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Endophytes versus biotrophic and necrotrophic pathogens—are fungal lifestyles evolutionarily stable traits?

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Abstract Endophytes infect living plant tissues without causing symptoms of disease. Indeed, many of them contribute to the resistance phenotype of their host. However, fungal endophytes are generally closely related to plant pathogens, fungi that either develop within living host tissue (biotrophic fungi) or that kill the host cells and then live in the dead tissue (necrotrophic fungi). We adopted a phylogenetic approach to investigate whether these strategies represent evolutionarily stable lifestyles and to elucidate their general phylogenetic relationships. We analysed 163 fungal strains for which we found information on the sequence of the 5.8S rRNA gene and the flanking internal transcribed spacer regions, the identity of the host plant and the concrete phenotypic outcome of the infection. A Maximum-Likelihood analysis combined with ancestral character mapping by maximum parsimony revealed that some fungal lineages had switched multiple times between a necrotrophic and an endophytic lifestyle. Ancestral character mapping indicated a minimum of four changes from an endophytic to a necrotrophic lifestyle, four changes in the opposite direction and eight changes among these lifestyles for which the direction could not be determined unambiguously. By contrast, biotrophs formed five clusters that did not contain necrotrophs or endophytes. Once biotrophy evolves there is apparently no regression to one of the other two lifestyles. We conclude that biotrophy usually represents a derived and evolutionarily stable trait, whereas fungi easily can switch between an endophytic and necrotrophic lifestyle at the evolutionary and even the ecological timescale. Future experimental studies should focus on the environmental or genetic changes that cause the rapid switches between these two phenotypically different lifestyles.

Keywords Disease expression · Fungi · Plant pathogens · Phylogenetic effects

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Introduction

Microfungi comprise a heterogeneous group of organisms with diverse lifestyles, which vary in traits such as dispersal mechanism, type of reproduction, growth, nutrient assimilation and parasitism (Burdon 1993; Gilbert 2002; García-Guzmán and Morales 2007; Dickman and Figueiredo 2011; Porras-Alfaro and Bayman 2011; Martin et al. 2013). Furthermore, plant-infecting fungi in particular affect their host plant in many ways, and establish associations ranging from mutualistic to parasitic (Schulz and Boyle 2005; Kogel et al. 2006; Rodriguez et al. 2009; Saikkonen et al. 2010; Partida-Martínez and Heil 2011; Junker et al. 2012; White and Bacon 2012). The type of association established with their host plant represents a major life history strategy that seemingly differs greatly among fungal species. Whereas mycorrhiza and asymptomatic endophytes exemplify the mutualistic or commensalistic side of this spectrum of associations, other fungi behave as latent and virulent pathogens that kill their host plant, or at least strongly reduce its performance and, ultimately, fitness (Jarosz and Davelos 1995;

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Schulz and Boyle 2005; Brown and Tellier 2011; Kolb 2012; Swinfield et al. 2012).

Strictly speaking, endophytes are all types of microorganisms that live entirely within plants (Partida-Martínez and Heil 2011). Most commonly, however, the term 'endophyte' is used for fungi that infect living tissues without causing any concurrent symptoms of disease (Wilson 1995; Rodriguez et al. 2009; Purahong and Hyde 2011), and this definition will be used throughout the present work. This definition is strictly operational and thus context-dependent, because it considers only the current effects of a specific fungus in a specific host plant under the concurrent environmental conditions. Therefore, several endophytes have been considered as latent pathogens (Carroll 1988; Begoude et al. 2011; Goodwin et al. 2011; Andrew et al. 2012; Sanchez-Marquez et al. 2012). Below, we will discuss more examples of fungi that have been isolated as symptomless endophytes but then behaved as pathogens under changed environmental conditions. Endophytes are extremely common and diverse (Arnold et al. 2000; Saikkonen 2007; Albrectsen et al. 2010; Gazis and Chaverri 2010; Ghimire et al. 2011). Indeed, it has been predicted that no wild plant is entirely free of them (Arnold 2007; Promputtha et al. 2007; Hyde and Soytong 2008; Rodriguez et al. 2009; Aly et al. 2011; Porras-Alfaro and Bayman 2011). Two major groups of endophytes (clavicipitaceous and non-clavicipitaceous) have been distinguished based on phylogeny and life history traits (Rodriguez et al. 2009). Clavicipitaceous fungal endophytes are restricted to certain grass species, but non-clavicipitaceous fungal endophytes have a wide range of both nonvascular and vascular host plant species (Rodriguez et al. 2009). However, recent studies suggest that members of the non-clavicipitaceous group should be separated into three subgroups according to host range, tissue colonized, transmission, in planta colonization, biodiversity, and fitness benefits conferred to hosts (Rodriguez et al. 2009; Purahong and Hyde 2011).

An infection by endophytes might affect the phenotype of the host in various ways, including the resistance of the host to pathogens (Arnold et al. 2003; Lehtonen et al. 2006; Herre et al. 2007; Mejia et al. 2008), herbivores (Meister et al. 2006; Hartley and Gange 2009; Vega et al. 2009; Albrectsen et al. 2010; Estrada et al. 2013) or abiotic stresses (Márquez et al. 2007; Hamilton and Bauerle 2012). However, the outcome of these interactions is highly context-dependent (Saikkonen et al. 2010). These fungi also play an important role in litter decomposition (Purahong and Hyde 2011; Peršoh et al. 2013).

