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Abstract As any epidemic on plants is driven by the amount of susceptible tissue, and the distance between organs, any modification in the host population, whether quantitative or qualitative, can have an impact on the epidemic dynamics. In this paper we examine using examples described in the literature, the features of the host plant and the use of crop management which are likely to decrease diseases. We list the pathogen processes that can be affected by crop growth and architecture modifications and then determine how we can highlight the principal ones. In most cases, a reduction in plant growth combined with an increase in plant or crop porosity reduces infection efficiency and spore dispersal. Experimental approaches in semi-controlled conditions, with concomitant characterisation of the host, microclimate and disease, allow a better understanding and analysis of the processes impacted. Afterwards, the models able to measure and predict the effect of plant growth and architecture on epidemic behaviour are reviewed.

Keywords Canopy structure · Disease transmission · Architectural traits · Microclimate · Host-pathogen models

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Introduction

A widely accepted definition of an epidemic is "a change in disease intensity in a host population over time and space" (Madden et al. 2007). This definition emphasizes that epidemics are dynamic processes with variable disease rates. How many of these disease rate changes are triggered by the host population is still an ongoing issue. As an epidemic is driven by the effective reproduction number (Cintron-Arias et al. 2009), which is the product of the proportion of susceptible tissue, the disease transmission rate, and the infectious period duration, any modification in the host population, whether quantitative or qualitative (distribution of plants or of susceptible organs), can have an impact on the epidemic dynamics.

The host population growth is modified during the growing season by architectural features or by cultural practices. The production of new organs continuously modifies two aspects: the plant or canopy porosity, as well as the plant's level of susceptibility when organ susceptibility changes with age (ontogenic resistance or receptivity). Indirectly, plant growth and structure modify the microclimatic environment inside the canopy, generating favourable or unfavourable conditions for pathogens. Organ susceptibility or the susceptibility period may also be modified by cultural practices disrupting crop-pathogen synchronisation. Spatial heterogeneity in the host population can also be generated by differences in phenology (e.g. variety, pruning, sowing date) or growth rate (e.g. soil variation, vigour, rootstock). There is evidence that these variations within host populations do impact on disease incidence, severity or spread. However, those effects have rarely been explicitly taken into account in epidemiological models. Tracking back the impacted pathogen processes from the global dynamics of an epidemic, and then ranking the host traits involved in their modifications, constitute a challenge.

In this paper, we: 1) review evidence of epidemic variations attributed to plant growth and architecture in main pathosystems; 2) identify the pathogen processes impacted, and assess them in different epidemic contexts; and 3) explore the models able to measure and predict the effect of plant growth and architecture on epidemics.

Evidence of epidemic variation attributed to plant growth and architecture

Plant growth and architecture: the context What kind of growth and architectural changes in the host population can we expect? The architecture of crop plants is influenced by endogenous factors (hormone signals, trophic competition between organs) as well as by exogenous factors (light distribution, soil water, nutrients of organs, temperature, wind). For perennial plants, the crop density and the pruning type are defined at planting time, while significant changes in growth and architecture can be generated during the vegetative season by cultural practices. Some of these practices act on exogenous factors; that is the case for irrigation, fertilisation, root-stock and low density of planting, which have a positive effect on primary growth and ramification. In contrast, cover-cropping has a negative effect due to root competition for water and nutrients. Water supply and soil type modulate those effects. Cultural practices such as shoot pruning, topping, thinning and training will have an effect on endogenous factors modifying plant ramification and reiteration (morphogenetic process through which the organism duplicates its own elementary architecture in response to environment or damage (see this issue, Costes et al. under review)), thereby affecting the balance of young vs. older leaves, and exogenous factors, such as light distribution through an indirect effect on leaf density. For annual plants, agronomic practices play an important role in plant growth and architecture, e.g. sowing density and date, nitrogen fertilization, or the use of a growth regulator. But more variation in architecture can be variety-specific, since the endogenous regulation of architecture is under genetic control (Fig. 1) (Reinhardt and Kuhlemeier 2002).

Variables such as 'architectural diversity', 'cultural practices', 'primary growth', 'ramification', 'plant and canopy porosity' or the 'amount of susceptible tissue', which are the key variables driving the epidemic dynamic, are latent variables. Although not directly observable, such latent variables can be derived from a large array of observed variables (Fig. 1). Architectural diversity and cultural practices determine the level and dynamic of primary growth and ramification which together modify plant porosity and the amount of susceptible tissue. Most of these observed variables can be measured at different times or plant stages. The experimenter has to choose or test what is the most relevant

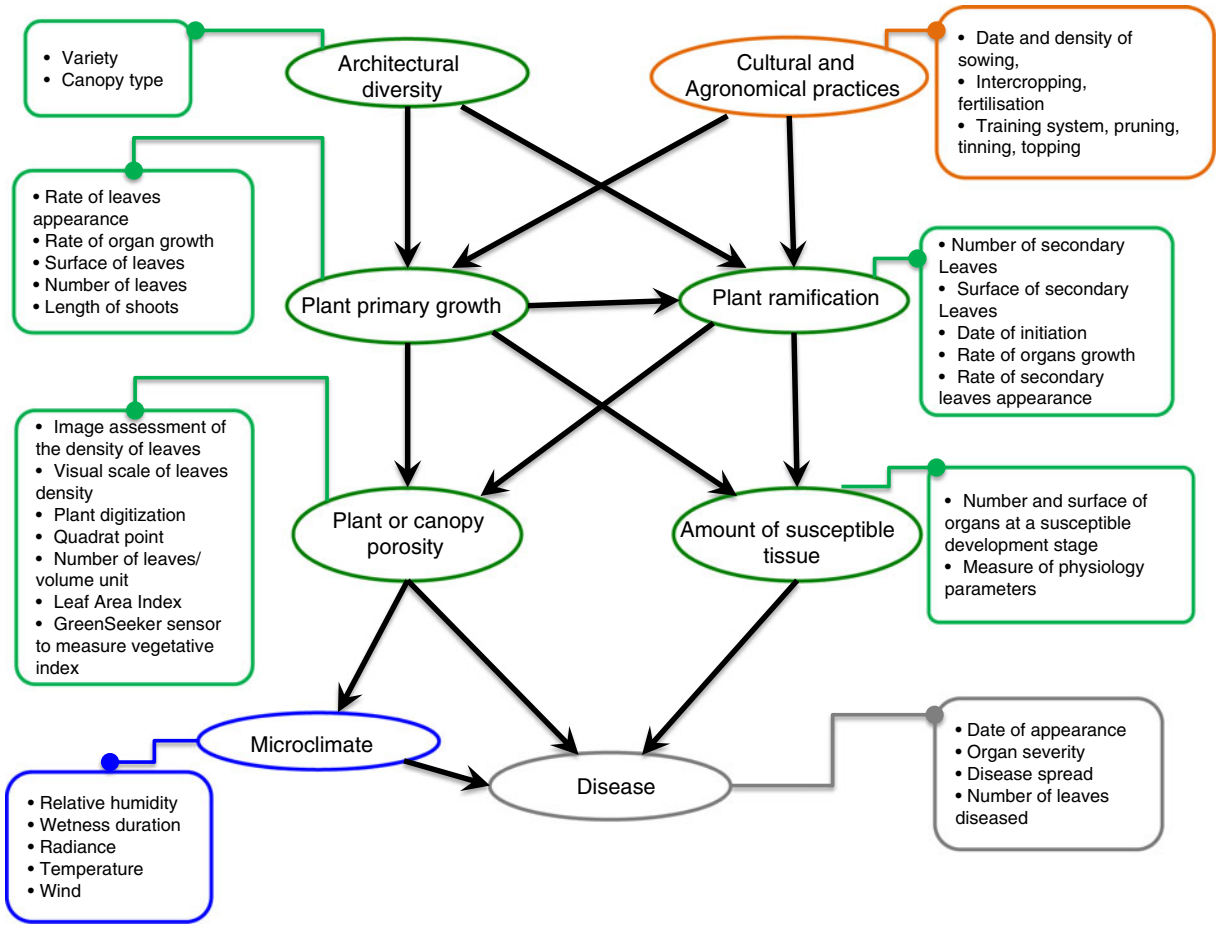

Fig. 1 Potential relationships between latent variables such as 'Architectural diversity', 'Cultural practices', 'Primary growth', 'Ramification', 'Plant or canopy porosity', 'Amount of susceptible tissue' with 'Microclimate' and 'Disease'. Each latent variable is characterised by numerous observable variables

