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Concepts, approaches, and avenues for modelling crop health and crop losses

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ABSTRACT

This article addresses the modelling of crop health and its impact on crop losses, with a special emphasis on plant diseases. Plant disease epidemiological models have many different shapes. We propose a summary of modelling structures for plant disease epidemics, which stem from the concepts of infection rate, of site, of basic infection rate corrected for removals (R_c), and of basic reproductive number (R_0). Crop losses, the quantitative and qualitative impacts of diseases and pests on crop performances, can be expressed along many different dimensions. We focus on yield loss, defined as the difference between the attainable yield and the actual yield, in a production situation. The modelling of yield loss stems from the concept of damage mechanism, which can be applied to the wide range of organisms (including pathogens, weeds, arthropods, or nematodes) that may negatively affect crop growth and performances. Damage mechanisms are incorporated in crop growth models to simulate yield losses. In both fields, epidemiology and crop loss, we discuss the process of model development, including model simplification. We emphasize model simplification as a main avenue towards model genericity. This is especially relevant to enable addressing the diversity of crop pathogens and pests. We also discuss the usefulness of considering differing evaluation criteria depending on the stage of model development, and thus, depending on modelling objectives. We illustrate progress made on two global key crops where model simplification has been critical; rice and wheat. Modelling pests and diseases, and of the yield losses they cause on these two crops, lead us to propose the concept of crop health syndrome as a set of injury functions, each representing the dynamics of an injury (such as, for example, the time-course of an epidemic). Crop health in a given context can be represented by the set of such injury functions, which in turn can be used as drivers for crop loss models.

1. Introduction

What is the importance of crop diseases and pests today? How to define “importance”? – Should “importance” refer only to the amount of crop yield that is lost to disease or pests? These losses may somehow matter to economic fabrics, or to the state of global food security, but by how much? Should the calculation also include the waste of agricultural land, water, fertilizer, human labour, and many other environmental and social resources, due to untaken harvests? Climate change, world population, and globalisation are driving changes in the world's

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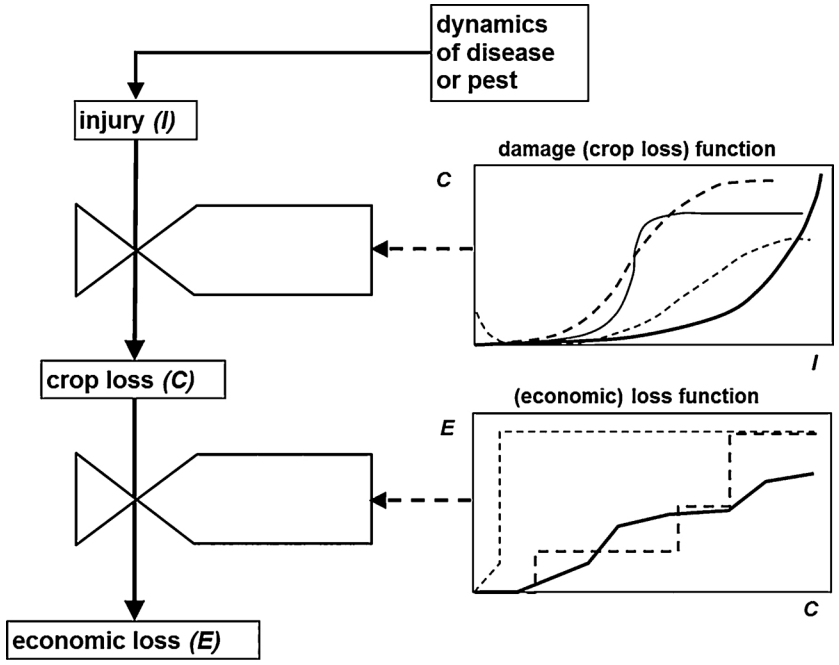

Fig. 1. Diagrammatic relationships between the dynamics of disease or crop pests, injury, crop loss, and economic loss (After Esker et al., 2012, modified).

agricultural systems – to what degree are crop pests and diseases affected? Will the world's most important crops be more vulnerable to pests and diseases tomorrow than they are today, and if so, with what impacts?

These questions have been addressed by many reviews because of their importance to the plant science community. Instead of presenting a compilation of the literature, we propose some elements for an overall methodological framework, we illustrate with a few examples how some of the conceptual difficulties may be addressed, and we highlight some areas where progress is critically needed. Of particular interest are approaches which might bring together very diverse fields of research. In this respect, this article is a position paper, a conceptual roadmap for further thoughts and research.

Despite the long reference list accompanying this article, we conceptually draw strongly on a handful of references. Departing from custom, we feel important to list them here. First is a book chapter by Zadoks and Rabbinge (1985), with insights on the objectives of modelling and the nature of model coupling. Second is a critical review by L.V. Madden (2006) on recent advances in botanical epidemiology. The article by M.J. Jeger et al. (2004) provides both a perspective of modelling of insect-transmitted plant viruses, and a vision on the application of modelling to identify and assess disease control strategies. From the crop modelling standpoint, the reviews by Jones et al. (2001, 2003, 2017) provide an overview of approaches, including the evolution of modelling platforms and the importance of modularity. Two articles by Teng (1981), Teng (1985a) provide critical elements regarding the background of modelling in plant pathology, and key aspects of model evaluation, respectively.

We first consider the modelling of the dynamics of crop harmful agents. We do so from a botanical epidemiology viewpoint, where many questions are shared with other contiguous fields, including entomology or weed science. We look at a few generic questions: What are the main modelling approaches, and to what extent are they connected? What are the biological entities and processes modelled? Beyond the diversity of organisms and interactions considered; what are the common frames and concepts?

In the second section we look at crop losses: the concepts, the definitions, and the approaches to modelling the impacts of pests and diseases on crops. Among the questions we address are: Should crop loss be restricted to yield loss? What are the mechanisms through which harmful agents inflict injuries to growing crops, and how to model them? Can we bring yield loss modelling into the broader framework of natural resource management through the multidimensionality of crop yield (and crop) losses?

In the third section, we address the central question of model evaluation. We step back and look at the nature of processes that are modelled in population dynamics. What are the process-components of plant disease epidemics? What are their spatial scales and time characteristics? We also consider the variable objectives modellers may have, and ask: What are the suitable approaches to model evaluation? We link these questions to the fundamental ones: What are the main purposes of modelling pests or diseases, and crop losses? To conclude this section we put forward an already existing framework to organize these objectives.

In a last section we address the question of linking disease (or pest) models to crop models. We revisit the earlier questions, now from the perspective of a “crop-pest” linkage: What processes should be retained? Will spatial scales or time coefficients of processes constitute insurmountable issues? We illustrate recent progress made on two global key crops, rice and wheat, where model simplification has been critical to address the diversity of crop pathogens and pests. Modelling of pests and diseases, and of the yield losses they cause on these two crops, lead us to propose the notion of crop health syndrome, which can help synthesise the diversity of injuries a crop may encounter during its cycle. We finally discuss a few elements that the scientific community might be considering, in order to generate new insights in crop health, present and future.

2. Modelling pests and diseases – frameworks of thoughts

2.1. Disease and pest injuries; the resulting crop losses

The phrase ‘modelling pests and diseases’ covers a considerable range of objects and questions. A useful first step is to distinguish the causes and their consequences, that is, pests and diseases on the one hand, and the crop losses they cause on the other. The framework discussed by Esker et al. (Esker et al., 2012; Zadoks and Rabbinge, 1985; Teng, 1985b) summarizes the cascade of relationships (Fig. 1)

that successively link the dynamics of crop pests and diseases, causing injuries (*I*) to the crop physiology, development, or growth, leading to crop losses (*C*), and ultimately to economic losses (*E*).

In a given production situation, a given epidemic (a particular pest outbreak) may, or may not, lead to levels of crop injury that are sufficient to cause crop losses. In turn, a given level of crop loss may, or may not, be high enough to cause economic losses. Thus, not all epidemics (pest outbreaks) lead to crop losses; and not all crop losses result in economic losses. The translations of injury into crop loss, and of crop loss into economic loss, depend on transition functions, which have been respectively called crop loss (or damage) function and (economic) loss function (Fig. 1). The different damage functions of Fig. 1 can be associated with different diseases or pests, or with different attainable yields. Conversely, the different loss functions of Fig. 1 can be associated with different diseases or pests, or with different economic environments.

These two functions respectively correspond to thresholds for injuries to cause crop loss, and for crop losses to cause economic losses. Different shapes for these functions are sketched in Fig. 1 to show their variability (Zadoks and Rabbinge, 1985; Esker et al., 2012). Variation in the damage function incorporates, for instance, elements pertaining to the crop physiology, to the crop yield that would be achieved in the absence of injury, or to the occurrence of injuries caused by multiple diseases or pests. The (economic) loss function incorporates other elements, such as the investment to the crop, the crop value and its variability according to markets, accessibility to markets, and numerous opportunity costs. From a management standpoint, this threshold theory (Zadoks and Rabbinge, 1985) constitutes the basis of reasoning for integrated pest management (IPM, Stern et al., 1957; Zadoks and Schein, 1979; Jeger, 2000). From a modelling standpoint, this framework enables the analysis and linkage of the three systems components:

(1) the dynamics of pests and disease leading to injuries,
(2) the dynamics of injuries and their effects on crop growth and development that lead to crop losses, and
(3) the complex relationships between crop losses and economic losses.

In the context of this article, we focus on the two first points.

