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REVIEW

Landscape epidemiology of plant diseases

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Many agricultural landscapes are characterized by a high degree of heterogeneity and fragmentation. Landscape ecology focuses on the influence of habitat heterogeneity in space and time on ecological processes. Landscape epidemiology aims at applying concepts and approaches originating from landscape ecology to the study of pathogen dynamics at the landscape scale. However, despite the strong influence that the landscape properties may have on the spread of plant diseases, landscape epidemiology has still received little attention from plant pathologists. Some recent methodological and technological progress provides new and powerful tools to describe and analyse the spatial patterns of host–pathogen interactions. Here, we review some important topics in plant pathology that may benefit from a landscape perspective. These include the influence of: landscape composition on the global inoculum pressure; landscape heterogeneity on pathogen dynamics; landscape structure on pathogen dispersal; and landscape properties on the emergence of pathogens and on their evolution.

Keywords: plant pathology; disease emergence; pathogen dispersal; spatial heterogeneity; landscape pathology

1. INTRODUCTION

Potential losses of major world crops to pests, diseases and weeds have been estimated at approximately 70% (Oerke & Dehne 2004). However, the actual losses are approximately 30% due to efficient crop protection practices. The increase in human population density and the subsequent demand for meat are predicted to cause crop production to double in the next four or five decades (Tilman 1999). To achieve this objective, an improvement or at least maintenance of crop protection efficacy against agricultural pests, diseases and weeds is critical. The impressive progress in crop protection achieved in the past has, for a large part, relied on the use of synthetic pesticides. During the 1990s, the world use of pesticides increased by 4.4% annually (Oerke & Dehne 2004). However, crop protection strategies based upon chemicals have been questioned. Indeed, numerous negative effects have been documented on human health, natural flora and fauna or even on agricultural production and sustainability through the decimation of beneficial natural enemies of pests and parasites (Wilson & Tisdell 2001). Moreover, when used systematically, chemicals lose efficiency due to rapid selection of resistance in targeted organisms (Gullino *et al.* 2000; Ma & Michailides 2005; Urban & Lebeda 2006). Altogether, the need for improved control of agricultural pests and diseases and the necessity to reduce the use of chemicals require a change in the paradigm of crop protection.

In the past 40 years, intensive production practices tended to promote the conception that agriculture was in confrontation with wild nature. The new paradigm of agriculture will reintegrate agricultural production within its environment to achieve sustainability. As advocated by Tilman (1999), consideration of the principles governing ecosystems will provide precious insights to face the challenge of agriculture to improve productivity and sustainability while decreasing its environmental impact. It will require shifting the scale of crop protection investigations and strategies from the field to the agricultural landscape. Generally, agricultural landscape consists of a changing mosaic of cultivated and uncultivated habitats. Landscape ecology that focuses on interactions between spatial patterns and ecological processes (Turner 2005) appears relevant for providing concepts to build a new paradigm for agriculture with consequences on crop protection strategies.

The idea of mapping patterns of disease incidence has a long history. For example, Snow observed in 1854 that cholera cases were clustered around a pump in London supporting his view that cholera was caused by a water-borne pathogen. The concept of landscape epidemiology was first proposed in the early 1960s by a Russian scientist E. N. Pavlovski (Galuzo 1975). Landscape epidemiology aims at identifying factors