time to measure them. As an example in Fig. 1, manifest variables such as image assessment, visual scale of leaf density, plant digitization, quadrat point, number of leaves per volume, or LAI… can be used to measure plant or canopy porosity (ratio of pore space to the space occupied by plant organs). For a review of definitions in the literature see in this issue (Tivoli et al. 2012). The measurement of some of those manifest or observable variables allows us to test the correlations between variables describing plant growth or architecture with variables describing disease, and to test or predict which of them have the higher impact, and if they are not contradictory. It is, however, not always possible to clearly trace back what are the pathogen processes impacted, and sometimes only hypotheses can be proposed. The following section highlights examples of the effects of the architectural diversity or of cultural practices such as crop structure, density of plantation, training systems, management of crop phenology and mixed or cover-cropping on pathogen and on disease development.

Cultivars and architectural diversity Among the cultivated species, different cultivars may be characterised by architectural features, which are able (or not) to modify the development of epidemics. For example, in the *Mycosphaerella pinodes*/pea pathosystem (ascochyta blight), the comparison of different pea cultivars, belonging to three architectural types, showed that a large cumulative LAI above one diseased stipule, combined with a large distance between nodes within the canopy, favours disease development,

whereas a large internode length reduces disease severity. These results were explained by opposite effects: the direct effect of low canopy porosity favouring ascospore dispersal and disease development, and the indirect effect of larger LAI on the microclimate, increasing the wetness duration and disease severity (Le May et al. 2008). Similar effects on spore dispersal were observed in *Colletotrichum acutatum* on strawberry, a crop with a canopy structure very different from that of pea (Yang et al. 1990; Madden et al. 1993) and in *Mycosphaerella graminicola* on wheat in field conditions (Lovell et al. 1997).

Crop structure, plantation density and training system Agronomic practices such as row planting, training systems, pruning can generate various spatial canopy structures or the spatial distribution of organs at plant level, with consequences for epidemics. In grapevine, a perennial plant, the row structure of the crop was suspected to have an effect on the dispersal of the wind-dispersed, xerophilic spores of powdery mildew (caused by *Erysiphe necator*). The velocity of disease spread decreased along the row with an enhanced effect in highly vigorous plots (with a high visual density of leaves), whereas vigour was conducive to disease spread between rows (Calonnec et al. 2009). These results are consistent with a barrier effect ('fly paper') to spore dispersal on the row, due to decreased canopy porosity. At the plant scale, however, the porosity effect is no longer visible, or is counterbalanced by an increased inoculum production on vigorous vines. The rate of leaf emergence (characteristic of primary growth), and of diseased leaf emergence, followed an identical linear relationship for the various levels of plant vigour (Valdes-Gomez et al. 2011). For chickpea, row spacing and sowing density have an effect on ascochyta blight severity It was hypothesised that a combined effect of a high number of plants available to intercept inoculum (low canopy porosity) and a reduction in air movement within dense canopies that maintain a more humid microclimate, could explain the effect (Chang et al. 2007). A similar effect of canopy density was observed in several other pathosystems such as the common bean white mould caused by *Sclerotinia sclerotiorum* (Schwartz et al. 1978), and the sclerotinia blight of peanut caused by *Sclerotinia minor* (Butzler et al. 1998). In both cases, open, less dense canopies were less favourable to disease development, presumably because the microclimate was less conducive to disease. Less dense canopies lead to a microclimate which is less favourable to leaf wetness duration (Dalla Marta et al. 2005; Huber and Itier 1990) one of the key climatic parameters in disease development.

Training systems can have effects on both annual and perennial crops. On peanut, mechanical pruning affects soil temperature and helps to reduce disease. It is used in the integrated management of peanut blight (Butzler et al. 1998). In case of high disease pressure, mechanical pruning, combined with fungicide application, provides an efficient control of the disease. On carrots, lateral clipping influence the microclimate and development of apothecia of *S. sclerotiorum* (Kora et al. 2005). In apple orchards, the development of apple scab, caused by *Venturia inaequalis*, depended on the training system (Simon et al. 2006). When the Centrifugal system was compared with the Solaxe system, disease incidence on leaves decreased in the first part of the season, but no significant difference was observed for fruit damage. The effect of the training system might be caused either by inoculum removal during the thinning cut of fruiting spurs, or by a decrease in leaf wetness duration due to increased light penetration. On grapevine, two types of training system, resulting in two types of architecture, were compared. Free positioned top vines experienced a higher level of powdery mildew on bunches than on vertical shoot positioned vines (Zahavi et al. 2001). As irradiance was lower under the free positioned top vine system, the contrast in disease levels between the two systems was explained by a negative effect of sun radiation on infection. Similarly, vines with the highest level of disease on clusters were those that have a significantly lower proportion of clusters well exposed to sunlight (≥51 % photosynthetic photon flux) (Austin and Wilcox 2011). Additionally, clusters that were well exposed to sunlight were also well exposed to spray deposit. Thus, canopy management practices designed to optimize the sunlight exposure of grape clusters for fruit quality purpose should also significantly contribute to the management of the disease.

It is not easy, to disentangle an indirect effect of plant growth and architecture on the pathogen development through the micro-climate from a direct effect of the modification of plant susceptibility, through a physiological mechanism, especially in the case of biotrophic interaction. In fact, different types of

grapevine management (e.g. cover-crop, weed control, irrigation, and fertilization) showed significant positive correlations between the disease incidence of grey mould, caused by *Botrytis cinerea*, on bunches and key variables of shoot vigour (total leaf number, leaf layer number, leaf area per metre of row) and vine capacity (pruning mass, leaf dry matter, nitrogen accumulation) (Valdes-Gomez et al. 2008). The results were modulated by the climatic and microclimatic conditions of the year: in conducive years, grey mould developed in all experimental plots; whereas, in dry-summer years, disease developed only in the most vigorous vines, which were both irrigated and fertilised. However, the vines with a high canopy growth not only favoured higher humidity, but also developed very compact clusters and delayed fruit maturity, potentially increasing susceptibility to the disease.

Training systems or seasonal crop management may also affect the location and amount of primary inoculum, by reducing either disease severity the previous year or the surface of organs (e.g. trunk, shoots) harbouring the winter source of primary inoculum. There is, however, no firm evidence in the literature of such effects, whose practical significance remains a matter of speculation.