2.2. Components of models in botanical epidemiology

The review by Butt and Royle (1980) provides a very useful overview of the diversity of processes, entities, and concepts that botanical epidemiology addresses. A first element is the process of infection (Butt and Royle, 1980), which implicitly requires the definition of *sites*. Epidemiological models consider a crop of host plants as a population of sites with two possible states: healthy or infected. Infection is represented by the flow of sites from the healthy to the infected state. From an epidemiological standpoint, a site is thus the host unit where, for a given disease, infection may take place, and produce a lesion. A *lesion*, in turn, is an infected site where propagules of the pathogen may be produced (Van der Plank, 1963; Zadoks, 1971; Djurle and Yuen, 1991; Savary et al., 1990, 2012, 2015).

The nature of the site chosen to represent individual host units in a population (i.e.; a crop) depends on the disease considered. For instance, entire plants constitute suitable sites when dealing with a systemic disease; by contrast, a shoot, or a fraction of leaf area, or an individual fruit, are more suitable sites when dealing with a disease caused by a shoot-restricted, a leaf spotting, or a fruit-infecting pathogen, respectively. The choice of a site is one critical, yet often implicit, step in the definition of the epidemiological system to be modelled. The same model structure involving different site sizes may however be used for different versions of an epidemiological model. In the case of rice diseases, different site sizes may successively be considered for different diseases (Savary et al., 2012): disease-sites on a leaf (leaf blast and rice brown spot, two fungal diseases), whole leaves (bacterial leaf blight), tillers (sheath blight), or whole plants (rice tungro, a viral disease).

The succession of (sub-)processes following infection in an epidemiological process can be described as an infection chain (Kranz, 1974). Depending on the disease considered, elements of this chain include the production of propagules; and their liberation, transport, and deposition. Two time delays play critical roles in the dynamic of epidemics (Van der Plank, 1963; Zadoks and Schein, 1979; Madden et al., 2007): the duration between infection and production of propagules (latency period), and the duration of propagule production by an infected and infectious site (infectious period). As an epidemic unfolds, the increase in the number of infected sites enables the build-up of inoculum. Inoculum has a vast diversity of forms across plant pathosystems, from propagules that are specialized for survival, to short-lived ones that may enable rapid disease spread, or simply infected sites that may become infectious. The dynamics of a plant disease epidemic may be considered over the duration of a single growing season, or over many successive growing seasons. In that latter case, a polyetic process (Zadoks and Schein, 1979) is considered, where the dynamics of an epidemic in a given growing season depends on the epidemic of the preceding one: the inoculum produced in the previous season enables the carry-over of disease.

Vectored plant diseases, such as those caused by insect-transmitted viruses, represent a particular group of plant disease epidemics. However, the above terminology applies. Plant virus epidemiology focuses on systems attributes and components such as virus transmission, vector activity and behaviour, and vector immigration and emigration (Madden et al., 2000; Jeger et al., 2004). These pathosystems involve two infection processes by the virus; that of the host plant and that of the insect vector. Yet, the generic concepts of lesion and propagule apply to these systems, where they are respectively represented by a (systematically infected) whole host plant (Van der Plank, 1963), and a viruliferous vector.

2.3. Epidemiological modelling approaches in botanical epidemiology

2.3.1. Initial models

The following general form to summarize dynamic models in plant disease epidemiology was proposed by Madden (1980):

$$dx/dt = g\ f(x)\ h(x) \quad (1)$$

where:

- x represents the amount of disease in the host population (often, the fraction of diseased host tissue),
- t is time,
- g (often, a constant) is the apparent rate of disease increase,
- f(x) represents the multiplication of disease, especially, the re-mobilization of inoculum from infected tissues, and
- h(x) represents both the dynamics of host tissues and the consequences of disease on the host population, especially in terms of tissues that are still healthy, and thus available to new infections.

Eq. (1) is a generalisation of the models discussed by Van der Plank (1963) for

- exponential [f(x) = x; h(x) = 1],
- monomolecular [f(x) = 1; h(x) = 1-x], and
- logistic [f(x) = x; h(x) = 1-x] disease increase. Eq. (1) also embeds a fourth model, where:
- f(x) depends on infectious tissues at t; and
- h(x) depends on healthy tissues at t.

This fourth model corresponds to the integro-differential equation (Van der Plank, 1963) to represent the very important class of plant

disease epidemics where secondary infections occur (i.e. polycyclic epidemics). This model may be written as:

$$dx/dt = R_c\ f(x, p, i)\ g(x), \quad (2)$$

where:

- p and i represent the latency and infectious periods, respectively, and
- R_c is the basic infection rate corrected for removals (Van der Plank, 1963).

In this model, R_c has a particular weight, and its epidemiological meaning and dimensions need explanation. Following Van der Plank (1963) and Zadoks and Schein (1979), R_c represents the combination of two sets of processes; 1) the production of propagules that generate new infections, and 2) the efficiency through which each effective propagule establishes an infection. One can write:

$$R_c = N\ E, \quad (3)$$

where

- N is the number of effective propagules per unit time, that is to say, the number of propagules (dimension: $[N_{prop}]$) that are actually produced and dispersed per infectious lesion (dimension: $[N_{INF}]$), and which actually encounter host tissues (irrespective of their state, healthy or not) with a given probability (dimension: [1]), per unit time (dimension: [T]). The dimension of N therefore is: $[N_{prop}\ N_{INF}{}^{-1}\ T^{-1}]$.
- E is the infection efficiency (Butt and Royle, 1980), that is, the number of new (latent) lesions (dimension: $[N_{LAT}]$) generated per individual (effective) propagule (dimension: $[N_{prop}]$), with dimension: $[N_{LAT}\ N_{prop}{}^{-1}]$.

As a result, R_c, the daily flow of new (latent) lesions generated per existing (infectious) lesion has dimension: $[N_{prop}\ N_{INF}{}^{-1}\ T^{-1}]\ [N_{LAT}\ N_{prop}{}^{-1}]$, that is: $[N_{LAT}\ N_{INF}{}^{-1}\ T^{-1}]$.

2.3.2. Mechanistic simulation models for polycyclic epidemics

While building upon the foundations laid by Van der Plank (1963), the form of Eq. (1) synthesizes much of the modelling in plant disease epidemiology. Considering x, a first step is determining the way to represent disease in a population. One approach follows Van der Plank (1963), and considers proportions of host tissues (or host sites); another consists of sites as numbers. A second step consists of considering different, non-overlapping, categories of diseased sites, for instance, latent (infected but not yet infectious) sites, infectious sites, or removed (infected, but no longer infectious) sites. A third step concerns h(x), the variation in host tissues. Host (crop) growth, or more generally the fluctuation of host tissue associated with growth and physiological senescence, may be incorporated in h(x) in a summarized, or more complex, manner. The effect of disease on the amount of host tissues may also be introduced. Considering the meaning of 'disease multiplication' in Eq. (1) constitutes a fourth step which leads to a number of critical questions, because f(x) lumps together so many processes: the production of propagules, their dispersal, their survival and their efficiency, and the infection process itself. Expanding the f(x) term in Eq. (1) leads to issues pertaining to the spatial heterogeneity of host sites properties, and to pathogen survival and dispersal. This term also embeds the variability of the environment both physical (e.g. climate), and biological (host plant resistance, antagonists and natural enemies).

An equally generic and useful way of considering these questions in epidemiological modelling is via the concepts of contact distribution, time kernel, and gross reproduction (also referred to as the basic reproductive number), R_0 (Van den Bosch et al., 1988). These concepts respectively correspond to the spatial dispersion of propagules, to the production of inoculum over time by infected and infectious sites, and to the total number of infected sites associated with a single infectious site surrounded by healthy sites only (Madden et al., 2007). We provide below a short series of examples, beyond Van der Plank's models, illustrating how these questions have been addressed.

The mechanistic simulation model developed by Zadoks (1971) represents a key step to numerically integrate the integro-differential equation for polycyclic epidemics developed by Van der Plank. This model (1) considers numbers of sites (and not proportions of sites), (2) that are categorized in four non-overlapping classes (healthy, latent, infectious, and removed sites) representing state variables, (3) within a specified system, where space (i.e. 1 m^2) limits and a time step (i.e. 1 day) characteristic are explicitly set. The agricultural reference of the system is wheat stripe (yellow) rust, but simulations may also pertain (parameters and outputs) to wheat leaf (brown) rust or potato late blight. The approach allows for a quantitative assessment of the effects of:

(1) variable infectious period durations, i,
(2) variable latency period durations, p,
(3) variation in R_c, the relative rate of infection corrected for removals,
(4) variation in the host carrying capacity (i.e. number of healthy sites),
(5) the physical environment (daily variation of dew formation, daily variation in temperature),
(6) a chemical (e.g., a fungicide protectant, at variable levels of efficacy on the rate of infection, and for variable periods of time),

on disease progress curves (Zadoks, 1971; Zadoks and Schein, 1979). Hypotheses can be addressed through this sensitivity analysis, such as the epidemiological consequences of long infectious periods (their survival value for the pathogen), as well as of disruptions caused by environmental conditions that would temporarily be unfavourable (extended latency period, strongly reduced rate of infection) to the pathogen. It also generates insights on the potential effects of components of partial resistance using simulations to guide breeding for host plant resistance.

