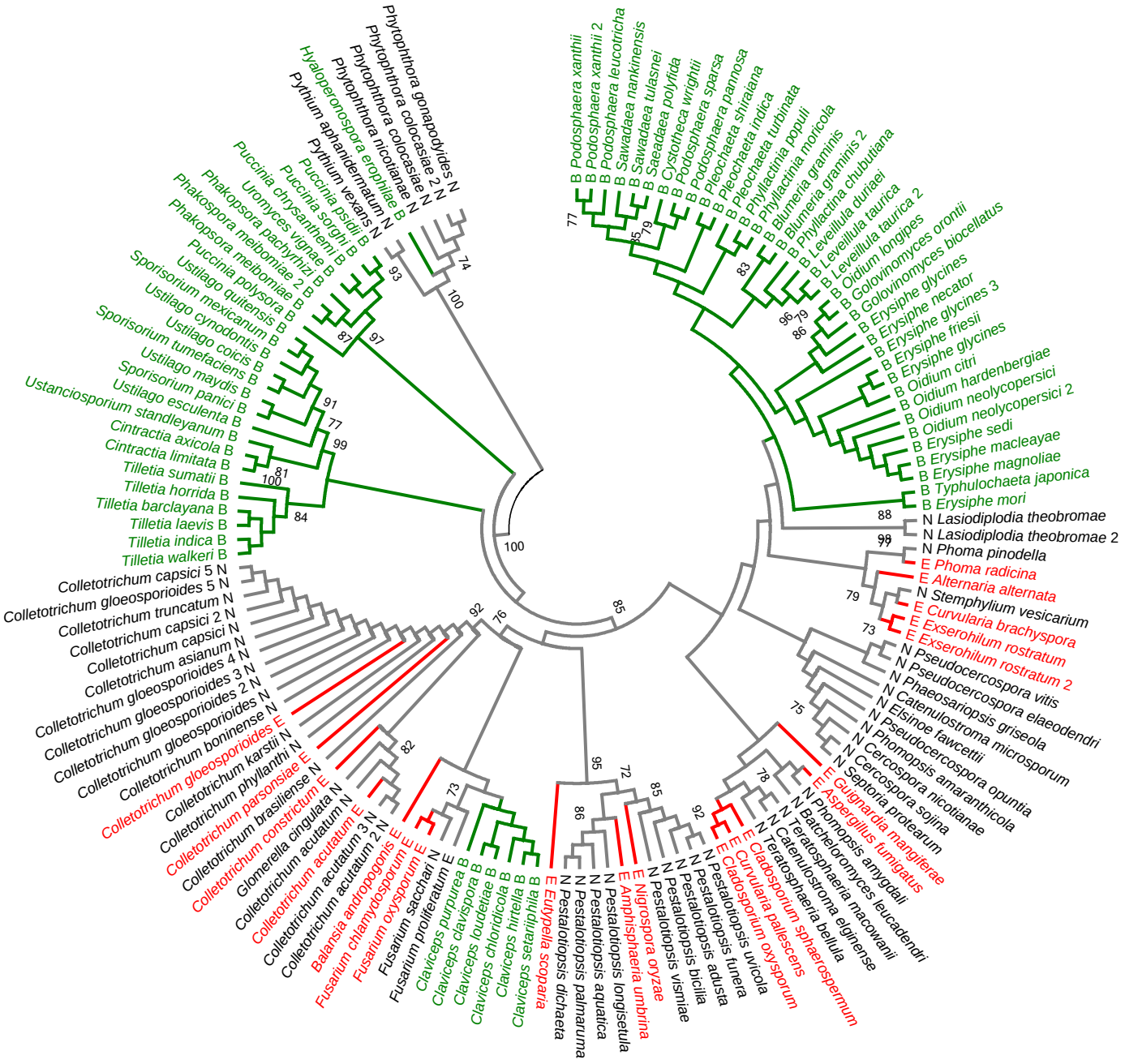

Fig. 7 Diagram summarizing the phylogenetic relationships among necrotrophic and biotrophic pathogens and symptomless fungal endophytes. A maximum-likelihood phylogeny based on the *5.8S* rRNA gene reveals that symptomless endophytes (shown in red) commonly cluster together with necrotrophs (shown in black), whereas biotrophs (shown in green) form independent clades. Only bootstrap values larger than 70 are shown. See Delaye *et al.* (2013) for details. Figure courtesy of L. Delaye and based on sequences used in Delaye *et al.* (2013).

