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REVIEW

Some reasons why the latent period should not always be considered constant over the course of a plant disease epidemic

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The latent period is a crucial life history trait, particularly for polycyclic plant diseases, because it determines how many complete infection cycles could theoretically occur during an epidemic. Experiments in controlled conditions are generally used to assess pathogenicity and host susceptibility, and also provide the opportunity to measure the distribution of latent periods in epidemiological systems. Once estimated for one or several pairs of host–pathogen genotypes, the mean value of this trait is usually considered to be fixed and is often used 'as is' in models. This review contends 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. Arguments are developed for four sources of variation challenging the assumption that the latent period remains constant: (i) daily fluctuations in host temperature (or other organ–environment factors); (ii) nature of inoculum; (iii) host stage or age of host tissues; and (iv) intrapopulation competition and selection for aggressiveness traits. The review is focused on the wheat pathogen *Zymoseptoria tritici*, making use of empirical datasets collected during the first author's research projects and a targeted literature review. Such empirical epidemiological knowledge is potentially important for epidemiological modellers. While some studies have demonstrated that the distribution of latent periods around the mean value has consequences for epidemiological dynamics, it is shown here that it might also be important for modellers to account for changes in this mean value during an epidemic. These results may be critical for improving epidemic forecasting.

Keywords: latent period, mathematical modelling, plant disease epidemiology, *Zymoseptoria tritici*

Introduction

The latent period is defined as 'the length of time between the start of the infection process by a unit of inoculum and the start of production of infectious units' (Madden *et al.*, 2007). It contributes to the generation time of the pathogen, i.e. the length of time between successive infections in a transmission chain, analogous to the age of reproductive maturity of nonparasitic organisms. The importance of the latent period for understanding and predicting pathogen development has long been recognized in plant disease epidemiology (van der Plank, 1963; Zadoks, 1972). It is a crucial life history trait and component of aggressiveness (Lannou, 2012), especially for polycyclic diseases, because it is one of the major determinants of the number of complete infection cycles that could theoretically occur during an epidemic in a single season, which in turn affects the final intensity of the epidemic. This view is an over-simplification in the case of pathogens for which disease cycles overlap. Other parameters (infection efficiency, infectious period, sporulation intensity) are also important in the adaptive value of a pathogen species and in the predictability of the disease dynamics, but the focus here is on the latent period because of its significant variation within epidemics caused by pathogens such as *Zymoseptoria tritici* (septoria tritici blotch of wheat). Some of the factors that drive temporal changes in the latent period vary systematically, potentially allowing for accurate predictions of variation in the latent period, and in the resulting epidemic dynamics, to be made.

The predictability of disease dynamics depends not only on the ability to assess accurately the mean length of the latent period, but also its variability (Cunniffe *et al.*, 2012; Thompson *et al.*, 2016). Ferrandino (2012) clearly showed that the simple use of a population average for the latent period, and also for the infectious

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period ('the length of time between the start of production of infectious units and the end of production of infectious units'; Madden *et al.*, 2007) is problematic. Using a theoretical model, it was demonstrated that the time course of a single annual epidemic does not only depend on the average values of the latent and infectious periods, but also on the variance in these quantities about their respective means and the covariance between them. This work was followed up by a more detailed analysis showing how the reproduction curves characterizing the production of progeny impact on the speed of an epidemic (Ferrandino, 2013).

Compartmental models used for simulating plant disease epidemics (Madden *et al.*, 2007) often include an exposed compartment, containing hosts that are in a latent stage (infected but not yet infectious). The length of time that infected hosts spend in this compartment is usually assumed to be exponentially distributed, although other distributions (e.g. gamma distributions) have also been considered in compartmental models (Cunniffe *et al.*, 2012; Thompson *et al.*, 2016). These distributions are usually assumed to account for random variation between hosts, rather than systematic differences in latent periods due to e.g. interactions between a pathogen genotype and a host genotype (Viljanen-Rollinson *et al.*, 2005). The mean value of the latent period is also usually assumed to remain constant throughout an epidemic. One exception is the model of citrus greening disease by Parry *et al.* (2014), in which the mean latent period is assumed to oscillate.