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that influence the spatial spread of diseases between subpopulations of hosts. Recent developments in this field have resulted from progress in technologies for gathering and analysing landscape structures combined with the realization of the importance of spatial structuring in epidemiology (Hess *et al.* 2002). The number of studies devoted to the impact of landscape properties on human or animal diseases is now growing rapidly. Examples include studies of Lyme disease (Brownstein *et al.* 2005), alveolar echinococcosis (Craig *et al.* 2000; Graham *et al.* 2004) and malaria (Sallares 2006) in humans, and of sylvatic plague in prairie dogs (Collinge *et al.* 2005) and the hantavirus in deer mice (Langlois *et al.* 2001). The same trends can be observed in studies devoted to agricultural pest dynamics and to the evaluation of habitat manipulation for pest management (see for reviews Landis *et al.* 2000; Bianchi *et al.* 2006). In surprising contrast, coupling landscape ecology and plant pathology has still received little attention, especially for economically important crop diseases. Nevertheless, some concepts originating from landscape ecology have been used in theoretical studies of plant disease dynamics. Metapopulation theory has been applied to theoretical studies of plant disease dynamics in a spatially structured host population (Alexander *et al.* 1996; Thrall *et al.* 1997; Park *et al.* 2001). Percolation theory has also been used to study the spread of the soil-borne fungal pathogen *Rhizoctonia solani* (Bailey *et al.* 2000; Otten *et al.* 2004). In both cases, the authors focused on the determination of thresholds for invasion and persistence of epidemics in metapopulations. Though the landscape approach is becoming more popular in forest pathology (Holdenrieder *et al.* 2004), empirical studies remain scarce.

From an applied point of view, landscape epidemiology studies could help identifying landscape characteristics impacting the *a priori* disease risk with two possible purposes: (i) designing crop protection strategies (e.g. treatment advice based on decision-support systems) taking into account this *a priori* disease risk and (ii) designing management strategies of landscape structures and agricultural systems with the aim to reduce the *a priori* disease risk. In both cases, a decrease in the need for curative interventions (like using chemicals) should be expected.

2. ASSESSING THE IMPACT OF LANDSCAPE PROPERTIES ON DISEASE DYNAMICS

Several new tools have been recently developed that allow analysing and integrating the spatial component in epidemiology among which are geographic information systems (GIS), global positioning systems, remote sensing and spatial statistics (Kitron 1998). Altogether, they allow mapping essential characteristics of a landscape and then testing for correlation between those characteristics and spatial patterns of disease dynamics. A nice example of the impact of landscape properties on the dynamics of an agricultural pest, the canola pollen beetle (*Meligethes aeneus*), has been provided by Thies *et al.* (2003). They used GIS to map major characteristics of 15 German landscapes. Then, they looked for a correlation between the local proportion of destroyed oilseed canola buds and the characteristics of the surrounding landscape in concentric circles ranging from 0.5 to 6 km in diameter. They showed that an increase in landscape complexity was associated with a decrease in damage caused to oilseed canola by *M. aeneus* and to an increase in parasitism rate of the pest by parasitoids, the correlation peaking at a spatial scale of 1.5 km diameter. Surprisingly, similar studies on crop diseases remain sparse. GIS and geostatistics have been applied to the description of spatial patterns of risk and incidence of tomato virus diseases in the Del Fuerte Valley (Mexico) and used to design and validate a regional management programme (Nelson *et al.* 1994; Barnes *et al.* 1999). A local risk index was assessed based on the description of the field environment (crops, weeds and insect vectors) and mapped using GIS. A good correlation was found between risk index and true disease incidence. Risk maps were then used as a decision tool for adapting disease management practices (mostly based on delaying the planting date and eliminating virus host weeds) to local production situations. It resulted in both a reduction of disease incidence and of the use of chemicals. Another example was provided by Fabre *et al.* (2005) who showed that the proportion of bird cherry–oat aphids (*Rhopalosiphum padi*) caught in suction traps and carrying barley yellow dwarf viruses (BYDV) increased with the ratio area sown to small grain : area sown to maize within a 50 km radius around the suction trap.

One of the major ecological processes concerning landscape ecology is dispersal. Unfortunately, classical methods such as mark–release–recapture or radio-tracking are inoperative for small organisms. This may partly explain the rarity of studies on landscape ecology of pests and pathogens (Lushai & Loxdale 2004). Important progress has been achieved in recent years on marking and tracking techniques for insects (see for a review, Lavandero *et al.* 2004) allowing the development of studies of landscape ecology of agricultural pests and their predators and parasitoids. Those methodologies are also useful for arthropod-vectored diseases but difficult to apply to the study of pathogens. This could partly explain why studies of landscape epidemiology of plant diseases mostly focused on animal-vectored diseases. Fortunately, powerful molecular markers such as microsatellites are also now available to indirectly assess patterns of dispersal through genetic structure of pathogens (e.g. Giraud 2004; Guérin *et al.* 2007) offering new opportunities to study landscape ecology of plant pathogens.

