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Does the Latent Period of Leaf Fungal Pathogens Reflect Their Trophic Type? A Meta-Analysis of Biotrophs, Hemibiotrophs, and Necrotrophs

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ABSTRACT

We performed a meta-analysis to search for a relation between the trophic type and latent period of fungal pathogens. The pathogen incubation period and the level of resistance of the hosts were also investigated. This ecological knowledge would help us to more efficiently regulate crop epidemics for different types of pathogens. We gathered latent period data from 103 studies dealing with 51 fungal pathogens of the three major trophic types (25 biotrophs, 15 hemibiotrophs, and 11 necrotrophs), representing 2,542 mean latent periods. We show that these three trophic types display significantly different latent periods. Necrotrophs exhibited the shortest latent periods (<100 degree-days [DD]), biotrophs had intermediate ones (between 100 and 200 DD), and hemibiotrophs had the longest latent periods (>200 DD). We argue that this relation between trophic type and latent period points to two opposing host exploitation strategies: necrotrophs mount a rapid destructive attack on the host tissue, whereas biotrophs and hemibiotrophs avoid or delay the damaging phase. We query the definition of hemibiotrophic pathogens and discuss whether the length of the latent period is determined by the physiological limits inherent to each trophic type or by the adaptation of pathogens of different trophic types to the contrasting conditions experienced in their interaction with the host.

Keywords: fungal pathogens, host resistance, incubation period, latent period, leaf crop pathogens, meta-analysis, pathogen trophic type

Leaf fungal pathogens can be differentiated by the nature of their trophic interaction with their host. Four types of trophic interactions are commonly distinguished: necrotrophy, biotrophy, hemibiotrophy, and saprotrophy (Divon and Fluhr 2007). Necrotrophic pathogens kill the host tissue before being able to exploit it as a resource. They only grow and sporulate on dead tissue (Horbach et al. 2011). By contrast, biotrophic pathogens require living host tissue to survive (Horbach et al. 2011; Mendgen and Hahn 2002). They feed through specialized trophic structures known as haustoria, which draw nutrients directly from living cell cytoplasm through a specialized extracellular matrix (Garnica et al. 2014). Hemibiotrophic pathogens are initially biotrophic (usually during a somatic growth stage) and then necrotrophic in a later stage (usually at the spore production stage), undergoing major transcriptomic reprogramming (Lee and Rose 2010; O'Connell et al. 2012; Palma-Guerrero et al. 2016). This group of pathogens can be perceived as less homogeneous than the other trophic types, because only some of them produce haustoria (Behr et al. 2010; Perfect and Green 2001), which differ structurally from those of biotrophs (Kemen et al. 2015). Finally, saprotrophic fungi feed on dead host tissue without killing it themselves. As such, they may not be regarded as true pathogens but rather as detritivores or scavengers. Because of our interest in true pathogens, we will hereafter focus on necrotrophic, biotrophic, and hemibiotrophic pathogens.

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Although the biological descriptions of fungal crop pathogens generally refer to their trophic type, little effort has been made to map general ecological traits to pathogen trophic types (Calhim et al. 2018). However, such ecological knowledge is useful from both a fundamental and applied point of view: increasing our basic understanding of fungi and being able to predict their population and epidemiological dynamics will help us to control them more efficiently. For instance, studying the relationship between a pathogen's trophic type and host resistance could prove useful. The aim of our article is therefore to use data from the scientific literature to explore the relationship between the latent period and trophic types of fungal pathogens attacking plant leaves. We further investigate the interaction between latent period, trophic type, and host resistance.

Our focus here is on the latent period (i.e., the period between infection and the onset of sporulation). The latent period is a well-studied epidemiological trait of fungal crop pathogens. The latent period is of key importance in their biology, as it separates a period of somatic growth from a period of spore production. It has a direct effect on the within-seasonal dynamics of the pathogen by determining the length of the infection cycle. Most fungal crop pathogens are polycyclic: they experience up to 30 infection cycles per year (Agrios 2005), thereby multiplying the quantity of inoculum released into the environment. An infection cycle starts with the deposition of pathogen spores on the host tissue. Mycelium emerges from germinating spores, invades host tissues, and eventually produces subepidermal reproductive structures. The maturation of these structures releases new spores, and then the infection cycle repeats itself as long as there is susceptible tissue to colonize. The latent period can thus be regarded as the minimum duration of an infection cycle. The latent period is an important aggressiveness trait in plant pathology: pathogen strains with shorter latent periods have been shown to cause more disease (Frenkel et al. 2010; Lehman and Shaner 1996; Milus et al. 2006; Pringle and Taylor 2002). This is partly attributable to the increased speed of canopy colonization (Beresford and Royle 1988; Lovell et al. 2004; Zearfoss et al. 2011). The latent period of the pathogen

also changes with the level of quantitative resistance of the host. Many studies have shown that a given fungal strain has an extended latent period when infecting a partially resistant cultivar compared with a susceptible one (e.g., see Czembor et al. 2003; Habtu and Zadoks 1995; Lehman et al. 2005). However, the relations between host resistance, pathogen trophic type, and pathogen latent period have not yet been investigated globally among pathosystems.

The latent period often consists of an asymptomatic phase, referred to as the incubation period, and a second phase during which presporulation symptoms (e.g., chlorosis; Robert et al. 2004; Teng and Close 1978) appear. The second phase may be absent if the first symptoms coincide with the appearance of reproductive structures, in which case the latent period is equal to the incubation period. In addition to the latent period, the incubation period can be considered to be an important aggressiveness trait (Setti et al. 2008, 2009; Suassuna et al. 2004). The link between incubation and latent periods, however, has rarely been studied. Here, we investigate the relations between these two periods of the infection cycle.

In this work, our main question is whether there exist any differences in the duration of the latent period among the three trophic types of pathogens. Knowledge about pathogen life history traits such as incubation and latent periods provides insights into the ecoevolutionary history of the different trophic strategies used by plant pathogens. We further explore the interactions between incubation period, latent period, host resistance, and trophic types. Knowledge about these interactions sheds light on how resistance is expressed in different ecological groups of pathogens, offering the potential to improve crop protection. To address these questions, we performed a meta-analysis of 103 published articles with data on 51 pathogen species (25 biotrophs, 15 hemibiotrophs, and 11 necrotrophs).

MATERIALS AND METHODS

Data. *Studies included in the meta-analysis.* A summary of the study selection process is given in Figure 1. To gather studies reporting quantitative data on the latent period, we searched the Web of Science and Google Scholar databases using the query 'fung* AND disease AND laten*'. This resulted in 538 different scientific articles (on 1 June 2016). Only articles dealing with fungi organisms and containing exploitable quantitative data relating to the latent periods were selected (286 articles). We excluded all articles on pathogens infecting organs other than leaves. We also excluded oomycete pathogens, because the search found only 10 articles dealing with six species, five of which were biotrophic and none necrotrophic. We considered that there were insufficient data to treat these pathogens as a separate category, which was necessary given their specific biology. We also excluded the few studies found on sexual spores, since the latent period of sexual inoculum may differ from that of asexual inoculum (Eriksen et al. 2001; Morais et al. 2015; but see also Karolewski et al. 2002). This resulted in a total of 144 articles reporting the quantitative latent period data of pathogenic fungi on plant leaves.

We further selected studies that used similar definitions of the latent period, as the time between infection and initial sporulation. Two main definitions of the latent period can be found in the literature. First, following Parlevliet (1979), some authors define the latent period as the time interval from inoculation to initial sporulation. From an epidemiological point of view, this corresponds to a latent period defined as the minimal possible duration of an infection cycle. Second, following Johnson (1980) and Shaw (1986), the latent period is also defined as the time required for 50% of the lesions to begin sporulation. From an epidemiological point of view, this corresponds to a latent period defined as the median duration of an infection cycle. Based on this dichotomy, we assigned the studies with slightly different definitions to one or the other group. For instance, latent periods identified as the time to appearance of 10% of the total number of sporulating structures (Flier and Turkensteen 1999) were deemed to be "minimal." Thus, only the minimal latent periods were considered for statistical analyses (third step of the study selection, see Fig. 1). Overall, 37 studies were excluded because they used "median" definitions of the latent period.

Because the latent period is known to strongly depend on temperature, we also excluded four studies without any information on temperature. We finally obtained 103 usable articles, dealing with 51 fungal pathogen species on 59 different host plants from 18 angiosperm families (encompassing both cultivated and wild pathosystems), corresponding to 2,542 latent periods. We found latent period data on 25 biotrophic (838 data), 15 hemibiotrophic (1,111 data), and 11 necrotrophic (593 data) pathogen species.

Response variables: latent and incubation periods. The latent period is the main variable studied in this article. Following Parlevliet (1979), the latent period is defined as the time interval from inoculation to initial sporulation.

Most studies report latent period measurements as the mean latent periods given for each tested experimental condition or for each cross-condition in a randomized (complete or incomplete) block design. These mean latent periods are calculated by averaging periods observed on several individual plants. The number of plants varies across studies and treatments. The 2,542 latent periods included in our analysis correspond to such mean values. For each of them, we recorded the size of the raw data sample used to generate them. Data in our analysis thus correspond to the mean values of latent periods, along with the number of measurements used to generate them.

The latent period is known to depend on the temperature of the pathogen's environment (Johnson 1980; Mersha et al. 2014; Shaw 1986). Several authors report a linear decrease in the latent period (in days) in line with the increasing temperature for several pathogen species (Figueroa et al. 1995a; Kolnaar and Van Den Bosch 2001; Lovell et al. 2004). However, studies on a wide range of temperatures have demonstrated that the relationship between latent period and temperature resembles more of a power function, with a possible linear approximation between 5 and 25°C (Bernard et al. 2013; Pedersen and Morrall 1994; Shearer and Zadoks 1972; Sosa-Alvarez et al. 1995). Several articles have used this linear approximation to model pathogen development (Garin et al. 2014; Précigout et al. 2017; Robert et al. 2008). In these models, the biology of the organisms is expressed in thermal time (Eriksen et al. 2001; Lovell et al. 2004; Zearfoss et al. 2011). In short, if a biological process requires 10 days to complete at 10°C, it will require only 5 days at 20°C; that is, it requires 100 degree-days (DD) to complete. This approximation is only valid in the temperature range where the relationship between latent period and temperature is approximately linear (i.e., often between 5 and 25°C). This temperature range is usually considered favorable to pathogen development. Hence, to make our latent period data more comparable, they are expressed in thermal time (keeping only those from the 5 to 25°C temperature range) in order to filter out the influence of variable temperatures in the different experimental studies. Data already expressed in DD in the original articles were used directly. Otherwise, thermal-time latent periods were calculated using the temperature indications available in these articles. Our data set is thus composed of "minimal" latent periods expressed in degree-days.