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 biotrophic pathogens (which obtain energy and nutrients from living plant tissue) or as necrotrophs (pathogens that kill the host tissue and then extract energy and nutrients from the dead tissue) (Kemen and Jones 2012). Faced with the multitude of possible outcomes of an infection by fungi, we still lack a convincing and general theory that could help to predict the outcome of a particular plant—microbe interaction (Kogel et al. 2006).

Which factors determine disease expression: the host, the fungus, both, the evolutionary history of one or both of them, or the environment? Is being a pathogen versus an endophyte an evolutionarily stable strategy, or can fungi easily change their lifestyle? The latter interpretation receives support from reports of individual fungal strains that can act as pathogens or endophytes in the same host (Álvarez-Loayza et al. 2011; Promputtha et al. 2010). More general considerations have also predicted that factors such as plant age and abiotic environment, rather than simply species identity, might influence whether a fungus behaves as a plant pathogen or as a symptomless endophyte, and a mutualist—parasite continuum has been proposed (Saikkonen et al. 1998; Schulz and Boyle 2005; Hyde and Soytong 2008; Porras-Alfaro and Bayman 2011). In this study we used a phylogenetic approach to investigate whether biotrophic, necrotrophic and endophytic lifestyles represent evolutionarily stable life history traits within the fungi.

Methods

Database To explore whether biotrophic, necrotrophic and endophytic lifestyles represent evolutionarily stable life history traits within the fungi, we carried out an extensive literature review. We conducted key-word searches in Web of Knowledge® (for 'endophyte', 'biotroph', 'necrotroph', 'fungal pathogen' and 'fung*' AND 'plant' AND 'life history') and in Google Scholar, Biological Abstracts, and GenBank records and analysed the respective studies and the references cited therein. To construct our database (Supplementary Table 1), we only considered those studies that reported the identity of both the host plant and the fungus, the phenotypic outcome of the infection (endophytic, or biotrophic or necrotrophic pathogens), and the sequence of the 5.8S ribosomal RNA (rRNA) gene and the flanking internal transcribed spacer (ITS) regions. Studies lacking information on the host plant, the realized lifestyle of the fungal species in the host or the sequence of the 5.8S gene and the ITS regions were strictly discarded.

Taxonomic names Throughout the manuscript, including Figures and Tables, we use all taxonomic names as they were used in the original studies, rather than current names, even for sequences that would give a different identification according to the current taxonomy. We have decided for this solution in order not to lose the direct connection among sequence, name and lifestyles of the fungus, as it has been reported in the original studies that form the basis of our analysis of the phylogenetic relations among the lifestyles of plant-infecting fungi.

Phylogenetic reconstruction The 5.8S rRNA and ITS sequences of 163 fungal strains with described lifestyle in planta (see above) were downloaded from the GenBank database (http://www.ncbi.nlm.nih.gov/nuccore/) during August and September 2012. Sequences were aligned with MUSCLE v3.8 (Edgar 2004), and an initial phylogenetic tree was reconstructed by Maximum-Likelihood with MEGA5 (Tamura et al. 2011). The tree was reconstructed by using the model of evolution with the largest *lnL* (GTR + G + I), and with 100 bootstrap replicas. Hereafter we will refer to this tree as *initial-t*.

This *initial-t* showed several polytomies (internal branches showing bootstrap values lower than 50), which is likely due to the low number of aligned gap-free positions (only 174 sites). Therefore we implemented a 'divide and conquer' approach to resolve the phylogenetic history of this group of fungi. To divide the initial set of 163 sequences into smaller groups of closely related sequences, we used the software CLANS (Cluster Analysis of Sequences) (Frickey and Weiller 2007). CLANS first uses BLAST (Altschul et al. 1997) to measure the similarity among sequences, and then to identify groups of related sequences with a specified similarity threshold based on e-values. We used the program to identify all clusters of sequences sharing an e-value $<1.0e^{-100}$. This led to seven clusters of sequences (named clusters I to VII) comprising 147 sequences. The remaining 16 sequences were too divergent to be included in any of the above clusters. Interestingly, with the exception of cluster I, all clusters were supported by bootstrap values of at least 50 in *initial-t* (Fig. 1). This strongly suggests that the clusters identified by CLANS consisted of group of closely related sequences. Then we proceeded to reconstruct the evolutionary history within each cluster.

For each cluster (I to VII), we aligned again the sequences with MUSCLE and reconstructed a Maximum-Likelihood phylogenetic tree with the model of evolution GTR + G + I and with 100 bootstrap replicates as implemented in MEGA5. Owing to the larger number of gap-free aligned positions, several clades that were previously unresolved in *initial-t* had bootstrap values larger than 50. These trees, that we will call *cluster-trees*, were used to map the evolutionary history of lifestyle traits.

Rooting the trees To identify the position of the root in trees from clusters I to VI (*cluster-trees*), we repeated again the same phylogenetic analyses described in the previous section now using sequences from cluster VII (which consisted of sequences from Oomycetes) as external group. These trees were not used for tracing the evolution life traits. They were used only as a guide to place the branch on the *cluster-trees*.