3. IMPACT OF LANDSCAPE COMPOSITION ON GLOBAL PATHOGEN PROPAGULE PRESSURE

Landscape composition refers to the relative amounts of each landscape element. A simple metrics of landscape composition is the relative frequencies of different types of patches. This composition determines the local abundance of potential reservoirs of inoculum that may obviously influence the global propagule pressure and hence the risk of infection of a plant. Those reservoirs may, in particular, consist of diseased individuals of the same host species or of alternative, either cultivated or

wild, hosts. The reservoirs may play different roles. They may simply influence the regional density of inoculum. They may also provide a refuge for pathogen during seasons or years unfavourable to disease spread. Finally, in the case of heteroecious pathogens, the presence of alternative hosts may be required to allow the pathogen to locally complete its life cycle.

Host density is recognized as a major factor driving disease epidemics and theoretical studies have focused on determining a threshold below which a pathogen cannot invade a population of susceptible individuals (McCallum *et al.* 2001; Otten & Gilligan 2006). The dependence of plant disease incidence on the local density of the host is documented in forests (Gilbert *et al.* 1994; Bell *et al.* 2006; Emiko Condeso & Meentemeyer 2007) or in grasslands (Knops *et al.* 1999; Mitchell *et al.* 2002). Moreover, a recent study of Webb *et al.* (2006) suggests that disease incidence could also depend on the abundance of phylogenetically related species in the nearest considering that the probability for two host species to share a pathogen increases with their phylogenetic proximity. Such a phylogenetic effect is likely to occur in cultivated plants in which closely related species, either cultivated or uncultivated, can be locally abundant. Despite being universally regarded as a chief driving factor, the effect of host density on disease dynamics has received little attention in crops even at the field scale (Garrett & Mundt 2000) and empirical studies remain scarce. In their review, Burdon & Chilvers (1982) found a tendency towards increased disease severity at higher densities, but this trend was not very consistent. For instance, a decrease in barley powdery mildew severity with increasing host density has been recorded (Finckh *et al.* 1999).

The effect of the presence in the surrounding landscape of an alternative host can be diverse. In the simplest case, the alternative host does not differ in its characteristics in terms of pathogen dynamics from the focal host. In this case, the presence of an alternative host is equivalent to an increase in the patch occupancy of the focal host. However, in general, the effect of the presence of alternative hosts depends on the properties of the focal species relative to the entire community of alternative hosts (Keesing *et al.* 2006). For example, if the focal host is a poor reservoir that does not multiply or transmit the pathogen effectively, then the presence of a more competent alternative host can largely increase the disease prevalence in the focal host. Power & Mitchell (2004) illustrated this effect by showing that the presence of a highly susceptible host (*Avena fatua*) increased the prevalence of BYDV, a generalist virus, in several other species of annual wild grasses in experimental fields. Such a source–sink interaction is also operating in a well-documented example: the invasion of bean dwarf mosaic virus (BDMV) in Argentina. In the 1970s, the soya bean acreage in Argentina increased sevenfold leading to the emergence of BDMV that threatened production of common bean (Morales & Anderson 2001). BDMV is vectored by the whitefly (*Bemisia tabaci*) and causes a severe disease on common bean (*Phaseolus vulgaris* L.) while being able to infect soya bean (*Glycine max* L.) without inducing symptoms (Wang *et al.* 1999). The expansion of soya bean, a suitable reproductive host for *B. tabaci*, caused BDMV emergence in bean. Whereas common bean is not a preferred host of *B. tabaci*, it can feed and reproduce on this legume when populations are high or in the absence of its preferred breeding hosts. More generally, the indirect effects on the abundance of one species caused by the presence of a second one mediated by shared pathogens or predators (apparent competition) is recognized as an important factor influencing community dynamics and vegetation patterns (Morris *et al.* 2004). It may account for the invasion success of exotic annual grasses in California Valley grasslands (Malmstrom *et al.* 2005). Conversely, an alternative host could act as a sink, contributing to the decrease in the prevalence in the focal species. Keesing *et al.* (2006) reviewed several cases in which a decrease in vector-borne pathogen prevalence is associated with the presence of an alternative host preferred by the vector.

