Death of the assumption that 'latent period' is fixed over the course of a plant disease epidemic

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Abstract – The latent period is defined as the time between infection and the onset of sporulation due to that infection. It is a crucial life history trait, particularly for polycyclic plant diseases, because it determines how many complete infectious cycles can occur during an epidemic and its final intensity. Many studies have focused on the variation of latent period with pathogen or host genotype, or its plasticity in response to environmental factors. The focus on these aspects is unsurprising, as these factors classically form the apices of the epidemiological triangle (host, pathogen, and environment). Experiments in controlled conditions are generally used to assess pathogenicity (virulence and aggressiveness) and host susceptibility. Once estimated for one or several pairs of host-pathogen genotypes, the value of the parameter 'latent period' is implicitly considered to be fixed and is used "as is" in epidemiological models. Paradoxically, most epidemiological studies do not consider the latent period of a pathogen population to be variable. My thesis here is that the latent period can display non-negligible variability over the course of a disease epidemic, and that this variability has multiple sources, some of which have complex, antagonistic impacts. I develop arguments for four sources of variation challenging the implicit assumption that the latent period remains constant: daily fluctuations in leaf temperature, nature of the inoculum, host stage or age of host tissues, intra-population competition and selection for aggressiveness traits. I focus on the wheat fungal pathogen Septoria tritici blotch (*Zymoseptoria tritici*), making use of empirical datasets collected during my own research projects and a targeted literature review. Finally I discuss in which case certain sources of these variation should be accounted for into epidemiological models.

Keywords – latent period, plant disease epidemiology, *Zymoseptoria tritici*

INTRODUCTION

In plant pathogens latent period is the time between host infection and the onset of pathogen sporulation from that infection (Pariaud *et al.*, 2009). It determines the interval between successive generations of pathogens, analogously to age of reproductive maturity in nonparasitic organism. The importance of the latent period for the understanding and prediction of pathogen development has long been recognized in plant disease epidemiology (Vanderplank, 1963; Zadoks, 1972). It is a crucial life history trait and a component of aggressiveness (Lannou, 2012), especially for polycyclic diseases, because it determines how many complete infectious cycles can occur during an epidemic and the final intensity of the epidemic. Several other monocyclic parameters (infection efficiency, infectious period, sporulation intensity) are of great importance, but my objective is not here to discuss about their relative impact.

Many plant pathology studies have investigated variations of the latent period with pathogen or host genotype, or its plasticity in response to environmental factors, such as temperature and humidity (Davis & Fitt, 1994; Shaw, 1990; Tomerlin & Jones, 1983; Webb & Nutter, 1997). Such approaches are relevant, because these factors lie at the apices of the epidemiological triangle (host, pathogen, environment; Zadoks, 1972). Paradoxically, because the common definition of latent period relates more to individual infection rather than dynamic population processes, these approaches mainly focused on the mean value and rarely on the variance within pathogen population. There is very little information about the expected magnitude and between-individual variability of the latent periods of foliar fungal plant pathogens. Such as variability was detected over a longer period in rare cases, for example in poplar rust: the latent period of five *Melampsora larici-populina* pathotypes collected in 1998 on Beaupré, a largely cultivated poplar cultivar, was 7 days, compared to 8 days required by the older isolates of the same pathotype (Pinon & Frey, 2005). Focusing on soilborne plant pathogens, Leclerc *et al.* (2014) similarly noticed that there is little information about how the incubation period (the time between host infection and the expression of first disease symptom, so shorter than latent period) varies within host populations. Finally, no study has ever considered the possibility that the latent period or the incubation period of a pathogen population may display variability over the course of a single annual epidemic.