In practice, the length of the latent period changes based on a number of factors. From experimental data, three origins of variability have been identified: (i) experimental uncertainty in the assessment (measurement errors and biases); (ii) phenotypic heterogeneity between individuals within a population (interindividual variance, due for instance to the inherent range of virulence or aggressiveness within the pathogen population); and (iii) variation in the conditions of disease expression, including those due to environmentally induced changes (phenotypic plasticity, whose expression can be amplified for instance by somatic differences in host tissue or differences in microclimate within the plant canopy). Many experimental studies in plant pathology have investigated variations in the latent period with pathogen or host genotype, or its plasticity in response to climatic factors, such as temperature and humidity (e.g. Shaw, 1990; Davis & Fitt, 1994). Such approaches are relevant, because these factors lie at the corners of the epidemiological triangle (host, pathogen, environment; Zadoks, 1972). Most of these studies focused on a mean latent period with statistical features such as standard error (related to the definition of latent period that is used; see below) in order to reduce the uncertainty of the measure, but rarely on the extrinsic variance, i.e. not due to measurement biases, but due to the interindividual variability or the expression of phenotypic plasticity. Pariaud *et al.* (2012) showed that, even in a clonal lineage population (e.g. *Puccinia triticina*), differences can exist in the latent period within the same pathotypes (pools of individuals with the same combination of qualitative virulence factors).

There are few empirical data about the time periods over which the latent period of a plant pathogen population changes. However, changes have been detected over pluriannual scales in some cases, for example in poplar rust (Pinon & Frey, 2005). Focusing on soilborne plant pathogens, Leclerc *et al.* (2014) also noticed that there is little information about how the incubation period (the time between infection and symptom expression) varies temporally in a pathogen population. Interestingly, in that study, the latent period is fixed at zero. A similar observation could be made for the latent period – very few studies consider the possibility that the latent period of a pathogen population may display variability during an annual epidemic.

The goal here was to highlight key sources of short-term variability in the latent period that cause the length of the latent period to vary within a single epidemic season. To this end, four sources of variability in the development of *Z. tritici* are considered, making use of empirical datasets collected during the first author's own research projects and also a targeted literature review. This fungal disease is particularly suitable for this analysis because the effects of several factors are now well documented. *Zymoseptoria tritici* is a polycyclic, 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 season (Suffert & Sache, 2011). The pathogen population displays a high degree of genetic diversity and there may be considerable phenotypic variability in the latent period between strains (Morais *et al.*, 2015). Wheat has a long growth cycle and infections occur from early autumn to late spring, under the influence of heterogeneous environmental selective pressures driven by abiotic conditions such as temperature (Lovell *et al.*, 2004), but also biotic conditions such as the physiological stage of wheat or its fertilization regime (Robert *et al.*, 2005). As septoria tritici blotch epidemics are polycyclic and result from the integration of many overlapping infection cycles, the latent period is a crucial fitness trait. The latent period is long, facilitating the quantification of any differences by *in planta* experiments and reducing uncertainty in those measurements. Moreover, it may display signs of local adaptation to climatic conditions (Suffert *et al.*, 2015). The four drivers of variability in the latent period considered here are described below, where it is also demonstrated that accounting for such variability can change pathogen dynamics as predicted by mathematical models.

Operational definitions of the latent period can be inconsistent

The latent period is regularly measured in conflicting ways by different plant disease experimenters. This contributes to the variability in the published literature, and makes it impossible to directly compare results. Aligning

experimental procedures to allow for direct comparison between experiments might be assumed to be an important first step, and would be advisable in experiments that are similar to each other. However, complete homogenization is neither possible nor desirable. For example, certain definitions are better adapted than others to particular experimental setups because they make it possible to overcome methodological constraints. 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) 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). Nevertheless, when several lesions rather than a single lesion are considered, particularly when methodological constraints make it necessary (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 often estimated at the scale of a leaf, as the time between inoculation and the appearance of half of the eventual number of sporulating lesions (Shaw, 1990; Lovell *et al.*, 2004). Studies are often conducted by modellers who 'search the literature' for experimental parameters to use. It is therefore recommended that the operational definition used in a particular experiment is considered by modellers before experimental data are used to infer model parameters.

The latent period can vary with fluctuations in host 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) is widely preferred over physical time for assessing and modelling disease development. This preference is frequently seen in studies of septoria tritici blotch. 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.*, 2004) typically corresponds on average to 33 days in early spring (April) but only 22 days in late spring (June) in France (average monthly temperature in Poissy, Yvelines; see https://en.climate-data.org). However, taking into account the impact of temperature in this way is not completely adequate, because relationships between temperature and the efficiency or duration of a given epidemiological process are usually nonlinear and often not even monotonic. Consequently, the latent period, while assessed using thermal time, should not be considered constant in time, particularly if the time-step used for the calculation is large (e.g. daily), for at least two reasons.