Work on this modelling structure has been expanded in several directions. We briefly discuss below the spatial work it led to. It also provided the background for much experimental work and several modelling applications (e.g., Teng, 1985a; Savary et al., 1990; Djurle and Yuen, 1991; Rossi et al., 1997). Luo and Zeng (1995) independently used an approach very similar to that of Zadoks (1971), with a focus on the modelling of partial host plant resistance. Their work entailed very detailed experimental work on monocyclic processes in controlled conditions, and heavy field testing of simulation outputs. Their model, SRESM (Slow-Rusting Epidemic Simulation Model), which incorporates stochastic elements to account for variation about mean values of the partial resistance components, led to conclusions in close agreement with the simulations discussed by Zadoks (1971). Because of its stochastic features, SRESM further enabled Luo and Zeng (1995) to conduct statistical analyses of simulation outputs, constituting in effect one of the first simulated experiments (Teng, 1985a) in this field.

2.3.3. Linked differential equation models

The use of linked differential equations represents a major current approach to address the modelling of plant disease epidemics (Madden et al., 2007). In his review, Madden (2006) summarizes the simplicity and flexibility of the approach with a four-equation differential model:

$$\begin{aligned} dH/dt &= -\beta HI \\ dL/dt &= \beta HI - \omega L \\ dI/dt &= \omega L - \mu I \\ dR/dt &= \mu I \end{aligned} \quad (4)$$

where:

– H, L, I, and R are densities of healthy, latent, infectious, and removed host plant entities,
– ω is the inverse of the mean latency period duration,
– μ is the inverse of the mean infectious period, and
– β is the per capita transmission rate
– while H_0 is the sum H + L + I + R.

In this model, β obviously carries a particular weight. It is useful to briefly link this model with the former modelling approaches. The term 'per capita transmission rate' is borrowed from medical epidemiology (Madden et al., 2007), and is the product of three terms:

$$\beta = \alpha\ \theta\ \psi, \quad (5)$$

where (Madden et al., 2007):

– α is the amounts of propagules produced per unit time by each infectious entity (dimension: $[N_{prop}\ N_{INF}{}^{-1}\ T^{-1}]$),
– θ is the 'probability' (dimension: $[N_H{}^{-1}]$) of a propagule reaching a healthy entity, and
– ψ is the fraction of propagules that may lead to an infection (dimension: $[N_{LAT}\ N_{prop}{}^{-1}]$).

As a result, β has dimension:
$[N_{prop}\ N_{INF}{}^{-1}\ T^{-1}]\ [N_H{}^{-1}]\ [N_{LAT}\ N_{prop}{}^{-1}]$,
that is: $[N_{LAT}\ N_{INF}{}^{-1}\ N_H{}^{-1}\ T^{-1}]$.

In plant disease epidemiology words, the per capita transition rate β therefore expresses the number of newly infected (thus, latent) host entities per infectious entities, per healthy entities, per unit time, and the product $\beta\ H_0$ is analogous to the basic infection rate corrected for removals, R_c. Furthermore, R_0 can be expressed as $\beta\ H_0/\mu$, and is analogous to the product R_c i. Compared to the logistic model discussed by Van der Plank (1963), the first line of the set of differential Eq. (4) above therefore expresses in a different way the flow of infections occurring in a pathosystem, where new infections may constantly (within the limits of the model) occur, and where delays for the latency and the infectious periods are explicitly expressed.

The model represented by the set of differential Eq. (4) above can be expanded to incorporate a number of epidemiological features (Madden, 2006), such as:

(1) the dynamics of primary infections, through which the density of healthy entities declines (equation for dH/dt), and the density of latent entities increases (equation for dL/dt);
(2) host mortality, with a death rate parameter introduced in the equations for the four components, H, L, I, and R; or
(3) host growth, with a growth rate incorporated in the equation for H.

This modelling approach has many applications. An important one is the modelling of vector-borne epidemics, with two steps:

– first, the replacement of the first equation: $dH/dt = -\ \beta\ H\ I$ in the system of Eq. (4) above by:

$$dH/dt = -\ \beta\ H\ Z, \quad (6)$$

where Z is the density of infective vectors, and

– second, a new set of differential equations to represent the dynamics of the density of vectors, healthy, latent, and infective (Madden et al., 2000; Jeger et al., 2004).

This approach is conceptually very close (Madden, 2006) to the modelling framework developed by Gilligan (1990, 1994) for diseases caused by soil-borne pathogens, and more generally, for epidemics where a dual process of infection occurs (e.g., from a variable primary inoculum, and a dynamic secondary inoculum), such as rice sheath blight (Savary et al., 1997).

2.3.4. Diversity of modelling approaches and of objectives in botanical epidemiology

In this section, our objective is only to provide an overview of the diversity of approaches and objectives in botanical epidemiology.

- Spatial aspects are critical in the understanding of plant disease epidemics. One important step is materialized by the EPIMUL framework (Kampmeijer and Zadoks, 1977), where a mechanistic simulation model is operated within an explicit representation of space. The framework represents plant disease epidemics in space using a very simplified representation of disease spread. The framework also addresses fundamental questions regarding the adaptation of pathogens to host plant resistances, and practical questions regarding the genetic vulnerability (Zadoks and Kampmeijer, 1977) of crops to plant pathogens. This framework has been extended considerably, regarding the sizes and spatial distribution of genotypic area units of host plant resistances (Mundt et al., 1986) or the implications of specific epidemiological characteristics (Lannou et al., 1994), and their effects on epidemiological patterns.
- The genetic structure of plant pathogens and the types of reproduction (Eriksen et al., 2001) are seldom explicitly incorporated in simulation models. However, the multiple linkages within the matrix of pathosystem components: [physical space – host genetic diversity – pathogen variability – pathogen dispersal] constitutes a major research theme (Garrett and Mundt, 1999) where elements such as the nature of host plant resistance, the genetic variability of the pathogen, the modes of pathogen reproduction, the modes of pathogen dispersal, the shape and size of host plants, landscape patterns and diversity, are jointly investigated. A range of modelling approaches (e.g., matrix population models) is being used to address these questions (e.g., Papaïx et al., 2014).
- The implications of global changes, including especially climate change, on plant pathosystems have become important aspects for plant disease epidemiology. Questions (Garrett et al., 2006; Chakraborty and Newton, 2011) include: the effects of climate change on the life cycles of pathogens (duration, number of cycles, fraction of sexual reproduction), the implications of climate change on host-pathogen interactions and gene (host and pathogen) expression and physiology; epidemiological and biological characteristics that are required to assess the risks of invasion and persistence (Gilligan and Van den Bosch, 2008) of plant pathogens. Epidemiological modelling is necessary to explore these questions, identify knowledge gaps, and guide research.

2.4. Modelling structures in plant disease epidemiology

A number of different modelling structures have been developed to address different pathosystems. Fig. 2 is a graphical summary, using the conventional symbols of systems analysis. These symbols are convenient to convey model structures; their use does not imply that only mechanistic simulation models are considered: the above discussion indicates that there are many bridges between, for example, linked differential equation, matrix population, and mechanistic simulation approaches. Fig. 2A summarizes models for polycyclic epidemics, with four categories of sites: healthy (H), latent (L), infectious (I), and removed (R) sites. This structure mirrors the linked differential equations described in Eq. (4) above. In this case, the rate of infection (RI) is initially fed from the primary inoculum (P), while RI is then mainly fed by a secondary inoculum represented as a feed-back loop from I to RI, therefore capturing the polycyclic nature of epidemics. The other figures represent only two categories of sites: healthy and infected. Fig. 2B depicts the structure of epidemics where only the primary inoculum (P) determines disease progress. Fig. 2C accounts for epidemics with dual,