(PRRs) to mount PAMP-triggered immunity (PTI) – a response that usually leads to a general resistance phenotype. However, pathogens have evolved molecular mechanisms to evade or suppress host resistance with the help of effectors (i.e. molecules that are released from plant enemies to manipulate the host) (Jones & Dangl, 2006; Pieterse *et al.*, 2009; Dodds & Rathjen, 2010; Hogenhout & Bos, 2011; Schulze-Lefert & Panstruga, 2011). Many hosts have, in turn, acquired the capacity to recognize either the changes that are inflicted by the action of these elicitors ('modified-self recognition'), or to directly and specifically recognize the effectors via their interaction with plant receptor proteins that contain nucleotide-binding (NB) domains and leucine-rich repeat (LRR) receptor kinases. The recognition of effectors by plant NB–LRR proteins results in further layers of more specific ('gene-for-gene') resistance responses denominated effector-triggered immunity (ETI) (Jones & Dangl, 2006; Dodds & Rathjen, 2010).

Both PAMPS and PRRs are highly conserved among species (Zipfel, 2009). By contrast, the effector repertoire of pathogens can be highly variable, particularly among species and host-specific

lineages (Schulze-Lefert & Panstruga, 2011). Similarly, NB–LRR resistance proteins are variable among species (Stahl *et al.*, 1999). Schulze-Lefert & Panstruga (2011) hypothesized that the interplay between highly conserved PRR-triggered immunity and highly specific (and evolutionarily derived) NB–LRR protein-triggered immunity can explain the general phylogenetic conservatism of plant enemies (see section V. 'Endophytes as a reservoir of necrotrophic pathogens?' and Fig. 7). For example, in the interaction between flax (*Linum usitatissimum*) and its fungal pathogen *Melampsora lini* (Fig. 1a), several interacting host r-genes and pathogen effector gene loci have been identified and provide alternate resistance and virulence (Dodds & Rathjen, 2010). Importantly, allelic variants at the *AvrP123* effector locus that escape recognition by one r-gene usually confer recognition by a different r-gene (Dodds & Rathjen, 2010). Analogous trade-offs might also mediate broader patterns of resistance specificity, such as resistance to insect herbivores vs microbial pathogens (Erb *et al.*, 2011; Thaler *et al.*, 2012), and biotrophic vs necrotrophic pathogens (Kliebenstein & Rowe, 2008).

One of the most important trade-offs applies to the resistance of plants to biotrophic pathogens, on the one hand, vs necrotrophic pathogens and chewing herbivores, on the other. As with pathogens, plants can recognize the action of herbivores – in a similar way to the above-mentioned 'modified-self recognition' strategy – by perceiving fragmented or delocalized molecules as damage-associated molecular patterns (DAMPs). The responses that are elicited by plant 'damaged-self recognition' are general ones and are commonly based on JA induction (Heil, 2009; Heil *et al.*, 2012). Necrotrophic pathogens lead to the death and disruption of multiple cells and, thus, like chewing herbivores, their action releases DAMPs. Therefore, necrotrophic pathogens usually also activate JA-dependent signalling pathways and are controlled by JA-dependent genes (Pieterse *et al.*, 2009; Thaler *et al.*, 2012). By contrast, biotrophic pathogens in general establish long-lasting interactions with living host cells and usually cause minimal physical damage. Plants resist these pathogens with SA-dependent resistance traits whose activation is commonly called 'systemic acquired resistance' (SAR). Hallmarks of SAR are local cell death and the systemic induction of phytoalexins and pathogenesis-related (PR) proteins, such as chitinases and glucanases (Dong, 2001; Métraux, 2001; Durrant & Dong, 2004; van Loon *et al.*, 2006).

VII. Plant defence theory

Plants differ in their resistance level depending on their species and the environmental conditions. Multiple theories have been formulated to predict and explain general patterns in the type and distribution of plant defence traits. These theories usually consider herbivores rather than pathogens, because plant pathologists traditionally focus on crops, whereas ecologists more commonly work on plant–animal interactions. Therefore, we briefly mention some of the theories that have been formulated in the context of plant anti-herbivore defence and then discuss how they can be used to interpret the infection of tropical plants by pathogens, in spite of their focus on a different group of organisms. All of these theories assume that resistance traits are costly and, therefore, that plants cannot produce unlimited amounts of resistance components. This general assumption has been reviewed previously (see Heil & Baldwin, 2002; Walters & Heil, 2007; McCall & Fordyce, 2010; Agrawal, 2011; Endara & Coley, 2011) and is not discussed further here.