In compartmental models SIR and SEIR (Kermack & McKendrick, 1927; van der Plank, 1963; Madden *et al.*, 2007) used for simulating polycyclic plant disease epidemics, the latent period in the worst case is considered to be fixed (e.g. *Plasmopara viticola* [Rossi *et al.*, 2009], *Phytophthora infestans* [Andrade-Piedra *et al.*, 2005], *Puccinia arachidis* [Savary *et al.*, 1990]), and in the best case is considered to be specific to an interaction between a pathogen genotype and a host genotype (e.g. Viljanen-Rollinson *et al.* 2005). The simplest case is the SIR model, in which the compartments represent susceptible, infectious and removed individuals, while the SEIR model also includes an exposed compartment, containing hosts who are in a latent stage (infected but not yet infectious). Several standard SEIR formulations in botanical, but also veterinary and medical epidemiology, assume that the stage durations (including length of infectious and latent periods) are exponentially or gamma distributed. However, real distributions are narrower and centred around the mean, and one can imagine that these distributions change over time. Effects of the infectious and/or latent period distribution on prediction concerning epidemiological transitions were rarely investigated: for instance in childhood disease dynamics by Krylova & Earn (2013), in plant disease dynamics by Parry *et al.* (2014) who includes seasonal and host age effects in their model of the latent period of Huanglongbing, and by Cunniffe *et al.* (2012) who considers flexible distributions of latent periods in generic SEIR models and demonstrates how mis-specificated or time-varying latent periods can lead to incorrect assessment of final intensity of an epidemic.

Experiments in controlled conditions are generally performed to assess the pathogenicity of the pathogen (separated into a qualitative term, virulence, and a quantitative term, aggressiveness; Lannou, 2012) or the susceptibility of the host. Once estimated for one or several pairs of host-pathogen genotypes, the value of the parameter 'latent period' is considered to be fixed and is introduced into the model "as is" (except for temperature, which is usually taken into account - imperfectly, as explained below - through the use of a thermal time scale). This approach is entirely appropriate if there is no great heterogeneity in the pathogen population, the host population or the environmental conditions during an epidemic. However, in practice, the pathogenicity of the active part of a pathogen population (e.g. in the case of a polycyclic epidemic, the strains responsible for secondary infections), the susceptibility of a host population (e.g. landraces or varietal mixtures), and the environmental conditions (e.g. diurnal or seasonal temperature variations) may vary in significant proportions. The values taken by the parameters 'pathogenicity', 'susceptibility', and 'temperature' may therefore be heterogeneous locally at a given time step and may vary over time. In such conditions, one may wonder if the variability of the latent period, as important as its mean value, should not be better estimated and taken into account in models for which it is important. My goal is here to convince the reader that the short-term variability of the latent period has numerous sources, some of which are antagonistic, and is sometimes not negligible. I have identified four sources of variability that challenge the implicit assumption that the mean latent period of a local pathogen population remains constant over the course of an epidemic. To present this view, I focus on the wheat pathogen Septoria tritici blotch (*Zymoseptoria tritici*), making use of empirical datasets collected during my own research projects and a targeted literature review. This fungal disease is particularly suitable for this analysis because the effects of several factors have been highlighted and are now well documented. *Z. tritici* is a cyclic heterothallic pathogen reproducing both sexually and asexually, resulting in infections initiated by two types of spores (ascospores and pycnidiospores), with relative contributions to the epidemic that change over the course of the year (Suffert *et al.*, 2011). The pathogen population displays a high degree of genetic diversity (Linde *et al.*, 2002) and there may be considerable phenotypic variability in latent period between strains (Morais *et al.*, 2015; 2016). Wheat has a long growth cycle and infections occur from early fall to late spring, under the influence of heterogeneous environmental selective pressures driven by abiotic conditions such as temperature (Lovell *et al.*, 2004a), but also biotic conditions such as the physiological stage of wheat or its fertilization regime (Robert *et al.*, 2005; Précigout *et al.*, 2017). As Septoria tritici blotch epidemics are polycyclic and results from an integration of many overlapping infection cycles, the latent period is a crucial fitness trait. It is quite long, facilitating the detection and quantification of any differences by *in planta* experiments, and it may display signs of local adaptation to climatic conditions (Suffert *et al.*, 2015).