Some studies considered in this meta-analysis report not only latent periods but also incubation periods. Incubation is defined as the time from inoculation to the appearance of the first symptoms (possibly nonsporulating symptoms such as chloroses). It is consequently shorter than, or at most equal to, the latent period. In our work, similarly to the latent period data, the incubation period data are expressed as mean values associated with the number of measurements used to generate them. They are reported in degree-days. Overall, 29 studies reporting incubation period data on six biotrophic (84 data), seven hemibiotrophic (343 data), and five necrotrophic (350 data) pathogen species are included in the analysis. A list of selected studies and the associated pathogen(s) can be found in Table 1.

Explanatory variables. *Trophic type.* We sought to investigate whether there is a relation between the latent period of a pathogen and its trophic type, hence the due place of trophic type among explanatory variables. The first step of our analysis was to classify the pathogens according to their trophic type. Some species are universally recognized as members of a given trophic group. This is true for the biotrophs and most necrotrophs included in our analysis. However, the status of other species was more problematic, especially among those termed hemibiotrophic in the literature. For instance, it is not clear whether the early asymptotic phases of *Zymoseptoria tritici* (or *Parastagonospora nodorum*) truly correspond to a "biotrophical feeding" stage (Sánchez-Vallet et al. 2015). For this reason, in this article, hemibiotrophs are considered to be pathogens displaying a two-stage infection cycle composed of an asymptomatic colonization of the living host tissues followed by a necrotrophic stage with asexual reproduction (regardless of whether a true biotrophic interaction was demonstrated during the asymptomatic period). Supplementary Table S1 lists the 51 pathogen species included in this study as well as the trophic type attributed to each of them. It also provides the literature references that guided our choices where relevant. These studies demonstrate a biphasic infection cycle of hemibiotrophic pathogens as defined above. Trophic type thus

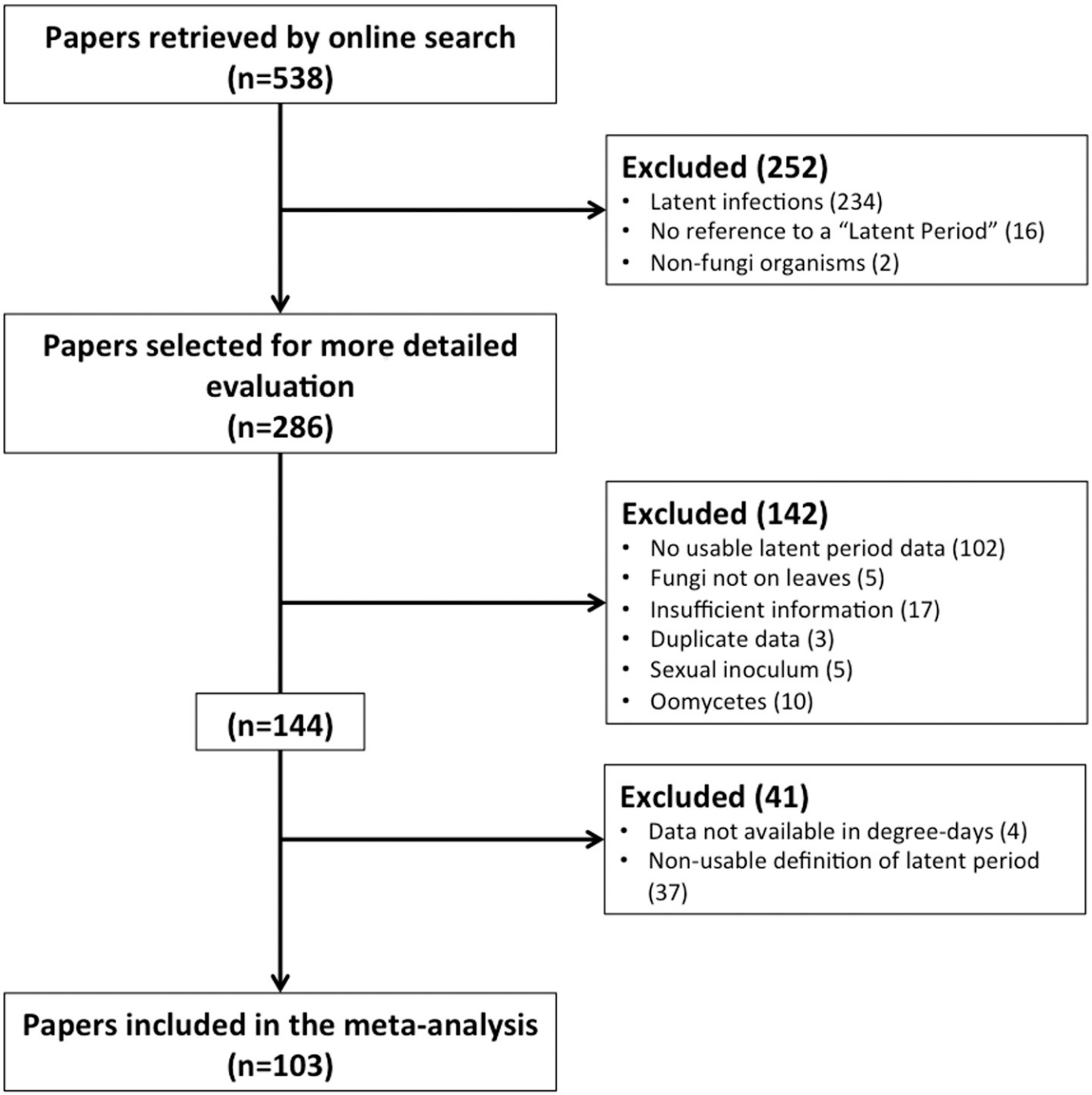

Fig. 1. Study selection flow diagram. From the initial 538 different articles found on Web of Science and Google Scholar using the query 'fung* AND disease AND laten*', 250 were immediately excluded because they did not mention any latent period (234 dealt with latent infections; 16 did not refer to any sort of latency). Two articles were also excluded because they did not focus on fungi organisms. Of the 286 remaining articles, we excluded those without usable latent period data (n = 102) and those not dealing with the latent period of asexual reproduction cycles of fungal pathogens infecting crop leaves (n = 30). We also excluded studies on oomycetes (n = 10). Among the 144 remaining studies, those with data not expressed in degree-days (n = 4) and with data corresponding to "median" latent periods (n = 37) were excluded.

corresponds to a categorical variable with the following levels: biotrophic (B), hemibiotrophic (H), and necrotrophic (N).

Resistance. In this meta-analysis, 52 studies deal with quantitative partial resistance, with the resistance level of the host plant thus being recorded as an explanatory variable. Depending on the studies, it is taken as a binary (susceptible versus partially resistant), categorical (highly susceptible, susceptible, moderately resistant, or resistant), or an ordinal variable (resistance level between 1 and 10). Since most studies on resistance presented three cultivar categories, we opted for a categorical variable with three levels: susceptible (S), moderately resistant (M), and resistant (R). Wild plants and cultivars with no resistance rating were considered

TABLE 1. List of the studies included in this meta-analysis[a]