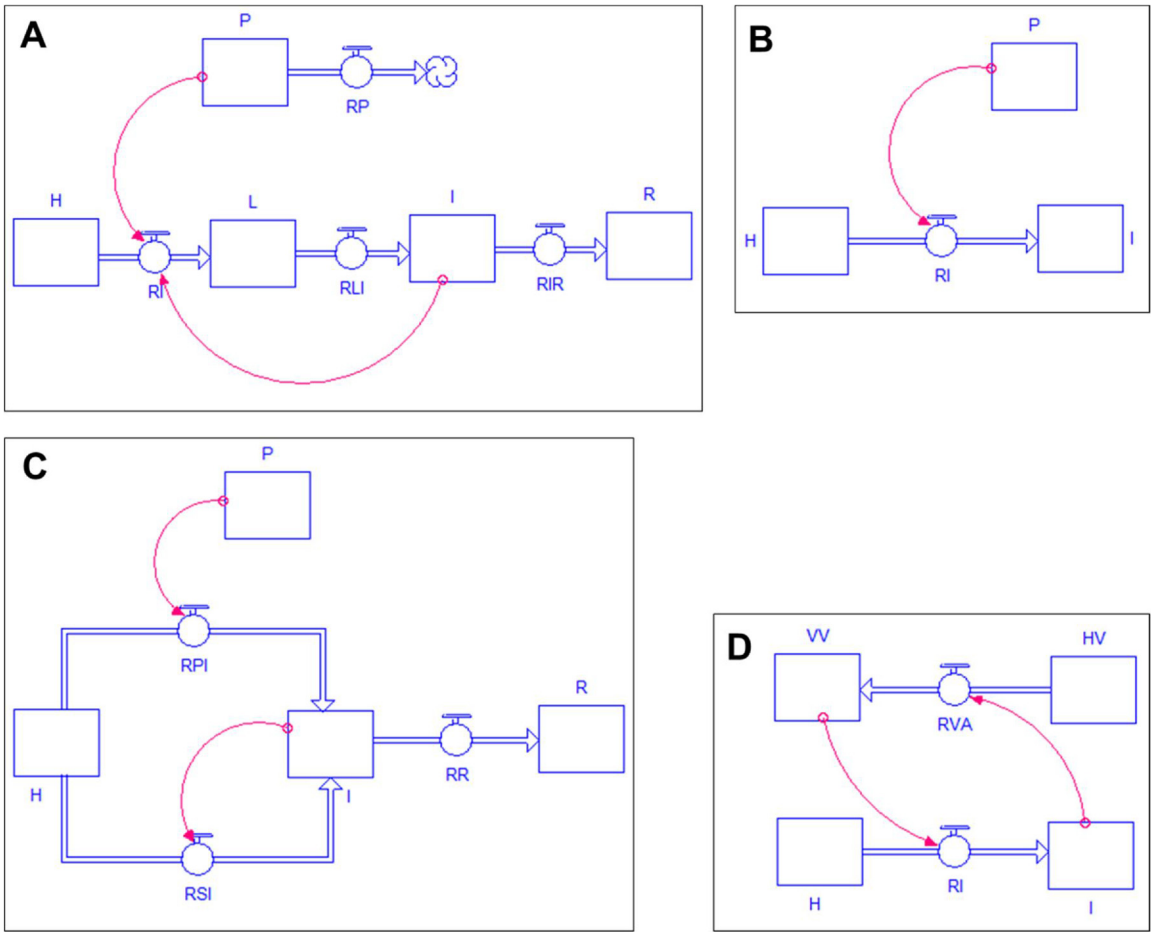

Fig. 2. Some typical modelling structures used in botanical epidemiology.
In all model structures, the host population is expressed as sites. Rectangles represent quantities and double arrows represent flows of quantities. Only a few essential quantitative relationships are indicated (red arrows).
A: Polycyclic disease. P: primary inoculum; H: healthy sites; L: latent sites; R: removed sites; RP: rate of elimination of primary inoculum; RI: rate of infection; RLI: rate of transfer from latent to infectious; RIR: rate of removal of infectious sites.
B: Monocyclic epidemic. P: primary inoculum; H: healthy sites; I: infected sites; RI: rate of infection.
C: Epidemic with dual (primary and secondary) infection processes. P: primary inoculum; H: healthy sites; I: infected sites; R: removed sites; RPI: rate of primary infection; RSI: rate of secondary infection.
D: Epidemic of vector-borne disease. VV: viruliferous (or infectious) vector; HV: healthy vector; H: healthy site; I: infectious site; RVA: rate of virus(or pathogen) acquisition; RI: rate of infection.

primary (RPI) and secondary (RSI) infections which both govern the increase in infected sites over time. Fig. 2D represents the case of epidemics caused by vector-borne diseases, where two (very simplified) chains of processes are shown, for the vector (healthy, HV, or infectious VV) and for the host crop (healthy, H or infected and infectious, I). The two chains are coupled via the rates of pathogen acquisition by the vector (RVA) and of infection (RI) of host plant entities. As discussed in the previous sections, there are many ways to expand these structures in order to account for additional processes, or to modify them and develop hybrid structures among these patterns.

3. Modelling crop losses caused by crop pests and diseases

The diversity of organisms, including pathogens, weeds, insects, or nematodes, which affect negatively crop performances is extremely large. Generic concepts, which may be applied to very different biological systems, are critical to progress in this area. In this section, we especially highlight how (1) well-established definitions of crop loss and yield losses, (2) the concept of “pest-crop” coupler, and (3) the concept of damage mechanism, have brought about a cohesive framework to model yield losses.

3.1. Definition of crop losses and of yield losses

Crop loss due to plant diseases is the “raison d’être” for plant pathology (James, 1974). The term ‘crop loss’ includes qualitative and quantitative losses (e.g., Zadoks and Schein, 1979; Savary et al., 2006). We focus in this section on quantitative yield losses caused by crop harmful organisms: animal pests, plant pathogens, and weeds. The study of yield losses is grounded on a framework which was derived from production ecology (van Ittersum and Rabbinge, 1997), and which in turn borrowed concepts from many works, including Chiarappa (1971), and Zadoks and Schein (1979). This framework considers three different levels of crop growth and yield, and has been successfully used by a number of scientists with very diverse objectives (Chiarappa, 1971; Zadoks and Schein, 1979; Rabbinge et al., 1989; Campbell and Madden, 1990; Rabbinge 1993; van Ittersum and Rabbinge, 1997; Savary et al., 2006; Madden et al., 2007; Savary and Willocquet, 2014).

The three yield levels considered in this framework are:

- The potential (or theoretical) yield, which is defined by radiation, temperature, and the genetic characteristics of the variety grown.
- The attainable yield, which is determined by the above defining

Table 1
A typology[a] of damage mechanisms caused by pests, weeds, and disease.

Damage mechanism	Physiological effect	Effect in a crop growth model	Examples of pests, pathogens, weeds
Light stealer	Reduces the intercepted radiation	Reduces the green LAI	Pathogens producing lesions on leaves, weeds
Leaf senescence accelerator	Increases leaf senescence, causes defoliation	Reduces leaf biomass by increasing the rate of leaf senescence	Foliar pathogens such as leaf spotting pathogens, downy mildews
Tissue consumer	Reduces the tissue biomass	Outflows from biomasses of the injured organs	Defoliating insects
Stand reducer	Reduces the number and biomass of plants	Reduces biomass of all organs	Damping-off fungi
Photosynthetic Rate reducer	Reduces the rate of carbon uptake	Reduces the RUE	Viruses, root-infecting pests, stem infecting pests, some foliar pathogens, weeds
Turgor reducer	Disrupts xylem and phloem transport	Reduces the RUE, accelerates leaf senescence	Vascular, wilt pathogens
Assimilate sapper	Removes soluble assimilates from host	Outflows assimilates from the pool of assimilates	Sucking insects, e.g. aphids, some plant hoppers, biotrophic fungi exporting assimilates from host cells

[a] Rabbinge and Vereyken, 1980; Rabbinge and Rijsdijk, 1981; Boote et al., 1983.

factors, and limited by water and nutrients.

- The actual yield, which is determined by the above defining and limiting factors, on which a number of yield-reducing factors are superimposed: plant pathogens, animal pests, weeds, and other factors such as pollutants and calamities.

Using this framework of three yield levels, yield loss is defined as the difference between the actual yield and the attainable yield. The actual yield is *de facto* harvested in a given field, whereas the attainable yield is the yield that would have been achieved if the same crop had not been exposed to reducing factors. The attainable yield is the reference yield in studies focusing on yield losses, and the difference between the attainable and the actual yield is the international yardstick to measure progress (Zadoks, 1981). Because it involves the combination of defining factors and limiting factors of crop production, the attainable yield is a reflection of a production situation (Rabbinge et al., 1989; Savary et al., 2006).

3.2. Multidimensionality of crop losses and its implications on systems performances

The typical dimensions for measuring quantitative crop loss (i.e., yield loss) is biomass (dimension: [M]), or biomass per production area [$M.L^{-2}$]. Yet, the untaken harvest (Ordish, 1952) might be scaled along a number of other dimensions. These include labour time [T], energy ([$M.L^2.T^{-2}$]; spent into e.g., fertilizer, or traction), money [$] as well as in the dimensions of environmental resources (topsoil, water) invested into agricultural production. Considering the multidimensionality of crop losses also brings about important questions about the role of crop health in relation to ecosystem services (Cheatham et al., 2009). The concept of crop loss, encompassing yield and quality reductions, actually is very current, dealing with very pressing questions (e.g., FAO, 2007).

3.3. The crop seen from the pathogen or pest perspective, and the concept of coupler

The previous discussion on epidemiological modelling did not consider in much detail the crop where disease, and also animal pests and weeds, may develop. The physiological state where the crop is, its status with respect to nutrients, water, and weather has a strong effect on the dynamics of pests and diseases, and in their translation into injuries (Zadoks and Rabbinge, 1985). This has been quantitatively documented in detail on both arthropod pests and plant diseases (Rabbinge et al., 1989; Rabbinge, 1993).

Elements of the crop canopy architecture may also affect dynamics. For plant pathogens (Zadoks and Rabbinge, 1985), one may for instance consider the leaf layer area index (Djurle and Yuen, 1991), the date (expressed in time or degree days) of appearance and disappearance of successive leaves, or the leaf angle, which may affect epidemiological components such as the infection efficiency or the sporulation intensity. Linking very detailed epidemiological models with very detailed crop growth models has been considered for a long time. Zadoks and Rabbinge (1985) provide the concept of coupler, which can be very useful to link components of a model with variables that have different dimensions: leaf area (with lesions), [L^2], and fungal spores, [N_{spore}]; or plant biomass, [M] and aphid numbers [N_{insect}].

The effect of the crop physiology on the life cycle of animal pests and plant pathogens is a well-established, documented fact. The question was once whether one can technically model in a dynamic and mechanistic fashion these effects on population dynamics, and ultimately, on injuries to the growing crop; but the question is still asked of whether and when this is necessary. We address this question below.

3.4. Damage mechanisms as disease-to-Crop coupling instruments

Switching perspective, modellers also look at pests and diseases from the plant standpoint. Table 1 reproduces a typology of damage mechanisms, as defined by Boote et al. (1983). This typology is supported by a very large series of different studies, especially for plant pathogens and insects (Rabbinge and Vereyken, 1980; Rabbinge and Rijsdijk, 1981). The unifying concept of damage mechanism is the basis of the coupling of crop models with pest and disease models (e.g., Pinnschmidt et al., 1995; Luo et al., 1997), or with pest and disease driving functions. This is addressed in the later part of this article.