Alternative hosts, especially long-lived ones, can provide a refuge for pathogens during periods of time when the focal plant is unsuitable (overwintering or oversummering) or during years when conditions are unfavourable for disease spread. Henry & Dedryver (1991) have shown that the incidence of BYDV increased with pasture age in several grass species. This suggests that perennial pasture grasses could act as refuges for BYDV during years unfavourable to pathogen spread in annual cereals.

The role of alternative hosts on pathogen dynamics can be drastic when it governs pathogen survival at some critical point of its cycle. This effect is particularly obvious in the case of heteroecious pathogens that require two unrelated host plants to complete their life cycle as many rust species do. García-Guzmán & Wennström (2001) have shown that the occurrence of *Ochropsora ariae*, a heteroecious rust fungus, on *Anemone nemorosa* was strictly dependent on the presence in the nearest of the long-lived primary host *Sorbus aucuparia* (no diseased *A. nemorosa* was found farther than 90 cm away from *S. aucuparia*). They suggested that the short life duration of *A. nemorosa* infected by *O. ariae* associated to the low dispersal capacity of the fungus resulted in the need for frequent reinfections from the alternative host to maintain disease in *A. nemorosa* populations. Wennström & Eriksson (1997) have shown that the heteroecious rust, *Gymnosporangium cornutum*, could not survive during winter in its secondary host *S. aucuparia* and that infection on this species relied on spore dispersal from its primary host *Juniperus communis* (in this case, spore dispersal is effective up to 50 m away from the primary host). The role of alternative hosts of cereal rusts (leaf rust caused by *Puccinia triticina*, stripe rust caused by *Puccinia striiformis* and stem rust caused by *Puccinia graminis*) on summer or winter survival of the pathogens has been considerably studied in North America but remains unclear (Eversmeyer & Kramer 2000; Line 2002). A well-studied example is the case of the heteroecious stem rust in the USA. *Puccinia graminis* is a major pathogen of small grains worldwide. It also infests *Berberis* and *Mahonia* spp. where the sexual phase of its cycle occurs (Leonard & Szabo 2005). The plantation of common barberry (*Berberis vulgaris*) as an ornamental shrub throughout the north

central USA has resulted in the initiation of devastating epidemics of stem rust on small grains in the early 1900s. A barberry eradication programme initiated in 1919 extended to 18 states and was phased out during 1980. It resulted in stem rust becoming a minor problem on wheat and other small grains in North America (Peterson *et al.* 2005). The white pine blister rust (*Cronartium ribicola*) is another heteroecious pathogen that alternates between its primary hosts, *Ribes* spp., and pines. A strong influence of the presence of *Ribes* spp. on the disease incidence of blister rust on white-bark pine (*Pinus albicaulis*) in British Columbia, Canada, has been revealed (Campbell & Antos 2000).

