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Current knowledge on plant/canopy architectural traits that reduce the expression and development of epidemics

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Abstract To reduce the use of pesticides, innovative studies have been developed to introduce the plant as the centre of the crop protection system. The aim of this paper is to explain how architectural traits of plants and canopies induce a more or less severe epidemic and how they may be modified in order to reduce disease development. In particular, it focuses on three key questions: i) which processes linked to epidemics can be influenced by architecture ii) how can architecture be characterized relative to these modes of action, and iii) how can these effects be explored and exploited? The roles of plant/canopy architecture on inoculum interception, on epidemic development via the microclimate and on tissue receptivity are discussed. In addition, the concepts of disease avoidance, canopy porosity and an ideotype unfavourable for disease development are described. This paper shows that many advances have already been made, but progress is still required in four main fields: microclimatology, mathematical modelling of plants, molecular genetics and ideotype conception.

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Introduction

The increase in agricultural production over the past 50 years has been attributed to the improvement of yield potential of major crop cultivars, the improvement of seed quality and the massive use of plant health products during the cropping season. Alternative control methods to reduce the number of pesticide applications have not been extensively developed, and often resistant cultivars have enjoyed a limited success because of the breakdown of major-gene resistance and of the usually lower yields of resistant cultivars compared with their susceptible counterparts sprayed with pesticides. The very high success of controlling plant pests through pesticide applications has limited the amount of attention paid to cultural practices in crop protection systems, in spite of the development of pest genotypes resistant to major pesticide families. The complex problems for human health and environmental protection generated by crop protection systems that have primarily relied on pesticides over the past 30 years, have led to the development of products with significantly reduced toxicity by the pesticide industry and growers' organisations since the turn of the millennium, but also to increased efforts to reduce their overall use. These developments have been strengthened by a global movement on regulation and legislation towards stricter criteria for pesticide

registration, and by setting ambitious goals for a reduction of pesticide applications. For example, Europe has adopted a revised legislative arsenal on pesticides including several Directives on pesticide registration, marketing (Regulation 1107–2009) and sustainable use (Directive_2009_128_EC), and the French government currently enforces a national action plan for pesticide reduction (the Ecophyto 2018 plan) which aims at halving pesticide applications over a 10-year period. This legislation also withdraws from the market the most toxic molecules (Butault et al. 2010; Ecophyto 2018: http://agriculture.gouv.fr/Ecophyto-in-English). Consequently, low-pesticide systems based on the development of innovative control methods need to be developed and their performance evaluated.

Setting up and implementing alternatives to the 'pesticide only' approach requires research to improve currently existing methods (decision support systems, longer rotations, more robust cultivars), to assess their applicability in practice, but also to develop new knowledge that can be put to use in integrated and innovative crop protection and crop production systems. Among these methods, the exploitation of the architectural features of plants and canopies to reduce disease development has been initiated, but remains insufficiently analysed. Research developed on this topic aims at revisiting the plant - disease interactions within host canopies, in order: i) to analyse how plant architectural traits modify the epidemiological processes, ii) to define which of these traits (alone or in combination, at the scale of the plant or the canopy) can reduce epidemic progress, and iii) to jointly model plant and epidemic disease development.

Plant architecture is a term applied to the organization of plant components in space, which can change with time (Godin et al. 1999). Plant characteristics, mainly plant height, the number of branches, the leaf area, the respective position of the organs (leaf, stem, flowers and fruits), are genetically controlled, but also depend on environmental influences and/or human interventions. In addition, these architectural characteristics and their general form vary during the growth/development stages of plants (Costes et al. 2003; Renton et al. 2006; Barthélémy and Caraglio 2007). They can be modified by human intervention at sowing or planting (plant density, date of sowing (or planting), row spacing, etc), as well as during the vegetative period (irrigation, fertilisation, pruning and trimming, etc), resulting in a multiplicity of plant and canopy architectures.

The aim of this review, which will focus on aerial diseases caused by fungi, is to describe and explain from a global perspective how plant architectural traits induce a more or less severe epidemic, and how pathogens may have adapted to exploit these traits, but not to give an exhaustive presentation of the mechanisms involved in disease reduction in different canopies. In particular, it will focus on three key questions: i) which processes linked to epidemics can architecture influence, ii) how can architecture be characterised relative to these modes of action, and iii) how can its effect be explored and exploited?

Which processes linked to epidemics can architecture influence?