First, 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 more difficult to measure than air temperature, but it can be estimated indirectly from soil–vegetation–atmosphere transfer (SVAT) models, including data recorded at standard weather stations (Xiao *et al.*, 2006). Note that, while the focus here is on leaf temperature, due to its relevance to epidemics caused by *Z. tritici*, other environment–organ factors are also likely to apply to other pathogens, such as root- and soilborne pathogens.

Second, 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 the mean temperatures were equal (18 °C) but daily temperature ranges differed (±2 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 & van Bruggen, 1994). The differences in pathogen development between constant and fluctuating environments are partly due to 'rate summation' or the Kaufmann effect, a mathematical consequence of the nonlinear shape of thermal performance curves (TPCs). The length of the latent period under fluctuating temperatures can be predicted by integrating constant-temperature developmental rates over the fluctuating temperature regime (Xu, 1996; Shakya *et al.*, 2015),

$$S(y) = \int_0^y R(T(t))dt,$$

where S is the accumulated development over the time interval $[0, y]$, $T(t)$ is the temperature as a function of time t and $R(T(t))$ is the development rate as a function of temperature. S is dimensionless and defined as zero initially and one at the completion of a process (i.e. appearance of the first pycnidia).

Finally, degree-hours should be preferred over degree-days post-inoculation (ddpi) 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) and controlled (Bernard *et al.*, 2013) conditions. The variability in the latent period between pathogen populations of different geographic origins has never before been characterized in detail. The latent period TPCs presented in Figure 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