The latent period for Septoria tritici blotch is usually estimated at the scale of a lesion, as the time between inoculation and the appearance of the first pycnidium (Shearer & Zadoks, 1972; Armour *et al.*, 2004) or, for the sake of convenience, 5% of the final number of pycnidia or 5% of the maximum percentage of area covered by pycnidia (Suffert *et*

al., 2013). When a population of lesions, rather than a single lesion is considered, particularly if this is due to methodological constraints (e.g. impossibility of replicating individual inoculation with a given *Z. tritici* genotype using the ascosporic form, contrary to the conidial form; Morais *et al.*, 2015), the latent period is estimated at the scale of a leaf, as the time between inoculation and the appearance of half the final sporulating lesions (Shaw, 1990; Lovell *et al.*, 2004a).

THE LATENT PERIOD CAN VARY WITH FLUCTUATIONS IN LEAF TEMPERATURE DESPITE AN IDENTICAL DAILY MEAN AIR TEMPERATURE

The development of plant pathogens responds strongly to the temperature of the surrounding environment. The effects of temperature are so well recognized in plant epidemiology that linear thermal time (referring to the accumulation of degrees above a given base temperature over a specified period of time; Lovell *et al.*, 2004b) is widely preferred over physical time for assessments and the modeling of plant pathogen development, particularly for *Z. tritici*. Consequently, the latent period is usually expressed in degree days rather than as a number of physical days. This accounts, for example, for the decrease in the latent period of *Z. tritici* estimated as a number of days over the spring epidemic period: a 350 degree-day latent period (with a base temperature of -2.4°C; Lovell *et al.*, 2004a) typically corresponds in average to 33 days in early spring (April) but only 22 days in late spring (June) in France (average monthly temperature in Poissy, Yvelines; https://en.climate-data.org). Taking into account the impact of temperature by this way is however not completely adequate because relationships between temperature and the efficiency or duration of a given epidemiological process are actually not linear and not monotonous. Consequently the latent period, while assessed using thermal time, should not be considered fixed, particularly if the time step used for the calculation is large (e.g. daily), for at least two reasons.

Firstly, thermal time is usually calculated from air temperature, whereas the development of foliar fungal pathogens, including *Z. tritici*, reacts more directly to leaf temperature (the temperature actually perceived by the fungus), which can be very different from air temperature (Bernard *et al.*, 2013). Leaf temperature is harder to measure than air temperature, but it could be estimated indirectly estimated from soil-vegetation-atmosphere transfer (SVAT) models including data recorded at standard weather stations (Xiao *et al.*, 2006).

Secondly, the latent period is usually assessed under fluctuating temperature regimes, with a thermal scale based on the accumulation of daily mean temperatures. The effects of diurnal fluctuations are, therefore, not taken into account. Bernard (2012) established the impact of two patterns of leaf temperature variation, in which mean temperature was equal (18°C) but daily temperature range differed (±2°C and ±5°C), on the latent period of *Z. tritici*: the larger temperature range increased the latent period by 1.3 days on average. Similar results have been obtained for other plant pathogens (Scherm *et al.*, 1994; Xu, 1999). The differences in pathogen development between constant and fluctuating environments are partly due to 'rate summation' or the Kaufmann effect (Ruel & Ayres, 1999), a mathematical consequence of the nonlinear shape of thermal performance curves (TPCs). The length of the latent period under a fluctuating temperature regime can be predicted with the rate summation scheme (Hau *et al.*, 1985):

$$S = \sum_{i=1,Y} [R(T)\Delta t]$$

where Δt is the integration step (e.g. 1 hour); S, the accumulated development, is dimensionless and defined as zero initially and one at the completion of a process (i.e. appearance of the first pycnidia), and $R(T)$ is the rate function of this process.

Finally, degree-hours should be preferred over degree-days once the TPC of the latent period is available.