Study identifier	Article	Pathogen(s)	Trophic type[b]	Host(s)
ARM04	Armour et al. (2004)	*Zymoseptoria tritici*	H	*Triticum aestivum*
ARS04	Arseniuk et al. (2004)	*Parastagonospora nodorum*	H	*Triticum aestivum*
ASA16	Asalf et al. (2016)	*Podosphaera aphanis*	B	*Fragaria* × *ananassa*
AUS12	Austin and Wilcox (2012)	*Erysiphe necator*	B	*Vitis vinifera*
BAS75	Bashi and Rotem (1975)	*Stemphylium botryosum*	N	*Solanum lycopersicum*
BOI95	Boiteux et al. (1995)	*Podosphaera fuliginea*	B	*Cucumis melo*
BOU03	Bouhassan et al. (2003)	*Botrytis fabae*	N	*Vicia faba*
BOU04	Bouhassan et al. (2004)	*Botrytis fabae*	N	*Vicia faba*
BOU07	Bouhassan et al. (2007)	*Botrytis fabae*	N	*Vicia faba*
BRO97	Broers (1997)	*Puccinia striiformis*	B	*Triticum aestivum*
BRO04	Browne et al. (2005)	*Monographella nivalis*	N	*Triticum aestivum*
BRO06	Browne et al. (2006)	*Monographella nivalis*	N	*Triticum aestivum*
BRC04	Browne and Cooke (2004)	*Monographella nivalis*	N	*Triticum aestivum*
CAR94	Carson and Van Dyke (1994)	*Exserohilum turcicum*	H	*Zea mays*
CAS93	Casela et al. (1993)	*Colletotrichum graminicola*	H	*Sorghum bicolor*
CHH12	Chhikara et al. (2012)	*Alternaria brassicae*	N	*Brassica juncea*
CHO00	Chongo and Bernier (2000)	*Colletotrichum truncatum*	H	*Lens culinaris*
CHO14	Choudhury et al. (2014)	*Erysiphe necator*	B	*Vitis vinifera*
CRO92	Cromey (1992)	*Puccinia striiformis*	B	*Triticum aestivum*
CUN88	Cunfer et al. (1988)	*Parastagonospora nodorum*	H	*Triticum aestivum*
CZE03	Czembor et al. (2003)	*Parastagonospora nodorum*	H	*Triticum aestivum*
DAN16	Danelli and Reis (2016)	*Phakopsora pachyrhizi*	B	*Glycine max*
DEH02	Dehghani et al. (2002)	*Puccinia striiformis*	B	*Triticum aestivum*
DEH04	Dehghani and Moghaddam (2004)	*Puccinia striiformis*	B	*Triticum aestivum*
DEW03	Dewdney et al. (2003)	*Venturia inaequalis*	H	*Malus domestica*
DIA99	Diamond and Cooke (1999)	*Monographella nivalis*	N	*Triticum aestivum*
DIA03	Díaz-Lago et al. (2003)	*Puccinia coronata*	B	*Avena sativa*
DOW03	Dowkiw et al. (2003)	*Melampsora laricis-populina*	B	*Populus deltoides* × *trichocarpa*
DU014	Du et al. (1999)	*Parastagonospora nodorum*	H	*Triticum aestivum*
EAT84	Eaton et al. (1984)	*Puccinia graminis*	B	*Triticum aestivum*
ENG96	Engels and de Waard (1996)	*Blumeria graminis*	B	*Triticum aestivum*
EVE80	Eversmeyer et al. (1980)	*Puccinia recondita*	B	*Triticum aestivum*
FIG95	Figueroa et al. (1995a)	*Pyrenopeziza brassicae*	H	*Brassica napus*
FIG95b	Figueroa et al. (1995b)	*Pyrenopeziza brassicae*	H	*Brassica napus*
FIS08	Fisher et al. (2008)	*Puccinia hieracii*	B	*Centaurea solstitialis*
FRE10	Frenkel et al. (2010)	*Erysiphe necator*	B	*Vitis vinifera* *Vitis vinifera* × *Vitis labrusca*
GAL88	Galea and Price (1988)	*Microdochium panattonianum*	N	*Lactuca serriola*
GHA95	Ghannadha et al. (1995)	*Puccinia striiformis*	B	*Triticum aestivum*
GIL00	Gilles et al. (2000)	*Pyrenopeziza brassicae*	H	*Brassica napus*
GIN95	Gingera et al. (1995)	*Puccinia sorghi*	B	*Zea mays*
GIO96	Giorcelli et al. (1996)	*Melampsora laricis-populina*	B	*Populus* × *euramericana*
GRA11	Graichen et al. (2011)	*Puccinia coronata*	B	*Avena sativa*
HAM92	Hamelin et al. (1992)	*Melampsora medusae*	B	*Populus deltoides*
HAM94	Hamelin et al. (1994)	*Melampsora medusae*	B	*Populus deltoides*
HAZ98	Hazra (1998)	*Colletotrichum graminicola*	H	*Sorghum bicolor*
HER14	Hernandez Nopsa and Pfender (2014)	*Puccinia graminis*	B	*Triticum aestivum*
JAC04	Jacobsen et al. (2004)	*Cercospora beticola*	H	*Beta vulgaris* *Triticum aestivum* *Triticum compactum* *Triticum dicoccoides* *Triticum dicoccum* *Triticum durum*
JEG82	Jeger et al. (1983)	*Parastagonospora nodorum*	H	*Triticum monococcum* *Triticum mutica* *Triticum polonicum* *Triticum spelta* *Triticum timopheevi* *Triticum turgidum*
JOR05	Jorge et al. (2005)	*Melampsora laricis-populina*	B	*Populus deltoides* × *Populus trichocarpa*

(*Continued on next page*)

[a] Entries include citations of the articles from which the latent period data were taken, the pathogens included in the study, their trophic type, and the hosts on which they were grown.

[b] H = hemibiotrophic, B = biotrophic, and N = necrotrophic.

susceptible when not otherwise specified. None of the selected studies dealt with complete (gene-for-gene) resistance.

Study environment. Experimental conditions have been shown to influence the infection cycle of leaf fungal pathogens. In this respect, one could cite climatic parameters, inoculation techniques, or the environment in which the plants are grown. Because these experimental conditions were only partially documented for a large proportion of the articles, we kept only the latter. It was defined as a synthetic categorical environmental variable with four levels: detached leaves, leaflets, or leaf disks (D), controlled environment chamber (C), glasshouse (G), and field (F) experiments.

Statistical methods. *Latent period.* To investigate the putative effect of explanatory variables on the latent period, we used the following linear mixed model (model 1; equation 1):

TABLE 1. (*Continued from previous page*)

Study identifier	Article	Pathogen(s)	Trophic type[b]	Host(s)
KAN02	Kanrar et al. (2002)	*Alternaria brassicae*	N	*Brassica juncea*
KAR10	Karaoglanidis et al. (2011)	*Alternaria alternata*	N	*Pistacia vera*
KAR93	Kari and Griffiths (1993)	*Rhynchosporium secalis*	H	*Hordeum secalinum*
KAR02	Karolewski et al. (2002)	*Pyrenopeziza brassicae*	H	*Brassica napus*
KOC75	Kochman and Brown (1975)	*Puccinia graminis*	B	*Avena sterilis*
				Hordeum vulgare
KOL01	Kolnaar and Van Den Bosch (2001)	*Puccinia lagenophorae*	B	*Senecio vulgaris*
LAS96	Lascoux et al. (1996)	*Melampsora ribesii-viminalis*	B	*Salix viminalis*
LEC10	Lecompte et al. (2010)	*Botrytis cinerea*	N	*Solanum lycopersicum*
LI006	Li et al. (2006)	*Erysiphe pulchra*	B	*Cornus florida*
LI007	Li et al. (2007)	*Puccinia hemerocallidis*	B	*Hemerocallis* sp.
LI009	Li et al. (2009)	*Erysiphe polygoni*	B	*Hydrangea macrophylla*
LOU96	Loughman et al. (1996)	*Zymoseptoria tritici*	H	*Triticum aestivum*
		Parastagonospora nodorum	H	
LOV04	Lovell et al. (2004)	*Zymoseptoria tritici*	H	*Triticum aestivum*
MIL09	Milus et al. (2009)	*Puccinia striiformis*	B	*Triticum aestivum*
MON08	Montarry et al. (2008)	*Erysiphe necator*	B	*Vitis vinifera*
MOR15	Morais et al. (2015)	*Zymoseptoria tritici*	H	*Triticum aestivum*
MWA06	Mwakutuya (2006)	*Stemphylium botryosum*	N	*Lens culinaris*
NEW98	Newcombe (1998)	*Melampsora medusae*	B	*Populus deltoides*
PAR89	Park and Rees (1989)	*Puccinia striiformis*	B	*Triticum aestivum*
PED94	Pedersen and Morrall (1994)	*Ascochyta fabae*	N	*Lens culinaris*
PEE94	Peever and Milgroom (1994)	*Pyrenophora teres*	H	*Hordeum vulgare*
PRA89	Prakash and Thielges (1989)	*Melampsora medusae*	B	*Populus deltoides*
PRA13	Prasad et al. (2013)	*Nothopassalora personata*	H	*Arachis hypogaea*
		Puccinia arachidis	B	
RAZ05	Razavi and Hughes (2005)	*Zymoseptoria tritici*	H	*Triticum aestivum*
ROD95	Roderick and Clifford (1995)	*Blumeria graminis*	B	*Avena sativa*
ROG99	Roger et al. (1999)	*Didymella pinodes*	H	*Pisum sativum*
				Digitaria sanguinalis
				Festuca ovina
				Festuca rubra
ROS05	Rossi et al. (2005)	*Stemphylium vesicarium*	N	*Lolium perenne*
				Poa pratensis
				Pyrus communis
				Setaria glauca
				Trifolium repens
ROT89	Rotem et al. (1989)	*Alternaria brassicae*	N	*Gossypium barbadense*
SET08	Setti et al. (2008)	*Didymella pinodes*	H	*Pisum sativum*
SET09	Setti et al. (2009)	*Didymella pinodes*	H	*Pisum sativum*
SHA03	Sharma and Sharma (2003)	*Melampsora ciliata*	B	*Populus deltoides*
SHA90	Shaw (1990)	*Zymoseptoria tritici*	H	*Triticum aestivum*
SHE73	Shearer and Zadoks (1973)	*Parastagonospora nodorum*	H	*Triticum aestivum*
SOM09	Sombardier et al. (2009)	*Podosphaera aphanis*	B	*Fragaria* sp.
SOS95	Sosa-Alvarez et al. (1995)	*Botrytis cinerea*	N	*Fragaria* × *ananassa*
SOS05	Sosnowski et al. (2005)	*Plenodomus lingam*	H	*Brassica napus*
SOU12	Souza et al. (2012)	*Cercospora coffeicola*	H	*Coffea arabica*
STO87	Stooksbury et al. (1987)	*Parastagonospora nodorum*	H	*Triticum aestivum*
SUF13	Suffert et al. (2013)	*Zymoseptoria tritici*	H	*Triticum aestivum*
SUF15	Suffert et al. (2015)	*Zymoseptoria tritici*	H	*Triticum aestivum*
TEN78	Teng and Close (1978)	*Puccinia hordei*	B	*Hordeum vulgare*
TIE92	Tiedemann (1992)	*Bipolaris sorokiniana*	H	*Triticum aestivum*
		Parastagonospora nodorum	H	
TOL85	Tollenaar (1985)	*Puccinia striiformis*	B	*Triticum aestivum*
TOM83	Tomerlin and Jones (1983)	*Venturia inaequalis*	H	*Malus domestica*
TRA92	Trapero-Casas and Kaiser (1992)	*Ascochyta rabiei*	N	*Cicer arietinum*
VAN89	Vanniasingham and Gilligan (1989)	*Plenodomus lingam*	H	*Brassica napus*
VIL98	Viljanen-Rollinson et al. (1998)	*Erysiphe pisi*	B	*Pisum sativum*
VIL05	Viljanen-Rollinson et al. (2005)	*Zymoseptoria tritici*	H	*Triticum aestivum*
WOL16	Wolfenbarger et al. (2016)	*Podosphaera macularis*	B	*Humulus lupulus*
XU999	Xu (1999)	*Podosphaera pannosa*	B	*Rosa* sp.
XUR00	Xu and Robinson (2000)	*Podosphaera clandestina*	B	*Crataegus monogyna*
XUR01	Xu and Robinson (2001)	*Erysiphe polygoni*	B	*Clematis* sp.
YAN06	Yan et al. (2006)	*Podosphaera pannosa*	B	*Rosa* sp.
ZEA11	Zearfoss et al. (2011)	*Parastagonospora nodorum*	H	*Triticum aestivum*