1. The growth–differentiation balance hypothesis (GDBH)

Herms & Mattson (1992) suggested that the synthesis of defensive compounds represents a differentiation process and, thus, is subject to a general physiological trade-off between growth and differentiation. This trade-off can limit the expression of resistance traits as soon as resources become limited that are shared by growth and resistance-related processes. Herms & Mattson (1992) stated that nutrient-rich conditions favour growth and result in a low relative availability of carbon for secondary metabolism. By contrast, any factor that limits growth more than photosynthesis will free up resources for allocation to secondary metabolism.

Based on these assumptions, the GDBH 'provides a framework for understanding phenotypic variation in secondary metabolism' (Herms & Mattson, 1992). First, the described trade-off limits the defence of young, fast-growing plant parts, which causes ontogenetic differences in defence allocation. Second, inherently fast-growing species (growth-dominated plants) are predicted to have lower levels of defence than in-herently slow-growing species (differentiation-dominated plants). Third, competition is more severe under nutrient-rich conditions (thus favouring growth-dominated plants), and lost growth and competitive ability represent more severe costs than the loss of some tissue due to consumption by herbivores. These conditions constrain the evolution of high constitutive defence levels but can favour the evolution of inducible resistance, which 'may be an adaptation minimizing the cost of defence' (Herms & Mattson, 1992).

2. The resource availability hypothesis (RAH)

Coley *et al.* (1985) wanted to understand 'why plant species differ in their commitment to defences and hence in their susceptibility to herbivores'. The resource availability hypothesis (RAH) (Bryant *et al.*, 1985; Coley *et al.*, 1985) predicts the type and amount of defence based on inherent growth rates and leaf lifespans, which have evolved as an adaptation to the resource availabilities at the typical growing sites of the plant species. Therefore, whereas the GDBH also applies to within-species differences (i.e. ontogenetic effects and site effects), the RAH explicitly compares different species.

Plants growing at resource-poor sites are characterized by inherently slow growth rates, low maximum photosynthetic rates and low turnover rates (e.g. long-lived leaves) (Bryant *et al.*, 1985; Coley *et al.*, 1985; Arendt, 1997). The opposite traits are characteristic of plants at resource-rich sites that, moreover, should respond flexibly to fluctuations in the available resources and, thus, should exhibit a high morphological and biochemical flexibility. Coley *et al.* (1985) supposed, 'that the optimal level of defence

investment increases as the potential growth rate of the plant decreases'. This prediction is based on three main arguments. (1) Replacement of plant parts lost to herbivores is more costly when nutrients are more limiting for future growth. (2) The relative impact of herbivory increases with decreasing inherent growth rate because a given rate of herbivory represents a larger relative proportion of the biomass production of a plant. (3) Similarly, a percentage reduction in growth rate due to the cost of producing defences represents a greater absolute growth reduction for fast-growing species than for slow-growing species.

3. The plant apparency hypothesis (PAH)

Feeny (1970) compared early successional crucifers with oaks that form a climax stage in vegetation development and found that the first group are defended mainly by glucosinolates whereas oaks are defended by tannins. Feeny (1975) defined the first group of compounds as 'qualitative defences', which are characterized by the fact that they act at low concentrations. Dose dependency plays a role in the effects of these compounds but it is their mere presence that leads to resistance against most generalist insects. However, specialists have evolved to overcome these defences. Tannins, by contrast, were assumed to act strictly in a dosage-dependent manner and via a biochemical mechanism (the precipitation of proteins) that hardly allows counter-adaptations. Therefore, tannins and similarly acting compounds such as lignins and many mechanical defences were termed 'quantitative defences'. The main reason for using the 'quantitative–qualitative' terminology was 'to contrast the scales of evolutionary counter-adaptations: with the right detoxification mechanisms, herbivores can potentially evolve to overcome qualitative defences at relatively little cost. When faced with quantitative resistance, by contrast, an herbivore is still faced with poor food and must typically adapt to slow growth' (P. Feeny, pers. comm.).