Management of crop phenology Plant phenology can be manipulated to disrupt crop-pathogen synchronisation. As observed with climate change (Cleland et al. 2007), crop management can induce a shift in plant phenology, especially in the timing and duration of bud burst and blossoming. The effect can be due to: a decrease in the amount of primary inoculum when the amount of susceptible organs is reduced at the time of primary spore release, and/or a shorter period of exposure of susceptible tissue to the pathogen. There is evidence of disease dynamics being impacted by modification of sowing or planting time, or by the use of early season varieties (e.g. (Navas-Cortés et al. 1998)). A June bearing strawberry cultivar producing a single crop of berries during the early summer is rarely attacked by powdery mildew (caused by *Podosphaera aphanis*) in the field, in spite of its high disease susceptibility in a tunnel (Carisse and Bouchard 2010). The ontogenic resistance of such a cultivar is certainly similar to a day-cultivar highly susceptible to disease. In the June bearing cultivar grown in the field, the pathogen population is low during the berry susceptible period, which is not the case in tunnels, due to the higher temperature and low leaf wetness. Cultural tactics (date of pruning, topping, inter-cropping) can be further implemented to desynchronise the periods of high plant susceptibility and of maximum pathogen development or release. On grapevine, bud break can be delayed and flowering shortened by cultural practices such as late pruning or cover-cropping; minimal pruning can have an effect on the canopy closure before bloom. Such practices could be used to better control pathogens, based on good knowledge of the pathogen cycle and of its response to environment.

Mixed cropping and cover-cropping With mixed or cover-cropping systems, another crop is used to modify the microclimate of the main crop (mixed crop) or directly influence its growth and structure (cover-crop). A reduction of disease severity in mixed cropping systems is usually attributed to either the barrier effect or microclimatic changes. In the pea/wheat intercropping system, under field conditions, the disease severity of ascochyta blight on pea pods and stems was substantially reduced in the intercrop, compared to the pure pea stand, when the epidemic was moderate to severe (Schoeny et al. 2010). In a two-species mixture such as pea-wheat, reduction of disease severity could occur on the two components of the mixture. A substantial reduction of severity of septoria tritici blotch (caused by *M. graminicola*) was assessed on wheat (Lebon et al. 2012). Disease reduction is due to the barrier effect of the immune species which reduces the splash dispersal of the companion species disease causal agent (Wolfe 1985). Modification of the microclimate within the intercrop canopy, in particular by a reduction in leaf wetness duration, during and after flowering, partially contributes also to disease reduction. Whereas the barrier effect is beneficial for all components, modification of the microclimate might not be beneficial for all intercropping components. Mixed cropping with cereals also contributed to the slowing of chocolate spot (caused by *Botrytis fabae*) epidemics on faba bean (Sahile et al. 2008). In faba bean/maize and faba bean/barley intercropping, free air circulation and less humid conditions might have been less favourable for chocolate spot development. Intercrops with oats, barley and wheat showed low to moderate suppressive effects on pea ascochyta blight (Fernandez-Aparicio et al. 2010). Suppressive effects can be ascribed to a combined reaction of host biomass, altered

microclimate and a physical barrier to spore dispersal. In cultivar mixtures, disease and plant-plant interactions all affect one another with a great epidemiological (e.g. distance, barrier effect) and ecological (e.g. plant nutritional status and microclimate) complexity (Finckh et al. 1999). Cover-cropping, especially associated with perennial crops, has a negative effect on plant growth through competition for water, radiation and minerals (Ripoche et al. 2011), although the effect may be time-dependent and vary with the type of inter-crop culture (Celette et al. 2009). An effect of inter-cropping on the development of grapevine powdery mildew (Valdes-Gomez et al. 2011) and grey mould (Valdes-Gomez et al. 2008) was demonstrated, but the processes involved are still the subject of debate.

Description and quantification of the pathogen processes impacted in relation to the plant variables and epidemic types

Each component of the fungal infection cycle (infection, mycelium growth, sporulation, dispersal) (Fig. 2), and the resulting component of aggressiveness (i.e. infection efficiency, mycelium growth, latent period, sporulation rate) (Pariaud et al. 2009) can be directly influenced by the host growth, the nutrient status of the organs, and by climatic or microclimatic factors. Host primary growth and ramification will have an impact on leaf density, and, therefore, on the plant and canopy porosity and the amount of susceptible tissue at each time step and plant level. Plant growth variables such as leaf density and organ age can have an indirect impact on microclimatic factors (Fig. 2). At each time step of the epidemic, the disease transmission rate r (infected surface/infectious surface/day) is defined as the product of the contact probability between the spores and the host, times the infection efficiency, and times the inoculum produced by infectious tissue. The effective reproduction number $\left(R_{eff}(t) = r\,i\,S(t)/N(t)\right)$ can then increase or decrease, depending on the evolution of the different components of host and pathogen, and predict the qualitative characteristics of the host-pathogen system dynamics (Burie et al. 2011).

What should be measured in the host and pathogen in order to quantify and rank those processes? Is the development and architecture of the plant likely to have the same impact, or does it depend on the intrinsic pathogen characteristics (e.g. water-dependent infection or not, wind- or splash-dispersed, biotrophic or necrotrophic life style), on the epidemic characteristics (type and distribution of primary inoculum and type of spores released) and on the plant species (annual or perennial and resistant or not) and training system or conduct? The interactions scheme is complex but may be simplified, bearing in mind Liebig's Law of the Minimum: only some of the interactions are strong and important, with relatively few of them limiting the growth of the pathogen population at any given time and space (Berryman 2004). A good understanding of the pathosystem is required to select which of those interactions should be addressed, and when, in order to provide the best cost/benefit ratio for controlling disease development.

Canopy structure, pathogen development processes and microclimate Infection is most likely impacted by plant or canopy porosity and organ susceptibility as it occurs at the surface of susceptible organs. Plant or canopy porosity (e.g. LAI, internodal length and leaf spatial distribution) affects four micro-climatic variables (linked by the organ energy budget) critical for spore infection: *radiance*, *temperature*, *relative humidity*, and *wetness duration* (Fig. 2). If the effects are pathogen-dependent, microclimatic conditions before and after inoculation may all greatly influence the reaction to biotrophic pathogens.

An increase in radiance consecutive to increased porosity will generally decrease spore germination, but pathogens penetrating the plant through stomata, such as *Leveillula taurica*, can compensate for that effect (Rapilly 1991). Solar radiation has been found to affect spore survival of several airborne fungi (Aylor and Sanogo 1997; Ulevicius et al. 2004). After inoculation on grapevine leaf disks, spore germination and mycelium growth of powdery mildew were decreased by UVB radiation. Consequently, in the field, shaded exposure was more favourable to pathogen development than sunny exposure, while sunny, UVB-protected exposure was more favourable to disease than shaded exposition (Willocquet et al. 1996). The exposure of tomatoes to low light intensity predisposes leaves to the development of both the *B. cinerea* pathogen and its fungal biocontrol agent, *Clonostachys rosea*, (germination and mycelium growth are increased) without decreasing the ability