The mean TPC of the latent period for *Z. tritici* was established empirically, with a limited number of fungal isolates, in natural (Shaw, 1990) or controlled conditions (Bernard *et al.*, 2012). The variability of the latent period between pathogen populations of different geographic origins has never before been characterized in detail. The latent period TPCs presented in Fig. 1a were obtained from two groups of nine *Z. tritici* samples collected from two regions of France with different climates (Brittany and Burgundy). The thermal optimum differed between the two populations: <20°C for the isolates from Brittany, >21°C for the isolates from Burgundy. Thus, the effect of temperature on latent period can differ between pathogen populations expressing local patterns of climatic adaptation.

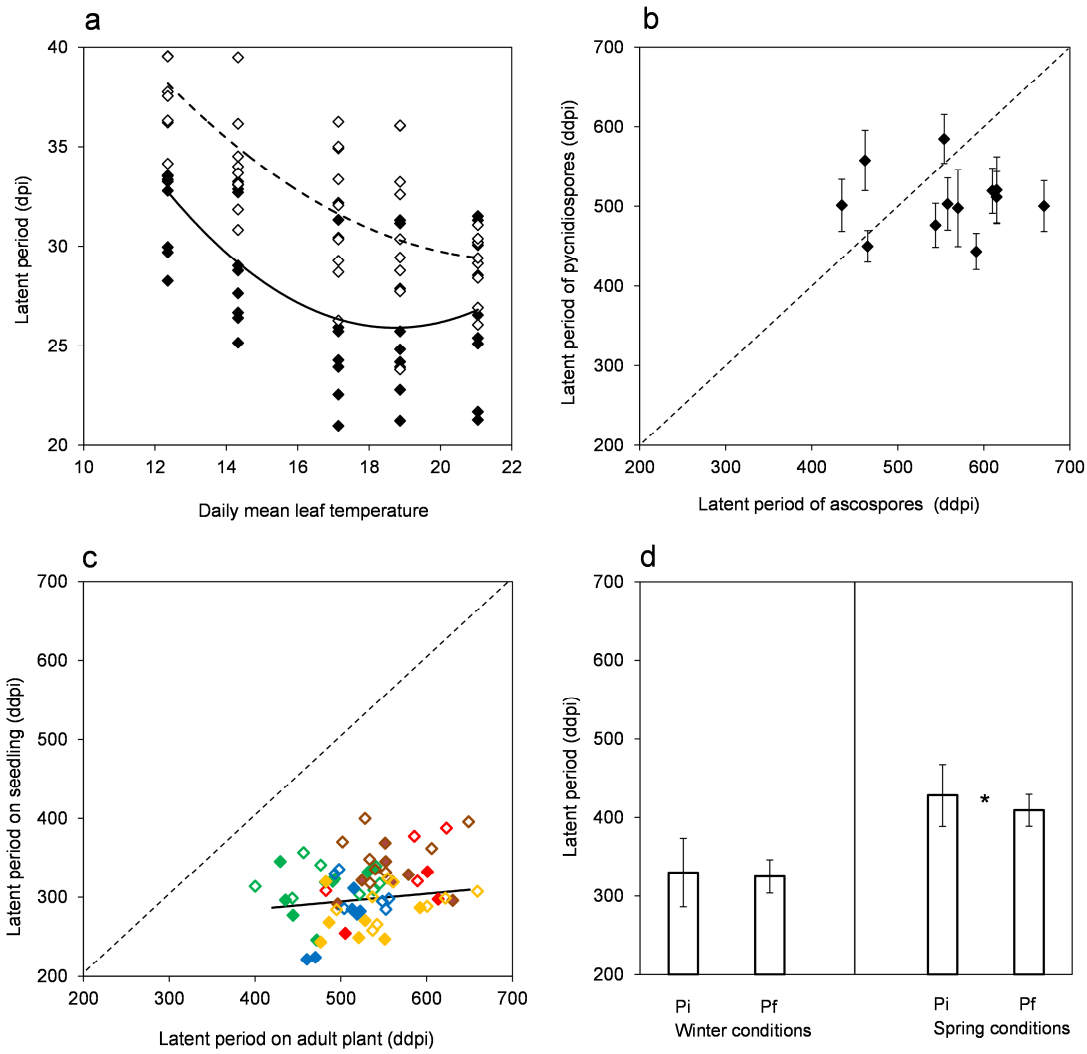

Figure 1 – Illustration of four sources of variability of the latent period (expressed in days post-inoculation [dpi] or in degree-days post-inoculation [ddpi] with a base temperature of 0°C) in the wheat pathogen *Zymoseptoria tritici* over the course of an annual epidemic. Latent period was calculated in cases 1a, 1c and 1d as the time between inoculation and the appearance of 5% of the maximum percentage of area covered by pycnidia, calculated by fitting a Gompertz growth curve to experimental data as described in Suffert *et al.* (2013), and in case 1b as the time between inoculation and the appearance of 5% of the maximum number of pycnidia in each individual lesion as described in Morais *et al.* (2015).

1a. Effect of daily mean wheat leaf temperature on the latent period of two *Z. tritici* populations (2 × 9 isolates collected from cv. Apache in two French regions; black diamond = Dijon in Burgundy; white diamond = Ploudaniel in Brittany) assessed after pycnidiospore inoculation on wheat adult plants cv. Apache. The thermal performance curve (order 2 polynomial) was adjusted with six replicates per temperature.