Tracing the evolution of lifestyle traits To trace the evolution of the lifestyles 'biotroph' (B), 'endophyte' (E) and 'necrotroph' (N) along each of the six trees we used a maximum parsimony algorithm implemented in MESQUITE (Maddison and Maddison 2011) on the trees reconstructed from sequences from each of the clusters I to VI (*cluster-trees*). We counted the number of changes in lifestyles in all branches with a bootstrap of at least 50. The root from each of the *cluster-trees* wasn't included in these counts.

Results

Our divide and conquer approach to phylogenetic reconstruction led to the identification of seven clusters of closely related sequences, six of them belonging to in-groups and one formed by the out-group (Fig. 1). Species belonging to the Ascomycota were found in clusters I, II and III, whereas species belonging to the Basidiomycota were found in clusters IV, V and VI. Most importantly in the context of the present study, biotrophic fungi formed five discrete monophyletic groups (clusters II, IV, V, VI and a clade within cluster I) that did not contain any necrotrophic or endophytic species. By contrast, necrotrophic and endophytic fungi appeared together in clusters I and III. Cluster I was made up of endophytic and necrotrophic species, as well as biotrophic species from the Hypocreales (Fig. 2). Only biotrophic species from the *Erysiphaceae* family were found in cluster II (Figure S 1). Cluster III contained necrotrophs and endophytes from the Pleosporaceae and *Didymellaceae* families (Fig. 3). Clusters IV, V and VI contained biotrophic pathogens from the Basidiomycota (Figures S 2–4). Species from the Ustilaginaceae were grouped in cluster IV and cluster V comprised species from the *Tilletiaceae* family. Finally, the *Uredinales* were grouped in cluster VI.

Maximum-Likelihood phylogenetic analysis combined with ancestral character mapping by parsimony indicated four evolutionary changes from an endophytic lifestyle to a necrotrophic lifestyle, four changes in the opposite direction, and eight nodes for which it was not possible to determine the ancestral character state (i.e., whether the lifestyle was endophytic or necrotrophic, see Table 1). Our data show that once biotrophy has evolved, there is no regression to the other two lifestyles.

Discussion

Our results indicate at least 16 changes between an endophytic and a necrotrophic lifestyle among the 163 fungal strains that were analysed in our study. Changes in both directions (endophyte to necrotroph and *vice versa*) occurred at equal frequency. By contrast, we found four independent biotrophic clades and only one change from an endophytic lifestyle to a pathogenic biotrophic lifestyle