$$\log\left(Y_{s,i,j,k,l}\right) = \mu + \alpha_i + \beta_j + \delta_k + \gamma_{i,j} + \gamma_{i,k} + \gamma_{j,k} + P_s + \varepsilon_{s,i,j,k,l} \quad (1)$$

$$\text{with } \left\{\varepsilon_{s,i,j,k,l}\right\} \sim N\left(0, \sigma^2_{s,i,j,k,l}\right)$$

where Y is the latent period, α is the main effect of trophic type, β is the main effect of host resistance level, δ is the main effect of the study environment, γ is the first-order interaction term between explanatory variables, and ε is the model residual. Second-order interactions could not be included in the model. P represents the random effect of the study. For all data, index i denotes the trophic type, j the host resistance level, k the study environment, s the study, and l the repetitions. In our model, the number of repetitions l corresponds to the number of mean latent period values given in the original studies for each experimental treatment (the "weight"): that is, each study s ($s \in [1 : 103]$) dealing with one or more pathogens of trophic type i ($i \in \{N, B, H\}$) infecting a host of resistance level j ($j \in \{S, M, R\}$) and grown in an environment k ($k \in \{D, C, G, F\}$) (see the section on explanatory variables).

Statistical analyses were performed with R 3.3.3 software (R Core Team 2013). The models were fitted with the lmer function from the lme4 package (Bates et al. 2015). Parameters of the model were estimated by the restricted maximum likelihood method. Data were log-transformed to improve the distribution of the model residuals (Supplementary Fig. S1). Because our data are mean values of several individual plants, the residual variance in our model was defined as proportional to the inverse of the number of plants used to generate these means, using the "weights" argument function of the lmer R function.

Latent period observed data are presented in Figures 2 and 3 (boxplots). A type III analysis of variance (ANOVA) was first performed using the model (equation 1) to test the significance of main effects and interactions (Table 2). Estimated latent periods for trophic types, resistance levels, environmental conditions, and their interactions were produced using the emmeans R package (Lenth 2016). These estimated means were back-transformed and expressed in degree-days before being presented in Figures 3 and 4 (filled circles and 95% confidence intervals). To assess the influence of each explanatory variable on the latent period, we compared the values of the Akaike information criterion (AIC) of models including the variables to those not including them (Supplementary Table S6). The AIC is a standard criterion to compare models of different complexity (Claeskens et al. 2010). We computed the AIC of the null model, of the model containing only one of the explanatory variables, and finally of the full model (equation 1).

Incubation period. To investigate the effect of trophic type on the length of the incubation period, we used a similar statistical approach (equation 2) while considering only two explanatory variables: trophic type and level of host resistance. Because there were fewer data on moderately resistant cultivars in this new reduced data set, the host resistance level categories M and R were merged into a new R category. The effect of the environment was not tested, because we lacked incubation period data for hemibiotrophs on detached leaves, both biotrophs and necrotrophs in the glasshouse and in the field, and biotrophs in controlled environment chambers.

We use the following linear mixed model (model 2; equation 2):

$$\log\left(Y_{s,i,j,l}\right) = \mu + \alpha_i + \beta_j + \gamma_{i,j} + P_s + \varepsilon_{s,i,j,l} \quad (2)$$

$$\text{with } \left\{\varepsilon_{s,i,j,l}\right\} \sim N\left(0, \sigma^2_{s,i,j,l}\right)$$

where Y is the incubation period, α the main effect of trophic type, β the main effect of host resistance level, and γ the interaction term between trophic type and resistance. P represents the random effect of the study. For each data point, index i denotes the trophic type, j the host resistance level, s the study, and l the repetitions. Statistical analyses were performed with the same tools as for the latent period. Data were log-transformed to improve the distribution of the model residuals (Supplementary Fig. S1). As for the latent period, the residual variance of the model was weighted by the inverse number of measurements used to generate data in the articles using the "weights" argument of the lmer function from the lme4 R package.

Incubation period observed data are presented in Figures 5 and 6 (boxplots). A type III ANOVA was performed to test the significance of main effects and interactions. Estimated incubation periods for trophic types and resistance levels were produced using the emmeans R package (Lenth 2016). These estimated means were back-transformed and expressed in degree-days before being presented in Figure 6. To complete this analysis, we compared incubation and latent period data on susceptible and resistant cultivars for each pathogen species in each article (Fig. 7).

RESULTS

Variability in the latent period on susceptible hosts. Figure 2 shows latent period data (mean values ± standard errors) only for susceptible hosts in each study on each pathogen species. The latent period ranged from 45 ± 7.0 DD for the necrotrophic *Stemphylium botryosum* to 623 ± 65 DD for the hemibiotrophic *Cercospora coffeicola*. Except for the biotrophic *Erysiphe graminis*, all of the pathogens with a mean latent period of <100 DD were necrotrophs. The longest latent period among necrotrophs was 134 ± 34 DD (*Ascochyta fabae*). Regarding biotrophs, 23 of 25 species had a mean latent period between 100 and 200 DD. Among them, seven of eight pathogens with the shortest latent periods were mildews (genera *Erysiphe* and *Podosphaera*), whereas seven of eight pathogens with the longest latent periods were rusts (genera *Puccinia* and *Melampsora*). The longest latent period among biotrophs was 322 ± 30 DD (groundnut leaf rust *Puccinia arachidis*). Among hemibiotrophic pathogens, only *Colletotrichum graminicola* had a mean latent period of <150 DD (110 ± 39 DD). Most hemibiotrophs had a mean latent period of more than 200 DD. It even exceeded 600 DD for *Cercospora coffeicola.* A broader range of latent periods was observed among hemibiotrophs than among biotrophs and necrotrophs.

Significant differences between trophic types. The ANOVA on model 1 (equation 1; a table of estimates is given in Supplementary Table S2) showed the significant effects of the explanatory variables: trophic type, host resistance, and study environment (Table 2). All interactions were significant. Figure 3 and 4 show both the observed data (boxplots) and the estimated latent periods (full circles with 95% confidence intervals). The statistical model established that the estimated latent period of necrotrophs ($LP_N = 103.3 \pm 11.5$ DD) was significantly shorter than that of biotrophs ($LP_B = 169.5 \pm 13.2$ DD, $P = 0.0006$), which was in turn significantly shorter than that of hemibiotrophs ($LP_H = 270.2 \pm 17.3$ DD, $P < 0.0001$; Fig. 3A). The statistical model also showed an effect of the level of host resistance on the latent period. The estimated latent period was significantly shorter for susceptible cultivars ($LP_S = 151.0 \pm 7.3$ DD), intermediate for moderately resistant cultivars ($LP_M = 167.9 \pm 8.7$ DD), and maximal for resistant cultivars ($LP_R = 186.6 \pm 10.4$ DD, $P < 0.0001$ for both S-M and S-R differences and $P = 0.0012$ for the M-R difference; Fig. 3B). Estimated latent periods showed no significant differences between the different experimental environments ($LP_D = 153.3 \pm 9.4$ DD, $LP_C = 178.4 \pm 14.6$ DD, $LP_G = 161.9 \pm 10.9$ DD, and $LP_F = 179.4 \pm 21.0$ DD; Fig. 3C). The estimated latent periods of each modality of the main effects are presented in Supplementary Table S3. AIC values showed that resistance is the most influent factor to explain the latent period, followed by the trophic type and then the environment (Supplementary Table S4).

Figure 4 shows the interactions between trophic type, host resistance, and study environment. The environment of the study did not change the ranking of the trophic type for the latent period (Fig. 4A). For all environmental conditions, the estimated latent period of necrotrophs was significantly shorter than that of hemibiotrophs. It was also less than for biotrophs in the detached leaf and glasshouse conditions. Biotrophs had a shorter estimated latent period than hemibiotrophs in both the controlled environment and glasshouse. Resistance impacted the latent period in all trophic types (Fig. 4B). The estimated latent period of biotrophs and hemibiotrophs was longer with moderately resistant and resistant hosts compared with susceptible ones, whereas the estimated latent period of necrotrophs was the same for susceptible and moderately resistant hosts but longer for resistant ones. Differences in the latent