Feeny (1976) used these considerations to discuss how plants should use different kinds of chemical defence with respect to their life history and their resulting danger of being discovered and attacked by herbivores. Feeny (1976) used the term 'apparency' to describe 'the susceptibility of an individual plant to discovery by its enemies in ecological time' and predicted that (1) very apparent species should allocate higher proportions of metabolic resources to defence than less apparent species; and (2) unapparent plants should contain mainly qualitative defences, whereas apparent plants should be defended by quantitative compounds.

VIII. General patterns in fungal infections of tropical plants

We searched for reports on the hosts and the distribution of – and symptoms caused by – fungal plant pathogens in tropical areas using Google Scholar, Biological Abstracts and Current Contents. In order to obtain clear patterns, we focused on the end-points of the continuum in the infection strategies of fungi and, hence, considered only reports that used phenotypic symptoms to clearly characterise the infection as either 'biotrophic' or 'necrotrophic'. We determined plant life form (annual or perennial herbs or grasses, and evergreen or deciduous trees), site (open areas, open forest, tropical rain forest, deciduous forest, cerrado, savanna, etc.) and plant guild (shade-tolerant or light-demanding) from the respective publications and, when this information was missing, by consulting the Missouri Botanical Garden's VAST (VAScular Tropicos) nomenclatural database (http://www.tropicos.org/), Flora of Australia Online (http://www.anbg.gov.au/abrs/online-resources/flora/), FloraBase, The Western Australia Flora (http://florabase.calm.wa.gov.au), and the Instituto Nacional de Biodiversidad, Costa Rica database (http://www.inbio.ac.cr/es/default.html). Fungal life form (biotrophic or necrotrophic) was confirmed using MycoBank databases (http://www.mycobank.org), as well as phytopathological literature, but we restricted our analysis to cases in which clear symptoms were described in the original publication. In addition, we investigated whether any pathogenic fungal species found in our search had also been recorded as an endophyte.

We identified 493 tropical plant–fungal associations that comprised 203 annual and perennial herbs and grasses and 290 woody plant species. The fungi comprised necrotic pathogens which were described as causing leaf spots, stem cankers and damping-off diseases, and biotrophic pathogens which were described as tar spots, mildews, rusts and smuts (Fig. 1, and Supporting Information Table S1). Among the fungi that we found to be reported as necrotrophs, two species were more recently considered to be hemi-biotrophs and 28 species belong to genera that are now commonly treated as hemi-biotrophs. Interestingly, 14 out of the 493 pathogenic fungal species were also reported as endophytes, and all of these fungi behave as necrotrophs in their pathogenic state (for more examples of fungal species switching between the endophytic and the necrotrophic lifestyle, see Delaye *et al.*, 2013). With respect to host life-history, we found more reports on fungal infections in evergreen than in deciduous species (Fig. 4), more reports on infections in light-demanding than in shade-tolerant hosts (Fig. 5), and a higher frequency of infections by necrotrophic pathogens in shade-tolerant trees, whereas light-demanding herbaceous plants suffered relatively more from biotrophic infections (Fig. 6).

IX. Explaining patterns

1. Infections in evergreen vs deciduous hosts

We identified three times the number of reports on compatible infections by fungal pathogens in tropical evergreen than in deciduous host species, which differs significantly from the expectation of equal numbers of cases ($\chi^2 = 45$, d.f. = 1, $P < 0.001$) (Fig. 4). This observation supports a central assumption of the PAH because it demonstrates that the continuous presence of the foliage of evergreen species makes it more predictable from the perspective of the pathogens. These 'apparent' leaves are, thus, under a higher enemy pressure. Similarly, the number of pathogens found in a plant species increases with the area that is inhabited by this particular species (Miller, 2012), which indicates a higher disease pressure on plant species that cover large areas (and, hence, are more apparent). However, interestingly, PAH fails to predict the consequences: the evergreen species do not appear to show an

adequate evolutionary response (an enhancement of their resistance levels). Have pathogens won the race in tropical ecosystems because hosts are evolving too slowly to fulfil the predictions made by the PAH? Interestingly, the opposite pattern was detected in a preliminary screening of reports on temperate systems (Fig. S1, Table S2), which indicates that higher infection rates in evergreen trees might be a characteristic of certain tropical ecosystems.