1b. Relationship between the latent period of 12 *Z. tritici* isolates assessed after ascospore and pycnidiospore inoculation on wheat adult plants cv. Apache (from Morais *et al.*, 2015). Each point corresponds to the mean of several values for pycnidiospore inoculation (vertical bars represent the standard deviation) and a single value for ascospore inoculation.

1c. Relationship between the latent periods of *Z. tritici* populations (2 × 9 isolates collected from cv. Apache in two French regions; colored diamond = Dijon in Burgundy; white diamond = Ploudaniel in Brittany) assessed after pycnidiospore inoculation on wheat seedlings and wheat adult plants cv. Apache for five different wheat cultivars: Apache (green), common to both Brittany and Burgundy; Altamira (red) and Paledor (yellow), mostly cultivated in Brittany; Arezzo (blue) and Altigo (brown), mostly cultivated in Burgundy. Each point is the mean value for assessments on six replicates.

1d. Mean latent period of two *Z. tritici* subpopulations (2 × 15 isolates collected on seedlings cv. Soissons very early [Pi] in the epidemic and the upper leaf layers at the end of the same epidemic [Pf]), assessed under winter (on wheat seedlings cv. Soissons at 8.9°C) and spring (on adult plants cv. Soissons at 18.1°C) conditions (from Suffert *et al.*, 2015). Asterisks indicate that the mean differs significantly ($P < 0.1$) between Pi and Pf.

Several published models of Septoria tritici blotch development consider the latent period to be proportional to temperature: 330 dd in Robert *et al.* (2008); 250, 350, 450, and 550 dd in Eriksen *et al.* (2001); 256 dd in Lovell *et al.* (2004a); 310 dd in Baccar *et al.* (2011). The mean TPC of the latent period is thus considered to be a decreasing linear function, with base temperature estimated at -2.4°C (Lovell *et al.*, 2014a) or 0°C (Armour *et al.*, 2004). The assumption of linearity for the suboptimal part of the TPC (from 10°C to 18°C) is acceptable, but not taking the supraoptimal part of the temporal TPC correctly into account can have deleterious consequences, particularly in fluctuating thermal regimes