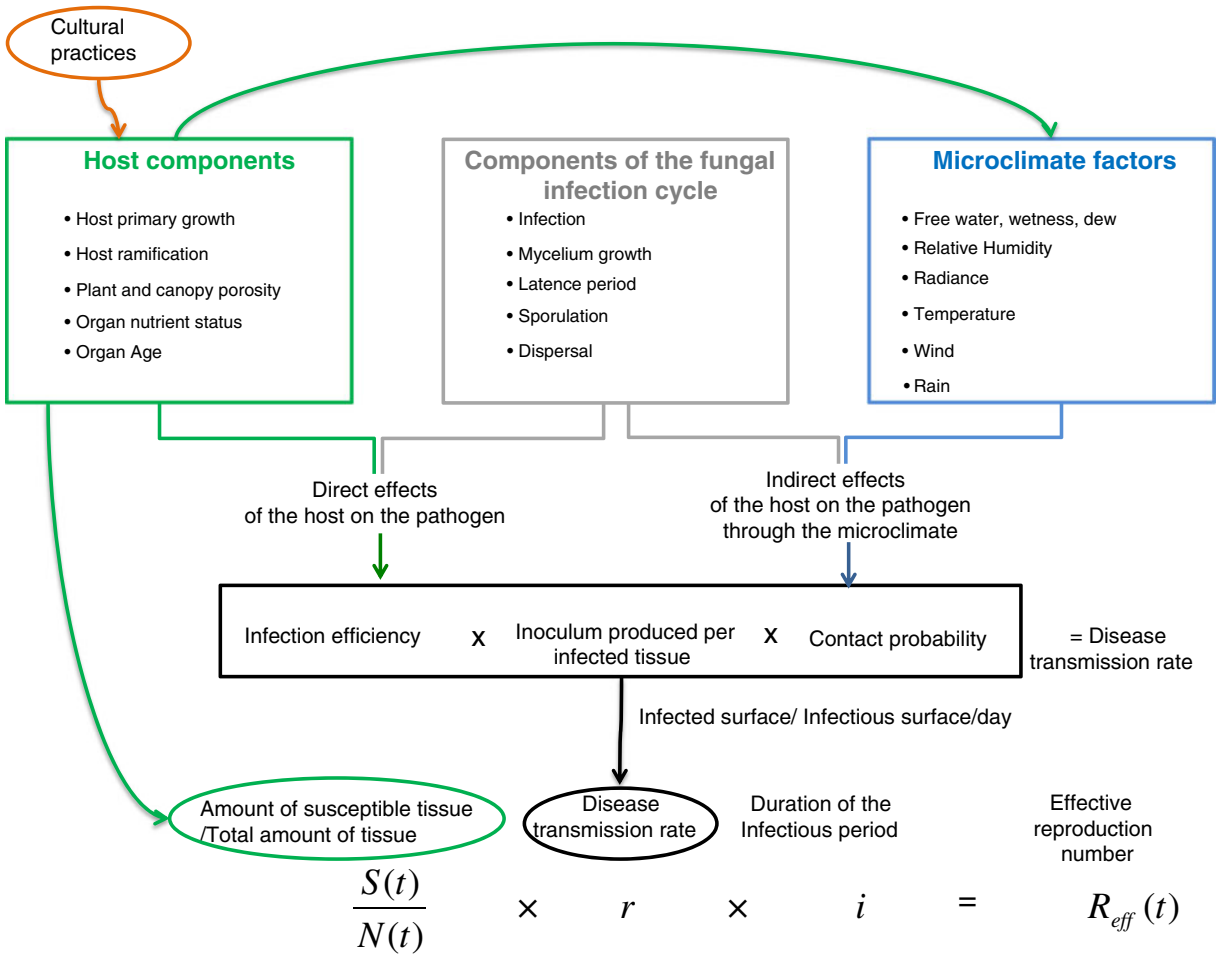

Fig. 2 Effect of host components, fungal infection cycle components and microclimate factors on epidemiological parameters (disease transmission rate and effective reproduction number)

of the biocontrol agent to control spore production in the pathogen (Shafia et al. 2001). Light could have indirectly decreased pathogen infection on berries through modifications of plant physiology, such as a decrease in pH and K concentration and an increase in polyphenol and anthocyanin concentration (Zahavi and Reuveni 2012). Indeed, berries picked in the field from two vine training systems were inoculated and incubated in controlled conditions and those that had been collected under the system which received 50 % less radiation intensity had higher disease incidence. After inoculation, berries incubated again under that system had a slightly higher disease incidence. In that study, plant leaf phenology, berry diameter and radiation were measured, enabling differences in disease incidence to be linked to architecture and microclimate rather than to global differences in phenology between the two training systems. On the contrary, light quantity and duration increased wheat infection efficiency to *Puccinia striiformis* by up to 36 % (de Vallavieille-Pope et al. 2002). A measurement of plant physiological status with optical sensors (chlorophyll fluorescence, nitrogen status, phenolic contents) can be useful to assess the average sun exposition of the organs and their nitrogen status (Tremblay et al. 2011) but also their age (Meyer et al. 2003).

What about modifications of temperature? Most plant pathogens are mesophilic fungi, with optimum temperatures for germination and development in the 18–25 °C range thereby exhibiting a wide range in development rate corresponding to the Boltzmann-Arrhenius model (Dell et al. 2011). In general, water-dependent pathogens such as downy mildews have lower optimum temperatures. As host management

and canopy structure have consequences on temperature modification that will generally impact the proportion of spores germinating and cause infection (Calonnec et al. 2008), with species- and even isolate-dependent cardinal temperatures. A highly variable temperature was measured and modelled within an apple tree (Saudreau et al. 2009), even if the average fruit temperature on the daily time scale corresponded to the average daily air temperature. Linking such a model with a virtual plant model (Saudreau et al. 2011) can be useful to test which tree architecture would be less conducive to infection by apple scab (increase in the number of latent periods with high temperature). However, the literature suggests that, for pathogens which are not dependent on free water for infection, an increase in temperature consecutive to a more aired canopy will not be a sufficiently strong limiting factor to control disease increase.

Wetness duration, expressed by the presence of free water on the leaf surface in the form of dew droplets, is a key variable, as free water is required for spore germination in many biotrophic pathogens, such as rusts (de Vallavieille-Pope et al. 2000), septoria tritici blotch (Shaw 1990) and downy mildews, as well as in many necrotrophic or hemibiotrophic pathogens, such as *Botrytis* or *Colletotrichum* species. Although it is often overlooked, dew that is usually formed during the night, can play a critical role, in particular in geographical areas where nightly temperatures may still be warm enough to allow fungal spores to germinate and take advantage of long periods of leaf wetness (Payne and Smith 2012). In such cases, dew-induced wetness periods may even be significantly longer than rain-induced wetness periods (Guyader and Bussière 2012). When comparing the canopy structure of yam plants between staking and ground growth, more dew deposition was measured on plants growing to up to 2 m high than on leaving vines growing directly on the ground, leading to an increase in severity of anthracnose caused by *Colletotrichum gloeosporioides*.

The pattern of variation in leaf wetness duration inside crops was shown to differ according to crop characteristics and shape (Sentelhas et al. 2005). In mature apple orchards and tall mature annual crops like corn, dew-induced leaf wetness duration was longer at the top than inside the canopies. In young coffee plants, and in a very well-aired grapevine, no significant differences were observed, either on dewy or rainy days, for leaf wetness duration between top and inside positions. On pea, leaf wetness duration due either to dew or rainfall was significantly modified by the architectural features of the canopy (Richard et al. under review). However, leaf wetness duration showed less variation between top and inside positions during rainy days in apple orchards and maize crops.

Microclimatic models have been used to estimate dew or leaf wetness, using energy balance equations. Physical leaf wetness simulation models (Dalla Marta et al. 2005; Huber and Gillespie 1992; Magarey et al. 2006) are complex and difficult to parameterise in a heterogeneous canopy but can be simplified by splitting the canopy into layers (Norman 1982). The number of days conducive to successful infection can be calculated during the whole season, or at a critical period of time for the epidemic, and can be compared between different canopy structures.