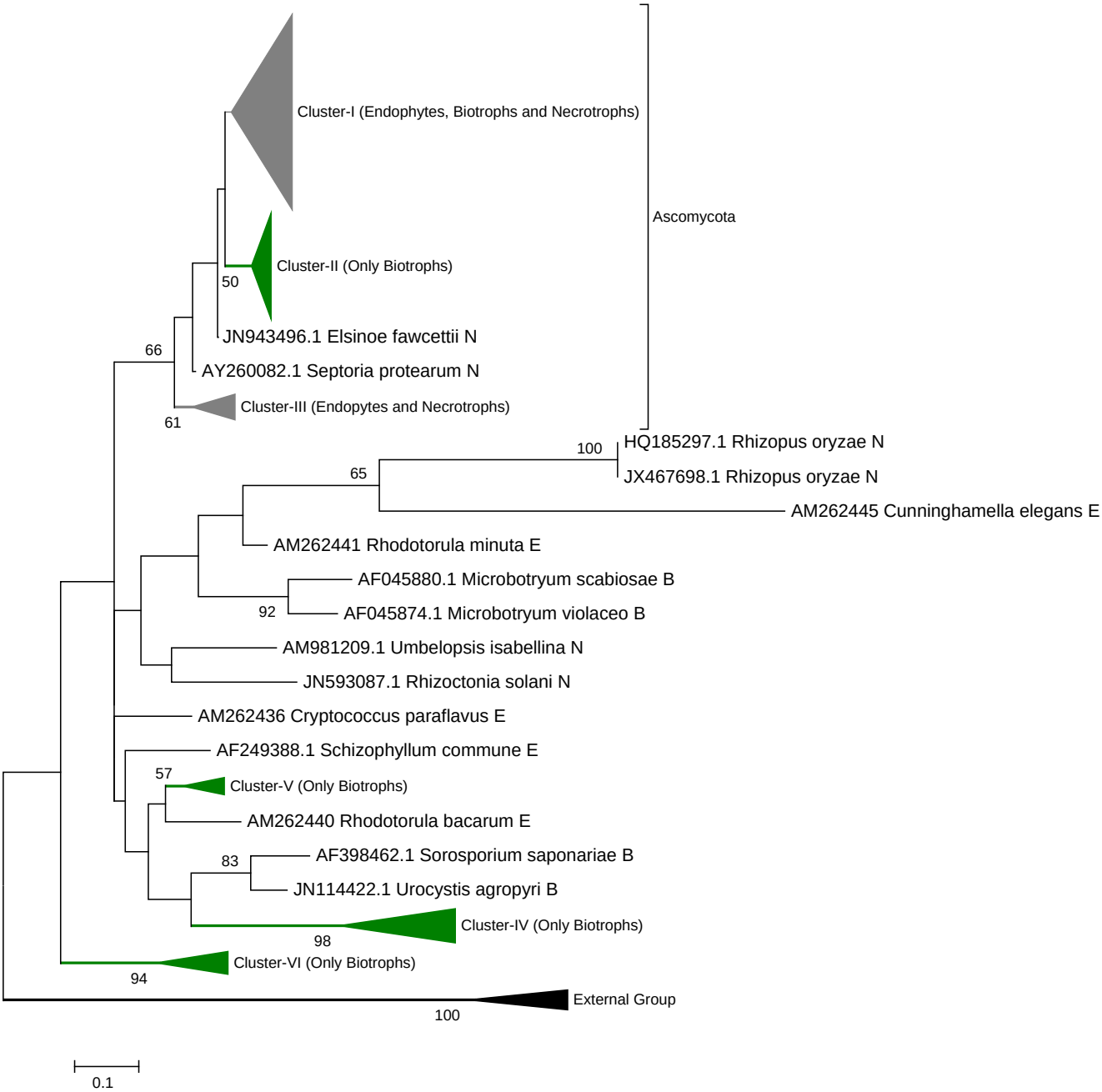

Fig. 1 Maximum-Likelihood tree (*initial-t*) of 5.8S rRNA from diverse necrotrophic (N), endophytic (E), and biotrophic (B) fungi. Clusters from I to VII are indicated. Bootstrap values ≥50 % are shown. Names comply with the original sequence depositions

among the *Clavicipitaceae* in cluster I, and we found no case of a change from a biotrophic to one of the other lifestyles. This suggests that once biotrophy has evolved, there is no easy return to the other lifestyles, whereas fungal lineages frequently switched between an endophytic and a necrotrophic lifestyle.

At first glance, this result appears counterintuitive. Among the three fungal lifestyles considered here, endophytes have the lowest negative effects on the health of their host whereas necrotrophic pathogens kill the cells of their host immediately and, hence, have the most dramatic effects on the performance of their host (Burdon 1993; Jarosz and Davelos 1995; Gilbert 2002; García-Guzmán and Morales 2007; Oliver and Solomon 2010; Talbot 2010; Horbach et al. 2011). Indeed, endophytes can positively affect host resistance to disease and abiotic stress, or increase the vigour of the infected plants (Clay 1991; Saikkonen et al. 1998; Frölich et al. 2000; Sieber 2007; Aly et al. 2011; Aschehoug et al. 2012), although evidence of their mutualistic effects under field conditions remains inconclusive (Faeth and Fagan 2002; Schulz and