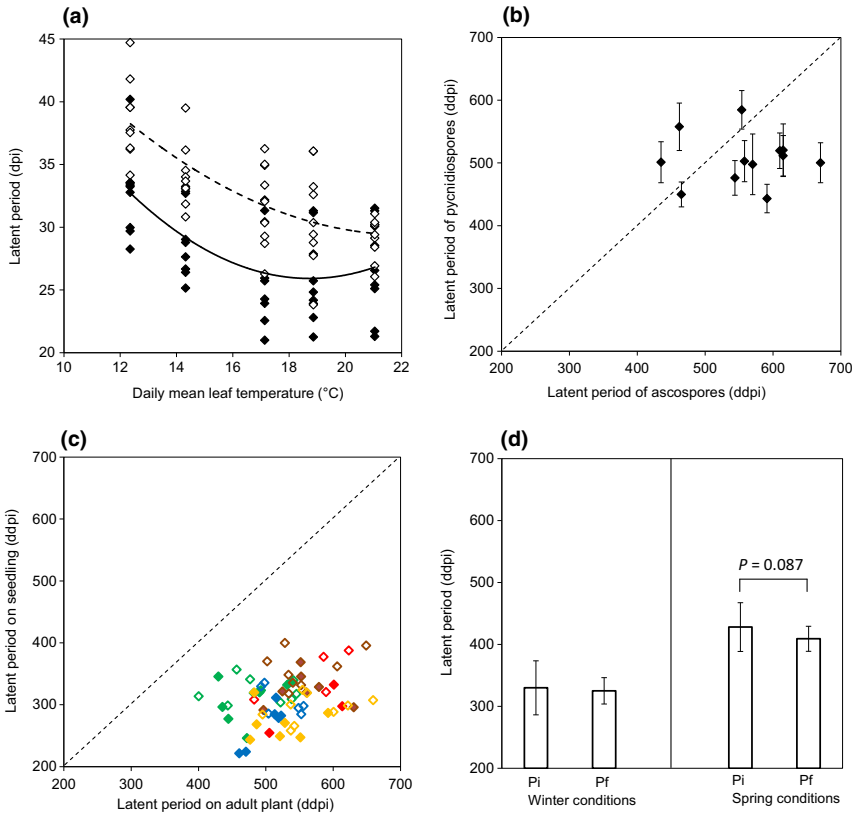

Figure 1 Illustration of four sources of variability in the latent period (expressed in days post-inoculation [dpi] or in degree-days post-inoculation [ddpi] with a base temperature of 0 °C) of the wheat pathogen *Zymoseptoria tritici* over the course of an annual epidemic. The experiments and datasets underlying panels (a)–(d) are independent. The latent period in panels (a), (c) and (d) is the time between inoculation and the appearance of 5% of the maximum area covered by pycnidia, calculated by fitting a Gompertz growth curve to experimental data as described by Suffert *et al.* (2013). In panel (b) the latent period was characterized as the time between inoculation and the appearance of 5% of the maximum number of pycnidia in each individual lesion, as described by Morais *et al.* (2015). (a) Effect of the daily mean wheat leaf temperature on the latent period of two groups of nine *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 adult wheat plants. The thermal performance curve (the quadratic $y = ax^2 + bx + c$, with: a = 0.17, b = −6.28, r^2 = 0.358, optimal temperature = 18.7 °C for Dijon; and a = 0.09, b = −4.10, r^2 = 0.548, optimal temperature = 22.1 °C for Ploudaniel) was adjusted using six replicates per temperature. (b) Length of the latent period of 12 *Z. tritici* isolates (collected from cv. Soissons in Grignon, Paris basin, France) assessed after ascospore and pycnidiospore inoculation on adult wheat 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. This heterogeneity is due to the impossibility of replicating individual inoculation with a given *Z. tritici* genotype using the ascosporic form, in contrast to the conidial form (Morais *et al.*, 2015). Inclusion of replicates for the assessment of the latent period after ascospore inoculation would have made the differences that are currently being displayed far less evident. (c) The latent periods of two groups of nine *Z. tritici* populations (the same isolates as in panel (a); black diamond = Dijon in Burgundy; white diamond = Ploudaniel in Brittany) assessed after pycnidiospore inoculation on wheat seedlings and adult wheat plants cv. Apache for five different wheat cultivars: Apache (triangle/green diamond on-line), common to both Brittany and Burgundy; Altamira (diamond/red diamond on-line) and Paledor (circle/yellow diamond on-line), mostly cultivated in Brittany; Arezzo (cross/blue diamond on-line) and Altigo (square/brown diamond on-line), mostly cultivated in Burgundy. Each point represents the mean value from six replicates. The mean latent period was 298 ± 41 ddpi on seedlings and 535 ± 66 ddpi on adult plants. Such differences should be taken into account carefully, especially for multifactorial modelling purposes, as the latent period definitions were not the same because of methodological constraints: the latent period (5% of the maximum percentage of area covered by pycnidia) was assessed for adult plants by fitting a logistic model (Suffert *et al.*, 2013) to 17 points, but was obtained for seedlings using raw data (5 points) without fitting a model. This difference could explain the low variance in latent period values assessed on seedlings compared to the equivalent measurements on adult plants. (d) The 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 for a fixed spore type (pycnidiospores). See Suffert *et al.* (2015) for further information on the interactions between the effect of plant growth stage and the effect of temperature. Error bars represent the standard deviations.

derived from the TPCs was <19 °C for the isolates from Brittany and >22 °C for the isolates from Burgundy. The effect of temperature on the latent period can differ between pathogen populations expressing local patterns of climatic adaptation.

The latent period is affected by the type 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. However, Morais *et al.* (2015) showed that the latent period of *Z. tritici* was significantly longer (about 60 degree-days, i.e. 3–4 days in late spring) and more variable (standard deviation 68.4 vs 38.0 ddpi) 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 (e.g. Karolewski *et al.*, 2002). For *Z. tritici*, one concrete consequence of this difference is that the mean latent period early in the epidemic, when lesions are predominantly caused by wind-dispersed ascospores, is typically longer than that during the spring epidemic stage, when infections are caused mostly by splash-dispersed pycnidiospores (Suffert & Sache, 2011). This is in contrast to Figure 1d, in which only pycnidiospores are considered. Hypothetical, theoretical distributions of the length of the latent period at different stages of an epidemic, due to the numbers of new lesions induced by different *Z. tritici* spore types, results in the superimposition of two unimodal distributions centred around the mean latent period value of each type of spore (Fig. 2). Note that, in the particular example considered here, ascospore induced infections appear earlier in the epidemic despite being associated with a longer latent period than pycnidiospore induced infections. This is because all primary infections are caused by ascospores, whereas pycnidiospores only cause secondary infections. The resulting distribution may or may not be bimodal, depending on the relative contributions of the two types of spore to the infection. The latent period of a plant pathogen with both sexual and asexual reproduction modes can therefore vary over the course of an epidemic.