3.5. Examples of approaches in modelling crop losses

The concepts of damage mechanisms and coupler were implemented towards the end of the last century. These concepts enabled developing agrophysiological models that incorporate damage mechanisms, to reflect the physiological effects of diseases and pests on crop growth and yield build-up processes. General features of the studies listed here include: (1) a very strong reliance on detailed field experiments designed to quantify yield losses, and monitor processes; (2) the importance of experimental quantification of physiological processes, especially meant to measure damage mechanisms and their parameters; and (3) the use of mechanistic simulation modelling, involving variable levels of detail, to assess the overall effects of damage mechanisms, their relevance and their contributions to measured and simulated reductions in crop performances. One overall conclusion these examples lead to is that the concept of generic damage

mechanism (and the list of Table 1) developed by Rabbinge and Vereyken (1980); Rabbinge and Rijsdijk (1981) and Boote et al. (1983) constitutes a robust basis to represent "pest" → crop relationships. The following (non-comprehensive) list provides some highlights of these efforts.

- The effects of diseases and insects on crop growth and yield build-up were analysed and modelled in a number of cases, for example on aphids on wheat (Rossing, 1991), rice blast (Bastiaans, 1993), groundnut rust (Savary et al., 1990), rice weeds (Graf et al., 1990; Justeen and Tammes, 1992), crop weeds (Kropff and van Laar, 1993), verticillium wilt in cotton (Gutierrez et al., 1983), using models with various levels of complexity (Rossing et al., 1995). These studies allowed the quantification and analysis of the damage mechanisms involved in each system and the assessment of their relative impact on growth and yield. This led to modelling analyses in view of a better understanding of systems' behaviour, and also provided a basis for strategic decisions in disease and pest management.
- Modelling yield losses also addressed the effects of partial resistance on yield loss reduction, as for example in the case of wheat leaf rust (Van Roermund and Spitters, 1990) and potato late blight (Van Oijen, 1992).
- Johnson (1992) incorporated the effects of multiple pests in a crop growth and yield model, considering the effects of a foliar disease (early blight), a soil borne disease (verticillium wilt) and an insect pest (leafhopper) on potato (Johnson, 1992). Multiple diseases were further considered in the case of rice, using the CERES model (Pinnschmidt et al., 1995).
- The effect of photosynthesis impairing around foliar lesions was measured and modelled as the concept of the virtual lesion (Bastiaans, 1991), which represents the area around a (visible) lesion where photosynthesis does not occur. This concept was applied to quantify and analyse the effects of virtual lesions in several foliar diseases (e.g., Bassanezi et al., 2001; Robert et al., 2004), but was actually seldom integrated into crop growth models incorporating the effects of diseases on crop growth and yield losses, as it was, e.g. in the case of rice blast (Luo et al., 1997).
- Detailed, modular crop growth models primarily designed to address potential and attainable growth were recently coupled to diseases and pests models using simplified (Whish et al., 2015) or complex damage mechanisms (Bregaglio and Donatelli, 2015).

4. Model evolution and model evaluation

4.1. Overview of purposes in crop health modelling

Fig. 3 offers a simplified view of objectives in plant disease epidemiology. We believe this view is shared by the disciplines dealing with crop health in general. A first objective is to understand epidemics (Fig. 3). This domain of epidemiological research deals with concepts such as "infection", "latency period", "infectious period", "lesion expansion", or "spore dispersal", as well as "viruliferous vector". Many of these concepts refer to the sub-processes which constitute the building blocks of epidemics, seen as overarching processes. The methodology, in terms of definition, experimentation, measurement, and analysis, has been extensively discussed, with reference books such as Kranz and Rotem (1988) and Campbell and Madden (1990).

Building blocks need assembling in order to understand epidemics. This is where other concepts, such as R_c, and R_0, as discussed earlier, are necessary. These concepts operate within (are inherently part of) modelling structures. In this area of research, the system under consideration is a simplification of reality (De Wit, 1982) and only a few attributes of the disease are considered. Critical to a theory is its parsimony (Penning de Vries, 1982; Teng, 1985a; Jeger, 1986; Jeger et al., 2004; Gilligan, 1994; Gilligan and Van den Bosch, 2008; Madden, 2006; Madden et al., 2007). In a very simple model, for instance, perhaps only healthy sites and diseased sites are considered; the crop may not even grow; and the environmental conditions are assumed constant throughout the epidemic. In this area of epidemiological research the meaning of "understanding" is very close to the ability, with these concepts and models, to predict the behaviour of systems. A typical example is the article by Madden (2006) where there is a constant dialogue between equations, reasoning and hypotheses, and (graphic) outputs. Similar are the articles by Zadoks (1971) or Jeger (1986), and many others, where adhesion to the theory which is developed comes not from numerical or statistical comparisons with a measured reality, but from the conformity of the behaviour of the model to what is expected as logical: the model is judged from the quality of its predictions.

As the theory becomes acceptable, it may be tested against more complex systems, where additional elements are included, and where additional (sub-) processes of the epidemiological process are considered. The article by, Madden (2006), which we chose as a guide, shows this path. As the considered system becomes more complicated, more hypotheses are incorporated in the model, because more processes are assumed to account for the overall system behaviour. Even when vectors, in terms of dynamics, and pathogen acquisition/transmission, are included in such a model, the overall behaviour can still be tested against the reasoning of scientists and their experience of the epidemiological reality. In their analysis of the behaviour of insect-transmitted plant virus epidemics, Jeger et al. (2004) bring about convincing hypotheses such as the mathematical form of the basic reproductive number, R_0, for a vector-transmitted virus disease.

A second objective of botanical epidemiology, and of the crop health sciences in general, is to predict crop losses. This area of research is driven by a set of concepts and processes which differ from those which prevail in the first ("understanding epidemics") domain, as suggested by Fig. 3. In Fig. 3 we use the key words "production situation" or "attainable yield" for concepts; and "damage mechanisms" for processes. Beyond the essential field-experimental work (which we did not discuss here); the models that can be used in this domain represent bridges between the discipline-oriented fields (entomology; weed science; plant pathology) of crop health and the fields of plant physiology and agronomy. Theories are derived from environmental physics (Monteith, 1972); and implemented in a wide range of models; from very simple to very detailed. A critical objective of the domain is to predict measured yield losses using models that summarize the crop physiology; and the behaviour of the [crop – pest(s) – environment] system.

A third objective is crop health (diseases, pests, weeds) management. To represent this area, we chose to illustrate "managing epidemics" with names of diseases affecting different crops. This is to suggest that the requirements to manage grapevine downy mildew differ from those needed to manage rice tungro. But this is not meant to say that no common concepts and models could apply. Jeger et al. (2004) provide a fine example to show how a generic approach can be used to define overall strategies to manage plant virus diseases, even though the viruses are quite different from one another, and the crops considered are widely different in their cycles. As in the other two domains, much detail can be incorporated into the models. In the management domain, accumulation of details reflects the need to address questions which are quite specific and extremely localized, as research in grapevine disease management shows (Caffi et al., 2010).

As more processes, and more parameters, are incorporated in models, several issues emerge. The conceptual issues and the practical consequences of model enrichment have been discussed many times. We address it again, from a slightly different perspective.

4.2. Which processes? – spatial and temporal scales of epidemiological processes

The dynamics of crop pests, diseases, and weeds, involve a wide

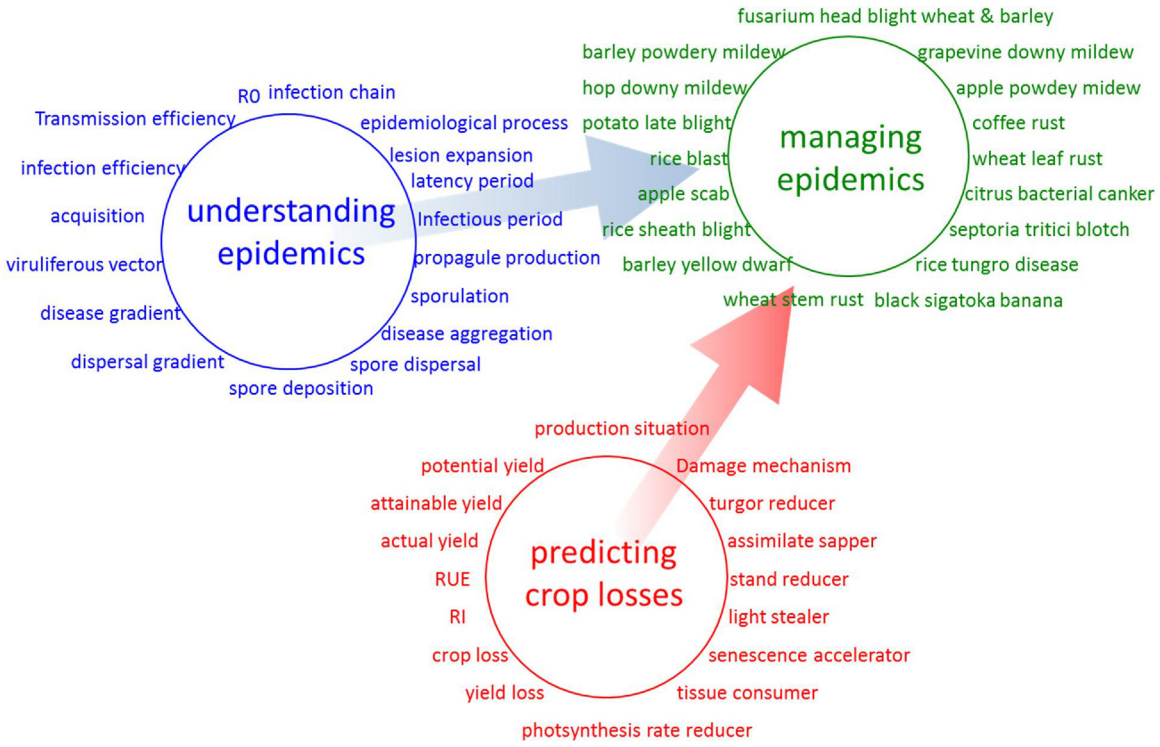

Fig. 3. Modelling objectives in plant disease epidemiology and management.
Under "understanding epidemics", a set of key parameters (e.g., R_0), processes (e.g., infection efficiency), and concepts (e.g., disease gradient) are listed. Under "predicting crop losses", a few key concepts (e.g., attainable yield), processes (e.g., radiation use efficiency, RUE), and concepts are given. Under "managing epidemics", only the names for key plant diseases for botanical epidemiology (e.g., apple scab, wheat stem rust) are indicated.

array of processes, which can all differ in terms of spatial and time characteristics. Fig. 4 displays the ranges of time constants and spatial characteristics that are encountered amongst a few epidemiological processes. If these processes are to be incorporated in a model, the issue of the choice of suitable time and spatial scales arises.