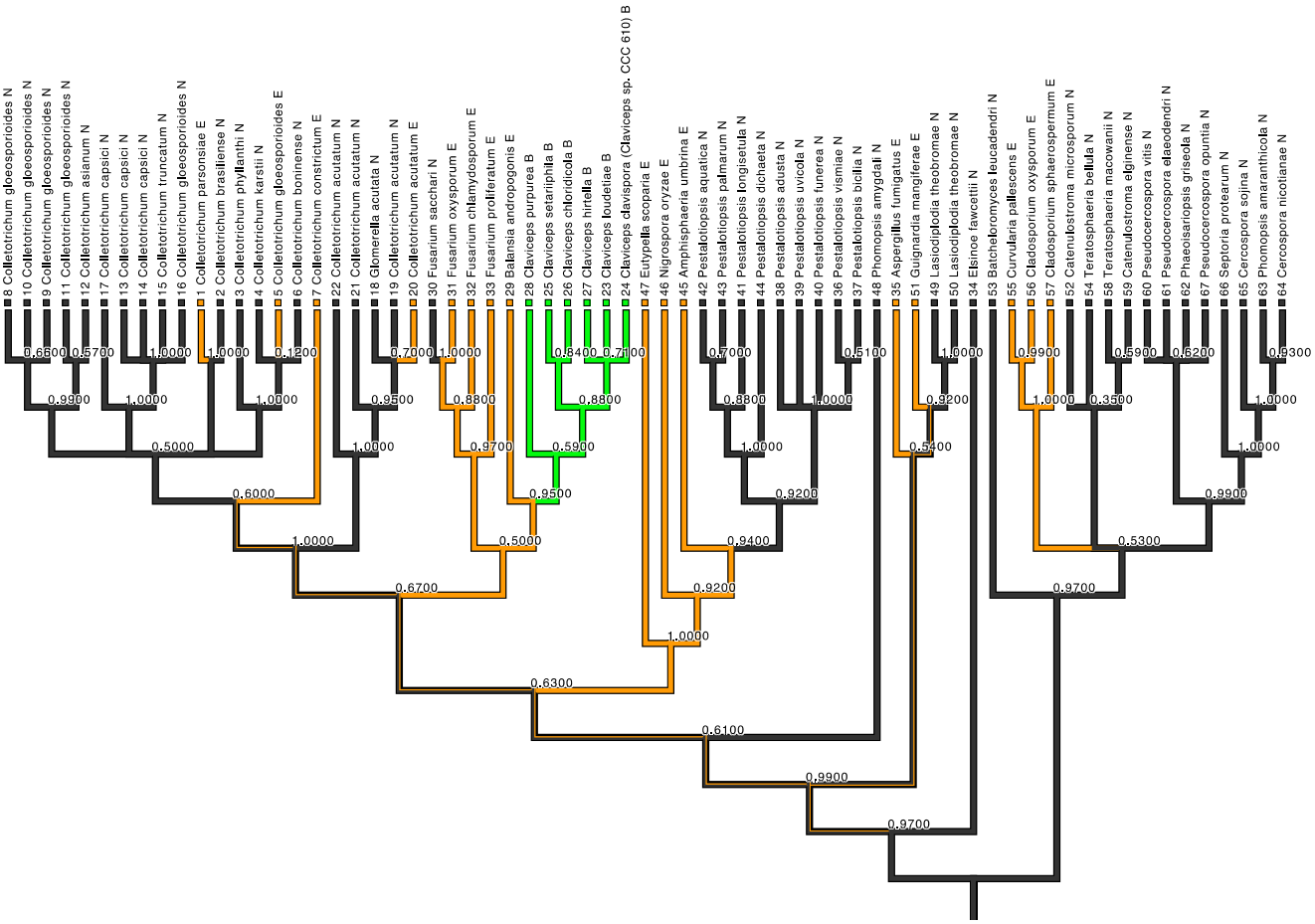

Fig. 2 Cluster I comprises endophytic and necrotrophic species, as well as biotrophic species from the Hypocreales. The ancestral lifestyles: necrotrophy (*N*, black), endophyte (*E*, orange) and biotrophy (*B*, green) were reconstructed by maximum parsimony. The phylogenetic tree was reconstructed by Maximum-Likelihood from 5.8S rRNA molecules. Bootstrap values are shown in the nodes. Nodes with bootstrap values<0.5 (i.e., <50 %) were collapsed. Names comply with the original sequence depositions; updated names are included in *brackets*

Boyle 2005; Sieber 2007; Hyde and Soytong 2008; Partida-Martínez and Heil 2011; Peršoh 2013).

Why are fungi able to switch easily between two lifestyles that appear to be the most different at the phenotypic level, and are these results likely to represent the general case? A shortcoming of our study is the relatively low number of cases that could be included for every lifestyle. Unfortunately, historical differences among research disciplines made it difficult to find studies of fungi for which all the required information was available. For example, studies on endophytes and those on biotrophic pathogens traditionally have used different marker genes (the 5.8S gene together with the more variable ITS1 and ITS2 regions vs. the 18S gene, see Guo et al. 2000, 2003; Schulz and Boyle 2005). Thus, more studies using balanced datasets are required. Nonetheless, we were able to include 21 endophytes, 47 necrotrophs and 68 biotrophic pathogens in our phylogenetic analysis. The major characteristics of the resulting phylogenetic tree coincide with other studies that in part have used different molecular markers. For example, Zhuang and Liu (2012) used the large subunit of ribosomal DNA (nucSSU and nucLSU rDNA) from 12 classes of Ascomycota and also placed *Aspergillus* (Eurotiomycetes) close to the *Dothideomycetes*, as shown by our cluster I. Similarly, cluster III is congruent with the phylogeny of *Pleosporales* presented by Zhang et al. (2009; 2012). We are therefore confident that the major structure of the presented tree is stable and that the major message—endophytic and necrotrophic lifestyles cluster together and are subject to frequent evolutionary and even ecological changes—represents a reliable discovery.

Endophytic and necrotrophic lifestyles were present in most of the major clades that were formed in the six phylogenies of the 5.8S rRNA genes. Thus, fungal lineages can switch between these two lifestyles at the very short evolutionary timescale. Indeed, our survey of the literature revealed that both lifestyles have been reported for six species: *Alternaria alternata* (Huang et al. 2009; Yu et al. 2011; Park et al. 2012), *Colletotrichum acutatum* (Than et al. 2008;