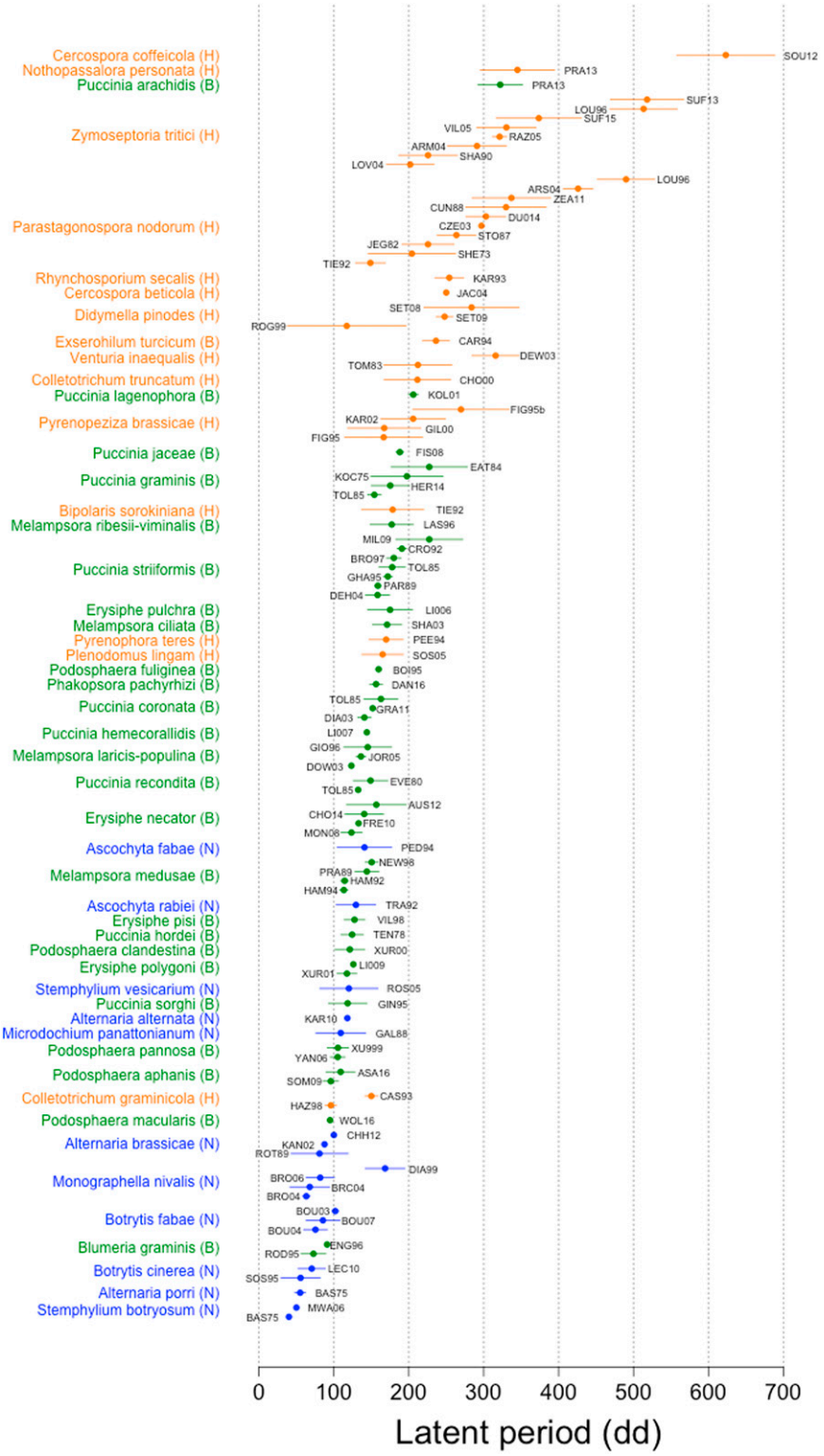

Fig. 2. Mean observed latent periods (expressed in degree-days [dd]) for the 53 pathogen species included in the meta-analysis. Pathogen species are ordered bottom-up according to their increasing mean latent period. For pathogens present in several studies, data are plotted separately for each study and labeled with the study identifier (listed in Table 1). Bars represent standard errors. (N), necrotrophs; (B), biotrophs; and (H), hemibiotrophs. Only data from susceptible cultivars are plotted here.

period between trophic types could be detected in all classes of partial resistance, with the exception of resistant necrotrophs and biotrophs, which had similar latent periods (Fig. 4B). On average, the estimated latent period increased by 22 DD (21%), 32 DD (22%), and 63 DD (27%) between susceptible and resistant hosts for necrotrophs, biotrophs, and hemibiotrophs, respectively. This suggests that the latent period increases in similar proportions on resistant hosts, regardless of the trophic type of pathogen, although the absolute increase is higher for hemibiotrophs.

Incubation versus latent period. On susceptible hosts, all biotrophs except *Puccinia arachidis* displayed a rather homogeneous incubation period of around 100 DD, whereas all necrotrophs except *A. fabae* had an incubation period of <100 DD (Fig. 5). The incubation period of necrotrophic pathogens ranged from 7.9 ± 1.8 DD for *Botrytis fabae* to 125.4 ± 35.0 DD for *A. fabae*; for biotrophic pathogens, it ranged from 77.6 ± 8.9 DD for *Melampsora ciliata* to 253.0 ± 37.6 DD for *Puccinia arachidis*. For hemibiotrophs, it ranged from 100.2 ± 7.6 DD for *Didymella pinodes* to 430.9 ± 44.0 DD for *Cercospora coffeicola*.

The latent period data of this subset show the same relation to trophic type and host resistance as the whole data set (data not shown). Results for the incubation period from the ANOVA on model 2 (equation 2; table of estimates in Supplementary Table S5) are given in Table 3 and show the significant effects for both trophic type and host resistance level on the incubation period of the pathogen. Figure 6 shows both the observed data (boxplots) and the estimated incubation periods (full circles with 95% confidence intervals). Figure 6 also shows that necrotrophs (IP_N = 39.3 ± 10.6 DD) had a significantly shorter estimated incubation period than biotrophs (IP_B = 115.1 ± 34.5 DD, P = 0.0144) and hemibiotrophs (IP_H = 179.6 ± 36.5 DD, P = 0.0001), but there was no significant difference between biotrophs and hemibiotrophs (P = 0.2191). Partial host resistance increased the incubation period (IP_S = 86.3 ± 13.4 DD and IP_R = 100.8 ± 17.5 DD; Fig. 6), but the difference was not significant (P = 0.0772). Incubation period estimates and statistical contrasts are presented in Supplementary Table S6.

There is a substantial diversity among pathogens in terms of the relative importance of the incubation period versus the period of symptom development that determines the latent period (Fig. 7). The time needed for the appearance of symptoms (incubation) ranged from 7.2 to 89.0% of the latent period of *B. fabae* (Fig. 7C) and *A. fabae*, respectively (Fig. 7B and C). The incubation period of *Parastagonospora nodorum* varied from 56 ± 2.8 DD to 280.5 ± 63.0 DD in different studies, respectively, equivalent to 18.5 to 86.6% of the latent period (Fig. 7M to Q). On average, for all of the data, the incubation period represented about 50% of the latent period for both susceptible and resistant host cultivars (*data not shown*). Therefore, the period of symptom development corresponded to half of the duration of the latent period. However, the ratio between incubation and latent periods was 36.5, 60.1, and 51.5% for necrotrophs, biotrophs, and hemibiotrophs, respectively (*data not shown*), suggesting a shorter relative incubation period for the necrotrophs. The impact of host resistance on the relation between incubation and latent periods is variable. The latent period may increase in resistant hosts for a variety of reasons: the incubation period alone increases (with a negligible increase in the period of symptom development, as in Fig. 7K and Q), the period of

TABLE 2. Results of the statistical test (analysis of variance on model 1) presenting the main effects, interactions, and contrasts between trophic type, culture environment, level of host resistance, and latent period (expressed in degree-days)

Effect and interaction	χ^2	df[a]	P value
Intercept	6,672.340	1	<0.0001
Trophic type	32.373	2	<0.0001
Environment	16.427	3	0.001
Resistance	62.735	2	<0.0001
Trophic type–environment	30.461	6	<0.0001
Trophic type–resistance	22.289	4	0.0002
Environment–resistance	87.264	6	<0.0001

[a] df = degrees of freedom.

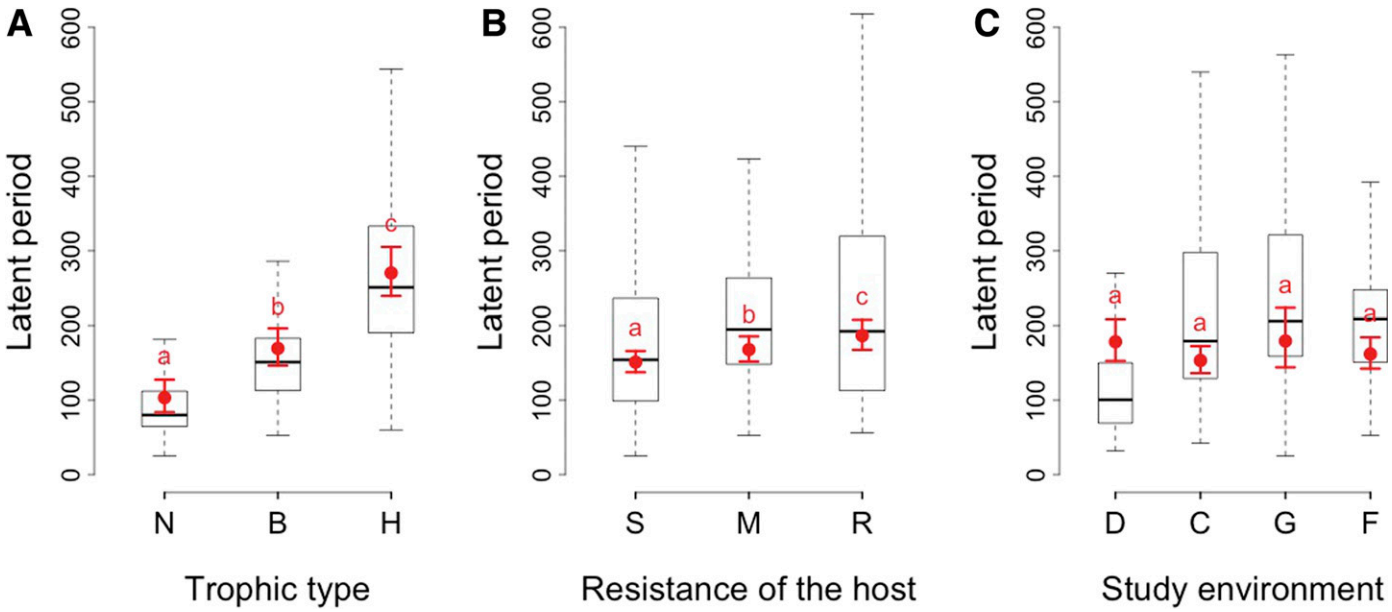

Fig. 3. Observed and estimated latent periods (expressed in degree-days) for each level of the three factors (trophic type, host resistance, and environment). The boxplots describe the distributions of the observed data for each level of the factors. Filled circles and bars show estimated mean values of the latent periods with their 95% confidence intervals. Mean latent periods and confidence intervals were estimated by fitting model 1 to log-transformed data. Estimated values were back-transformed into degree-days values. The following factors were considered: **A,** trophic type (N = necrotroph, B = biotroph, and H = hemibiotroph; contrasts: N-B, P = 0.0006; B-H, P < 0.0001; and N-H, P < 0.0001); **B,** level of host resistance (S = susceptible, M = moderately resistant, and R = resistant; contrasts: S-M, M-R, and S-R, P < 0.0001); and **C,** study environment (D = detached leaves or leaf disks, C = controlled environment cabinet, G = glasshouse, and F = field; contrasts: C-D, P = 0.4179; C-F, P = 0.8739; C-G, P = 1.000; D-G, P = 0.1464; D-G, P = 0.2715; and F-G, P = 0.6675). Within the same graph, different lowercase letters indicate significant contrasts (P < 0.05).

symptom development alone increases (with a negligible increase in the incubation period, as in Fig. 7C and P), or both the incubation period and period of symptom development are longer (as in Fig. 7A and G). All of the cases in which the increase in the latent period was only attributable to an increase in the incubation period were found among hemibiotrophs. Reciprocally, biotrophs and necrotrophs always display an increase in the period of symptom development in resistant hosts.