Higher infection rates in evergreen hosts also contradict the GDBH and the RAH. Evergreen trees can photosynthesize year round and invest in tough leaves that persist through long periods (Kikuzawa, 1991). By contrast, deciduous species have a limited period of the year available for photosynthesis and require high contents of nitrogen and water to make this process as efficient as possible (Mooney & Dunn, 1970; Reich *et al.*, 1991, 1992; Poorter *et al.*, 2009). Moreover, relative growth rates in general decrease with increasing leaf lifespans (Reich *et al.*, 1992). Thus, limitations as expressed in the GDBH should constrain the possibilities for defending the fast expanding and metabolically active short-lived leaves of deciduous plants more than the long-lived leaves of evergreen species; also considerations relating to the RAH predict that (slow-growing) evergreen species should exhibit enhanced resistance levels. Whereas this prediction has been confirmed for herbivory (Coley & Aide, 1991) and seems to apply also to the infection of temperate trees by fungal pathogens (Fig. S1), we found the opposite pattern in the reports on fungal infections in the tropics (Fig. 4). Thus, although the RAH, PAH and GDBH in this context successfully predict the general patterns in herbivory, they fail to predict the frequency of infections by fungi in tropical plants.

This contradiction might result in part from the trade-off between induced plant resistance to herbivores and pathogens (Heil & Bostock, 2002; Thaler *et al.*, 2012), which, if applicable, would simply predict contrasting general patterns in damage caused by herbivores and pathogens. However, several constitutive defensive traits such as lignified cell walls and thick cuticles help plants to resist both herbivores and pathogens, and these 'quantitative' resistance traits in particular should be enhanced in evergreen species. Why then are evergreens still more frequently infected than deciduous species despite these defensive traits? Interestingly, the observed pattern is fully consistent with the hypothesis that deciduous species reduce the population size of their enemies by depriving them of their host during part of the year (Coley, 1998; Karban, 2007, 2008).

The interpretation of our data is limited by the fact that the standing damage level of a plant depends on both its levels of resistance and the current or past severity of the enemy pressure. Simply speaking, low levels of damage can result from an efficient resistance or just from the absence of enemies, whereas under high enemy pressure, even a reasonably resistant plant will suffer from damage. Thus, defining the mechanism that causes the described pattern would require systematic quantifications of the true enemy pressure and of the resistance levels, a task that is hardly ever fulfilled for any resistance trait. However, it appears that the deciduous habit deprives tropical fungal pathogens of suitable hosts during a significant part of the year and thereby reduces the pressure that is exerted by these enemies.

2. Infections in fast-growing vs slow-growing hosts

We found twice as many reports of fungal infections in tropical fast-growing, light-demanding hosts than in slow-growing, shade-tolerant species (Fig. 5), which is consistent with earlier observations that light-dependent tree species in the tropics are more affected by leaf fungal pathogens than shade-tolerant species (García-Guzmán & Espinosa-García, 2011). Similarly, in a survey of temperate systems we found more infections in light-demanding than shade-tolerant hosts (Fig. S2). Here, an interaction between the growing site and the host lifestyle became apparent: the light-demanding hosts among the herbaceous species were three times more commonly reported to be infected by fungi than shade-tolerant hosts, whereas this difference was much less pronounced for woody hosts (193 cases of infection in light-demanding species vs 101 cases in shade-tolerant species, see Fig. 5). This distribution differed significantly from a homogenous distribution ($P < 0.01$).

'Canopy light and plant health' (Ballaré *et al.*, 2012) interact in multiple ways. For example, UV-B radiation in general increases plant resistance to microbial pathogens (Kunz *et al.*, 2008; Demkura & Ballaré, 2012). Therefore, this effect should lower the disease incidence in light-demanding species or, at least, in the light-exposed parts of plants, whereas our analysis of the literature suggested the opposite pattern. Care must be taken when interpreting the patterns reported here at the physiological level because our analysis concerns only numbers of reported cases. Detailed information is usually lacking regarding the disease severity or the abiotic conditions under which these observations were made. However, interestingly, this general pattern is consistent with the prediction made by both the RAH and the GDBH that light-demanding, fast-growing species invest less in defence because they suffer from greater trade-offs between growth and defence (GDBH) and/or because they are under greater pressure from competing neighbours and can replace damaged tissue more easily (RAH). In this case, classical plant defence theory is a better predictor of general patterns in the literature on the infection of tropical plants by fungi than physiological studies on model plants.