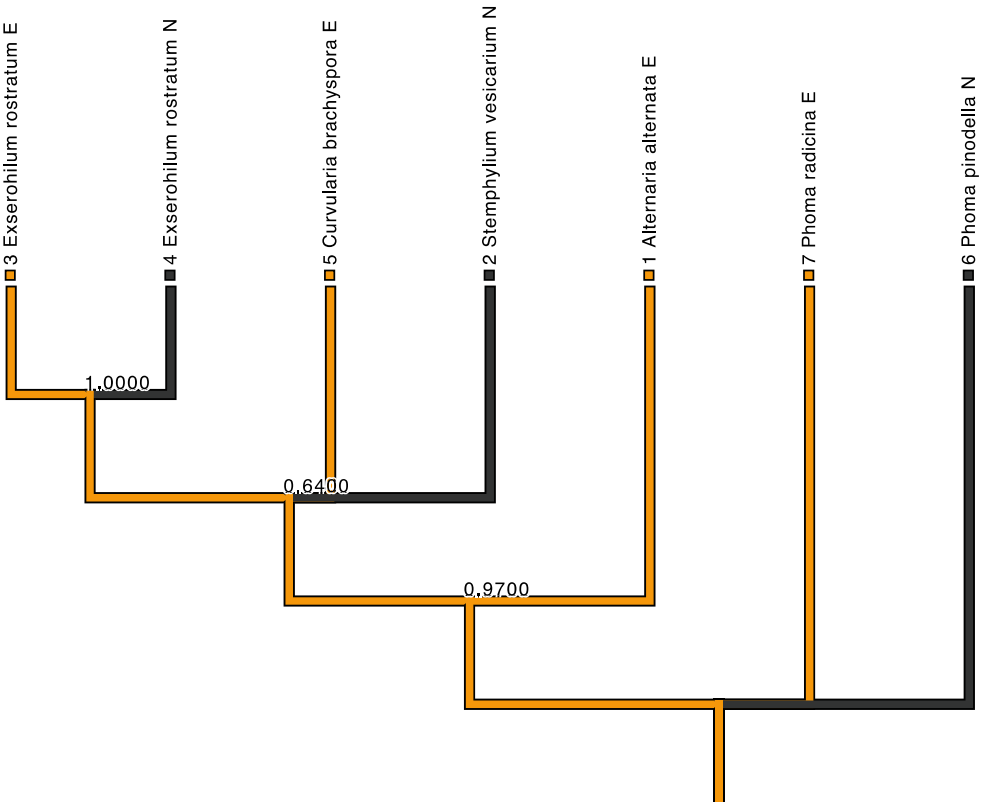

Fig. 3 Cluster III contained necrotrophs and endophytes from the Pleosporaceae and Didymellacaea. The ancestral lifestyles: necrotrophy (*N*, black) and endophyte (*E*, orange) were reconstructed by maximum parsimony. The phylogenetic tree was reconstructed by Maximum-Likelihood from 5.8S rRNA molecules. Bootstrap values are shown in the nodes. Nodes with bootstrap values<0.5 (i.e., <50 %) were collapsed. Names comply with the original sequence depositions

Glenn and Bodri 2012), *Colletotrichum gloeosporioides* (Promputtha et al. 2007; Than et al. 2008; Yan et al. 2011; Choi et al. 2012), *Colletotrichum musae* (Photita et al. 2005; Promputtha et al. 2007), *Exserohilum rostratum* (Lin et al. 2011; Loro et al. 2012) and *Glomerella cingulata* (Sette et al. 2006; Kwon et al. 2012). Recent phylogenetic revisions indicate that some of these species might represent groups of cryptic species (see, e.g., Silva et al. 2012 and Weir et al. 2012 for *Colletotrichum gloeosporioides* and Damm et al. 2012 for *Colletotrichum acutatum*). Thus, the reported lifestyles in fact might have been performed by different (although closely related) species. However, in other studies, both lifestyles were reported for the same strain. For example, the same strain of *Leptosphaeria maculans* was isolated from completely healthy *Arabidopsis thaliana* plants in their natural environment but behaved as necrotrophic pathogen in a more stressful (laboratory) environment (Junker et al. 2012). Infecting *Arabidopsis* roots with endophytes that had been isolated from healthy plants in the field was even more detrimental: colonisation by *Stagonospora* sp. 9722, *Geniculosporium* sp. 9725, *Phoma* sp. 9728 and *Chalara* sp. 9731 resulted in strong disease of stressed plants (Junker et al. 2012). Although endophytes can protect their host against abiotic stress, stressful conditions can clearly shift the interaction along the 'fine line' (Junker et al. 2012) between mutualism and parasitism. For example, high light intensity caused *Diplodia mutila*, which represents a common symptomless endophyte of the tropical palm, *Iriartea deltoidea*, to switch to pathogenicity and cause necrosis (Álvarez-Loayza et al. 2011). Changed abiotic conditions or single mutants can even cause endophytes of the family *Clavicipitaceae* to switch to proliferative pathogenic growth (Eaton et al. 2011). Similarly, the common endophyte, *Lasiodiplodia theobromae*, was identified as a significant source of (necrotic) disease symptoms in baobabs (*Adansonia gregorii*) growing at certain sites (Sakalidis et al. 2011), *Deightoniella torulosa,* an endophyte in *Musa acuminata*, can cause leaf spots in the same host species (Photita et al. 2004), and climatic conditions determined whether *Discula quercina* behaved as a pathogen or an endophyte in Oak (Moricca and Ragazzi 2011). In these reports the same strain behaved as endophyte and pathogen and in all cases the pathogenic behaviour was described as necrotrophic (see also Andrew et al. 2012).

Table 1 Transitions in lifestyles inferred from Clusters I and III

	Cluster	
Type of transition	I	III
From endophyte to necrotroph	2	2
From necrotroph to endophyte	4	0
From endophyte to biotroph	1	0
Not determined (endophyte versus necrotroph)	8	0

Context dependency is a crucial parameter in the outcome of the endophyte-host interaction (reviewed in Schulz and Boyle 2005; Partida-Martínez and Heil 2011; Junker et al. 2012). Many endophytes have been regarded as dormant pathogens (Mostert et al. 2000; Photita et al. 2004; Hyde and Soytong 2008; Hyde et al. 2009; Kleczewski et al. 2012) and mutations at a single genetic locus can change a fungus from a pathogenic to a non-pathogenic lifestyle, with no effect on host specificity (Tanaka et al. 2006; Eaton et al. 2011). According to Heller and Tudzynski (2011), most necrotrophic pathogens asymptomatically colonize the host tissues during the earliest phases of the infection. For example, *Botrytis cinerea*, which is regarded as a model necrotrophic pathogen, survives as an endophyte prior to causing host-cell death (Sowley et al. 2010), and the same type of 'stealth pathogenicity' has also been described for *Mycosphaerella graminicola* and congeneric species (Goodwin et al. 2011). Thus, the separation between the endophytic and necrotrophic pathogen life histories is much more diffuse at ecological and evolutionary timescales than would be assumed when observing the fully developed phenotypic symptoms.

