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Local adaptation and gene-for-gene coevolution in a metapopulation model

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SUMMARY

In several reciprocal cross-infection experiments parasites were found to be significantly more adapted to their local host populations than to hosts from distant populations. We developed a metapopulation model, taking explicit account of both population densities and gene frequencies, to determine the influence of ecological and genetical parameters on the local adaptation of the parasites and on the spatial distribution of resistance and virulence genes. Our results point to the predominant effect of ecological parameters such as parasite growth rate and host and parasite migration rates on coevolutionary outcomes. In particular, the parasites are more likely to be adapted to their local host population than to allopatric hosts when the parasite migration rate is larger than the host migration rate. The opposite should be observed whenever hosts migrate more than parasites.

1. INTRODUCTION

Natural populations of hosts and parasites tend to be unevenly distributed in time and space (Burdon *et al.* 1989; Thompson & Burdon 1992). This patchiness, by influencing the distribution of disease resistance alleles among host populations and virulence alleles among parasite populations, is likely to greatly affect the extent of adaptation of parasites to sympatric hosts. Several authors found that parasites were locally adapted. For instance, transplant experiments, involving various organisms, showed that parasite populations were more infectious or pathogenic on sympatric than on allopatric host populations (e.g. the fungus *Synchitrium decipiens* on the legume *Amphicarpaea bracteata* (Parker 1985); the digenic trematode (*Microphallus sp.*) on the snail *Potamopyrgus antipodarum* (Lively 1989); the microsporidian *Pleistophora intestinalis* on *Daphnia magna* (Ebert 1994)). Similarly, Burdon & Jarosz (1991, 1992), studying several populations of the *Linum marginale–Melampsora lini* system, found that there was locally no correspondence between host resistance genes and pathogen virulence genes (so that most plants were susceptible to local strains, as in Parker's system) whereas such a correspondence could be found at the regional level.

All these results suggest that spatial heterogeneity has a strong effect on the antagonistic interaction between the host and the parasite. The way host and parasite traits, such as their respective migration rates, affect the spatial structure in host–parasite coevolution is still largely unknown. In this paper we use a metapopulation model to study the effect of these parameters on the degree of local adaptation of the parasites and the spatial distribution of resistance and virulence genes. Our model uses population densities and gene frequencies as dependant variables (Frank 1991, 1993*a*, 1994). First, we begin with an analytical characterisation of the host–parasite interaction at the scale of the population and, second, we develop a simulation approach to describe the coevolutionary process in a metapopulation.

2. THE MODEL

We assumed a Matching Allele Model (MAM) type of interaction as described by Frank (1991, 1994). Under this model hosts resist when a matching between alleles in the host and in the parasite occurs (see table 1). Therefore, we assumed that resistance in the host and virulence in the parasite were determined by a single locus with n alleles.

Both hosts and parasites are haploid and reproduce asexually. Because we assume haploidy and that the determinism of the interaction is governed by a single locus, sexuality should not have a strong, if any, effect. We also assume discrete, overlapping generations of both the host and the parasite, with no age-structure. Both interacting species evolve in a landscape constituted of discrete patches, among which migration occurs. We first describe local dynamics and provide analytical predictions at the local scale. We then

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Table 1. *Matching allele model with* n *types (alleles).* (+) *indicates resistance in the host and* (−) *indicates susceptibility*

		host types			
		*R*1	*R*2	...	*Rn*
parasite types	*V*1	+	−	−	−
	*V*2	−	+	−	−
	...	−	−	+	−
	Vn	−	−	−	+

outline the model at the metapopulation level and present simulation results at both local and metapopulation scales.