Disease is the consequence of the dynamic interaction between the plant and the pathogen under the influence of the environment and human actions. Due to the plant/canopy plasticity during crop development and the localisation of inoculum sources within the canopy, it is important to consider the simultaneous dynamics of plant/canopy architecture and epidemic development during the cropping season, rather than a snapshot of crop/plant architecture at a single moment or developmental stage. The success of disease epidemics will depend on the initial inoculum pressure and the climatic conditions, but also on the dynamics of canopy architecture and of host receptivity to infection over time. The confrontation between the pathogen and the plant therefore raises three major issues: i) can architecture modify inoculum interception, ii) how does architecture drive the occurrence of microclimatic conditions favourable to epidemic development, and iii) can architecture change the dynamics of tissue receptivity?

Plant architecture and inoculum interception A pathogen's life cycle consists of several successive phases: inoculum interception by the plant, infection (germination, penetration), plant colonisation, spore production, and dispersion (wind or splash dispersion) to other organs, plants and/or canopies (Agrios 2005). Inoculum interception may be qualified as a passive phenomenon, which is not the case for insects where host choice is active. The pathogens may be characterised by several trophic status (from biotrophic to necrotrophic) and the observed symptoms may affect only one or several organs (leaf, stem, flower, fruit and

grains). Schoeny et al. (2008) showed the relationship between the leaf area index (LAI) and the horizontal dispersal gradient of *Mycosphaerella pinodes* pycniospores on pea. The leaves density measured through the LAI had an effect on the barrier rate increasing with canopy LAI. Similar effects were previously observed in the case of *Colletotrichum acutatum* on strawberry (Yang et al. 1990; Madden et al. 1993), suggesting that the barrier effect is a major contributor to architectural effects in a wide range of pathosystems. For ascochyta blight on chickpea (*Didymella rabiei*), Chang et al. (2007) showed that the high plant densities consistently associated with high levels of ascochyta blight are due on one hand to the high number of plants available to intercept inoculum, and on the other hand to the reduction of air movement within dense canopies, thereby maintaining a more humid microclimate that favours disease development.

Plant architecture and favourable microclimate for epidemic development Most fungal and bacterial pathogens need water (high level of relative humidity, dew, surface moisture of the organ) and certain temperatures to germinate at the surface of the host tissues and to penetrate the host tissues. Most pathogens also need water (rain, passive water movement) and/or wind for dispersal (Mc Cartney and Fitt 1998). In apple orchards, Simon et al. (2006) showed that some trimming systems led to a higher aeration within the trees and, therefore shorter periods of wetness, hence less apple scab infection (*Venturia inaequalis*).

The consequence of unfavourable effects of architectural features of the canopy on disease onset and development is described as 'disease avoidance' or 'disease escape'. This occurs when susceptible plants do not become infected because the factors necessary for disease do not take place at the proper time or for a sufficiently long period (Agrios 2005). Generally, a plant or canopy architecture unfavourable to an aerial epidemic may result in the total avoidance of disease expression, but more frequently reduces disease severity rather than preventing infection completely. Thus, Schwartz et al. (1978) and Park (1993) suggested that, in addition to genetic resistance, the use of a plant architecture which produces a less favourable microenvironment for fungal infection could significantly reduce disease. Coyne et al (1974) indeed observed that a microclimate unfavourable to the development of white mold (*Whetzelina sclerotiorum*) was generated within the canopy by the architecture of some dry bean cultivars. Blad et al. (1978) and Weiss et al. (1980) showed that a very low incidence of white mold is observed in the most open canopies characterised by a warm and dry microclimate, whereas dense canopies with temperatures below 30 °C and high leaf wetness duration and intensity, are highly conducive to the disease. This avoidance mechanism may be attributed to greater air movement within and beneath the canopy by way of a 'tunnel effect' above the open furrow, which contributes to faster drying of the foliage and the soil surface (Fuller et al. 1984). Disease avoidance is frequently described in intercropping systems. For example, mixed crops of faba bean and maize or faba bean and barley reduced the severity of chocolate spot on faba bean (*Botrytis fabae*), because of an increased air flow and consequently less humid conditions within the canopy in comparison to faba bean alone (Sahile et al. 2008).