An increase in the duration of the latent period (the time elapsed from infection to the production of the next generation of spores) will cause a decrease in the number of reproduction cycles per growing season. In biotrophs, the duration of latent period is temperature-dependent (Analytis 1980); as for infection efficiency, any canopy feature that can modify the local temperature can potentially act on the latent period. During the latent period, necrotrophic and hemibiotrophic pathogens growing within the leaf tissue remain sensitive to temperature, and as such are likely to have a latent period insensitive to relative humidity or wetness.

Canopy structure, pathogen developmental processes and organs susceptibility Infection efficiency may also be dependent on the host organ developmental stage in many plant-pathogen interactions (Develey-Rivière and Galiana 2007). Organ stage can potentially vary with plant developmental stage or cultural practices. The involved mechanisms of resistance behind the age-related resistance (ontogenic resistance or receptivity), still a matter of speculation, differ from those involved in response to infection in classical defence systems (Gee et al. 2008). Ontogenic resistance has been described for many plant-pathogen systems on perennial (strawberry, grapevine, apple tree) (Carisse and Bouchard 2010; Gadoury et al. 2003; Li and Xu 2002) or annual (cucurbits, tobacco) (Ando et al. 2009; Hugot et al. 1999) plants. In grapevine, ontogenic resistance can be expressed against

several key pathogens (*B. cinerea*, *E. necator*, *Plasmopara viticola*, and *Guignardia bidwellii*) (Deytieux-Belleau et al. 2009; Ficke et al. 2003; Kennelly et al. 2005; Molitor and Berkelmann-Loehnertz 2011). It is therefore important to assess whether plant stage or cultural practices could enhance its effect in semi-controlled experiments with artificial inoculations of detached organs varying in age. As an example, grapevine leaves detached from shoots and vines characterised for their rate of growth were inoculated in the laboratory with powdery mildew (Schnee et al. 2011) showing a positive correlation between shoot development (rate of growth, leaf area) and the average shoot infection and sporulation levels. The explanatory factors were that shoots coming from highly vigorous vines had more young leaves at a given time, and a slightly higher susceptibility. The young susceptible leaves were those still acting as sink organs but more investigations have to be done to understand if the balance of source/sink organs can be controlled. In apples, enhanced vegetative growth induced by high N-supply to the trees is correlated with an increasing susceptibility to scab and a reduced accumulation of total phenolic compounds (Leser and Treutter 2005). In pea, controlled experiments showed that organs entering the senescence process have an increased receptivity to ascochyta blight (Richard et al. 2012b). Thus, any situation that accelerates the yellowing of the organs, such as shading and plant-to-plant competition, can increase their receptivity.

Canopy structure - spore dispersal

Canopy structure may affect spore dispersal in different ways, depending on the main mode of dispersal (wind or rain splash) (Sache 2000). For several wind-dispersed spores, such as the rust asexual spores (urediniospores), dispersal requires the fruiting bodies to be dry in order for the wind gusts to detach spores (Aylor 1990; Geagea et al. 2000; Rapilly et al. 1970) but not for splash-dispersed spores (Suffert and Sache 2011) or spores actively discharged in the environment when the fruiting bodies are wet (Ingold 1971). Once the spores are discharged in the environment, the main effect of canopy architecture on spore dispersal process, however, is the limitation of spread distance via the barrier effect. Wind dispersal provides long distance dispersal whereas rain dispersal provides short distance dispersal and has the particularity of vertical and upward canopy dispersal (Saint-Jean et al. 2004). Parameters of interest are: wind intensity, direction and turbulence properties, for wind dispersal (Aylor 1990), and rain quantity and rain energy for rain splash dispersal (Saint-Jean et al. 2006). LAI, canopy height, canopy roughness, and the thermal stratification, directly affect the wind transfer and turbulence in the boundary layer. LAI, leaf posture, and internodal length will directly modify interception of the raindrop and the following splash-droplet production. Accordingly, modifications of the microclimate inside the canopy can indirectly affect spore dispersal.

Properties of dispersal in relation with the canopy structure can be analyzed with the concept of genotype unit area introduced by (Garrett and Mundt 1999). Large genotype unit area can offer to the dispersed pathogens large amounts of susceptible tissue. On other hand genotype unit area could be reduced with the use of cultivar mixtures (Gigot et al. 2012; Saint-Jean et al. 2008). Artificial inoculation of wheat field plots with leaf rust (caused by *Puccinia triticina*) allowed us to highlight the effect of the structure of wheat canopy on the dispersal of spores after a single disease cycle (Frezal et al. 2009). The canopy structure was defined by leaf layers (leaves of the same age and position on the plant) and plant rows, spaced 17 cm from each other. The higher leaf density restricted spore dispersal along the row more than vertical dispersal (between layers). The effect was also dependent on the vertical position of the spore source. Such variations could be due to the lack of precision in the physical definition of the canopy (distance, angle between leaves), or to the unmeasured microclimate; however, the effect on dispersal was mostly attributed to barrier effects within the canopy. In splash-dispersed fungi, canopy structure was also demonstrated to act as a barrier to spore dispersal. An increased canopy density, obtained by increasing planting density, resulted in shorter dispersal distances for the splash-dispersed spores of northern joint vetch anthracnose on rice, caused by *C. gloeosporioides* f.sp. *aeschynomene* (Yang and Te Beest 1991), of strawberry anthracnose (Madden and Boudreau 1997), and of pea aschochyta blight (Schoeny et al. 2008). Wheat varieties differing in canopy height had differential effects on the dispersal of *M. graminicola* rain-splashed spores (Lovell et al. 2004). When the distance between leaf layers was increased, either by using different genotypes or through hormonal

treatment, fewer spores from the lower layers were able to reach the upper layers. Accordingly, in addition to a barrier effect, canopy structure may allow larger areas of susceptible leaf tissue to escape infection. For the same pathosystem, in order to separate the canopy architecture from other factors that affect disease development, such as cultivar resistance, in controlled conditions it was possible to quantify the difference of spore dispersal in two canopies of quasi-isogenic lines of a wheat cultivar (Mercia) differing only by one dwarf gene: RHT. In 43 % smaller (and denser) wheat canopy, the disease severity increased between 7 and 10 times, depending on the kind of rain (Girardin et al. 2012). That work confirms that distances between organ played a major role in disease development. In a wheat-clover mixed stand submitted to simulated rain, the horizontal and vertical dispersal of spores of *M. graminicola* were significantly reduced, compared with a pure wheat stand (Bannon and Cooke 1998). In pea-wheat mixed field plots, dispersal of ascochyta blight was reduced by up to 78 % in the intercrop, compared to the pure pea stand at full density (Schoeny et al. 2010). Wheat plants, therefore, provided a physical barrier to spore movement within the intercrop canopy early in the cropping season.

Experiments, either in controlled or semi-controlled conditions, can give more insight into the processes involved. That should help the experimenter to choose the most appropriate timing and host and microclimate variables having an impact on disease or pathogen components. Statistical approaches, such as PLS-path modelling analysis, can then be used to analyse complex systems with many qualitative or quantitative host and microclimate variables (Burie et al. 2011; Tenenhaus et al. 2005). That type of analysis allows us to explore and predict the relationships between various latent variables, as well as to quantify the weight of each manifest variable and, therefore to test which are the relevant dates and variables (Fig. 3). Measures can be performed at different scales (crop, plant, organ). The analysis also distinguishes between direct and indirect effects, a convenient way of disentangling factors. As an example, cultural practices with no direct effect (correlation) on disease can actually have an indirect effect on disease, by modifying plant growth, porosity or physiology.