Several phylogenetic studies have found endophytism to be an evolutionarily unstable strategy and have indicated multiple switches from endophytism to saprophytism (Promputtha et al. 2007; Wang et al. 2009; Purahong and Hyde 2011) or to a life as lichenized fungi (Arnold et al. 2009). Multiple studies have reported shifts from an endophytic to a pathogenic lifestyle (Hyde and Soytong 2008; Álvarez-Loayza et al. 2011; Eaton et al. 2011) or vice versa (Carroll 1988; Freeman and Rodriguez 1993), and in most cases the authors described the pathogenic behaviour explicitly as being necrotrophic (Mostert et al. 2000; Photita et al. 2004; Slippers and Wingfield 2007; Sowley et al. 2010; Álvarez-Loayza et al. 2011). Therefore, the observation that fungi can easily change between an endophytic and a necrotrophic lifestyle is perhaps not as surprising as it appears to be at first glance.

Which traits, then make the biotrophic lifestyle so special that it is hardly ever lost once it has been gained? In our study, biotrophic pathogens clustered together in four major clades and biotrophy did not appear to have been lost secondarily from any of these clades. The only apparent exception to this rule is the *Clavicipitaceae*, the asexual states of which are usually reported as endophytes, whereas teleomorphs (e.g., *Claviceps*) behave as biotrophic pathogens (Faeth and Sullivan 2003). With this exception, being a biotrophic pathogen appears to represent an evolutionarily stable and derived strategy.

In an attempt to find causal explanations for this pattern, we searched for information on major traits that are characteristically related to the lifestyles that we have investigated (Table 2). All plant—infecting fungi possess a range of traits that are directly related to their mode of interaction with their host plants. Biotrophism in particular requires multiple sophisticated morphological, biochemical and genetic mechanisms to ensure the controlled and dynamic exchange of signalling molecules and nutrients with living host plant cells. Biotrophic pathogens characteristically depend on the formation of haustoria (Mendgen and Hahn 2002; O'Connell and Panstruga 2006). The haustorium allows the pathogen to obtain nutrients from the host cell and represents a key determinant of biotrophy (Horbach et al. 2011; Kemen and Jones 2012). To the best of our knowledge haustoria are notoriously lacking in necrotrophs (Horbach et al. 2011; Mengiste 2012), although they have been reported from some endophytes such as *Discula umbrinella* (Viret and Petrini 1994) and *Rhabdocline parkeri*, an endophyte of Douglas fir (*Pseudotsuga menziesii*) that produces haustoria from the intercellular hyphae at the onset of needle senescence (Stone 1988; Kriel et al. 2000). However, in the case of *D. umbrinella* and *R. parkeri*, haustorium formation usually results in the early death of the infected cells (Stone 1988; Viret and Petrini 1994), which allows distinguishing non-clavicipitaceous endophytic fungi from biotrophic pathogens. Furthermore, both *D. umbrinella* and *R. parkeri* lack the haustorial neckband characteristic of most haustorial fungi (Manners and Gay 1983). Neckbands are structures that increase the efficiency of haustorial nutrient uptake (Mendgen and Hahn 2002). In summary, most endophytes and seemingly all necrotrophs lack haustoria, whereas haustoria represent an integral aspect of the evolution of biotrophs.

Other important factors could be the secretion of hydrolytic enzymes, the specificity of the interaction and the systemic versus the local mode of infection (Table 2). Although it is still not understood how biotrophs avoid killing the cells of the host, several authors have suggested that secretory activity is required by both host and pathogen to form the interface layers that contribute to the preservation of compatibility and the weak or brief defence response of the host (Hahn and Mendgen 2001; Mendgen and Hahn 2002). By contrast, the necrotrophic mode of nutrition is linked to pro-death virulence strategies, which in turn influence the nature of effective host immune responses that have evolved to counter infection (Mengiste 2012). Necrotrophic pathogens produce a variety of phytotoxins and cell-wall degrading enzymes that induce cell necrosis and cause leakage of nutrients (Mengiste 2012). Interestingly, most endophytes also produce a variety of exoenzymes after germination that soften the cuticle and the wall of epidermal cells to ease penetration of the infection hyphae or, if an appressorium is formed, to facilitate breaching the plant cuticle by mechanical force (Sieber et al. 1991; Petrini et al. 1992; Schulz et al. 2002). The hyphae produced by foliar endophytes are thin and may develop coils to better absorb available nutrients (Schulz and Boyle 2005). After infection, a latent state is assumed, and induced defences are either not activated or the hypersensitive response kills only single