Plant architecture and tissue receptivity to infection Canopy architecture can modify the physiology of plant organs, leading to major changes in their receptivity to infection and disease. The term receptivity may have different levels of acceptability among authors. For example, for several rust researchers, low receptivity is associated with low-rusting that is partial resistance (Luke et al. 1981). In this paper, we use the term receptivity defined by Rapilly (1991) as the stage of a host tissue at which its level of susceptibility to a pathogen increases independently of all genetic resistance aspects. In particular, architecture impacts senescence, which is often a major factor for tissue receptivity to infection. However, accelerated senescence can either enhance or reduce epidemic development, depending on the pathosystem. For instance, faster senescence (measured by yellowing of the organs) due to shading and plant-to-plant competition is an important component of higher ascochyta blight severity in dense pea stands (Richard et al. 2012). By contrast, ontogenic resistance was defined by Agrios (2005) as an increase in the degree of resistance of a plant to a pathogen with age and the development stage of the plant. In the case of grapevine, this phenomenon which was linked to higher sugar contents in grape leaves, reduces the period of susceptibility to grape powdery mildew (*Erysiphe necator*) to the first few days following leaf emergence (Schnee et al. 2011).

How to characterise architecture relative to these processes?

As shown above, architecture primarily influences disease development through the spatial density and relative position of organs: it is those traits that determine inoculum interception, microclimate and physiological changes within canopies. These architectural traits relevant for epidemiological investigations could be best integrated into a single architectural variable, the canopy porosity. Several authors have used the integrative terms "porous" or "porosity" to express the ability of the air to go through the canopy and to dry the leaf area, but without giving a precise definition for porosity. Deshpande (1992) characterized the dry bean canopy porosity with a scale scored from 1 to 5 where 1=open canopy with the soil surface between rows completely visible and 5=completely closed canopy over the furrow, no soil visible. An interesting definition of 'optical porosity of a barrier' was made by Lazzaro et al. (2008) in the case of the hedgerows. These authors defined it as the percentage of silhouette that can be crossed by light. This is the sum of the 'holes' in the canopy that allows seeing beyond. As with the cultivated species, the hedgerow acts as a barrier to air flow and the spatial arrangement of branches and leaves may significantly modify the air flow in the canopy.

Porosity characteristics are not easy to describe. In the case of *Phaseolus vulgaris*, an attempt was developed by Campbell (1984), who defined field scoring scales to characterize the canopy components in terms of plant architecture: plant uprightness (from 1: most upright to 5: prostrate), branching over the tunnel formed by the canopy above the furrow (from 1: open to 5: no porosity), tunnel size within furrow (from 1: open rows to 5: no tunnel), or branch density over the row (from 1: highly porous to 5: extremely dense). Unfortunately, these scores are hardly cumulative, and therefore do not provide a quantitative, aggregated measure of porosity. An alternative is to measure crop porosity directly, either through diffuse non-intercepted radiation or from fractal analysis of digitalised 2D pictures of the canopy. Diffuse non-intercepted radiation (DIFN) is an accurate and direct measure of porosity; values range from 0 to 1, where 0 indicates that the sky is not visible to the sensor (entirely compact canopy) and 1 indicates that the foliage is not visible by the sensor (entirely open canopy). Campbell (1984) also used a fractal analysis to measure the light penetration. It consists of taking digital pictures of the top and of the front of the canopy, and to analyse these simultaneously with an image-fractal program, which calculates the count of the open areas in the canopy. The fractal dimension ranges between 1 and 2, where 1 corresponds to a canopy with no tunnel and no porosity and 2 to a totally bright canopy with very little foliage between the rows. This last method needs to be standardised. Several authors have also studied the light microclimate among vegetation components inside the canopy from 3D models (Sinoquet et al. 1998; Massonnet et al. 2008).

In epidemiology, the concept of canopy porosity is not only used to express disease avoidance under the influence of the air movement within the canopy, and consequently the rapid drying of the foliage (Fuller et al. 1984), but also to describe spore propagation inside the canopy. Thus, Deshpande (1992) concluded that a porous canopy would contribute to avoidance of white mold by maintaining effective air circulation in the dry bean canopy. The architectural trait of the canopy which is synonymous with 'porous' is 'upright and open', as opposed to 'prostrate and dense' (Deshpande et al. 1995). For the rust on soybean, Andrade et al. (2009) established a forecast model for escape rate of spores of *Phakopsora pachyrhizi* in relation to the wind flow within soybean canopies. These authors explain that the escape rate of spores depends on the interaction between spores and turbulence within and above an infected canopy, and on the filtering capacity of the canopy to trap upward-travelling spores. The 'barrier effect' described by numerous authors, generally due to high stem densities which prevent horizontal and vertical spore dispersal, clearly corresponds with non-porous canopies.