Epidemiological parameters such as the rate of disease transmission or the effective reproduction number are more difficult to assess in the field, because susceptible or diseased leaf surfaces are very difficult to accurately estimate. In contrast, those parameters can be approached by modelling.

Models to describe and predict the effects of plant growth architecture on epidemics

Models coupling host growth and pathogen dynamics are required to assess and quantify the complex interactions between plant growth and architecture, and disease dynamics.

Three types of model have been proposed for that purpose: 1) analytical models describing the time-evolution of the proportion of diseased tissue, 2) compartmental models (SIR or SEIR type) describing the time-evolution of the surface area of tissue, and 3) discrete mechanistic architectural models or process-based models coupling plant growth and pathogen processes.

Analytical epidemiological models In the first analytical model of (Van der Plank 1963) the rate of disease increase is proportional to the amount of infectious (sporulating) tissue multiplied by the amount of available tissue. That model assumes a constant infection rate (multiplication factor of disease per unit), a constant latent period and random inoculum dispersal between infected and susceptible hosts. Therefore, the rate of infection is a composite parameter that takes into account several processes, such as infection efficiency, sporulation rate, and spore dispersal. The analogy between the logistic equation of Verhulst (Verhulst 1845) describing the self-limiting growth of a biological population and that of Van der Plank describing the evolution of the proportion of diseased tissue, required the simplifying assumption of a constant carrying capacity (host tissue). That type of model could not take into account any change in the environment, such as plant growth and plant susceptibility modifications. However, fitting it to disease progress curves, allowed the variation of the apparent infection rate to be shown. (Hau 1990) reviewed various analytical models able to take into account changes in the environment. The apparent infection rate could be a function of time, describing an eventual decrease of infection consecutive to a change in host susceptibility. According to Hau, host change was the only way to account for a decreasing disease progress

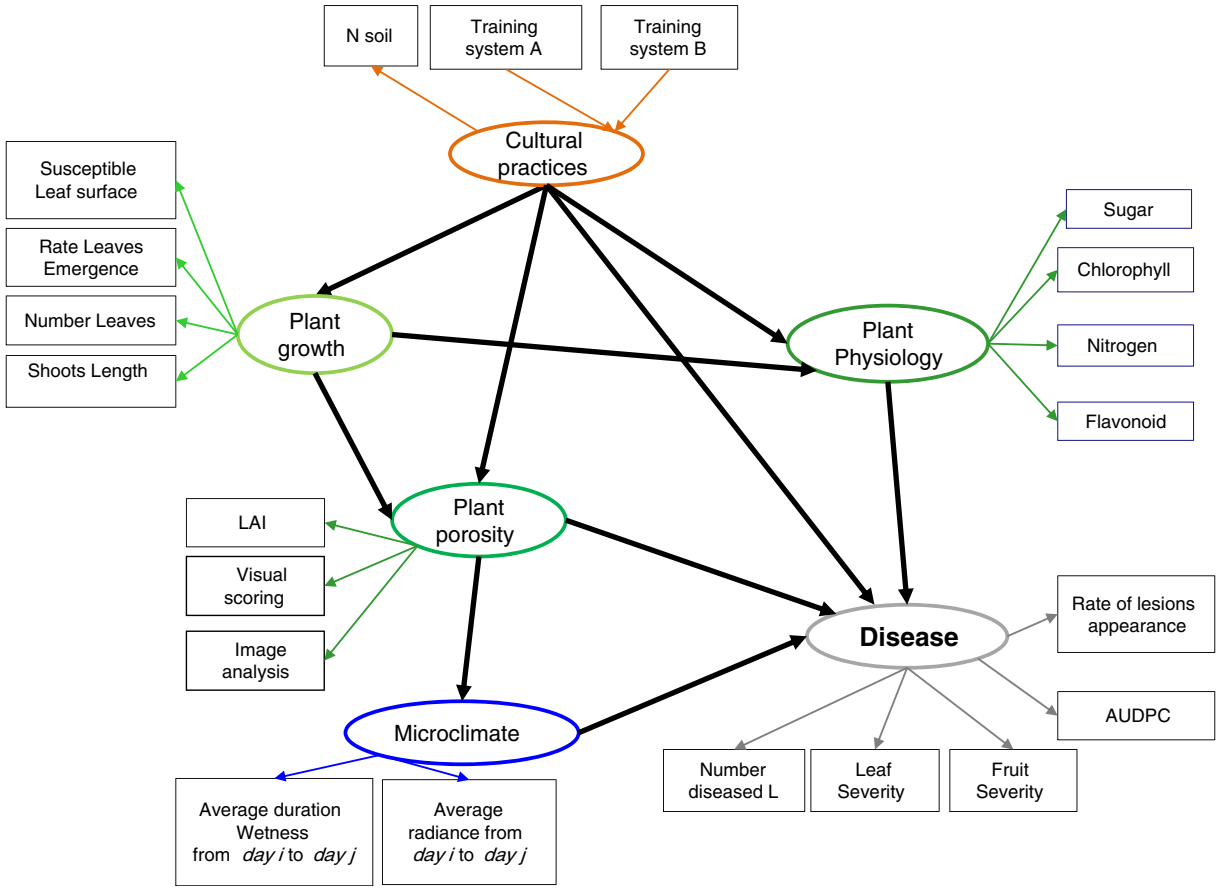

Fig. 3 Example of PLS-path model describing the relationships between the endogenous latent variable 'disease' and the other exogenous latent variables 'cultural practices', 'plant growth', 'plant or canopy porosity', and 'microclimate'. The model will evaluate the correlation between the different latent variables and the relative contribution of each exogenous variable to the endogenous disease variable as well as the weight of each observed variables (framed). Each observed variables can be measured at different phenological stages which may be relevant for the disease

curve, either by an increase in susceptible tissue (dilution effect) or by the loss of diseased tissue by defoliation (thinning-out effect). A more sophisticated analytical model was subsequently developed to take into account the crop density-dependent inoculum transmission (Ferrandino 2008). That approach incorporates a crop growth model, and an estimate for canopy filtration efficiency within the derivation of a basic epidemic model. The leaf area follows a logistic function of time, and the relationship of leaf area to canopy filtration efficiency is estimated using dimensional analysis and a gradient diffusion approximation for turbulent transport. The model does not take into account variation in host susceptibility. Three types of model are compared: frequency-dependent or *true mass-action* (the proportion of spores caught by the canopy is constant, or the rate of disease transmission is constant); density-dependent or *pseudo mass-action* (the proportion of spores caught is dependent on leaf density); and *canopy filtration* (pseudo mass-action at early crop growth moving to true mass action when canopy increases).

The impact of crop growth and canopy filtration on the basic reproduction ratio R_0 (Heesterbeek 2002; Van den Bosch et al. 2008), defined here as "the number of secondary cases produced, in a completely susceptible population, by a typical single infected individual during its entire period of infectiousness" was studied. Here, the unit of infection is one lesion. As a consequence of that definition, R_0 plays the role of an epidemic threshold. If $R_0>1$, then the epidemic will spread. On the other hand, if $R_0<1$, the epidemic

will die out. It appeared that the overall effect of increasing crop growth was to delay the normal initial exponential growth of the epidemic. The magnitude of that time delay was explicitly dependent on the crop growth rate and of the wind-dependent canopy filtration probability. In other words: the start of the epidemic was synchronised with the flush of crop growth, and the time at which the inoculum is introduced into the model was not important.