DISCUSSION

Trophic type, incubation, and latent periods. We performed a meta-analysis to investigate a putative correlation between the trophic type and latent period of pathogens. In this work, we use the division of the latent period into two stages that is common to most leaf fungal pathogens. During the first stage, the leaf appears untouched by the disease (incubation period). Then, during the second stage, nonsporulating symptoms such as chlorosis develop (period of symptom development).

One of the main results is that the three pathogen trophic types display significantly different latent periods (Fig. 3). Necrotrophs have the shortest latent periods (103 DD on average); biotrophs have intermediate latent periods (169 DD on average), whereas hemibiotrophs have the longest latent periods (270 DD on average). The incubation period also depends on the pathogen trophic type: as expected, necrotrophs have shorter incubation periods than biotrophs and hemibiotrophs (Fig. 6), although no significant difference was found between the latter. There is a huge variability between pathogens in terms of the proportion of the latent period that corresponds to the incubation period (from 10 to 90%). For all of the data, the incubation period corresponds to half of the latent period. However, the ratio between incubation and latent periods is shorter for necrotrophs than for hemibiotrophs and biotrophs. In necrotrophs, the incubation period probably corresponds to the time required for the pathogen to overcome immunity-triggered host cell necrosis and set its arsenal of lytic enzymes and toxins into motion (Kabbage et al. 2015). The variability of their incubation period may thus be attributed to twists and turns in the biochemical race for control of the cell death pathways between the host and the pathogen. For biotrophs and hemibiotrophs, the completion of the incubation period often signifies the appearance of chlorotic areas around the infection site (Perfect and Green 2001).

Host resistance, incubation, and latent periods. Several studies have already demonstrated an effect of cultivar resistance on the latent period, with host resistance increasing the pathogen latent period (e.g., Cromey 1992; Du et al. 1999; Roderick and Clifford 1995; Viljanen-Rollinson et al. 1998). However, no studies to date have examined the contribution of the incubation period to the increase in the latent period and its effect on resistant hosts or the interaction with pathogen trophic type. Our results show that all scenarios are possible: host resistance can extend (i) the incubation period only, (ii) the period for developing nonsporulating symptoms only, and (iii) both periods of time. Disentangling the effect of cultivar resistance on the incubation period and period of symptom development may open new perspectives for understanding resistance processes.

Choice of explanatory variables. The pathogen latent period is sensitive to many environmental factors. One could mention crop status such as plant age (Bouhassan et al. 2004; Giorcelli et al. 1996) or nitrogen status (Lecompte et al. 2010), origin of the host genotypes and pathogen strains (Broers 1997; Roderick and Clifford 1995; Setti et al. 2008), inoculation protocol (type of inoculum: Karolewski et al. 2002; Morais et al. 2015; inoculation technique: Giri et al. 2013; Miedaner et al. 2003; inoculum concentration: Karisto et al. 2018; Sosnowski et al. 2005; Xue and Hall 1992), and environmental climatic conditions such as temperature (Carson and Van Dyke 1994; Lovell et al. 2004), humidity (Gilles et al. 2000; Setti et al. 2008), and the plant growing conditions (Hernandez Nopsa and Pfender 2014; Ohm and Shaner 1976). Because data were missing from several articles, we only took into account three main explanatory variables: temperature, host resistance level, and study environment (i.e., detached leaves, growth chamber, glasshouse, or field). If data on more factors influencing the latent period were available, an interesting perspective would be to integrate these new variables into the analysis.

Defining and measuring the incubation and latent periods. Two main definitions of the latent period can be found

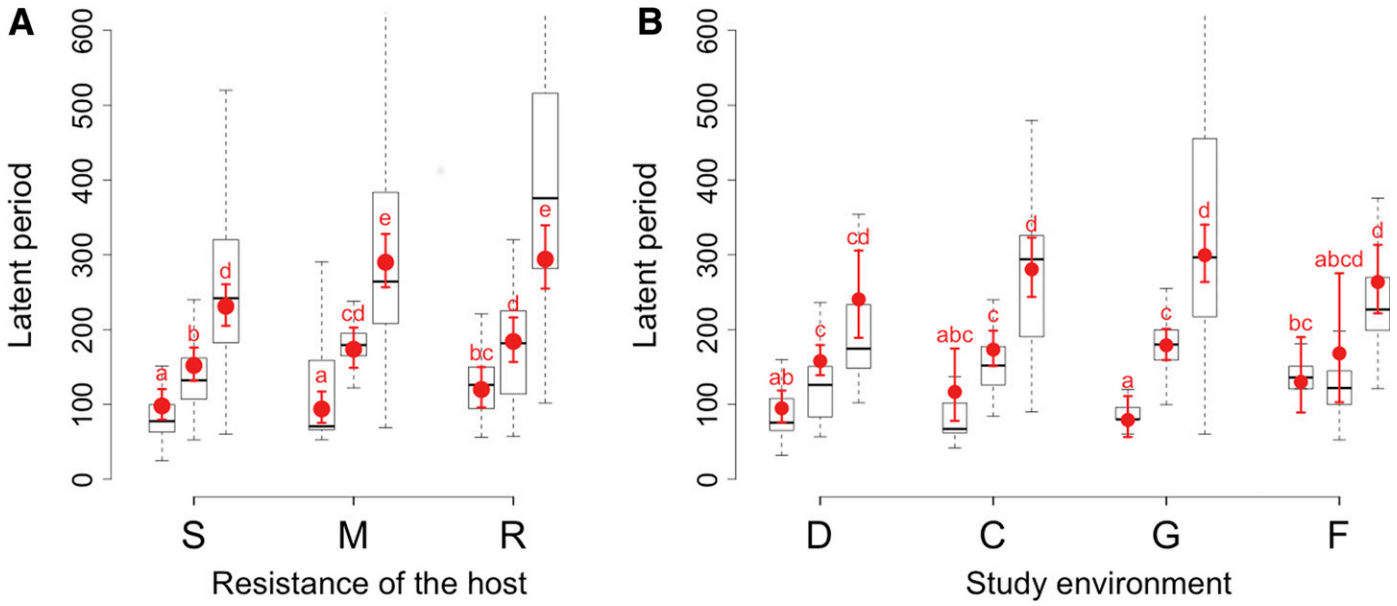

Fig. 4. Observed and estimated latent periods (expressed in degree-days) for the interaction between pathogen trophic type and the other two explanatory variables, resistance of the host and environment of the study. For each level of host resistance and study environment latent periods from left to right correspond to necrotrophs, biotrophs, and hemibiotrophs, respectively. The boxplots describe the distributions of the observed data for each level of the factors. Filled circles and bars show estimated mean values of the latent periods with their 95% confidence intervals. Mean latent periods and confidence intervals were estimated by fitting model 1 to log-transformed data. Estimated values were transformed back into degree-days values. **A,** Interactions between trophic type and resistance level of the host (S = susceptible, M = moderately resistant, and R = resistant). **B,** Interactions between trophic type and study environment (D = detached leaves or leaf disks, C = controlled environment cabinet, G = glasshouse, and F = field). Within the same graph, different lowercase letters indicate significant contrasts ($P < 0.05$).

in the literature. Some authors measure the latent period as the time interval between inoculation and the onset of reproductive structures (Parlevliet 1979; Shearer and Zadoks 1972). According to this definition, the latent period corresponds to the minimal possible duration for an infection cycle. Other authors measure the latent period as the time required for 50% of lesions to begin sporulation (Johnson 1980; Shaw 1986, 1990). In this case, the latent period corresponds to the average duration of an infection cycle. We refer to these two definitions as the "minimal" and "median" definitions of the latent period, which are both relevant when studying epidemiological dynamics. The onset of the sporulation period, and therefore the "minimal" latent period, is of great importance in the race between the plant and the pathogens (Robert et al. 2018). However, the date of the earliest sporulation