4. PATHOGEN DYNAMICS IN HETEROGENEOUS LANDSCAPES

Landscape ecology focuses on the influence of habitat heterogeneity in space and time on ecological processes. Populations under study are generally fragmented and may experience frequent events of local extinction and colonization (metapopulations). Obviously, agricultural pathogens frequently exhibit such features and metapopulation theory appears relevant to describe their dynamics. Despite being a natural scale for their study, metapopulation concept has received relatively little attention in plant–disease interactions (Park *et al.* 2001). Park *et al.* (2001) have studied the effect of the spatial structure of host population on invasion and persistence of plant parasites. They have shown that invasion at the metapopulation scale depended both on within-patch (basic reproductive number) and between-patch (range and strength of between patch interactions) components of parasite spread. The importance of the size and the isolation of patches of habitats have been highlighted by metapopulation theory. The general prediction is that patch occupancy should increase with patch size due to a reduction of extinction rate and should decrease with patch isolation due to a reduction of colonization rate (Carlsson-granér & Thrall 2002; Hanski & Ovaskainen 2003; Honnay *et al.* 2005). In agricultural landscapes, mean patch size depends on the level of landscape fragmentation. Mean patch isolation is related to landscape heterogeneity. Metapopulation theory then suggests a reduction of disease risk in highly fragmented and diversified landscapes. Indeed, several empirical studies revealed a reduction of disease incidence with decreasing host patch size (Ericson *et al.* 1999; Colling & Matthies 2004). Perkins & Matlack (2002) suggested that management practices reducing the natural fragmentation of pine forest in southeastern USA resulted in an increase in landscape connectivity favouring the spread of fusiform rust, *Cronartium quercuum*.

Fragmentation can also have a positive effect on disease dynamics by increasing the edge over surface ratio. Indeed, fragmentation exposes organisms that are close to the edge to the conditions of a different surrounding ecosystem (Murcia 1995). This edge effect may favour disease spread. This has been illustrated by the epidemiology of sudden oak death disease in Californian coast where the proximity to the forest edge is associated with an increase in infection probability (Rizzo & Garbelotto 2003). Similarly, a study of the infection of peach trees by the plum pox virus has shown that large blocks were generally less vulnerable than smaller ones because the perimeter-to-area ratio was lower, and consequently, the plants in the centre of the orchard block were at much less risk of infection from adjacent diseased orchards (Dallot *et al.* 2004).

Host genetic diversity may also constitute an important source of heterogeneity influencing disease dynamics. An impressive demonstration of its impact at the landscape scale has been provided by Zhu *et al.* (2000) who followed the effect of cultivating a mixture of highly susceptible and less susceptible rice varieties on the yield losses caused by the blast disease (due to the fungus *Magnaporthe grisea*). This strategy carried out on an area of several thousands of hectares was highly efficient in reducing disease severity for both varieties and especially for the more susceptible one. This effect was attributed to the dilution of the inoculum of a given pathogenic race due to an increased distance between plants bearing the same genotype. Another convincing support for the effect of regional management came from the former German Democratic Republic (Wolfe 1992) where barley cultivar mixtures gradually expanded from 0 to 92% of the total barley area during the 1980s. In the same time, the severity of powdery mildew (caused by *Erysiphe graminis*) declined from more than 50% to less than 10%, while fungicide use decreased about threefold. Similar decreased reductions of mildew severity did not occur in adjacent countries where diversification was not practised.

Agricultural landscapes are generally not static. Hence, landscape may influence disease dynamics not only through its structure but also through its own dynamics. This effect has long been recognized and used through the design of crop rotations. Long-term crop rotation prevents the build up of inoculum, especially in soil-borne diseases (Alabouvette *et al.* 2006). Moreover, it can favour an increase in beneficial organisms in the soil including those with a capacity to reduce the growth and activity of plant pathogens (Peters *et al.* 2003; Pankhurst *et al.* 2005). The stability of landscape elements that constitute a reservoir of pathogens could also play an important role. Henry & Dedryver (1991) have shown a rapid increase in BYDV infection levels in pasture grasses (brome, fescue and ryegrass) with increasing age of the pasture from a few per cent to approximately 90% or more after 6 years. Long-lived pastures may then constitute a much more important reservoir of virus than non-permanent ones. On the contrary, in long-term wheat monocropping, the rapid increase in take-all (caused by the soil-borne fungus *Gaeumannomyces graminis*) severity during the first years is followed by a progressive reduction (Lebreton *et al.* 2004). In this case, the development of soil suppressiveness is assumed to result from the selection of populations of antagonistic bacteria.