It is useful to provide an example. The process of spore dispersal in a fungal pathogen such as *Puccinia arachidis,* causing groundnut rust, involves two dispersal mechanisms: aerial dry spore dispersal following gravity-liberation, and dry spore dispersal following liberation through the transmission of kinetic energy of incident rain drops. A third process of spore displacement is the wash-off of the lesion spore contents of the canopy by heavy rain. Experimental measurements indicate that the ranges of time-constants and of spatial characteristics widely differ between these three processes (Savary et al., 1990). The fact that the dispersal processes are associated with very different efficiencies of disease transmission compounds the issue. The previous question of time and space scales for models is brought about again.

The notion of epidemiological sequence was introduced by Rapilly (1983) to represent sequences of sub-processes where the realization of each individual sub-process depends on the occurrence of the sub-process immediately preceding it. This leads to the idea of bringing together these sub-processes into broader processes, which are easier to manipulate in epidemiological reasoning. This approach was used in the case of groundnut rust modelling (Savary et al., 1990), where the sub-processes: spore liberation, spore transport, spore deposition, were combined. The combined processes were collectively parameterized from controlled experiment data, using categories of daily weather: "no rain – dry"; "no rain – humid"; "slight rain – dry"; "slight rain – humid"; and "heavy rain". These categories were defined using daily rainfall (with thresholds of 1 mm and 5 mm) and maximum daily relative humidity (with a threshold of 95%). Using this approach a single dispersal function was developed with time and space characteristics of 1 day and 1 m^2, respectively.

Merging sub-processes together can be an effective way to simplify modelling structures, to summarize the available (experimental, quantitative) information, and to synthesize sub-processes into a new one whose time and space characteristics are easier to handle. Another important and long-standing approach to simplification is through sensitivity analysis (Bregaglio and Donatelli, 2015). Early examples are Voltz and Rambal (1987) in crop science and Teng (1980) in plant disease epidemiology.

We shall come back again to the issue of model simplification. For now, a practical reason to simplify a model is shown in Fig. 4. Many (mechanistic, simulation) epidemiological models operate within the time and space characteristics of 1 day and 1 m^2, at least for annual crops. Using these characteristics to develop an epidemiological model will have the advantage of enabling model comparisons. It also corresponds to the characteristic used in most crop models, and this enables linkages between epidemiological and crop loss models (as in, e.g., Duku et al., 2016).

4.3. Modelling for a purpose

In his discussion on the development of models, Penning de Vries (1982) provides a description of model evolution, real or possible, in successive phases, which he called preliminary, comprehensive, and summary models. For the sake of this discussion we consider four stages: initial, preliminary, detailed, and simplified model. These four categories correspond to varying levels of conceptual innovation and of application. The initial model ("noon") is a breakthrough: this is an important new concept with a large conceptual value, which has yet little application, and so projects a short shadow along the applications axis. The preliminary model incorporates new elements, additional processes, which enable additional questions to be addressed through systems that are closer to reality. Innovation in preliminary models is lower, but their application value increases. Detailed models ("dusk") incorporate still more elements; their conceptual innovation content is further decreased, but their potential for application is highest.

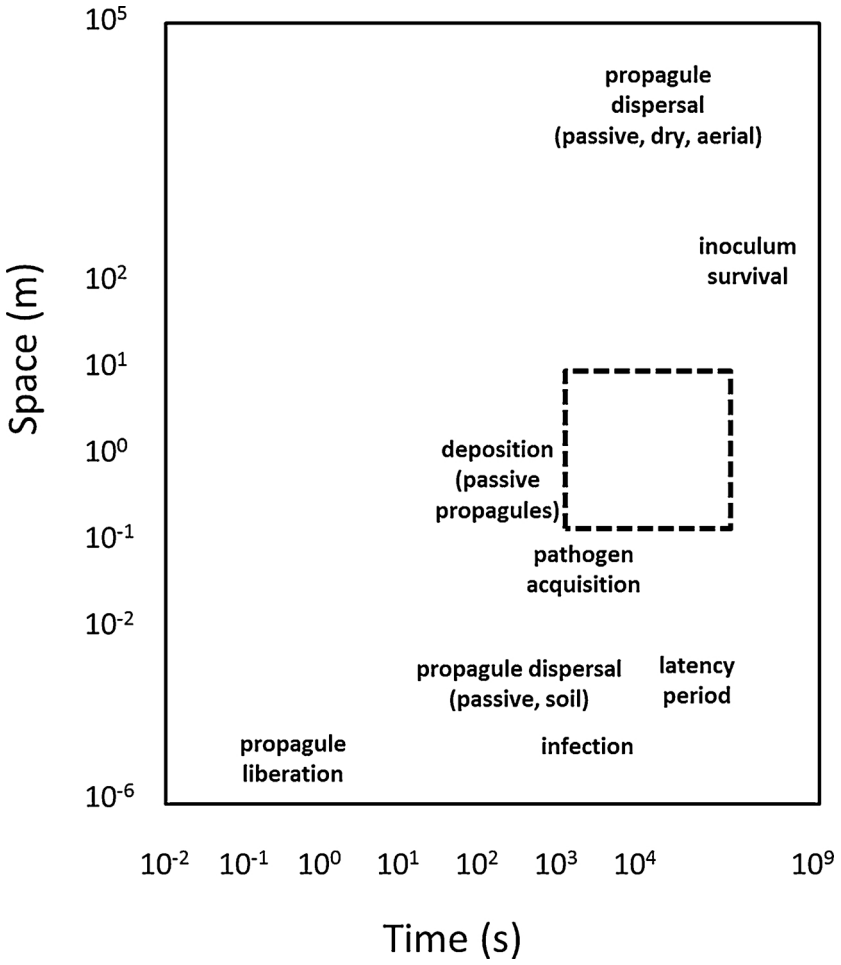

Fig. 4. Spatial scales and time characteristics of a few plant disease epidemiological processes.
Abscissa: time characteristics, seconds. Ordinate: spatial scale, meters. The dotted-line square represents typical space limits and time characteristics of many mechanistic simulation models (in the range of 1 day and of 1 m^2) in plant disease epidemiology.

Simplified models represent a new conceptual jump: simplification, through one approach or another, enables processes that do not contribute much to the behaviour of the considered system to be removed, and so brings forth again key processes. The conceptual value is increased, at the cost of application. The path described above is a simplification: as models evolve, so do the systems they address. Therefore, the meanings of "application" and of "prediction" vary, too, as models evolve. This has considerable importance in the objectives that are set in the modelling work, and also in the ways to assess if these objectives have been achieved – that is: in evaluating models (Zadoks and Rabbinge, 1985).

Table 2 displays four groups of objectives, hypotheses, and modelling approaches. Note that model objectives (Table 2) and model types (described above; derived from Penning de Vries, 1982) do not exactly coincide.

A first modelling objective is the development of a theory. The hypothesis is that a few processes are sufficient to account for observed patterns, or that the behaviour of a system is very strongly determined by a set of key processes and parameters. Van der Plank's (1963) models fall in this group, as does the set of linked differential Eq. (4) of Madden (2006) or again, the sets of differential equations for the host crop and the vector population of Jeger et al. (2004).

The objectives of models in the second group are associated with a better definition of the system under consideration, its components, and its characteristics. Some of these models often consider a few sub-processes of epidemics, as in Zadoks (1971). This is also the case in Madden (2006) where the initial theory (set of linked differential Eq. (4)) is progressively enriched with features such as the dynamics of primary inoculum, host mortality, or host growth. As the enrichment of processes continues, models of this group may become very large and complicated, as in e.g. Savary et al. (1990) and Djurle and Yuen (1991). Even more detailed and complex models can emerge from the coupling of modular crop growth models with pest and disease models (Jones et al., 2003, Bregaglio and Donatelli, 2015; Whish et al., 2015). Progressively, the objective of modelling changes, and becomes to:

(1) map the considered system in detail, and identify the knowledge gaps that hamper its quantitative analysis (Teng, 1985a), and
(2) produce a quantitative synthesis of the available (experimental) knowledge, to
(3) assess if this knowledge is congruent with quantitative measurements of observed processes.

This has particular value when new, quantitative epidemiological knowledge is recently gained on an emerging disease (as was the case of groundnut rust in West Africa, Savary et al., 1990), or when specific and experimentally challenging hypotheses need testing, such as the organization of epidemics amongst crop canopy layers (Djurle and Yuen, 1991).