THE LATENT PERIOD IS AFFECTED BY THE FORM OF INOCULUM: ASCOSPORES VS. PYCNIDIOSPORES

Models of Septoria tritici blotch development considering both ascospores and pycnidiospores in an explicit manner, either throughout the cropping season (Eriksen *et al.*, 2001) or solely at the onset of the epidemic (Robert *et al.*, 2008), assume that the infection process after spore deposition is the same for both types of spore. Morais *et al.* (2015) showed however that the latent period of *Z. tritici* was significantly longer (about 60 degree-days, i.e. 3-4 days in late spring) after infection with ascospores than after infection with pycnidiospores (Fig. 1b). This empirical result is consistent with results obtained for other plant pathogens in studies considering the efficiency of different types of spore without specifically focusing on the latent period (Gilles *et al.*, 2001; Karolewski *et al.*, 2002; Li *et al.*, 2004). In *Z. tritici*, one concrete consequence of this difference is that the mean latent period early in the epidemic, when first lesions are caused by wind-dispersed ascospores, is intrinsically longer than that during the spring epidemic stage, when infections are caused mostly by splash-dispersed pycnidiospores (Suffert *et al.*, 2011). A hypothetical, theoretical distribution of the number of new lesions induced by a pathogen population consisting of different *Z. tritici* strains according to their latent periods results in the superimposition of two unimodal distributions centered around the mean latent period value of each type of spore (Fig. 2). The resulting distribution may or may not be bimodal, depending on the relative contributions of the two types of spore to the infection. This example reveals another reason for which the latent period of a plant pathogen with both sexual and asexual reproduction modes should not be considered to be fixed or unique over the course of an epidemic.

THE LATENT PERIOD DEPENDS ON HOST STAGE AND HOST TISSUE AGE

An increase in the latent period with increasing host development is classically observed for several plant pathogens, such as *Puccinia hordei* (Parlevliet, 1975) and *Puccinia striiformis* (Tomerlin *et al.*, 1983). This finding is consistent with the lack of univocal relationship between seedling and adult plant resistance, in wheat rusts for example, because many resistance genes are expressed in adult plants but not in seedlings (McIntoch *et al.*, 1995). I assessed the latent periods of two groups of *Z. tritici* isolates collected in two climatically different regions of France (Brittany and Burgundy), on both seedlings and adult plants. I found a large difference between plants of different ages, with a mean latent period of 301 dd for seedlings and 534 dd for adult plants (Fig. 1c). Moreover, other experimental studies have suggested that the susceptibility of wheat tissues varies with leaf layer for synchronous measurements (i.e. on the same date) on adult plants, probably due to differences in leaf age (interactions between the susceptibility of host tissues, natural senescence and nitrogen status; Ben Slimane *et al.*, 2012; Bernard *et al.*, 2013; Suffert *et al.*, 2015). The increase in latent period with developmental stage (seedling vs. adult plant), and, more generally, with leaf age (time between leaf emergence and leaf infection), has been investigated in detail for *Puccinia arachidis* (Savary, 1987). These findings provide further support for the contention that the latent period of a plant pathogen should not be considered to be fixed or unique over the course of an epidemic.

THE LATENT PERIOD IS STRAIN-DEPENDENT AND, THEREFORE, AFFECTED BY COMPETITION WITHIN A LOCAL PATHOGEN POPULATION

As mentioned above, latent period depends on pathogen genotype. Variability within a local pathogen population may be high or low, according to the intrinsic structure of the population (sexual reproduction leading to high levels of variability or clonality). Locally, at the scale of a single annual epidemic, some authors consider average aggressiveness, and, thus, latent period, to be stable (for a given type of spore). This assumption is usually implicit, but might be erroneous. I showed that the mean latent period of *Z. tritici* pycnidiospores can vary significantly during a single annual epidemic (Suffert *et al.*, 2015): isolates collected on the upper leaf layers of wheat at the end