The Latent Period Depends On Host Stage And Host Tissue Age

An increase in the latent period with 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 a univocal relationship between wheat seedling and adult plant resistance. This age-related (or ontogenic) resistance is, for example, clearly established in wheat rusts: many resistance genes are expressed in adult plants but not in seedlings (McIntosh *et al.*, 1995). The latent periods of two groups of *Z. tritici* isolates collected in two climatically different regions of France (Brittany and Burgundy) were assessed, on both seedlings and adult plants. A large difference was found between plants of different stages, with a mean latent period of 298 ± 41 ddpi for seedlings and 535 ± 66 ddpi 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; e.g. Bernard *et al.*, 2013; Suffert *et al.*, 2015). The increase in the latent period length with developmental stage (young vs adult plants), 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 can vary over the course of an epidemic.

The latent period is strain-dependent, and therefore affected by competition within a local pathogen population

As mentioned in the introduction, the latent period depends on pathogen genotype. Genetic and phenotypic variability within a local pathogen population may be high or low, according to the inherent structure of the population (clonality vs sexual reproduction that typically leads to high levels of variability). 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). Suffert *et al.* (2015) showed that the mean latent period of *Z. tritici* pycnidiospores can vary during a single annual epidemic: isolates collected on the upper leaf layers of wheat at the end of an epidemic have a shorter latent period than those collected from seedlings very early in the same epidemic. This difference in the latent period between strains, expressed under spring conditions (adult plants, warm temperature) but not under winter conditions (seedlings, cold temperature), suggested that strains with shorter latent periods may be selected during the second part of the epidemic (spring), when the disease is propagated by the upward splash dispersal of spores, although note the limited statistical support here (Fig. 1d; for additional details, see Suffert *et al.*, 2015). During this stage of the epidemic, a short latent period appears to be a key fitness trait conferring a competitive advantage. These conclusions were corroborated by the significant decrease in between-genotype variance for the latent period over the course of the epidemic. The decrease in the mean latent period of a pathogen population is consistent with the increase in other aggressiveness traits recorded for various fungal pathogens after some cycles of asexual reproduction (e.g.