As indicated above, model simplification leads to simpler, leaner, structures. Simplification aims to regain grip on key processes generated by the initial theory, together with additional processes that have proven to be of critical value to understand and predict the behaviour of the system. EPIRICE and EPIWHEAT (Savary et al., 2012, 2015), two

Table 2
Hypotheses and approaches in plant disease epidemiological modelling.

Objectives	Model type	Hypotheses	Modelling approaches
Theory development		• Some few sub-processes[a] and the associated parameters are sufficient to account for observed process patterns. • Patterns (spatial, temporal) can critically change as responses to variations of a given, few set of sub-processes (and their parameters).	– Simple, parameter-sparse models. – May be solved analytically. – May involve stochasticity. – May be grounded on, but do not critically depend, of experiments at the systems scale.
Systems definition and characterization		• The considered collection of sub-processes is sufficient to account for the bulk of observed process variability (spatial, temporal). • The available (experimental) knowledge on sub-processes is congruent with quantitative measurements of the observed process.	– Models with more parameters, including large, complicated, multi-(sub-) processes models. – Cannot be integrated analytically. – Based on experiments and parameter quantification at the sub-process level. – Tested against observations at the process level.
Systems simplification		• Reduction of an already existing systems-design to a simpler system, involving fewer sub-processes, is sufficient to account for key features of the (spatial, temporal) process behaviour. • The simplified system conforms to an existing theory.	– Medium-size models with fewer selected sub-processes. – Reduction of the number of considered sub-processes (and parameters) results from elimination, merger, combination, or reformulation of sub-processes. – Based on experiments and parameter quantification at the sub-process level. – Tested against observations at the process level.
Systems control, management		Intervention points correspond to control or management actions that efficiently drive the considered process to satisfactory systems performances or outputs.	– Medium to large-size models. – Emphasis on intervention points linked to parameters or driving functions corresponding to human action.

[a] A plant disease epidemic is the (main) process considered; its constitutive parts are referred to as sub-processes.

generic models to simulate the dynamics of several diseases of rice and wheat, respectively, belong to this group. Another important objective in developing these models is also communication and sharing.

Following Fig. 3, a fourth group is indicated in Table 2, with systems control and management as objectives. Models in this group may be fairly detailed, since they incorporate intervention points for disease management and they often include features of the host population as well as elements of the physical environment. Models for the management of leaf rust (Rossi et al., 1997) and powdery mildew (Rossi and Giosuè, 2003) in wheat exemplify this group.

4.4. Model evaluation

In his discussion on the philosophy of model evaluation, Teng (1981) considers three possible views. The rationalist view holds that a model is a system of logical deductions made from a set of premises. The empiricist view contends that only proven facts, not assumptions, should be involved in the development of models. And the positivist view holds that a model is valid only if it is capable of accurate predictions, irrespective of the structure or logic of the model.

It appears that all groups of models in Table 2 involve these views in their evaluation. However, the weights of the three views differ according to the groups we discussed. When theory development is the objective, the rationalist view predominates. By contrast, when the definition of a system is the objective, the empiricist view will assume greater importance. If system simplification becomes the objective, a combination of the rationalist and of the empiricist views is implied, because this objective derives in part from the first (development of a theory) and the second (inclusion only of verified, falsifiable, elements) views. The objective of systems control and management, where accuracy of predictions is critical, leads in turn to an increased weight of the positivist view.

As explained by Teng (1981), and later discussed by Zadoks and Rabbinge (1985) and Savary and Willocquet (2014), approaches to model evaluation therefore depend on the modelling objectives. This is logical, since the hypotheses associated with the different groups in Table 2 are different. Borrowing from Teng (1981), Rabbinge (1993), and Thornley and France (2007), one can suggest the following steps (Savary and Willocquet, 2014) for model evaluation:

- Model verification: checking that the programming structure and the computations are performed as expected;
- Visual assessment of the outputs of the model and its behaviour: assessing whether the model's outputs conforms with the expected overall behaviour of a system;
- Quantitative assessments of the model's outputs against numerical

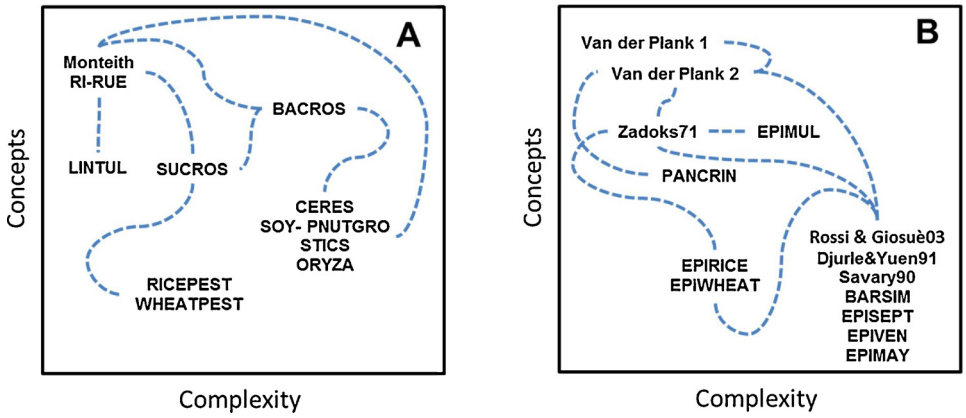

Fig. 5. Patterns of model evolution: crop models and epidemiological models.
Distribution of a few mechanistic crop models (A) and epidemiological models (B) according to levels of concepts (vertical axis) and of complexity, i.e., the number of processes (parameters, horizontal axis) they involve. The list of models shown is very incomplete, and model names may refer to groups of models. Examples have only been chosen to illustrate a distribution along the two axes. Dashed lines only suggest conceptual linkages, not necessarily phylogenetic relations or chronology.
A: Crop models: Monteith RI-RUE: Monteith, 1977; LINTUL: Spitters and Schapendonk, 1989; SUCROS: Van Keulen et al., 1982; BACROS: Penning de Vries and Van Laar, 1982; CERES: Jones et al., 2003; SOYGRO: Wilkerson et al., 1983; PNUTGRO: Boote et al., 1986; STICS: Brisson et al., 2003; ORYZA: Kropff et al., 1994; RICEPEST: Willocquet et al., 2002; WHEATPEST: Willocquet et al., 2008.
B: Epidemiological models: Van der Plank 1, Van der Plank 2: Van der Plank, 1963; Zadoks71: Zadoks, 1971; EPIMUL: Zadoks and Kampmeijer, 1977; PANCRIN: Zeng, 1991; Djurle&Yuen91: Djurle and Yuen, 1991; Savary90: Savary et al., 1990; Rossi&Giosuè03: Rossi and Giosuè, 2003; BARSIM: Teng et al., 1980; EPIVEN: Kranz et al., 1973; EPIMAY: Waggoner et al., 1972; EPISEPT: Rapilly and Jolivet, 1976; EPIRICE: Savary et al., 2012; EPIWHEAT: Savary et al., 2015.

observations, with a large number of procedures. A starting point is to consider that common statistical testing aims at rejecting the hypothesis H_0 that the distributions of observed and simulated values are identical. Testing H_0 is opposite, however, to the issue of validation, where one wants to establish the sameness of outputs and observations. H_1 is the hypothesis that the two distributions are different. The error of rejecting, wrongly, H_1 is the main concern in model evaluation, and entails a number of approaches, for which Thornley and France (2007) provide a starting point.

4.5. Patterns of model evolutions: crop models and disease models

Fig. 5 displays the evolution of crop models (Fig. 5A) and epidemiological models (Fig. 5B) along the two axes of model complexity (the detail of processes that are incorporated, with increasing number of model parameters) and conceptual innovation (the implementation of theories, and the operationalization of over-arching systems principles). A few selected models are displayed in the two plots, and some connections are indicated to show conceptual linkages. The both fields, crop modelling and epidemiological modelling, experience similar pulsations, from concepts to increased details, and to simplification.

5. Simplification of models as a means for progress

5.1. The [crop health – production situation] framework

In the last part of this article, we return to some of the questions of the Introduction: (1) What are the evolutions in crop diseases and pests to be expected from agricultural shifts? (2) What are the main changes to expect with respect to crop and disease, as a result of global and climate changes? and (3) What are the expectable changes in pest and disease impacts, in terms of crop losses?

These questions represent major challenges for the plant sciences. Several authors (e.g., Coakley et al., 1999; Chakraborty and Newton, 2011) have pointed to the risks of addressing questions for which data (both experimental data and field data) are largely missing, and warned about the dangers of exploring phenomena that are driven by factors with confounding effects (for instance population growth and climate change) and/or by factors which are largely unaccounted for in current analyses (such as biological invasions in interactions with many other driving factors of change). While acknowledging these difficulties and risks, we believe that some progress can be made if a suitable framework to address these questions is set.

Multiple studies on a number of annual and perennial crops (Savary et al., 1988, 2006; Daamen et al., 1989, Willocquet et al., 2008; Reddy et al., 2011; Allinne et al., 2016) have shown that a framework that considers both production situations and crop health syndromes enables the:

- pooling of individual crop fields into production situations, that is, into groups that share common attributes pertaining to production environment and crop management, thus, sharing the same level of attainable yield;
- grouping of individual crop fields that share the same crop health (animal pests, diseases, weeds) syndromes, corresponding to injury profiles;
- detection of associations between crop health syndromes and production situations.

For a given crop, the [crop health-production situation] framework enables to focus on a limited set of combinations of production situations and crop health syndromes, within which the modelling of crop losses is undertaken. For instance, six production situations may be distinguished for lowland rice in tropical Asia, which can be linked to five distinct crop health syndromes (Savary et al., 2006). Similarly, three crop health syndromes linked to three production situations may be considered in Western Europe (Willocquet et al., 2008), or four crop health syndromes of coffee distributed across four crop management patterns in three topoclimates in Costa Rica (Allinne et al., 2016).