3. LOCAL POPULATION DYNAMICS

Local population dynamics follow a Lotka-Volterra model, as modified by Frank (1991):

$$\Delta h_i = h_i\left[r_h\left(1-\sum_{j=1}^{n}\frac{h_j}{K}\right)-d\sum_{j,j\neq i}^{n}p_j\right]$$

$$\Delta p_i = p_i\left[-s+r_p\sum_{j,j\neq i}^{n}h_j\right]. \qquad (1)$$

In the above system, h_i and p_i are population sizes of hosts and parasites of type i; Δh_i and Δp_i are variations of population sizes between two subsequent generations; n is the number of alleles (or types); r_h and r_p are their respective growth rates; K is the host carrying capacity; s is the death rate of the parasite; d is the pathogenicity of a parasite on a susceptible host. We define pathogenicity as the intensity of the deleterious effect of the disease on the host whereas the virulence is the capacity of the parasite to grow on a host. Therefore, in our model, there is a qualitative diversity (i.e. all the parasites are not virulent on the same host), but no quantitative diversity (i.e. when they are virulent, all parasites have the same level of pathogenicity; Frank 1993*b*). We may thus define parasite adaptation as the level of infectivity (i.e. the proportion of the parasite population which is virulent on a given host population). Parasites will be considered locally adapted when they are more infectious on sympatric than on allopatric host populations. We also assume discrete generations of the same length for both the host and the parasite. Competition occurs before reproduction among hosts and has a deleterious effect on fecundity but not on the survival rate. Competition occurs among parasites of the same type when they develop on a susceptible host. Intertype competition occurs only indirectly through the regulation of host density. We also assume that the deleterious effect of parasitism is density dependent which implicitly means that there is a homogeneous distribution of both organisms within each deme and that infection occurs randomly as in an air-borne disease (Thrall *et al.* 1995).

It is possible to study analytically the dynamics of such an antagonistic coevolutionary system at the scale of the population, without migration and mutation. Frank (1993*c*) found that when all types of hosts and parasites are present there is no stable equilibrium point. The interaction between hosts and parasites is highly unstable and leads to the extinction of some types. Coexistence of both organisms can be maintained through limit cycles between matching types of hosts and parasites. However, for the parameter values we used this coexistence is much more likely in the absence of any matching between host and parasite types. This situation implies that the host is totally susceptible to all the parasites present in the population. Therefore, in this case, the effective coevolutionary process, which can be defined as a durably evolving interaction, is irremediably stopped. Indeed, in the absence of any migration or mutation, the allele frequencies of both the host and the parasite do not change. However, population sizes can vary through time and we found that the level of demographic stability of such a system is governed by the quantity $(Kr_p - s)$ (cf. Appendix).

This result is consistent with experiments in natural populations that have shown a local adaptation of the parasite (Parker 1985; Lively 1989; Ebert 1994). Thus, if we consider several populations evolving independently, in each population the parasite will be adapted to the hosts with which it coevolved. However, there are many chances to find a resistant type in some other population. A similar result was found by Morand *et al.* (1996) for a different ecological and genetical model. However, such a system cannot explain the increasing gradient of resistance with geographic distance that has been found in natural populations. Moreover, the robustness of this prediction (i.e. local adaptation of the parasite) when migration occurs must be tested. We thus now turn to a metapopulation model in which populations do not coevolve independently but are connected by migration.

4. METAPOPULATION DYNAMICS

We consider a landscape made of N discrete patches of equal carrying capacity organized in a two-dimensional torus. Migration between demes occurs independently for hosts and parasites in a stepping-stone manner. Each deme sends migrants to the four neighbouring demes with a probability *migH* for the hosts and *migP* for the parasites. Such a model is difficult to analyse analytically and, therefore, we used a simulation approach.

At the beginning of a simulation, each type of host and parasite was introduced in a population with a probability of 0.5. This type of initialization creates a high level of diversity at the population level and allows coevolution to occur even in the absence of mutation. In the course of a simulation run, after 100 generations (to minimize the effect of the initialization), we collected summary statistics over all patches and all generations during 2000 generations. All statistics were then averaged over the 2000 generations interval. We ran ten replicates for each set of parameter values.

5. COMPUTER ANALYSIS

To study the effect of spatial structure we developed a hierarchical approach, studying coevolutionary outcomes at different scales. At each scale we asked different questions about the behaviour of the system.

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(*a*) *Local scale: average level of host resistance to sympatric parasites*

The local probability of resistance was quantified by:

$$P_{\text{loc}} = \frac{1}{N'} \sum_{i=1}^{N'} \sum_{k=1}^{n} (f_{ki}^{h} f_{ki}^{p}),$$

where f_{ki}^{h} and f_{ki}^{p} are the frequencies of the k^{th} type in the i^{th} population for the host and the parasite respectively. In calculating P_{loc} we considered only the N' populations where parasites and hosts coexisted.