3. Biotrophs vs necrotrophs and the light environment

Contingency analysis detected contrasting frequencies in the reports on biotrophs and necrotrophs that affect light-demanding vs shade-tolerant tropical hosts and, again, this pattern was also dependent on host life history (i.e. herbaceous vs woody host; see Fig. 6). The light-demanding herbaceous species were mostly affected by biotrophs ($\chi^2 = 31.8$, d.f. = 1, $P < 0.01$) and light-demanding woody species were slightly more affected by necrotrophs ($\chi^2 = 4.74$ d.f. = 1, $P < 0.05$) (Fig. 6). By contrast, the shade-tolerant herbaceous species were affected at equal frequencies by necrotrophic and biotrophic pathogens, whereas shade-tolerant woody species were mostly affected by necrotrophs (Fig. 6; $\chi^2 = 46.3$, d.f. = 1, $P < 0.01$). We detected surprisingly similar patterns in our preliminary survey of reports from temperate systems: biotrophs were most frequently reported from light-demanding herbaceous hosts whereas necrotrophs were most frequently reported from shade-tolerant and woody hosts (Fig. S3).

In summary, necrotrophs more commonly affect shade-tolerant plants and woody species whereas biotrophs are reported more commonly from light-demanding and herbaceous hosts.

The different hormonal pathways controlling plant resistance to necrotrophs and biotrophs in combination with the inhibitory effects of far-red perception on certain plant resistance traits are likely to provide us with a physiological explanation of this ecological pattern. Plants perceive current and future competitors via phytochrome B, which responds to the ratio of red to far-red (R : FR) radiation that reaches a plant. Because chlorophyll absorbs more R than FR radiation, plants their resource allocation from defence to growth in accordance with the RAH shift when they receive low R:FR values (Izaguirre *et al.*, 2006; Ballaré, 2009; Ballaré *et al.*, 2012). FR perception inhibits JA signalling (Izaguirre *et al.*, 2006; Ballaré, 2009; Moreno *et al.*, 2009) and plants resist infection by necrotrophs mainly by using JA-dependent responses (Pieterse *et al.*, 2009). Indeed, *Arabidopsis* plants pre-treated with FR radiation impaired resistance to *Botrytis cinerea*, a necrotrophic pathogen (Cerrudo *et al.*, 2012), and de Wit *et al.* (2013) have confirmed that disease resistance is inhibited by a low R : FR ratio. Both light quality and quantity are crucial regulators of JA signalling (Moreno *et al.*, 2009; Demkura *et al.*, 2010; Robson *et al.*, 2010) and plant defence (Roberts & Paul, 2006; Ballaré, 2011; Kazan & Manners, 2011; de Wit *et al.*, 2013). In summary, the impaired JA signalling caused by low R : FR ratios in the understorey is likely to reduce resistance to necrotrophs in shaded plants and in shaded parts of large, modular woody pants and climbers (Izaguirre *et al.*, 2013).

Further considerations will require systematic research into the physiological mechanisms that cause phenotypic susceptibility to pathogens. One is that many fungal pathogens release carbohydrate-active enzymes (CAZymes; see www.cazy.org) for the degradation of plant polysaccharides and the breakdown of the plant cell wall (Cantarel *et al.*, 2009). Interestingly, biotrophic fungi tend to have fewer CAZymes than necrotrophic fungi (Horbach *et al.*, 2011; Zhao *et al.*, 2013). A recent study by Kitajima *et al.* (2012) suggests that carbon allocation to cellulose contributes to leaf toughness, particularly within the shade-tolerant plant species, but less so in light-dependent plants. A second consideration is that several plant immune responses are exploited by necrotrophic pathogens (Mengiste, 2012). For example, cell death associated with the hypersensitive response impairs pathogenic growth by stopping nutrient allocation; however, cell death has different roles in plant responses to necrotrophic and biotrophic pathogens. Whereas fast and local cell death can effectively trap a biotroph in a ring of dead (and, thus, unsuitable) tissue (Kombrink & Schmelzer, 2001), local cell death occurs early during infection by necrotrophs and in this situation is an indicator of successful infection (Govrin *et al.*, 2006; van Kan, 2006). Mutants with enhanced cell death express increased resistance to biotrophic pathogens but enhanced susceptibility to necrotrophs (Govrin & Levine, 2000; Veronese *et al.*, 2004). For example, the *erecta* mutant of *Arabidopsis* is susceptible to many necrotrophs (Nühse, 2012), and this susceptibility is accompanied by increased amounts of cellulose in the plant cell (Godiard *et al.*, 2003; Llorente *et al.*, 2005; Sánchez-Rodríguez *et al.*, 2009). Along the same line, we observed that monocots (mostly represented by the group of annual and perennial herbs, *cf.* Table S1) are more commonly affected by biotrophs than dicots. Cell walls of dicots usually exhibit higher contents of pectin and hemicelluloses than cell walls of monocots (Vogel, 2008; Lagaert *et al.*, 2009; King *et al.*, 2011). Accordingly, necrotrophs attacking dicots (most of the woody species in our dataset) contain more pectinases than the pathogens that infect monocots (Zhao *et al.*, 2013). Thus, if shade-tolerant plants allocate more carbon to cellulose and pectin, necrotrophs might employ an enhanced secretion of diverse hydrolytic enzymes to attack shade-tolerant species more efficiently, whereas biotrophs in general might lack the adequate enzymatic equipment for successful invasion. This mechanism provides an alternative and nonexclusive explanation for the greater number of infections by necrotrophic pathogens in shade-tolerant plants (Fig. 6).