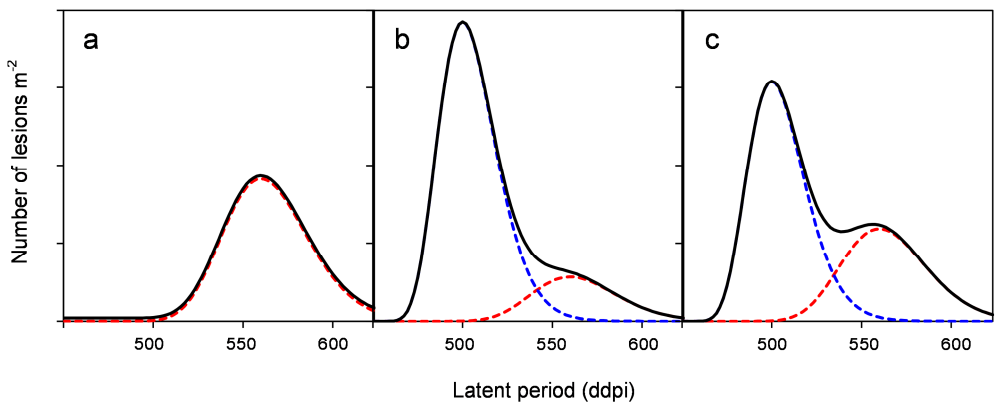

Figure 2 – Hypothetical, theoretical distribution of the number of new lesions (on wheat plants, per m^2 and per week) induced by a pathogen population consisting of different *Z. tritici* strains according to their latent period, taking into account the nature of the spores and the epidemic stage (a = early stage of the epidemic in December; b = intermediate stage of the epidemic in April, c = late stage of the epidemic in June).

Red dotted lines correspond to ascospore-initiated lesions; blue dotted lines correspond to pycnidiospore-initiated lesions; black lines are the cumulative curves. Curves were built with the hypothesis of the mean latent period is 505 ddpi for pycnidiospore infection and 557 ddpi for ascospore infection, based on the results obtained by Morais *et al.* (2015) and re-used in Fig.1b. Both latent periods have here a gamma distribution, with a similar range of variance for ascospores and pycnidiospores. The relative height of the curves does not rely on actual experimental values, which do not exist, and is to be considered as an order of magnitude. This order of magnitude is inspired by the relative importance of the two types of inoculum from early (a) to intermediate epidemic stage (b) estimated using data of inoculum availability (Suffert & Sache, 2011; Morais *et al.*, 2015) and data of simulated origin of inoculum causing lesions from intermediate (b) to late (c) epidemic stage (Duvivier, 2015). The effect of host stage (Fig. 1c), which would likely shorten latent period at the early stage of the epidemic (a), was here not taken into account.

of epidemic have a shorter latent period than those collected from seedlings very early in the same epidemic. This difference in latent period between disease expressed under spring conditions (adult plants, warm temperature) and under winter conditions (seedlings, cold temperature) and suggested that strains with a shorter latent period are selected during the second part of the epidemic (spring), when the disease is propagated by the upward splash dispersal of spores (Fig. 1d). During this period, a short latent period is a key fitness trait conferring a real competitive advantage. These conclusions were corroborated by the observed decrease in between-genotype variance for the latent period. The decrease in the mean latent period of a pathogen population over the course of the epidemic is consistent with the increase in other aggressiveness traits recorded in various fungal pathogens after a few cycles of asexual reproduction (Newton & McGurk, 1991; Villaréal & Lannou, 2000; Andrivon *et al.*, 2007; Le May *et al.*, 2012). Once again, these findings indicate that the latent period of a plant pathogen should not be considered to be fixed or unique over the course of an epidemic.

DISCUSSION

This dissertation asserts, through demonstrations based on scattered published results, that the mean latent period of the active part of a local pathogen population should not be considered constant over the course of the annual plant disease epidemic. Mean latent period is variable, and the sources of variation are numerous: daily fluctuations in leaf temperature, nature of the inoculum, host stage or age of host tissues, intra-population competition and selection for aggressiveness traits. Some of these sources of variation may have complex, antagonistic impacts.