Canopy porosity can also be approached with the notion of density. Thus in the case of white mold on dry bean, Schwartz et al. (1978) calculated the canopy density (cm/g) as total leaf area (cm^2)/average plant height (cm)×total dry weight (g). On five genotypes, the total canopy density ranging from 2.2 to 3.4, and genotypes with lower canopy densities were significantly less prone to infection. An alternative measure is Leaf Area Density (LAD), defined by Treuhaft et al. (2002) as the total one-sided leaf surface area per unit volume in the canopy. LAD is a good indicator of forest biomass. In plant disease epidemiology, more than plant biomass, this measure may be interesting to assess the canopy density and its impact for intra-canopy microclimate. In the case of grapevine, the strong increase in canopy density in the vigorous stands leading to

microclimatic conditions (leaf water potential and relative humidity) more conducive to grey mould (*Botrytis cinerea*) was called 'plant vigour' (Valdés-Gomez et al. 2008). Shoot vigour which is one feature of plant architecture related to microclimate, can be evaluated through the rate of leaf appearance, shoot extension and/or increase in cane diameter or biomass. Higher shoot vigour is associated with a significant increase in grey mould incidence at harvest, because leaf water potential and relative humidity inside the canopy increased as vines were more vigorous.

How to explore and exploit these mechanisms for better disease management?

Understanding the complex interactions between architecture and epidemic development obviously requires a true multidisciplinary approach, but also the building of new concepts and methodologies. Many steps forward have already been made, but progress is still required on three main fields: microclimatology, mathematical modelling and molecular genetics. Progress in these areas will contribute to define the plant ideotype unfavourable (or less favourable) to a disease epidemic.

Microclimatology As outlined above, architecture strongly impacts the microclimatic conditions within a canopy, but one important question is to know if it is necessary to consider the microclimatic variables to explain the epidemic development of the diseases, compared to the climatic ones measured outside the canopy (mesoclimate). An interesting example showing the importance of the microenvironmental factors on brown patch disease development on tall fescue was described by Giesler et al. (1996). These authors demonstrated how plant densities (low and high) may act on leaf wetness duration, relative humidity and temperatures (foliage and air), among the climatic conditions observed during three successive seasons. For example, they observed that leaf wetness duration averaged over 10 days was 0.8 h longer in high density than in low density and that relative humidity above 90 % was 2.3 h longer in the highest density. Fortunately, it is now possible to measure and predict most microclimatic variables within a crop canopy. The studies developed for instance by Huber and Gillespie (1992), Makowski et al. (2011) or Bregaglio et al. (2011) on leaf wetness duration and its integration in epidemiological models, are interesting examples. The next stage is now to analyse not only the microclimate within the canopy, but the phylloclimate, i.e. the climate each plant organ, and consequently each pathogen propagule, is exposed to (Chelle 2005). In this type of study, the question is to know how the microclimate may act on the disease development at the organ scale.

Mathematical modelling As shown above, architecture acts on epidemics by the way of complex and sometimes contradictory effects. This complex interplay of direct and indirect effects makes it hard to predict and optimise the architectural traits that will restrict most the disease dynamics from field experiments alone. This is why the development of mathematical models, allowing the quantification of these effects and interactions and the making of predictions through simulation, is of utmost importance. Several attempts in these directions have already shown the power of mathematical models. Ferrandino's (2008) paper is a good example of what mathematical modelling can contribute to the study of the effect of crop growth and canopy filtration on the dynamics of plant diseases spread by aerially dispersed spores. This author, by studying the simultaneous development of host and pathogen, showed that crop growth may have major impact on the development of plant disease epidemics occurring during the vegetative phase of crop growth. More complex mechanistic models have also been developed (Calonnec et al. 2008; Baccar et al. 2011). These models require 'virtual plants', i.e. a mathematical representation of the host structure. Generic 3D descriptions of plant structures are offered by the 'L system' algorithm developed by Prusinkiewicz et al. (1997) but can also be obtained by field measurements, combining description with digitization of plant architectures (Godin et al. 1999), which integrates topology and geometry. However, not all epidemiological models require such detailed descriptions of plant architecture. For instance, Wilson and Chakraborty (1998) developed a virtual plant model to study plant disease interactions. In addition, a new approach to investigate plant-pathogen interactions linked to canopy architecture was described by Robert et al. (2008) and Casadebaig et al. (2012). Finding the proper description of architecture – possibly through integrative variables such as canopy porosity (see above) – and of epidemiological mechanisms remains a major challenge for future work to keep algorithms tractable and

simulations possible within a reasonable time frame while maintaining biological relevance.