X. Conclusions and outlook

Our literature survey revealed that light conditions, host growth rates and fungal lifestyle represent important predictors of general patterns in the reports on fungal infection in tropical noncultivated plants. Interestingly, the greater number of reports on fungal infection in fast-growing and light-demanding hosts is fully consistent with predictions of classical plant defence theory, whereas both the RAH and the GDBH failed to predict correctly the infection frequencies reported in tropical evergreen vs deciduous hosts. By contrast, the higher infection rates of shade-tolerant hosts by necrotrophic pathogens are more likely to result from the physiological and molecular mechanisms that underlie specific resistance responses.

A shortcoming of our approach is its dependency on the number of reported cases. Thus, sampling biases might have contributed significantly to the reported patterns. Therefore, all our interpretations are based on the assumption that these patterns to some degree reflect the situation in nature. Future investigations should aim at balanced, unbiased censuses in order to corroborate these patterns and should experimentally separate resistance levels from pathogen pressure under the different environmental conditions that we discuss here. However, our study reveals concrete and testable predictions. (1) According to the PAH (and considering r-gene-mediated resistance as equivalent to 'qualitative defence', whereas thickened and more lignified cell walls should qualify as 'quantitative' defences), we can predict that unapparent host species should be more dependent on r-gene-mediated resistance and, thus, suffer more commonly from infection by specialists. Reliable and complete data on natural host ranges of pathogens and on the number of r-genes in the genomes of the hosts will be required to test this prediction and can be obtained by employing whole-genome sequencing of the host species and DNA barcoding of all the fungi that are present in plants. (2) According to both the GDBH and the RAH, fast-growing plants should exhibit lower absolute levels of resistance. This trade-off might explain in part the low resistance levels of crops, which to a large degree represent fast-growing and highly light-demanding species. Adequate empirical tests of this prediction will depend on the use of generalist

pathogens to quantify in a comparative manner the resistance levels of different plant species and under different growing conditions. (3) Our considerations concerning the effects of R:FR signalling have led to the prediction of lower concentrations of endogenous JA in shaded plants, or parts of plants, than in plants growing under full sunlight. Both predictions would be easy to test under natural conditions. Combining classical plant defence theory with information on the molecular and biochemical mechanisms that cause resistance vs susceptibility enables a better understanding of general patterns in fungal plant diseases.

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Supporting Information

Additional supporting information may be found in the online version of this article.

Fig. S1 Numbers of cases of fungal infections in deciduous vs evergreen trees found in the reports on temperate systems.

Fig. S2 Numbers of cases of fungal infections in herbaceous and woody hosts growing under different light conditions found in the reports on temperate systems.

Fig. S3 Numbers of cases of infections by biotrophs and necrotrophs in herbaceous and woody hosts growing under different light conditions found in the reports on temperate systems.

Table S1 Cases of fungal infections in tropical wild plant species that were used for the present analysis (Figs 4–6)

Table S2 Cases of fungal infections in temperate systems that were used for the present analysis (Supporting Information Figs S1–S3)

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