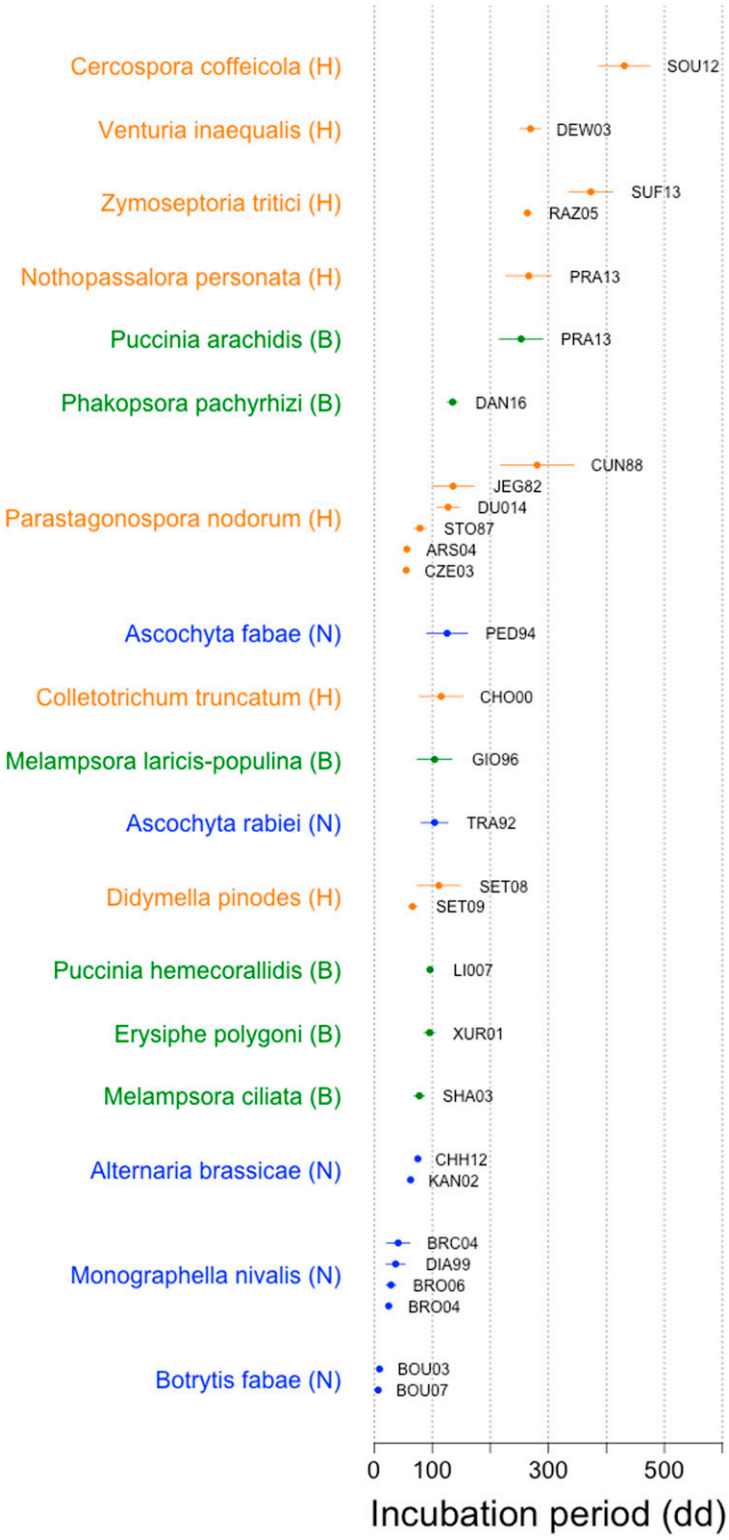

Fig. 5. Mean observed incubation periods (expressed in degree-days [dd]) for 18 pathogen species included in the meta-analysis for which both latent period and incubation period data are available. The pathogen species are ordered bottom-up according to their increasing mean incubation period. For pathogens present in several studies, data are plotted separately for each study and are labeled with the study identifier (Table 1). Bars represent standard errors. (N), necrotrophs; (B), biotrophs; and (H), hemibiotrophs. Only data from susceptible cultivars are plotted here.

event may not be related to the asymptotic intrinsic growth rate of the pathogen population (Shaw 1990), whereas the "median" latent period is analogous to the average generation time. As pointed out by Shaw (1990), a study of a pathogen's latent period should describe "both the temporal scale on which sporulating structures appear following infection at a single time, and the temporal pattern of that appearance" (p. 256). Although we initially looked for both types of data (minimal and median latent periods), we found no articles measuring the median latent period of necrotrophic pathogens; therefore, we did not include median latent periods in our analysis. Broers (1997) showed that minimal and median latent periods were highly correlated for the biotroph *Puccinia striiformis*. Similarly, Lovell et al. (2004) and Zearfoss et al. (2011), who measured both latent periods, showed similar responses of the minimal and medium latent periods to temperature and lesion development for both the hemibiotrophs *Z. tritici* and *Parastagonospora nodorum*. To check the robustness of our conclusions, we performed an analysis of the "median" latent periods reported in the selected studies (Fig. 1) and we found again that hemibiotrophs have a longer latent period than biotrophs (data not shown). Since our study focuses on average latent periods, it will be interesting to examine in more detail whether the ranges of variation of latent periods differ among trophic types, which may have profound consequences on the development of epidemics (Zearfoss et al. 2011).

Moreover, the assessment of the incubation and latent periods may depend on how the disease is monitored in the studies. For example, the visual assessment of disease symptoms can lead to substantial variations between individual raters and even between different measurements by the same rater (Bock et al. 2010; Mutka and Bart 2015; Nutter et al. 1993), thereby distorting incubation period measurements (but see Olmstead et al. 2001). New imaging techniques such as hyperspectral and thermal imaging (Chaerle et al. 2004; Mutka and Bart 2015) or molecular tools such as real-time PCR (Lievens et al. 2005, 2006; McCartney et al. 2003) could assist in the phenotyping of symptoms, lesions, and sporulation structures and lead to a more accurate estimation of both the incubation and latent periods. Using more precise and standardized imaging techniques is therefore an interesting perspective in order to make the studies on pathogen infection cycles more comparable.

Hemibiotrophy: Myth or reality? In this section, we discuss the definition of hemibiotrophy. Hemibiotrophs are usually defined as pathogens that establish biotrophically feeding interactions with their host during an initial period of tissue colonization before switching to necrotrophy (De Silva et al. 2016). Interpreted rigorously, this definition implies that a hemibiotrophic pathogen would derive nutrients from its living hosts during the initial colonization stage of the infection. Pathogens such as *Magnaporthe grisea*, *Nothopassalora personata*, and members of the genus *Colletotrichum* produce haustoria-like feeding structures or "intracellular" feeding hyphae (Behr et al. 2010; Perfect and Green 2001; Wambi 2014). It is therefore clear that such pathogens engage in a "true" biotrophic interaction with their host (i.e., they feed on the host at this stage).

The case of *Z. tritici* is subject to further controversy (Sánchez-Vallet et al. 2015). Some arguments are in favor of a strictly nonfeeding biotrophic stage. First, *Z. tritici* produces a limited amount of toxins and plant cell-wall degrading enzymes (PCWDEs) compared with other hemibiotrophs and necrotrophs (Brunner et al. 2013; Goodwin et al. 2011), whereas these PCWDEs are not expressed much during the asymptomatic phase (Yang et al. 2013). Second, few changes in the apoplastic nutrient content have been detected during the asymptomatic phase (which could suggest limited uptake by the pathogen), whereas the fungal biomass increases only slightly during the asymptomatic period (Keon et al. 2007; Palma-Guerrero et al. 2016). Third, meta-genomic data show that the fatty acid metabolism is dominant during the early stages of host colonization, suggesting that the pathogen uses the lipids stored in the spores (Palma-Guerrero et al. 2016; Rudd et al. 2015). Finally, many genes coding for nitrogen transporters or associated with nitrogen compound metabolic processes get upregulated only after the lifestyle transition (Palma-Guerrero et al. 2016). Based on such arguments, Sánchez-Vallet et al. (2015) proposed that *Z. tritici* should be considered a "latent" necrotroph rather than a hemibiotroph. However, there are also arguments in favor of "a biotrophic feeding" stage. First, the use of plant cell-wall carbohydrates by *Z. tritici* has been strongly suggested by Rohel et al. (2001). Second, despite the minimal increase in fungal mRNA during the asymptomatic phase, it supports spatially extensive tissue colonization by the pathogen (Steinberg 2015). Third, the mechanisms used by the pathogen to avoid the host immune response during the asymptomatic phase resemble those of biotrophic pathogens (Yang et al. 2013). Finally, exponential growth of the pathogen mycelium prior to asexual reproduction begins before the end of the asymptomatic phase (Steinberg 2015). *Z. tritici* is one of the most extensively studied pathogens, but despite all of the attention that it has received, we still lack physiological knowledge regarding its relation to the host. The same can be said for other highly studied pathogens, such as the oomycete *Phytophthora infestans* (De Silva et al. 2016; Fagard et al. 2014).

This raises difficulties regarding the classification of hemibiotrophs. Here, we chose to relax the definition of hemibiotrophy by defining hemibiotrophs as pathogens displaying a two-stage infection cycle characterized by contrasting interactions with the host: an asymptomatic colonization in the living host tissues followed by a necrotrophic stage with sustained asexual reproduction. This definition allowed us to bring together the 15 hemibiotrophic species of our analysis, including those displaying haustoria. Using this definition, we found that the latent period of hemibiotrophs is longer than that of biotrophs and necrotrophs. We also conducted a second statistical analysis that excluded the controversial pathogens *Z. tritici* and *Parastagonospora nodorum*. We once again found significant differences between the three trophic types (N:B, $P = 0.0012$; N:H, $P < 0.0001$; and B:H, $P = 0.0495$; data not shown). In any case, this would be of great interest to better understand the nature of the interaction during this long asymptomatic period of colonization in order to propose (or not) a new type of pathogens known as "long-latency necrotrophic pathogens." This raises various issues regarding the relevance of "hemibiotrophs" as a group. For instance, Parbery (1996) suggests dividing hemibiotrophs into those with short and long infection cycles.

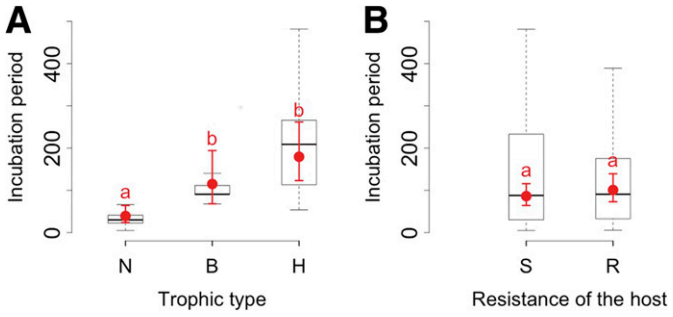

Fig. 6. Observed and estimated incubation periods (expressed in degree-days) for each level of two factors (trophic type and host resistance). The boxplots describe the distributions of the observed data for each level of the factors. Filled circles and bars show estimated mean values of the latent periods with their 95% confidence intervals. Mean latent periods and confidence intervals were estimated by fitting model 2 to log-transformed data. Estimated values were transformed back into degree-days values. Incubation periods are expressed in degree-days. **A,** Trophic type (N = necrotroph, B = biotroph, and H = hemibiotroph; contrasts: N-B, $P = 0.0144$; B-H, $P = 0.2191$; and N-H, $P = 0.0001$). **B,** Resistance of the host (S = susceptible and R = resistant together with moderately resistant; contrast: S-R, $P = 0.0772$). Within the same graph, different lowercase letters indicate significant contrasts. Data presented here come only from studies specifying both incubation and latent periods.