(*b*) *Regional scale: shape of the resistance gradient across distance*

We wanted to determine if parasites were locally adapted and if there was a resistance gradient of the hosts. In each generation, we calculated for all populations the probability of resistance $P(L)$ between hosts of each population and allopatric parasites which are located L demes away. We then studied the shape of the resistance gradient across distance.

(*c*) *Experimental design: choice of parameter values*

(i) Genetical parameters

In our $2n$-dimension system the number of alleles involved in the specificity of the interaction is expected to have a large impact on the coevolutionary process. In particular, because one parasite type is potentially virulent on $(n-1)$ host types, the asymmetry of the system increases with n. Therefore, we studied the effect of different n values ($n = 4$, 8 and 16) on host–parasite coevolution.

To maintain an effective coevolution between the host and the parasite, a high level of polymorphism should be conserved at the population level. To test the effect of mutation we used three mutation rates (mut = 0, where variability is introduced in the system only at the initial stage; $mut = 10^{-4}$; $mut = 10^{-3}$; mutation rates of parasites and hosts were equal).

(ii) Ecological parameters

For every simulation we fixed the host carrying capacity ($K = 200$), the host growth rate ($r_h = 0.5$), and the death rate of the parasite ($S = 0.9$). We used three levels of pathogenicity ($d = 0.001$, 0.005 and 0.01). In the absence of mutation and migration the level of stability of the system is governed by the value of $(Kr_p - s)$. To study the effect of the parasite's growth rate we chose the values of r_p such that we could study different dynamical cases. We used $r_p = 0.008$ to study a locally stable case, $r_p = 0.01$ for a globally stable case and $r_p = 0.012$ for an unstable case where the antagonistic interaction leads to parasite extinction.

Like mutation, migration may maintain a high level of polymorphism at the population level. To test the hypothesis that host and parasite migration between patches allow durable effective coevolution to occur we studied eight levels of migration rates between 0 and 100%.

6. RESULTS

(*a*) *Temporal dynamics*

In agreement with the analytical results on the local population dynamics as well as other studies (for examples, see Frank 1993*a*), we found that the stability of both the host and the parasite populations was mainly governed by the growth rate of the parasite and that the temporal dynamics was much more stable for the host than for the parasite. Furthermore, we found (results not shown) that intermediate migration and large mutation rates, by maintaining a higher diversity at the local scale, decreased the parasite extinction rate.

(*b*) *Coevolutionary outcomes at the population level*

(i) The average level of host resistance to sympatric parasites

The average probability of host resistance is governed by several parameters. When all types of hosts and parasites are present in the same frequencies in a population, the host probability of resistance is $1/n$ (for instance, probabilities of resistance for 4, 8 and 16 alleles are respectively 0.25, 0.12 and 0.06). Indeed, because of the intrinsic asymmetry of the matching allele model, as the dimension of the model increases hosts are susceptible to a growing number of parasite types. The host probability of resistance decreased as the number of possible types increased in all cases (results not shown).

Host and parasite migration rates and their interaction had a strong effect on local probability of resistance (see figure 1). In the absence of migration, parasites were virulent on all their sympatric hosts. The adaptation of parasites was mainly caused by the initialization where the presence or absence of each type of host and parasite was randomly drawn. Indeed, very often, at least one parasite type was not matched by any host type. Such unmatched types were strongly selected for, leading to full susceptibility of the host population (i.e. $P_{\text{loc}} = 0$). For heuristic reasons we are now going to consider the cases where either the hosts