Compartmental models The first SIR model (Susceptible–Infectious–Recovered), still the starting point of many modelling studies in human, animal or plant disease epidemiology, was developed to describe the evolution over time of the number of infected individuals in a closed population (Kermack and Mc Kendrick 1927). That model was designed for populations with highly–contrasted time scales between host and pathogen. It assumed a constant population size (no births, no deaths due either to disease or natural causes); no latent period (susceptible become instantaneously infectious after exposure to the pathogen); and a period of infectiousness equal to disease duration. The model further assumed a completely homogeneous population, with no age, spatial, or social structure in the target population. It consisted of a system of coupled nonlinear ordinary differential equations, describing the evolution over time of the number of susceptible, infected, and recovered individuals, featuring two parameters: infection rate, and recovery rate. The key value governing the disease dynamics is, again, the so-called epidemiological threshold R_0. From the second half of the 20th century, various structuring variables were added to that model, e.g. spatial distribution of hosts and their dispersal mode; chronological age of individuals; age of the infection and the status of the host. According to the studied epidemics, those variables could play an important role in the invasion and persistence of the epidemic. Those models have been introduced much more recently in plant disease epidemiology. Furthermore, as time is continuous, tissue generations in a given state are not separated by rate of change in the various states (healthy or susceptible, infectious, exposed and removed). Jeger (1986) was the first to propose a simple compartmental model with two differential equations, describing the growth of host tissue and the growth of diseased tissue, respectively. A stable endemic equilibrium was shown to exist, depending on the parameter set values. Then more sophisticated SIR (Susceptible–Infectious–Removed) or SEIR models (Susceptible–Exposed–Infectious–Removed) (also called HLSR for Healthy–Latent–Infectious–Post-infectious) were developed in plant disease epidemiology. Those models are reviewed in (Madden et al. 2007).

The key epidemiological (composite) parameter is the basic reproductive number, R_0, which predicts most qualitative characteristics of the host-pathogen system dynamics (it is closely related to the final size of the epidemic), and it can be used to devise and assess the efficacy of disease control measures (Segarra et al. 2001). R_0 also has a clear biological significance with a numerical value independent of the form of the model considered in this work, and is directly comparable for different pathosystems. For a non-growing host, its value is: $R_0=\beta H_0 i$, with β the disease transmission rate per capita (infection efficiency x contact probability x spores produced per infected tissue), H_0, the amount of primary healthy tissue, i, the infectious period.

Host growth, simply defined as an increasing number of leaflets at each time step, was introduced in SEIR models (Jeger and Vandenbosch 1994; Onstad 1992). It appears then that the growth of host tissue and host densities increase the likelihood of persistence (presence of a pathogen in an ecological proper spatial unit over many generations) (Gilligan and Van den Bosch 2008). The degree of persistence was directly related to the parameter of host growth and to the length of the infection cycle (latent period plus infectious period). In a recent paper (Burie et al. 2011), the SEIRT model was modified to include a logistic growth of the foliar area and a compartment to account for ontogenic resistance (a varying proportion of the healthy foliar area that is not susceptible or becomes resistant to the disease). That model also differs from those in (Segarra et al. 2001): it uses a frequency dependent rather that a density dependent assumption for the contact term to handle the host growth and the ontogenic resistance. $\left(R_{eff}(t) = r\,i\,S(t)/N(t)\right)$, the effective reproduction number, which depends on time, was considered instead of R_0 with N(t) being the total leaf area. Note that that term is also used in (Ferrandino 2008), but with a different meaning. In the grapevine-powdery mildew system, R_{eff} decreases during host growth (vine vigour) because the amount of susceptible tissue decreases in such a way that late epidemics have a lower chance of becoming

established. Agronomic practices such as shoot topping, which have an effect on ramification and reiteration, can again increase R_{eff} with variations due to climatic conditions acting on vine growth (Burie et al. 2011). In that example, the simulation of R_{eff} and the understanding of its behaviour, was possible because the pathogen processes and host growth had been previously quantified and modelled in a plant architectural–pathogen model (Calonnec et al. 2008). The ordinary differential equations SEIRT model is under extension at the plot scale into a partially differential equations model, building a Reaction–diffusion system in which the state variables are the leaf densities (leaf area index), in order to add the description of spore dispersal both at short and long ranges and to estimate biologically relevant parameters. Numerical simulations should allow us to explore the influence of heterogeneities within the plot and the plant (e.g. vigour), and between plots (e. g. phenology, density of plantation, spatial organization) on epidemic development (Burie et al. 2012).

Discrete mechanistic process based plant-pathogen model In the last decade, computer simulation models of plant functioning and growth have been developed, with the aim of simulating complex interactions between plant architecture, and the physical and biological processes that drive plant development at various spatio-temporal scales (Godin and Sinoquet 2005). That has led to the emergence of Functional-Structural Plant Models (FSPM) for annual crops (maize, (Fournier and Andrieu 1999), wheat, (Fournier et al. 2003), grapevine (Pallas et al. 2009), kiwi (Cieslak et al. 2011)) and fruit trees [peach (Allen et al. 2005; Lopez et al. 2008); apple (Costes et al. 2008)]. Those models have integrated different physical and physiological processes, and have varied in the level of details considered for the spatial representation of the plant (considering individual organs, set of organs or entire plants).

FSPMs coupled to models of pathogen development seem to be a key approach in investigating the complex relationship between host growth-pathogen-climate and crop management at different scales, and in exploring the potential of plant growth and cropping practices to become a lever to control disease outbreaks. Furthermore, functions and parameters in such models have the advantage of having a biological meaning. The first virtual plant growth to couple a disease damage model was devised 20 years ago (Wilson and Chakraborty 1998), opening up a still flourishing field of research (Calonnec et al. 2008; Robert et al. 2008; Pangga et al. 2011). Simulations performed with the vine-powdery mildew model (Burie et al. 2011; Calonnec et al. 2008) allowed us to assess the effect of vine vigour on the epidemics and to point out the existence of variation depending on climatic scenarios. Variations between years can mainly be explained by the variation in vine growth rather than by a direct influence of temperature on the pathogen. The results of those simulations were consistent with the field experiments. The severity of the disease is correlated with the rate of leaf emergence early in the season and the development of secondary shoots later on, combined with the number of infected leaves at flowering. However, the model indicates that potential variation of vine growth according to the climatic scenario could alter the synchronicity between disease and the production of susceptible organs, and possibly delay the severity of the disease. In the wheat–*M. graminicola* model (Robert et al. 2008) the rhythm of development of the phyllochron was shown to have a major effect on the course of the epidemic. Although it was able to simulate disease progress on the different leaf layers for contrasted sowing density treatments (Baccar et al. 2011), the model failed to predict the slightly higher rate of lesion progress observed at the lowest sowing density. The canopy reconstruction scenario, in which inter-plant variability was taken into account, yielded the best agreement between measured and simulated epidemics. It was possible to compare the predicted and measured epidemics on detailed variables. However, the complex and dynamic responses to sowing density made it difficult to test the model precisely, and to disentangle the various factors involved.