5. LANDSCAPE STRUCTURE AND DISPERSAL OF INFECTIOUS PROPAGULES

One key process influencing metapopulation dynamics in heterogeneous landscapes is dispersal and the effects

of landscape spatial structure are widely dependant on dispersal abilities of the organism under study (Burel & Baudry 1995). As a consequence, considerable attention has been paid to the study of landscape connectivity (i.e. the degree to which a landscape facilitates or impedes movement of organisms among resource patches) and of the role of some particular elements of habitat, especially linear structures (corridors and barriers) on dispersal (Tischendorf & Fahring 2000). The degree of connectivity among host populations is likely to influence spatial patterns of disease persistence and incidence (Thrall & Burdon 1997).

Pathogen propagules or arthropod vectors are generally small organisms that are passively transported by wind. As a consequence, any landscape characteristic that influences air motion may impact pathogen dispersal. Indeed, topographical effects and landscape structures have been shown to influence the landing places of small insects. In Japan, the eastern sides of hills exposed to westerly winds near the coast and the head of windward-facing valleys are favoured landing places of *Nilaparvata lugens* (Noda & Kiritani 1989), a planthopper vectoring several rice viruses. Similarly, in melon fields, plants in a strip along windbreaks of *Cupressus sempervirens* are significantly more infected by the cucumber mosaic virus (CMV). This strip was shown to be a preferential landing place for aphids mostly owing to lower wind speed (Quiot *et al.* 1979). In the same way, the effect of landscape structures on air movement is likely to impact spore deposition of fungal plant pathogens. Schmale *et al.* (2006) investigated the viable spore deposition of *Gibberella zeae*, the causal agent of fusarium head blight of wheat, over variable landscape environments. However, they showed that temporal patterns of viable spore deposition were identical over all landscape environments.

Various landscape elements may act as barrier for pathogen dispersal. Landscapes in southeastern France exhibit an exceptional density of windbreaks. They were planted to protect market gardens from the wind as irrigated horticulture proliferated in the mid-nineteenth century. In a 2-year study in Provence, Marrou *et al.* (1979) showed that windbreaks slowed down the migration of vectors of CMV from field to field. Similarly, Traore *et al.* (2005) analysed the spatial distribution of genetic diversity to elucidate the dispersal process of rice yellow mottle virus (RYMV), a major disease of rice in Africa. They showed a high differentiation between isolates collected in different valleys on the Tanzanian island of Pemba where rice fields are small, patchy and surrounded by forests and cultivated areas with hardly any RYMV host, habitats that act as efficient barriers to RYMV spread.

Alternatively, corridors, i.e. narrow continuous strips of habitat that structurally connect two otherwise non-contiguous habitat patches (Tischendorf & Fahring 2000), may facilitate disease spread. Thus, corridors are suspected to facilitate the recent emergence of RYMV in Africa. The wide adoption of irrigation had created corridors for virus spread. Indeed, all RYMV hosts are water-dependent species found mostly along riverbanks, lakeshores, swamps, temporary ponds and irrigation canals. For instance, wild or cultivated rice that grows along the shoreline of Lake Victoria plays the role of a corridor of susceptible hosts facilitating RYMV transmission and accounting for the close genetic relationship between isolates from Tanzania and Kenya around Lake Victoria (Traore *et al.* 2005). Other linear structures like rivers or roads may also facilitate the dispersal of pathogens. The recently emerged pathogen, *Phytophtora alni*, that has jeopardized the natural population of alders throughout Europe since the 1990s disseminates along rivers by producing a large quantity of waterborne zoospores (Ioos *et al.* 2005). In a study devoted to the spatial dynamics of *Podosphaera plantaginis*, an obligate pathogen of *Plantago lanceolata* in a fragmented landscape carried out in Finland, Laine & Hanski (2006) have shown a strong correlation between pathogen occurrence and proximity of a road suggesting an important role of roads in facilitating dispersal of the pathogen. Roads have also been shown to play a major role in the dispersal of spores of *Phytophtora lateralis*, a root pathogen of the Port Oxford cedar (*Chamaecyparis lawsoniana*) in Oregon and California (Jules *et al.* 2002).