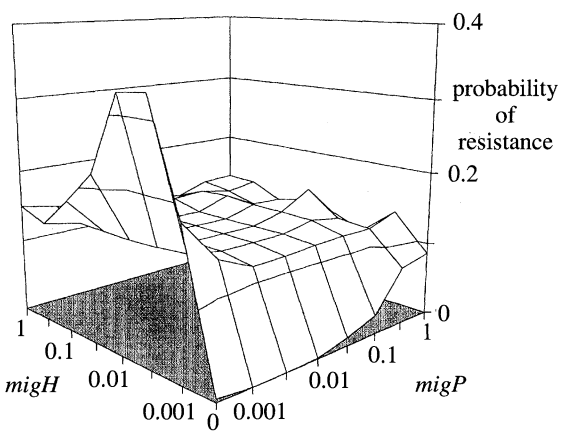

Figure 1. The effect of the host and parasite migration rates on the probability of resistance to sympatric parasites (for $n = 8$, $r_p = 0.008$, $mut = 0$, $d = 0.001$, $N = 100$). We considered here a case where the parasite growth rate was low. The coexistence between the host and the parasite was possible in the absence of migration, but the result of coevolution was that the hosts were all susceptible to the parasites.

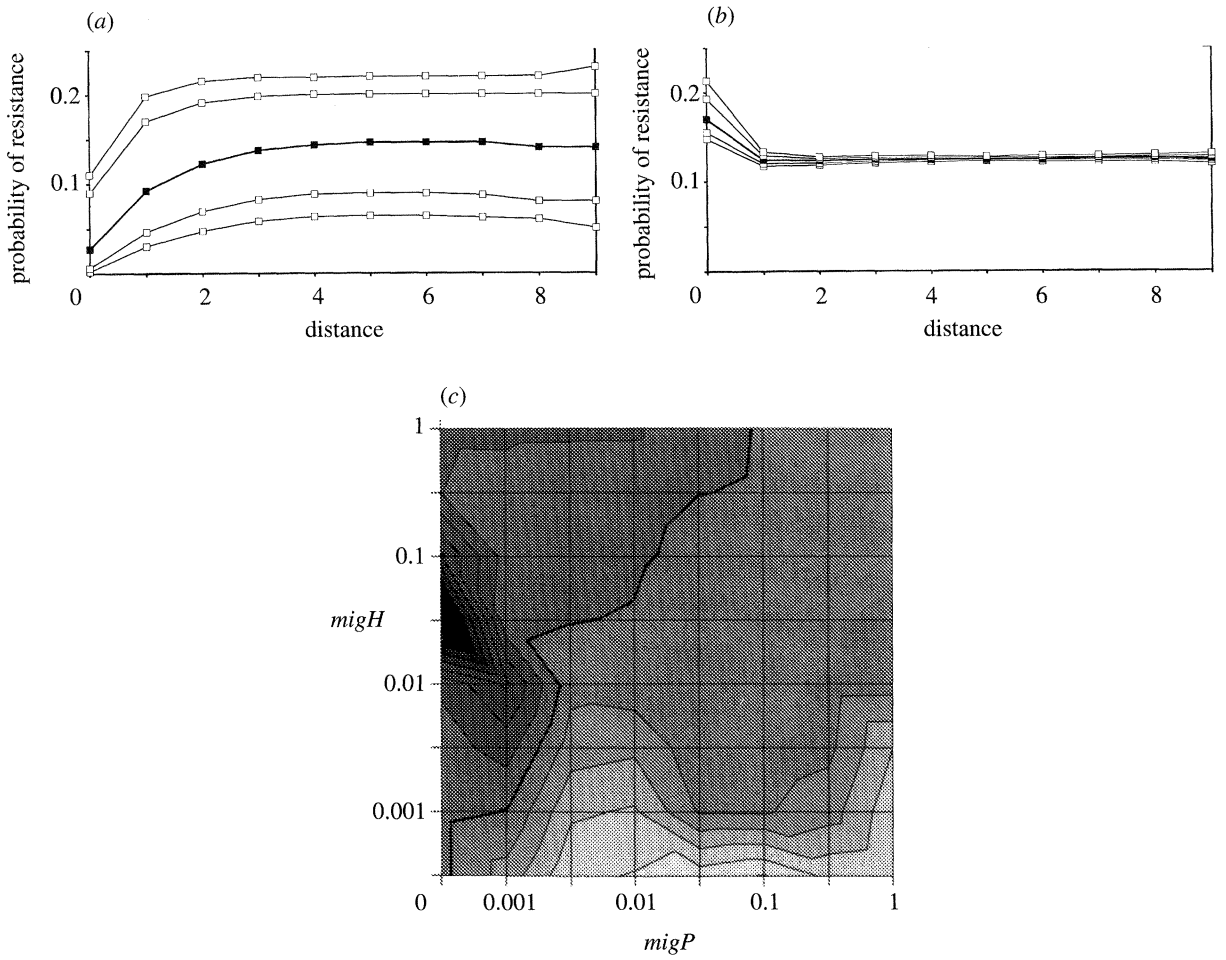

Figure 2. Probability of resistance across distance when: (*a*) $migP > migH$ (for $n = 8$, $r_p = 0.012$, $migH = 0.001$, $migP = 0.05$, $mut = 0$, $d = 0.001$, $N = 100$); and when (*b*) $migP < migH$ (for $n = 8$, $r_p = 0.012$, $migH = 0.1$, $migP = 0.001$, $mut = 0$, $d = 0.001$, $N = 100$). In (*a*) and (*b*) the bold line represents the median, the other lines, the minimum, the 5% percentile, the 95% percentile and the maximum over the 1000 generations of the simulations. In (*c*) the effect of the host and parasite migration rates on the difference between resistance to sympatric parasites and to parasites which are located one deme away (for $n = 8$, $r_p = 0.012$, mut = 0, $d = 0.001$, $N = 100$). Here, the bold line represents the points where there was no difference between resistance to allopatric or to sympatric parasites (the interval between two curves is 0.025). The dark shading (positive values) represents the area where the hosts were locally adapted to their parasites (like in (*b*)). The lighter shading (negative values) represents the area where the parasites were locally adapted (as in (*a*)).

or the parasites did not migrate at all. First, in the absence of parasite migration, a low level of host migration induced a high level of resistance and, therefore, many parasite extinctions. As the host migration rate increased host resistance decreased because host types susceptible to the local parasites were frequently introduced. Second, when hosts did not migrate, the probability of resistance increased with the parasite migration rate and reached an asymptote when all types of parasites were locally present. Thus, when either the host or the parasite had a very small migration rate, increase of the migration rate of one organism reduced the other organism's local adaptation. Finally, when both the host and the parasite migrate the probability of resistance reached a plateau at $1/n$.