Table 2 Major characteristics of life histories of plant-infecting fungi

Characteristic	Endophyte		Necrotroph	Biotroph	References
	Clavicipitaceous	Non-clavicipitaceous			
Host range	Mostly specialists (grasses)	Many generalists	Many generalists	Mostly specialists	Sieber 2007; Andrew et al. 2012; Kemen and Jones 2012
Haustorium	Absent	Absent	Absent	Present	Horbach et al. 2011; Kemen and Jones 2012
Secretion of lytic enzymes	[a]Limited	[a]Limited	Yes	[a]Limited	Petrini et al. 1992; Schulz and Boyle 2005; Schardl et al. 2009; Laluk and Mengiste 2010; Kemen and Jones 2012
Toxin production	Yes	Yes	Yes	No	Laluk and Mengiste 2010; Mengiste 2012
Host hypersensitive reaction	Suppressed	Suppressed	Promoted	Suppressed	Govrin and Levine 2000; Schulz and Boyle 2005; Jones and Dangl 2006; Laluk and Mengiste 2010; Mengiste 2012
General growth	Systemic	Local and systemic	Local	Systemic and local	Burdon 1993; Redman et al. 2002; Ernst et al. 2003; Schulz and Boyle 2005; Sieber 2007; Arnold et al. 2009; Yuan et al. 2010
Hyphal growth	Intercellular	Intercellular	Intracellular	Intracellular	Johnston et al. 2006; Sieber 2007; Arnold et al. 2009; Horbach et al. 2011; Tanaka et al. 2012
Sexuality	Asexual	Asexual and sexual	Sexual	Sexual	Faeth and Sullivan 2003; Bärlocher 2009
Effects on host reproduction	Stopped or reduced	Unaffected	Reduced	Stopped or reduced	Burdon 1993; Faeth and Sullivan 2003

[a] Does not result in tissue maceration

host cells, as demonstrated for *Rhabdocline parkeri* (Stone 1987). Endophytes have many of the same virulence factors that are present in pathogens, and produce the exoenzymes necessary to infect and colonize the host. For example, most endophytes can produce mycotoxins (Schulz et al. 2002; Wang and Dai 2011) and the hosts can react with the same defence responses to the presence of an endophyte or a pathogen (Schulz and Boyle 2005). Consequently, some authors (Arnold 2007, 2008; Schulz and Boyle 2006) have suggested that asymptomatic colonization is a balance of antagonisms between host and endophyte, but if this balance is destroyed, the endophyte becomes pathogenic. Indeed, a single mutation was enough to cause the filamentous fungal ascomycete *Colletotrichum magna* (the causal agent of anthracnose in Cucurbitaceae) to shift from a pathogenic to a symptomless, endophytic lifestyle (Freeman and Rodriguez 1993).

Finally, biotrophic pathogens commonly have lost certain biosynthetic pathways (Kemen and Jones 2012) and consequently lack the ability to synthesize important metabolites, which results in a high dependency on their specific host (Kemen and Jones 2012). For example, Spanu et al. (2010) analysed the genome of the biotrophic pathogen *Blumeria graminis* f. sp. *hordei* and showed massive retrotransposon proliferation, genome-size expansion and gene losses. The missing genes encode enzymes of primary and secondary metabolism, carbohydrate-active enzymes and transporters, probably reflecting their redundancy in an exclusively biotrophic lifestyle. Thus, pending future experimental proof, we assume that most biotrophic pathogens have acquired several specific infection mechanisms (the haustorium representing the most unique feature) and have lost essential genes. Both general characteristics make it difficult—or perhaps impossible—for them to return to alternative life histories.

The lifestyles of plant-infecting fungi (Table 2), form a continuum that ranges from necrotrophy on one extreme, to a life as a biotrophic pathogen, and finally to mutualism or commensalism as exhibited by symptomless endophytes. Necrotrophs are mostly non-systemic pathogens, which may be host specialists or generalists (Burdon 1993; Gilbert and Webb 2007; Hersh et al. 2012), and they usually have short generation times and a high fecundity per generation: selection for greater fitness of the pathogen might favour increasing aggressiveness (Jarosz and Davelos 1995; Cobb et al. 2010; Mordecai 2011). By contrast, biotrophic pathogens are usually systemic, have longer generation times and survive from year to year within infected plants. For biotrophic pathogens, increasing aggressiveness is unlikely to be favoured because this could kill the host, and hence the pathogen, before reproduction. Endophytes combine selective elements from necrotrophic and biotrophic pathogens. They may be systemic (e.g., grass

endophytes) or local (e.g., most foliar non-clavicipitaceous endophytes), and range from host specialists to generalists; however, the lack of reproduction within the host tissues could be considered as a unique trait that differentiates them from the pathogenic fungi.

Although our study is based on a limited number of sequences of plant-infecting fungi with known lifestyle as realized in the host, it reveals a surprising pattern that currently lacks a mechanistic explanation. Fungi can easily switch at the evolutionary or ecological timescale between the symptomless (and sometimes beneficial) endophytic growth in life host tissue and the life as a necrotrophic pathogen that kills its host, whereas they hardly ever switch from inhabiting life host tissue as a biotrophic pathogen to one of the other two lifestyles. Including the sequences of more strains with reported lifestyles would be required to corroborate the stability of this pattern but will crucially depend on the publication of the sequences of the same, standardised genetic markers for all fungi for which the lifestyle in a given host is being reported. We are optimistic that the advances in sequencing methods, and an increasing awareness among scientists who work with fungi to include detailed information on the host plants and the realized effects the sequenced fungi in a given host, should help to enhance our understanding of the evolution of plant—fungal interactions.

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