Due to the complexity and number of parameters of FSPM-disease models, upscaling simulation at the plot scale may be challenging. On the other hand, SEIR models may be too simple to link parameters to the various components of the system (e.g. plant-pathogen-cultural practices).

To overcome those difficulties, in vine powdery mildew (Burie et al. 2012, 2011) the SEIRT model was calibrated by using output from the architectural model (Calonnec et al. 2008). It then became possible to obtain a set of biologically meaningful parameter values for the rate of host growth and the rate of

disease transmission. Different scenarios of vine growth based on contrasting climatic scenarios could be modelled; various types of epidemics were generated using different initial conditions.

A generic model was recently developed to simulate air borne diseases taking into account the crop architecture (Casadebaig et al. 2012). The model combines the advantages of the biological significance of a 3D plant-pathogen model and the simplicity of an SIR model. Canopy structure is approximated as a network of functional units without geometric attributes. A functional unit represents a mean plant, individual plants or a sub-part of a plant depending on the pathosystem considered. Spore dispersal is represented by a network of connections between functional units. Functional units are mainly described by leaf area, but height and porosity attributes are also considered. As in SIR model types, the total leaf area in the functional unit is divided into compartments of Healthy, Latent, Infectious and Removed tissue. The active leaf area is defined as the tissue in expansion (E) and not yet senescent (S). Stem height elongation, leaf area expansion and the leaf senescence rate are modelled using a logistic equation driven by thermal time and three parameters. The model is still a research tool which needs additional features (crop management actions, microclimatic environment at the local scale…) to produce outputs likely to be used in agricultural development. However, the model based on functional units and a network of connections showed

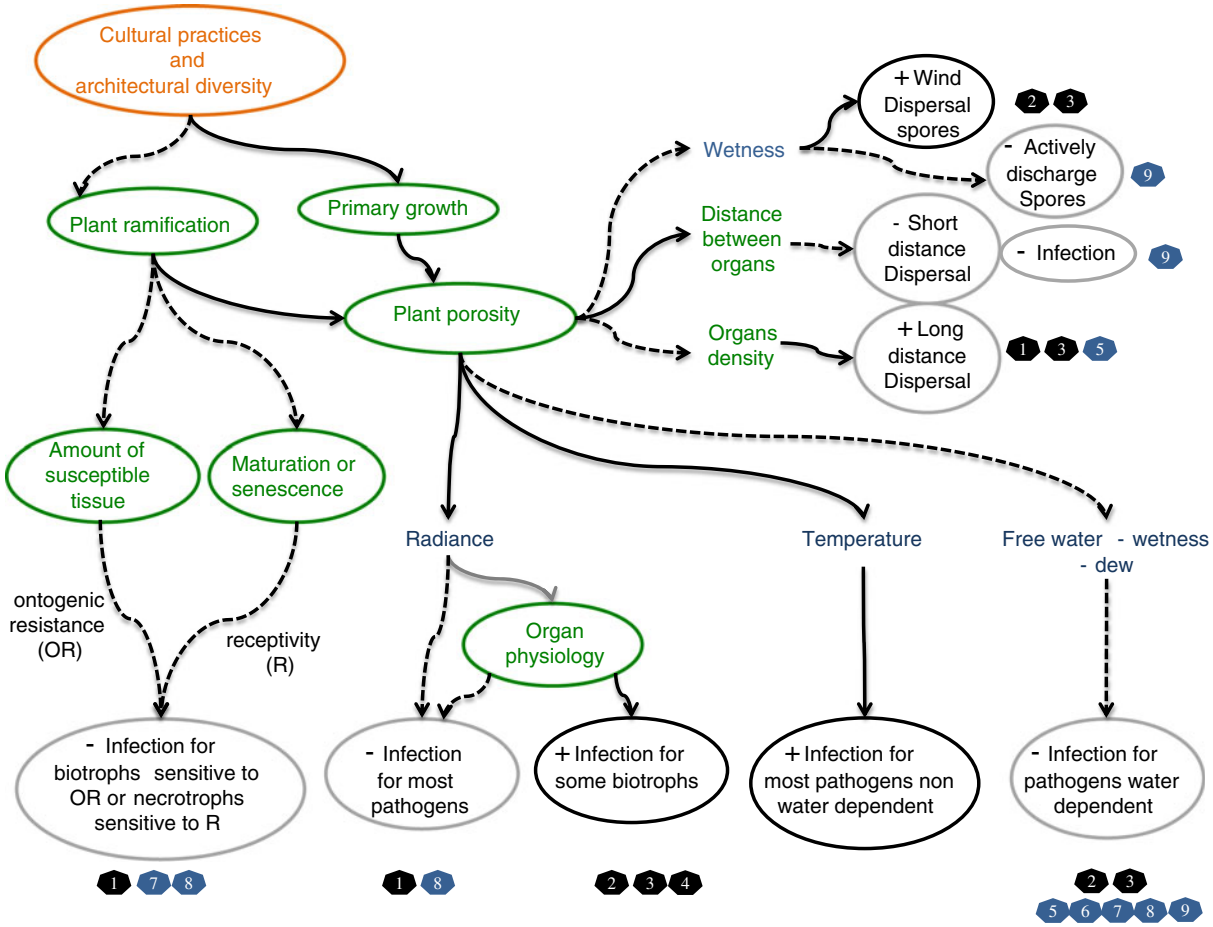

Fig. 4 Reported effects on pathogen processes of cultural practices acting on plant growth (primary growth and ramification) and increasing canopy porosity. Dashed black lines: negative effects, plain black lines: positive effects, grey line: little known effects. 1=*Erysiphe necator*, 2=*Puccinia striiformis*, 3=*Puccinia triticina*, 4=, *Leveillula taurica*, 5=*Colletotrichum* sp., 6=*Venturia ineaqualis*, 7=*Mycosphaerella pinodes*, 8=*Botrytis cinerea*, 9=*Mycosphaerella graminicola*. Water dependent pathogens are numbered in blue, non-water dependent in black

sufficient flexibility to represent different crop/pathogen systems with a single model structure, and to describe two types of epidemics.

Conclusion

In this review, we have discussed many examples of disease and pathogen processes that can be altered by modifying the plant/crop growth and architecture through genetic (architectural diversity) or cultural practices. Experimental approaches in semi-controlled conditions, with a concomitant characterisation of host, microclimate and disease, generally offer a better understanding and analysis of the processes impacted. In most cases, a reduction in plant growth, and an increase in plant or crop porosity, will reduce infection efficiency and spore dispersal (Fig. 4) and, consequently, disease development and spread. However, increasing porosity may have opposite effects, depending on the spore dispersal mechanism. Short-range dispersal by splashing may be decreased. In contrast long-range dispersal by wind may be increased consecutive to a decreased barrier effect. Pathogens dispersed by wind with dryer environmental conditions favouring their spore dispersal or biotrophs positively influenced by an increase in light and temperature, may gain from a reduction in plant growth and an increase in porosity. Biotrophs would however generally be disadvantaged by a lower production of young organs, whereas necrotrophs would be disadvantaged by any environmental conditions that delay organ receptivity. The result of these complex interactions with either positive or negative effects on the disease must be assessed by modelling. For this, modelling must, explicitly take into account the pathogen processes that are most impacted by the growth of the plant, to better assess and exploit their effect on the diseases. For that, we need to improve the current models, by taking into account, for instance, physiological processes. Improving the sensitivity analysis of those complex models is also critical in order to make them easily explorable and useful, so as to propose more environmentally friendly strategies of disease control.

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