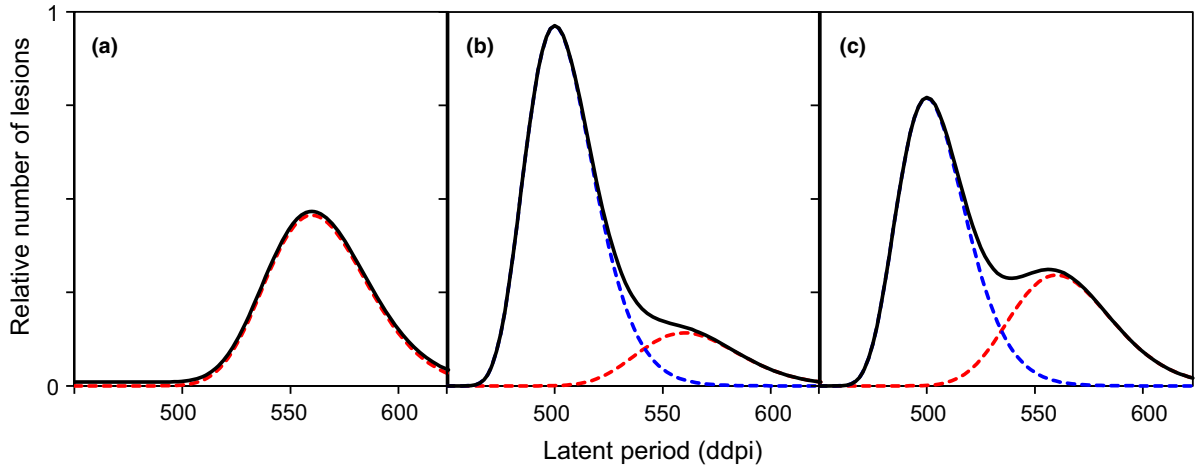

Figure 2 Proposed hypothetical distributions of the relative numbers of new lesions (on wheat plants, per m^2 and per week) with each latent period length, taking into account the nature of the spores at each stage of a *Zymoseptoria tritici* epidemic at early stage of the epidemic in December (a), intermediate stage of the epidemic in April (b) and late stage of the epidemic in June (c). Red dashed lines correspond to ascospore-initiated lesions; blue/dotted on-line lines correspond to pycnidiospore-initiated lesions; solid lines are the cumulative curves. Curves were built with the hypothesis that the mean latent period is 505 ddpi for pycnidiospore infection and 557 ddpi for ascospore infection (Morais *et al.*, 2015). Both latent periods are assumed here to have a gamma distribution, with a similar variance for ascospore and pycnidiospore infections ($\alpha = 23$ and $\beta = 1.3$ for ascospores, $\alpha = 11$ and $\beta = 1.3$ for pycnidiospores). The relative heights of the curves are not derived from experimental values, because such experiments do not exist, and should be considered as an approximate order of magnitude. This order of magnitude is inspired by the relative importance of the two types of spores to the epidemic over the growing season found in different studies based on experimental approaches (Suffert & Sache, 2011; Morais *et al.*, 2015), theoretical approaches (Eriksen *et al.*, 2001) or combined approaches (Duvivier, 2015). The numbers of ascospores trapped by Eriksen *et al.* (2001) and Duvivier (2015) suggest that ascospores can play an important role in late infection from mid-April to mid-June. The results of simulations by Eriksen *et al.* (2001) showed that the proportion of ascospore infection can reach 25% under the most favourable parameter combination. The results of simulations performed by Duvivier (2015), based on three dispersal mechanisms, suggested that 50–58% of infections can be explained by wind-dispersed ascospores.

Newton & McGurk, 1991; Le May *et al.*, 2012). Given the *P*-value in Figure 1d, these results should be interpreted with caution. However, these empirical findings add to the weight of evidence supporting the key conclusion – the latent period can vary over the course of a plant disease epidemic.

Temporal Variability In The Latent Period Impacts Epidemic Development

It has been demonstrated that the latent period can vary over the course of a plant disease epidemic, even within a single season. This was the main goal of this article. However, a key question is then whether or not this variability affects epidemic dynamics, and consequently whether or not temporal changes in the latent period should be included in mathematical modelling studies.

The development of a plant disease epidemic is therefore considered at the field scale, both within a season and over the course of multiple seasons (Fig. 3). The model tracks changes in the number of infected sites during the epidemic, where the term 'site' refers to a unit of plant tissue that can sustain an infection and further infect other plant tissue (Savary & Willocquet, 2014). In this analysis, it was not intended to replicate accurately the dynamics of successive septoria tritici blotch epidemics. Rather, the intention was to use as simple a model as possible to test whether or not latent period variation drives pathogen dynamics that are different to those that might be expected if a constant latent period is assumed. Even in the basic model, it was found that the number of infected sites each season can be different with variable and constant latent periods. This remained true even when the mean latent period averaged over the season was identical in each case (cf. Fig. 3c, d).

The mean length of the latent period in the – potentially more realistic – variable latent period case might be used in a model with a constant mean latent period if measurements of the latent period are taken at random time points throughout a season. However, as shown, this will lead to an incorrect representation of disease dynamics compared to using a model in which the length of the latent period varies temporally during the epidemic.

Discussion

This review has provided empirical evidence to support the suggestion that the mean latent period of a plant pathogen population can vary locally, in the short-term, and that changes in the latent period can impact on the development of epidemics. Consequently, the mean latent period should not automatically be assumed to be