Higher levels of parasite pathogenicity induced higher levels of host resistance. Indeed, high parasite pathogenicity induces a more effective selection for resistance in host populations. For instance with $r_p = 0.008$, $migH = migP = 10^{-2}$, $mut = 0$ and $N = 8$, we found $P_{loc} = 0.121$ for $d = 10^{-3}$ and $P_{loc} = 0.267$ for $d = 10^{-2}$.

(*c*) *Coevolutionary outcomes at the metapopulation level*

(*i*) *The shape of the resistance gradient across distance*

Our goal was to identify the parameters that mainly govern the spatial distribution of virulence and resistance genes. Our results (see figure 2), suggest that the shape of the resistance gradient is mainly governed by the ratio of the host and parasite migration rates. When $migP = migH$ or when host and parasite migration rates are too high (for example, in figure 2*c*, when $migH$ and $migP > 0.005$) we found no or small differences between resistance to sympatric or to allopatric parasites. When $migP > migH$ there was a gradient of resistance which increased with distance. When $migH > migP$ the host was more locally adapted, the gradient of resistance was less steep, and the gradient could even decrease with geographic distance.

7. DISCUSSION

In our model, coevolution is a transitory process that cannot be maintained without migration or mutation. The metapopulation structure of the host–parasite system has mainly two effects on coevolutionary outcomes. First, a demographic effect which stabilizes the interaction. Indeed, when the level of parasite growth rate is high the antagonistic interaction is highly unstable and can lead to parasite extinctions. This instability is mainly caused by the lack of an explicit density dependence in the parasite dynamics. In a metapopulation, however, for certain migration rates, parasite extinctions are decreased and the interaction is much more stable. This demographic effect, or buffer effect, was first described by Brown (1969) and later by Hassel *et al.* (1991). Second, a genetic effect, or gene storage effect, which can be explained by the maintenance of high levels of polymorphism at a regional scale (Judson 1995) and recurrent introduction of new resistant and virulent types (through migration or mutation). These recurrent events enable an effective coevolution to occur which is, from the host point of view, synonymous to a certain level of resistance.