6. LANDSCAPE CHARACTERISTICS AND EVOLUTIONARY DYNAMICS OF PATHOGEN

In addition to their influence on disease dynamics, landscape properties may also affect the genetic structure and functioning of pathogen populations. This may influence ecological and evolutionary genetics of pathogens and consequently the likelihood of disease (re)emergence. Analyses of causal mechanisms leading to disease emergence most often point out the simultaneous effects of ecological and evolutionary genetic changes in patterns of first appearance and spread (Real *et al.* 2005). This is an important issue in agricultural production as illustrated by the overcoming of disease resistance of plants by a virulent pathogen strain (Parlevliet 2002) or the spread of resistance of pathogens to pesticides (Oerke & Dehne 2004). Basically, landscape properties may influence these processes by favouring: (i) a large gene flow facilitating the spread of a virulent strain and (ii) a high pathogen genetic diversity resulting in a high probability of the appearance of a virulent strain.

Pathogens exhibiting a high gene/genotype flow pose a greater risk than pathogens with low gene/genotype flows for two reasons: (i) populations with high gene flow have larger effective size and thus a higher probability that a virulent (or resistant) mutant appears in the population and (ii) populations with high gene flow are more likely to transmit virulent mutants across a large geographical area (McDonald & Linde 2002). For instance, Hovmøller *et al.* (2002) have shown that long-distance migration of *P. striiformis* f. sp. *tritici* was responsible for several resistance genes becoming ineffective for the control of yellow rust in four northwest European countries. Similarly, long-distance dispersal has been proposed to be responsible for the rapid breakdown of *Rlm* resistance genes in oilseed *Brassica* crops by *Leptosphaeria maculans*, the causal agent of stem canker of crucifers (Gout *et al.* 2006).

It should then be theoretically possible to reduce the risk of emergence of a new virulent (or resistant) pathogen by designing management strategies that limits gene/genotype flows among pathogen populations. This would require identifying landscape features limiting gene flow and identifying the proper spatial scale at which to deploy management strategies. The proper spatial scale must delineate isolated pathogen populations or, at least, subpopulations that exchange migrants only occasionally. Assessment of dispersal distance and population subdivision may be derived using the tools of landscape genetics, an emergent discipline combining landscape ecology and population genetics (Manel *et al.* 2003; Holderegger & Wagner 2006).

Landscape composition that determines the range of host species available for pathogen colonization may influence pathogen genetic diversity. Indeed, a large genetic diversity of the host is expected to result in a large genetic diversity of the pathogen (Gérard *et al.* 2006). Accordingly, populations colonizing alternative hosts may provide a reservoir of genetic diversity to the populations developing on the focal host favouring the maintenance of a high evolutionary potential as long as there is gene flow between host populations.

Reproductive mode also influences pathogen evolutionary potential and consequently the likelihood of appearance of virulent strains. According to McDonald & Linde (2002), the highest risk of evolution is met in pathogens exhibiting mixed reproduction systems, i.e. an annual sexual outcrossing followed by the production of asexual propagules. Many pathogenic fungi alternate between sexual reproduction on one host species and asexual multiplication on another one (Gérard *et al.* 2006). Hence, landscape composition, that may or may not include the host on which sexual reproduction occurs, affects the reproductive mode of such pathogens and their evolutionary potential. For instance, the success in combating stem rust in North America is generally attributed to the use of resistant wheat cultivars associated with the eradication of the alternate host, barberry, limiting genetic diversity and thereby the appearance of new virulent races (McVey *et al.* 1997; Leonard & Szabo 2005; Peterson *et al.* 2005).