Molecular genetics The more intense exploration of plant genomes through better phenotyping techniques and the development of molecular markers and of whole genome sequences, helps in mapping the genes responsible for different architectural traits (Sakamoto and Matsuoka 2004; Ross et al. 2005; Turnbull 2005), but also those for partial resistance. In some cases, genes responsible for architectural traits and resistance QTLs co-localise. For example, Prioul et al. (2004) observed a high relationship between QTLs of pea for resistance to *M. pinodes*, plant height and flowering date. Similarly, Miklas et al. (2003) observed QTLs of resistance to white mold in snap bean associated with canopy height, lodging traits or internode length. The next challenge is to determine whether these co-locations indicate pleiotropic effects of the same genes, or result from insufficient resolution of QTL mapping. The advent of metabolomics, which allows a high-throughput analysis of physiological processes, will be also of great help to unravel the mechanisms behind tissue receptivity, senescence and ontogenic resistance, and to determine whether these mechanisms are also linked to genes involved in architecture.

Ideotype conception As a microclimate unfavourable to disease development may be induced by the canopy, several authors proposed the concept of an architectural ideotype unfavourable to disease epidemics. Coyne et al (1974) defined dry bean ideotypes suitable to limit white mold development to be of sturdy stiff upright determinate or short indeterminate plant habits, consisting of a few main stems long internodes, few short side branches, and small trifoliates. The canopies generated by such genotypes would favour a more rapid drying of the leaf surface, by way of an improved air circulation and a better penetration of light. These authors distinguished these ideotypes from the 'compact dense plants', as the 'dense determinate' dry bean varieties. Later, Schwartz et al. (1978) concluded that a cultivar designed to maximize disease avoidance should possess an upright growth habit, open plant structure, and low canopy density to induce microclimatic conditions within the canopy which are unfavourable to several stages of the disease cycle. Conversely, the genotypes with a significantly denser canopy are very favourable for white mold epidemics. The more critical determinant of disease severity was conferred by the distribution of the leaf area, especially near the ground. In the case of ascochyta blight on pea, Le May et al (2009) concluded that to reduce the wetness period, optimum canopy architecture to reduce the disease includes low LAI (mainly achieved by a low area of stipules), high internode distances and high mean distance between nodes. Such ideotypes, targeted to increase disease avoidance, are of great interest when they are combined with physiological resistance. Therefore breeding programs have been developed to combine disease avoidance and genetic resistance in new cultivars, for instance in dry bean (Coyne et al. 1977; Miklas et al. 2001) or in peanut (Coffelt and Porter 1982). These researches might rapidly allow to combine the architectural traits and other disease control methods (resistance, cultural practices) to reduce the use of pesticides.

Conclusion: an interdisciplinary approach

Research exploring the possibility of using canopy architecture for disease control has considerably progressed during the past 10 years. Currently, many authors do not hesitate to think of 'manipulating' architecture to design agronomical solutions to control diseases, in both annual and perennial crops (Bultzer et al. 1998; Simon et al. 2006; Ando et al. 2007). As shown in this review, the concepts and methods have been improved, and an integrative scientific strategy is now being followed. Two major fields have been covered by the scientists in the agronomy community. The first is to link the dynamics of plant development and of disease epidemics. Therefore, the complementarities among plant pathologists, entomologists, geneticists, agronomists, microclimatologists and mathematical modellers are now evident, and several groups who develop shared concepts, such as the EpiArch network in France, are implementing this multidisciplinary strategy into joint research projects. This perennial network is reinforced by the ARCHIDEMIO project which studies the plant - pathogen interactions within host canopies in four pathosystems: pea/ascochyta blight (*M. pinodes*), potato/late blight (*Phytophthora infestans*), grape/powdery mildew (*E. necator*), and yam/anthracnose (*Colletotrichum gloeosporioides*). The second field is to study the impact of this plant development/disease

epidemic interaction on yield components and on final yield. As described by Tivoli et al. (2010), this point needs to take into account the effects of key factors such as the time of infection, the site of infection and the intensity of infection.

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