Trophic types: what processes may influence the duration of their latent period? Pathogens can evolve to adapt to their environment through changes in their latent period (Milus et al. 2009). Variability of the latent period within and between populations has been reported (Lehman and Shaner 1996; Pariaud et al. 2012; Suffert et al. 2015). The latent period has been shown to be partially heritable (Carson 1995; Ghannadha et al. 2005; Lehman and Shaner 2007), thus making it a good candidate trait for pathogen adaptation. Furthermore, Lehman and Shaner (1997) demonstrated that the latent period responds to artificial selection.

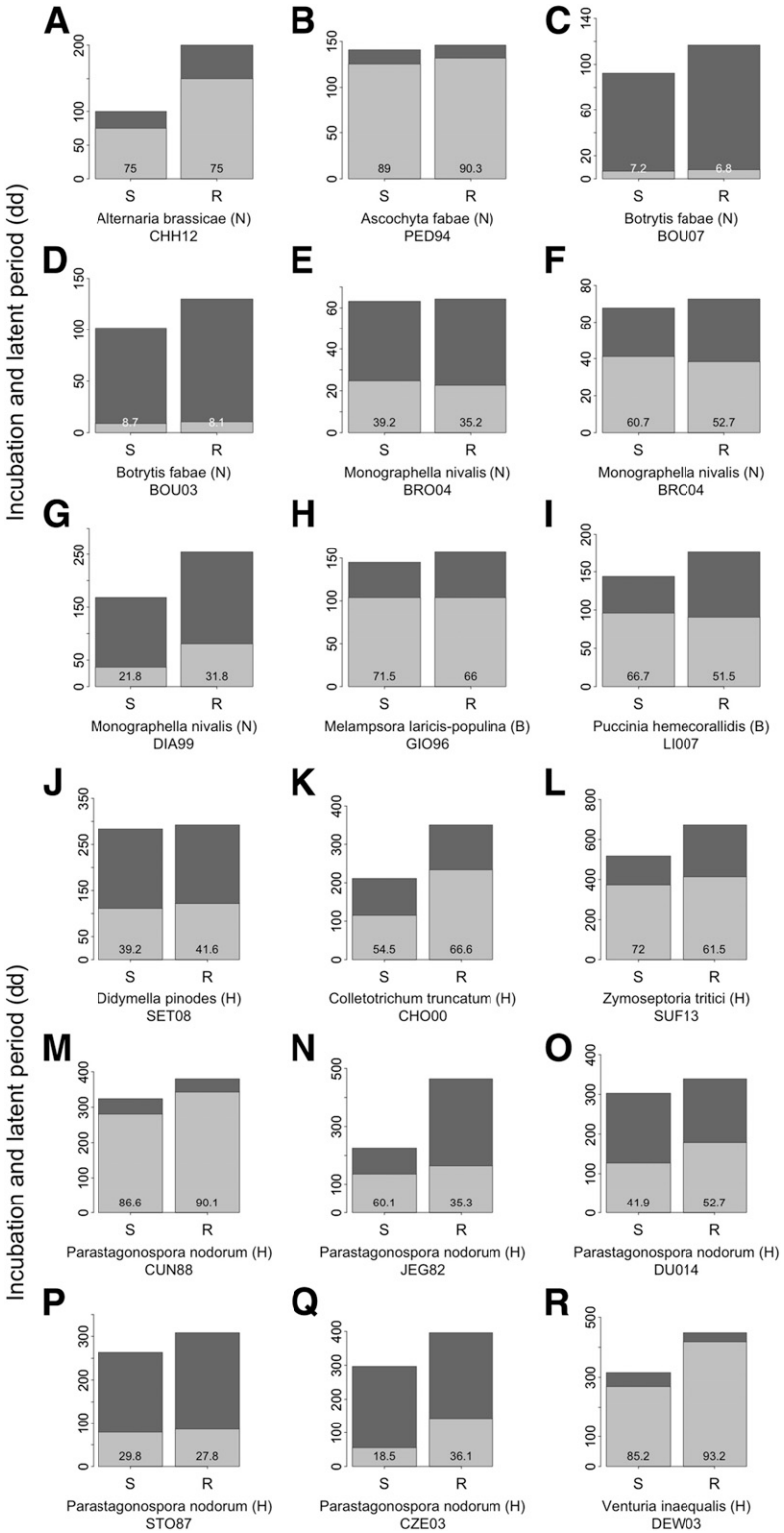

Fig. 7. Comparison of incubation and latent periods for necrotrophic (N), biotrophic (B), and hemibiotrophic (H) pathogens inoculated on susceptible (S) or resistant (R) plants. Light gray bars correspond to the incubation periods (expressed in degree-days [dd]). The sums of the light and dark gray bars correspond to the latent periods. Each graphic corresponds to a different pathogen from a specific study. **A,** *Alternaria brassicae*. **B,** *Ascochyta fabae*. **C** and **D,** *Botrytis fabae.* **E, F,** and **G,** *Monographella nivalis*. **H,** *Melampsora laricis-populina.* **I,** *Puccinia hemerocallidis*. **J,** *Didymella pinodes*. **K,** *Colletotrichum truncatum.* **L,** *Zymoseptoria tritici*. **M, N, O, P,** and **Q,** *Parastagonospora nodorum.* **R,** *Venturia inaequalis.* Numbers on bars indicate the percentage of the latent period that the incubation represents. Study identifiers are listed in Table 1.

Our data show that differences in pathogen trophic types are associated with differences in latent periods. Here, we discuss potential influential factors that could play a role in these differences. Our results suggest that there are two opposing host exploitation strategies: on the one hand, necrotrophs mount a rapid damaging attack on the host tissue, and on the other, biotrophs and hemibiotrophs carefully avoid or delay the damaging stage of the disease. Necrotrophs, by rapidly completing their infection cycle, access all of the resources of the necrotic tissue. By avoiding or delaying necrosis, biotrophic and hemibiotrophic pathogens establish a longer-lasting interaction with the host. We argue that this main difference can result from two different processes. The length of the latent period could be determined by physiological constraints set by the trophic types, such as physiological limits or genetic programs required for different trophic strategies (Lee and Rose 2010; O'Connell et al. 2012; Palma-Guerrero et al. 2016), and/or it could be the result of adaptation to contrasting conditions experienced by pathogens of different trophic types (Précigout et al. 2017). In other words, we discuss here whether the latent period of hemibiotrophs is long because it cannot be reduced for physiological reasons (physiological constraint processes) and/or because a longer latent period is optimal (adaptation processes).

Considering the physiological constraints, biotrophs and hemibiotrophs may need more time than necrotrophs to develop longer-lasting interaction structures such as haustoria and interfacial extracellular matrices (Garnica et al. 2014; Perfect and Green 2001; Yi and Valent 2013). Furthermore, it has been proposed that the long latent period of hemibiotrophs is necessary for them to undergo the profound transcriptomic rearrangements underlying the switch between biotrophy and necrotrophy (O'Connell et al. 2012; Oh et al. 2008). The diversity of processes that occur during the latent period probably reflects differences in host exploitation strategies. It is likely that these processes do not take the same amount of time for the different trophic types, meaning that they could lead to different latent periods for necrotrophs, biotrophs, and hemibiotrophs.

In terms of adaptation, we may argue that these contrasted host exploitation strategies appear to be analogous to the so-called "milker" or "killer" strategies with a prudent or aggressive exploitation strategy (van Baalen and Sabelis 1995). The advantage of the milker strategy is the expected benefit of continued host growth, whereas the advantage of the killer strategy is the immediate (and thus exclusive) access to the current host resource. This is consistent with the very low host damage caused by *Z. tritici* during the latent period (Robert et al. 2005). van Baalen and Sabelis (1995) showed that the evolutionary success of milkers and killers depends on the spatial population structure and, more generally, on the likelihood of sharing resources with competitors. A high risk of competition for resources (i.e., through coinfection) is predicted to select damaging strategies such as necrotrophs in our case. A low risk is predicted to select more prudent strategies such as hemibiotrophs. This idea is consistent with the work of van den Berg et al. (2013) who demonstrated that short latent periods are likely to be selected under conditions of autoinfection compared with alloinfection. High levels of autoinfection significantly increase host damage (Lannou et al. 2008), thereby reducing the amount of resources available to the pathogen (biotrophs need their host tissue to be alive). This increased level of damage is predicted to reduce the pathogen latent period as part of a virulence-transmission trade-off (Anderson and May 1982; van den Berg et al. 2013). From our results, this ecological "adaptation hypothesis" thus emerges for the relation between latent periods and trophic types. This hypothesis could be further tested by careful analysis of the phylogeny and ecological history of different pathogen species.

Another factor that could explain the differences in the latent periods between trophic types is related to the optimization of resource allocation between somatic growth (during the latent period) and reproduction (Gilchrist et al. 2006; Lika and Kooijman 2003; Précigout et al. 2017). Trade-offs that constrain the evolution of the latent period may very well differ for pathogens that do not continuously feed on living tissue such as necrotrophs and hemibiotrophs. One could expect the trade-offs to take different forms according to the pathogen trophic type (Gilchrist et al. 2006).

In conclusion, we found that pathogens of different trophic types display different latent and incubation periods and that the interaction between these traits and host resistance varies according to the trophic type. Such new knowledge on crop pathogen ecology opens new perspectives for crop protection.

TABLE 3. Results of the statistical test (analysis of variance on model 2) presenting the main effects, interactions, and contrasts between trophic type, level of host resistance, and incubation period (expressed in degree-days)

Effect and interaction	χ^2	df[a]	*P* value
Intercept	701.865	1	<0.0001
Trophic type	16.301	2	0.0003
Resistance	125.729	1	<0.0001
Trophic type–resistance	82.612	2	<0.0001

[a] df = degrees of freedom.

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