Further research (McMullen et al., 2012; Savary et al., 2005, 2011a, 2011b, 2017) has also led to the notion that, as global change occurs, production situations evolve, leading to (1) changes in the levels of attainable yield, and (2) evolution of crop health syndromes, which together translate in (3) different levels of crop losses. This framework may enable us to address the effects of global changes and its cascade of effects on crop health and on crop losses. This can be achieved through scenario analyses based on simulation modelling, where each scenario analysis focuses on specific [crop health-production situation] combinations.

The following points of this section pertain to technical and conceptual components, which we believe will be necessary for these analyses. These components include:

- a definition of crop health syndrome and its meaning in terms of crop injury;
- a definition of levels of injuries that mirror levels of disease, pest, or weed dynamics;
- epidemiological models that account for the main effects of crop

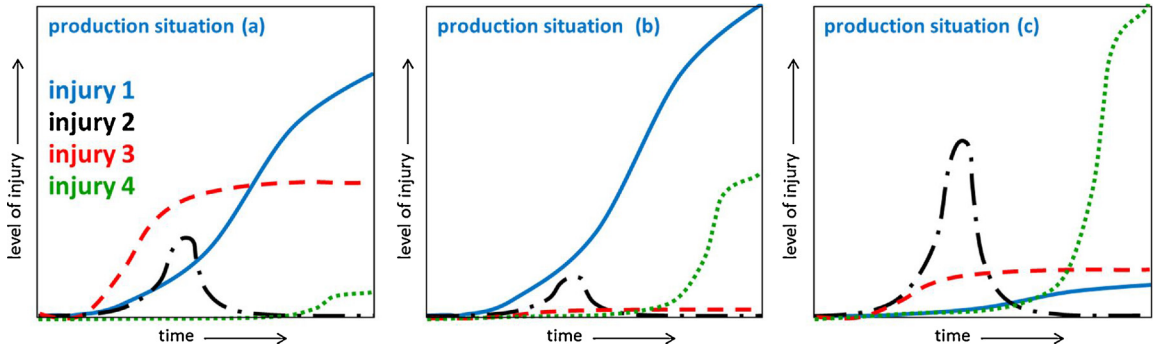

Fig. 6. Crop health syndromes and their variation across production situations.
Three crop health syndromes, corresponding to three production situations, are shown. Four injuries caused by diseases, animal pests, or weeds are indicated in each crop health syndrome. These injuries are represented by their dynamics, as would be observed in the field or simulated. Depending on the syndrome, the dynamics of each injury differs. These dynamics can be used as inputs (driving functions) in a crop growth model that incorporates damage mechanisms (Table 1).

management and of the physical (climate) environment pertaining to specified production situations in a simplified and generic manner, thus generating robust injury functions;

- crop growth simulation models that are detailed enough to (1) represent the dynamics of attainable growth and yield in a given production situation, and (2) simulate the effects of combined injuries on crop growth and yield.

5.2. Disease and pest syndromes

The definition of crop health syndromes for a given crop may be based on the literature pertaining to quantitative surveys in farmers' fields. Examples for wheat, rice, groundnut, and coffee are given in Savary et al. (2017). The pathogens, pests, and weeds to be included in the syndrome need to account for the most frequently harmful organisms encountered in the widest possible range of environments. Fig. 6 illustrates how the concept of crop health syndrome may be operationalized from a crop modelling standpoint, with variable dynamics of four arbitrary injuries caused by different pathogens, pests, or weeds. The patterns of such dynamics may be retrieved from the literature (see e.g. Savary et al., 2012), or generated by simplified models for the dynamics of diseases, pests, or weeds. An important departure from conventional population dynamics is that each syndrome corresponds in turn to driving functions (vertical axis of Fig. 6) expressed as injuries to crop growth models which incorporate damage mechanisms associated to these injuries.

5.3. Simplified, generic, epidemiological models: EPIRICE and EPIWHEAT

EPIRICE and EPIWHEAT are simplified models which simulate potential epidemics of crop diseases in the case of rice (Savary et al., 2012) and wheat (Savary et al., 2015). Both models use an SEIR structure (Susceptible, Exposed, Infectious, and Removed sites), following the seminal model of Zadoks (1971). They account for the host dynamics (growth and senescence) in a simple way and account for the effects of climate on diseases through the use of modifiers affecting R_c. Both models have a time step of one day, and consider a 1 m^2 crop system. Both models use daily mean temperature and rainfall as climatic inputs. These models allow estimating the epidemic risk, i.e., the probability of an epidemic occurring. Epidemics were simulated and mapped globally for five rice diseases (tungro, bacterial leaf blight, leaf blast, brown spot, sheath blight), and at the European scale for two wheat diseases (leaf [brown] rust and septoria tritici blotch).

Such simulation results can be used for priority setting. For example, regions can be grouped according to the mean and variance of disease intensity over years, generating a framework in three groups of areas to establish the most appropriate disease management strategies. The first group of areas to be considered as priorities for sustained disease management are those for which mean potential epidemics are high and variance is low (areas of disease endemicity), e.g., through host plant resistances, and/or collective crop health management at the landscape scale. The second set of areas is where mean potential epidemics are moderate-high and variance high, i.e., areas where acute disease epidemics – outbreaks – can occur. These areas will require adapted tactical decisions, possibly involving chemical control guided by warning systems. The third and last set of areas is where mean potential epidemics are moderate and variance is low, i.e., areas where chronic epidemics occur, and should be associated with long term management strategies involving partial resistances and decisions at the landscape scale.

5.4. Simplified, generic, crop loss models: RICEPEST and WHEATPEST

Simplified agrophysiological models accounting for damage mechanisms were developed for rice (RICEPEST; Willocquet et al., 2000, 2002, 2004) and wheat (WHEATPEST; Willocquet et al., 2008). Both models consider photosynthesis (using Monteith's framework; Monteith 1972, 1977), assimilates partitioning towards plant organs, translocation of starch from stems to storage organs, and leaf senescence. RICEPEST further includes the dynamics of the tiller population in order to explicitly account for tiller injuries such as tiller death caused by dead heart, or panicle elimination caused by white heads, two of the injuries implemented in the model. The crop physiological processes considered in these models are sufficient to accommodate the different damage mechanisms (Table 1) associated with the various harmful agents/yield reducers considered in both crops. As with the models for potential epidemics, these models have a time step of one day, and consider a 1 m^2 crop. Both models require daily mean temperature and global radiation. These models allow estimation of the risk magnitude, i.e., the impact (in terms of yield losses) of yield reducing factors. RICEPEST accounts for the effects of 11 crop harmful organisms (bacterial leaf blight, sheath blight, brown spot, leaf blast, neck blast, sheath rot, white heads, dead hearts, brown planthoppers, insect defoliators, and weeds). WHEATPEST accounts for the effects of BYDV, leaf (brown) rust, stripe (yellow) rust, powdery mildew, septoria tritici blotch, stagonospora nodorum blotch, take-all, eyespot, sharp eyespot, fusarium stem rot, fusarium head blight, weeds, and aphids.

A key feature in these models is that they consider production situation drivers and injury drivers as inputs. Production situation drivers as inputs include parameters that reflect the level of yield limiting factors, e.g., RUE, and therefore allow to simulate the attainable yield in a given production situation. Injury drivers correspond to crop health syndromes (or injury profiles), i.e., to combinations of dynamic levels of pest injuries, in the same way as illustrated in Fig. 6. Production situation and injury drivers were used to simulate yield losses in [production situation x injury profile] combinations identified from surveys

conducted in farmers' fields.

Such results can be used for research priority setting (e.g., Willocquet et al., 2004). For example, the ranking of pests according to the yield losses they cause in a given [production situation x injury profile] combination provides a basis to identify pests to address in priority in research programmes. Conversely, the ranking of risk magnitudes enables the assessment of the benefits of future research (e.g., improved resistances) or better crop health management in terms of yield gains.

6. Concluding remarks

This review contributes to highlight (Fig. 3) the diversity of organisms that can negatively affect agricultural crops, the diversity of life cycles of these organisms, and the very large number of interactions that potentially can be considered under the phrase "modelling pests and diseases". We have thus been seeking unifying concepts that enable genericity. This is for instance the case of the concepts of production situation, of attainable yield, and of damage mechanism, which we discussed. The concept of crop health syndrome, is further proposed as a contribution to a generic approach to crop health modelling.

The modular approach advocated by crop physiologists (McCown et al., 1995; Jones et al., 2001, 2003, 2017) enables the flexibility for disease and pest models to have their own evolution. Such an evolution is necessary. The modelling structures discussed here implicitly refer to a "mean-field" approach, where what is modelled is the average of what could be observed. In many plant disease epidemics, the average of processes may not well account for some critical properties of dynamics. This is especially the case for the beginning of epidemics, where for instance the environmental characteristics that enable epidemic onset, or the genetic nature of the primary inoculum, generate unique system properties. Examination of criteria for invasion and persistence allow insight into why some epidemics take place while others do not (Gilligan and Van den Bosch, 2008). Incorporating stochastic features in model structure is an important approach to account for such properties. A similar remark could be made about the spatial scale considered in models for epidemics and crop losses: the modelling structures discussed here address a plot/field scale, whereas the appropriate scale to address some processes in some systems is that of the agricultural landscape (Gilligan and Van den Bosch, 2008; Papaïx et al., 2014). Further progress needs to enable inclusion of such features.

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