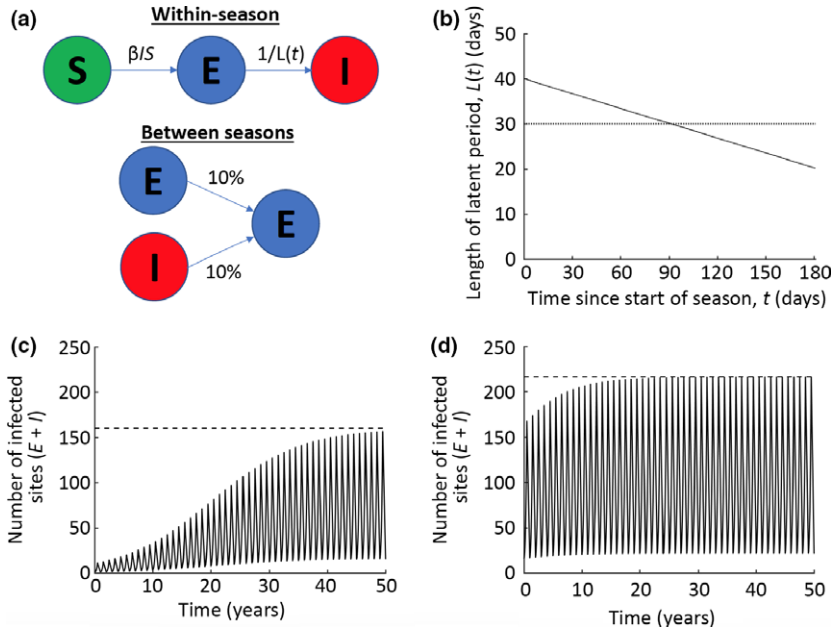

Figure 3 Impact of variability in the latent period on plant pathogen dynamics. An epidemic is modelled using the classic susceptible–exposed–infected (SEI) model in a host population consisting of $S + E + I = 1000$ sites. A site on a leaf can be susceptible (S) i.e. healthy, exposed (E) or infected (I) at any time. Between growing seasons, which lasted 0.5 years each, there were off-seasons of the same length. In each off-season, 10% of infectious sites from the end of the previous growing season were assumed to found the initial infections (in the E class) at the start of the following season. In the case of a disease with dual reproduction modes such as *Zymoseptoria tritici*, this means that 10% of isolates that induced lesions are then involved in sexual reproduction and generate recombinants that have the capability of causing infections the following season. (a) Model schematic. Equations for the within-season model are given by $dS/dt = -\beta IS$, $dE/dt = \beta IS - (1/L(t))E$, $dI/dt = (1/L(t))E$. The function $L(t)$ represents the length of the average latent period of the active pathogen population at time t days since the start of the season. This analysis uses the infection rate parameter value $\beta = 3 \times 10^{-5}$ per day. (b) The latent period lengths that were considered are: (i) variable latent period case, $L(t) = 40 - 0.11t$ days (solid black line); (ii) constant latent period case, $L(t) = 30$ days (dotted black line); in the variable latent period case, the function $L(t)$ is chosen so that the mean value is identical to that in the constant latent period case. Specifically, the length of the latent period decreases linearly between 40 and 20 days, which are values consistent with observed latent periods for *Z. tritici*. (c) The number of infected sites when the latent period varies linearly over the course of the season. The model is solved numerically over 50 seasons, starting from initial conditions $S = 999$, $E = 1$, $I = 0$. The black dashed line represents the number of infected sites at the end of each season in the long-term when the model settles into regular seasonal dynamics. (d) The number of infected sites when the latent period is constant. The model is solved numerically over 50 seasons, starting from initial conditions $S = 999$, $E = 1$, $I = 0$. The black dashed line represents the number of infected sites at the end of each season in the long-term when the model settles into regular seasonal dynamics. The number of infected sites at the end of each season is 35.2% greater using a constant latent period than when a potentially more realistic variable latent period is used (cf. panel c).

constant over the course of annual plant disease epidemics in future studies.

A significant part of the variability in the length of the latent period is due to the interaction between the between-genotype variance within a pathogen population and the expression of its phenotypic plasticity in response to environmental changes; in other words, it is biologically determined. Several empirical arguments justify this assertion as the sources of variation are numerous: daily fluctuations in leaf temperature, nature of the inoculum, host stage or age of host tissues, and selection for aggressiveness traits within a population to name but a few. Some of these sources of variation may have complex, antagonistic impacts. For example, the mean latent period may decrease over the course of an epidemic because of selection for aggressiveness traits driven by biotic or abiotic factors, for instance host stage and temperature (Suffert *et al.*, 2015) in the case of *Z. tritici*. The latent period may also increase at the very end of the epidemic due to changes in the ratio of the two spore types resulting from an increase in sexual reproduction before the end of the growing season (Eriksen *et al.*, 2001; Duvivier, 2015). Shaw (1990) suggested that the increase in the latent period that he observed at high mean temperatures reflected 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 conclusions of Suffert *et al.* (2015, 2018) that seasonal changes can drive short-term selection for fitness traits, recently confirmed by Anne-Lise Boixel (INRA BIOGER, France, personal communication). However, Shaw's results were obtained in field conditions, and are therefore also affected by a number of factors including host growth stage (the latent period

is 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).