On the one hand, mean latent period may decrease during the epidemic period because of intra-population competition driven by external factors, for instance temperature in the case of *Z. tritici*. On the other hand, it may decrease during the same period with changes in the ratio of the two spore types (pycnidiospores > ascospores), and finally increase as the ratio inverses (ascospores > pycnidiospores) during the late epidemic stage due to the occurrence of sexual reproduction before the end of the growing season. Shaw (1990) suggested

that the increase in latent period he observed at high mean temperatures reflects the adaptation of *Z. tritici* to local climatic conditions, such as the cool summers in the UK, and a physiological trade-off between an ability to grow rapidly at high temperatures and an ability to grow rapidly at low temperatures. This hypothesis is consistent with the conclusion of Suffert *et al.* (2015) that seasonal changes can drive short-term selection for fitness traits, recently confirmed by Boixel *et al.* (unpubl. data). However, Shaw's results were obtained in field conditions, and might therefore also be due to an artifact related to host stage effects (latent period shorter on seedlings than on adult plants), the use of air temperature rather than leaf temperature (Bernard *et al.*, 2013), and a greater amplitude of daily fluctuations during spring than during winter (Bernard, 2012). I suspect there are several causal factors involved, as is often the case in plant disease epidemiology.

The key message is that the mean latent period of a plant pathogen population can vary locally, in the short term. The direction (decrease or increase) and causes of this variation are difficult to determine, but plant pathologists need to be aware that the latent period is not fixed and that they may have an interest in incorporating, when necessary, a certain variability into epidemiological models. This variability should not be only described by a statistical distribution (e.g. exponential or gamma distribution) but also be structured by epidemiological processes as illustrated by the four sources of variation described above. The main difficulty is to know exactly in which case these sources of variation should be accounted for in a modelling work. The detail of the parameterization of any model should depend on the purpose of that model, considering that it cannot take into account too many unconstrained parameters. Sources of short term variability in latent period should be analyzed and potentially incorporated into three type of epidemiological models at least: in the case of *Z. tritici*, forecasting models used by growers to determine the proper timing for effective fungicide sprays (e.g. Audsley *et al.*, 2005; El-Jarroudi *et al.*, 2009), mechanistic models used as research tools for testing the impact of different epidemiological parameters on disease development (e.g. Eriksen *et al.*, 2017; Baccar *et al.*, 2011), and evolutionary models in which latent period is under selective pressure (Suffert *et al.*, 2015), is involved in a trade-off (e.g. Heraudet *et al.*, 2008; Pariaud *et al.*, 2012; Suffert *et al.*, subm.), or is expected to tend towards an evolutionary optimum (e.g. Précigout *et al.*, 2017). To justify this, I have identified only two examples in botanical epidemiology. In the first example, Cunniffe *et al.* (2012) proposed an extension to the generic SEIR model, splitting the latent and infection compartments and thereby allowing time-varying infection rates and more realistic distributions of latent and infectious periods to be represented. Their results demonstrated that extending a model that has such a simplistic representation of the infection dynamics may not, in fact, lead to more accurate results, and they suggested that modelers should carefully consider the underlying assumptions of the simplest compartmental models. In the second example, Leclerc *et al.* (2014) conducted experiments on the soilborne pathogenic fungus *Rhizoctonia solani* in sugar beet and used spatially-explicit models to estimate the incubation period distribution. They demonstrate the impact of differing distributional estimations about the incubation period (i.e. exponential vs. gamma) on the epidemiological understanding of the disease development. These two examples show that including additional biological realism in a model about the distribution or the variation in latent period can be more or less critical in assessing the disease development at the scale of a single annual epidemic.

The aim of this article was to highlight one thing that was largely ignored before: the latent period is not necessarily fixed in the short term. Having done this, it is up to each plant pathologist to analyze the conclusions he draws from "the death of this assumption". I hope this consideration will allow to justify the addition of useful biological knowledge into epidemiological models, since their precision or their robustness could be improved.

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