Finally, landscape composition may also influence the evolution of pathogen virulence. For instance, the gene flow between populations exploiting several host species may be responsible for 'maladaptation' in pathogens (Combes 1997) reducing their fitness. However, the relationship between host (mal)adaptation and virulence remains controversial. Though several theoretical works suggest that host adaptation should result in a decrease of virulence, most empirical studies indicate that the opposite is usually true (Ebert & Herre 1996; Dybdahl & Storfer 2003).

7. CONCLUSION

Several new and powerful tools have been recently developed allowing acquisition and analysis of the spatial component of ecological data. They have been successfully used to identify landscape properties influencing the dynamics of human or animal diseases or the dynamics of crop pest populations. However, though the importance of considering the influence of landscape composition and configuration on disease is now recognized (Ostfeld *et al.* 2005), empirical studies on plant disease dynamics remain scarce and mostly concern forest pathology. Consequently, whether or not landscape characteristics actually play a role in epidemiology of most crop diseases remains largely unknown.

Inoculum density is considered by plant pathologists as a major factor determining the probability of occurrence and the severity of disease epidemics. The local abundance of source habitats and refuges has been proven to strongly influence the prevalence of crop disease. This suggests the importance of landscape composition on the dynamics of at least some diseases. Dispersal is also a key feature of disease epidemiology. Plant pathogens exhibit very diverse dispersal mechanisms, from soil-borne pathogens to aerially dispersed fungi and bacteria to vector-borne viruses. In all cases, dispersal scale defines the hierarchical structure of host–pathogen associations, by coupling the host and pathogen populations of connected demes and decoupling the dynamics of more distant ones (Gilbert 2002). Landscape structure and heterogeneity are then likely to influence pathogen dispersal and hence disease dynamics. More specifically, some landscape elements may act as corridors facilitating the spread of plant disease or, conversely, as barriers limiting the dispersal of infective propagules. However, the sources, distance and routes of dispersal of infective propagules are poorly known for most crop diseases. Though developing new tools to track the movement of pathogens at the landscape scale would surely be useful especially for air- or water-borne pathogens, the use of existing ones, such as molecular tools, would allow a great improvement of our knowledge in this field.

Landscape properties may also influence evolutionary dynamics of pathogens. They may influence the evolutionary potential of pathogens by impacting population effective size and gene flow between local populations. Landscape composition is of special importance in heteroecious pathogens in which the reproductive mode and consequently the genetic diversity depends on the availability of the primary host. Finally, landscape characteristics may influence pathogen ecology and especially the range of hosts simultaneously or successively exploited and determine the evolution of important traits among which is virulence. Some of these questions have been addressed from a theoretical point of view; however, model predictions still remain to be validated on real data.

Hence, coupling landscape ecology, landscape genetics and epidemiology is a promising approach. Many concepts and tools are yet available to handle efficiently the questions addressed by landscape epidemiology of plant diseases. It requires applying them to a large range of pathosystems to better understand the actual impact of landscape properties on crop disease emergence and dynamics.

From an applied point of view, safeguarding crop production is obviously of major importance for human beings. Agriculture faces several important challenges. It will have to rapidly increase crop production in

response to increasing demand due to the growth of the human population. However, it will also at the same time have to adapt to global changes and to respond to the public demand for developing environmental friendly practices. To achieve these goals, a better understanding of the interactions between agriculture and environment and in particular of plant disease ecology is required. The landscape appears as a key scale for this purpose. The success will require the translation of knowledge in landscape epidemiology into agricultural practices. Modelling is a useful approach: (i) to provide tactic decision-support tools allowing optimization of crop protection in real time and considering the particular landscape context or (ii) to help selecting strategies of ecological engineering for crop disease management based on agricultural practices and habitat manipulation. The conceptual framework provided by landscape ecology has yet been used to model crop disease dynamics. The few existing studies are still preliminary and mostly theoretical. A lot of work is still needed to develop tools that are truly useful for managers. Finally, a multidisciplinary approach is needed to simultaneously consider the main crops and their pests and diseases that coexist in a given landscape, to include technical and socio-economical considerations and to consider the multifunctional role of landscape.

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