The arguments presented here challenge the assumption that the mean latent period of a local pathogen population remains constant over the course of an epidemic. While the direction (decrease or increase) and the biological causes of these variations are difficult to determine, and accurate characterization of variability in the latent period may require collection of additional data, modellers should consider that the mean latent period may not necessarily take a constant value throughout a plant disease outbreak. This may apply to epidemics at a range of spatial or temporal scales – including epidemics at the field or landscape scale, potentially including overlapping cohorts of plants planted in a region. Directional variability in the length of the latent period, driven by biophysical processes such as the four sources of variation identified here, could be built into epidemiological models.

Sources of short-term variability in the latent period could be analysed further and potentially incorporated in the case of *Z. tritici* into at least three types of epidemiological models: (i) forecasting models used to simulate the development of annual epidemics and improve wheat protection strategies, such as taking into account secondary inoculum pressure to determine the optimal timings for effective fungicide sprays (e.g. Audsley *et al.*, 2005; El Jarroudi *et al.*, 2009); (ii) mechanistic models used as research tools for understanding the impacts of different epidemiological parameters and processes in driving infectious disease outbreak dynamics, for example discerning the relative importance of pycnidiospore and ascospore infections (e.g. Eriksen *et al.*, 2001; Duvivier, 2015) or the dynamic interaction between plant architecture impacted by cropping practices (nitrogen fertilization, sowing density) and spore dispersal (Baccar *et al.*, 2011); (iii) eco-evolutionary models over several epidemic seasons in which the latent period might evolve in response to selective pressures, for example thermal variation (Suffert *et al.*, 2015; Anne-Lise Boixel, INRA BIOGER, France, personal communication). There may also be an evolutionary trade-off between intra- and interannual scales (Suffert *et al.*, 2018), or an evolutionary optimum driven for instance by the level of nitrogen fertilization (Précigout *et al.*, 2017).

This review has demonstrated the principle that including directional variability in the latent period, rather than making the common assumption that the mean latent period is constant throughout an epidemic, can change the behaviour of a mathematical model (Fig. 3). The simplest possible model was considered, in which there is a latent period, namely the susceptible–exposed–infected (SEI) model. However, forecasting would of course require a more detailed model adjusted for the specific system under consideration. Alterations might include features such as the spatial distribution of hosts and temporal changes in disease management strategies (control of inoculum sources, varietal diversification to limit adaptation of the pathogen population to the host, etc.). For *Z. tritici* specifically, more detailed models exist and could be used (e.g. Elderfield *et al.*, 2018). However, the concept that variability in the latent period should be considered in future modelling studies has been demonstrated.

Of course, the values of other epidemiological parameters are also likely to vary temporally (e.g. the infection rate and infectious period). In theory, it might be possible to include variability in those factors, as well as to model complex features in detail such as individual lesion growth dynamics and variability between different leaf layers. However, epidemiological modelling requires some simplifications to be made for tractability, and so that the model can be parameterized. Deciding which factors to include is therefore a challenging balancing act – including too little biological detail leads to a model that is unrealistic, yet including too many factors leads to an unparameterizable and intractable model. Here it has been shown not only that the latent period can vary, but also that this variation may alter disease dynamics significantly. As a result, the assumption that the latent period – as well as other epidemiological parameters – is constant in basic models should be considered further in future work. An interesting subsequent study might examine precisely how the exact form of the latent period distribution influences outbreak dynamics, including distributions with different means and variances but also different shapes.

Under some circumstances, including detailed descriptions of the latent period may not in fact increase the accuracy of model predictions. Cunniffe *et al.* (2012) proposed an extension to the generic SEI model, splitting the latent and infectious 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 might not always lead to more accurate results. However, including accurate representations of incubation, latent and infectious periods in models can be extremely important. 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 showed that accurate information about the incubation period distribution could be critical in assessing the current size of an outbreak and the probable efficacy of proposed control interventions.

Using *Z. tritici* as a case study, it has been demonstrated that the mean length of the latent period can vary during plant disease epidemics. Further sources of variability are likely to exist in addition to those considered here. However, it is hoped that this study will prompt more detailed quantification of the variability in the latent period for a wide range of pathogens, as well as more detailed testing of the circumstances in which this variability should be included in modelling studies. It is

contended that this might lead to more accurate characterization of pathogen dynamics, in turn potentially leading to more effective disease management.

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