Our model allows us to make some predictions concerning the level of host resistance to sympatric or allopatric parasites and, therefore, the ability of the parasite to be locally adapted. The shape of the resistance gradient, i.e. whether hosts resist more to sympatric or allopatric parasites, greatly depends on the ratio of the host and parasite migration rates. When both organisms have either similar or large migration rates we expect no local adaptation of either species. When parasites migrate more than the hosts, and when host migration rates are not very high the parasites are locally adapted. Finally, when hosts migrate more than the parasites, and when parasite migration rates are not very high the hosts are locally adapted.

In our knowledge there is no experimental system where both migration rates and local adaptation of parasites have been investigated. However, in the few cases where the parasites were shown to be locally adapted there are good reasons to believe that parasites migrate more than their hosts. For example, horizontal transmission of *Pleistophora intestinalis* occurs through free floating spores that can potentially migrate more than their host, the planktonic crustacean *Daphnia magna*. Similarly, the parasitic trematode *Microphallus sp.* probably migrates more than its snail host *Potamopyrgus antipodarum* because *Microphallus sp.* can reach another host population via intermediate hosts (vertebrate) such as ducks (Lively 1989). No attempt was made to estimate migration rates of *Melampsora lini*, a pathogen growing on *Linum marginale*, but the abrupt appearance of *M. lini* in individual populations in which they were previously absent suggests a relatively high level of migration (Burdon 1992). In two of these host–parasite systems an increasing gradient of resistance was found (Ebert 1994; Burdon *et al.* 1990). In the light of our model this gradient can well be interpreted as an average distribution of resistant and virulent genes which results from the differential rates of migration between hosts and parasites. At first glance this explanation does not seem to fit the *Amphicarpea bracteata* – *Synchitrium decipiens* system. Indeed, field transplant experiments showed an increasing gradient of resistance of *A. bracteata* even though the parasites apparently have very low dispersion rates (Parker 1985). However, our explanation may still be valid because as pointed out by Parker, *A. bracteata* presents high degree of self pollination and spatially restricted gene flow (Parker 1985, 1993).

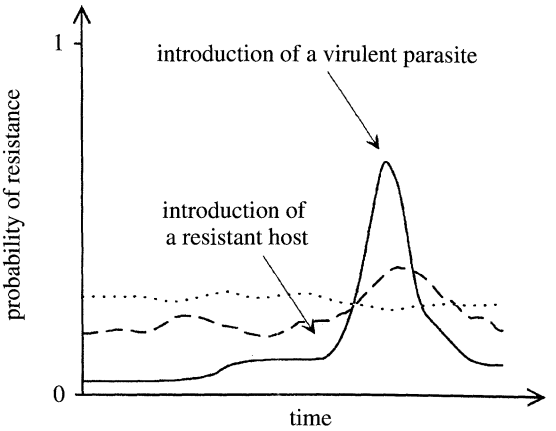

Figure 3. Temporary inversion of the resistance gradient when resistance genes were introduced in the population through immigration of a new type (for $n = 8$, $r_p = 0.01$, $migH = 0.001$, $migP = 0.01$, $mut = 0$, $d = 0.001$, $N = 100$). The solid line represents the probability of resistance to sympatric parasites, the dashed one represents the probability of resistance to allopatric parasites located 1 deme away and the dotted line represents the probability of resistance to allopatric parasites located 2 demes away.

It is likely that in general parasites migrate more than their hosts. One exception could be sexually transmitted plant diseases because in such systems parasites are transferred by pollinators only (e.g. the anther-smut disease of *Silene alba* caused by the fungus, *Ustilago violacea*; Antonovics *et al.* 1994). In this host–parasite system and in other examples of plant 'venereal disease' the migration rate of the host is potentially larger than the migration rate of the parasite because the plants migrate by both pollen and seeds. Because our model predicts that when the host migrates more than the parasite the resistance to sympatric parasites would be higher than to allopatric ones, cross-infection experiments on such systems could be used to test this prediction.

The predictions concerning local adaptation of the parasites must be treated with caution for two reasons. First, the effective coevolutionary process is characterized by complex temporal dynamics and the level of host resistance is highly variable through time (figure 2*a*). Indeed, the gradient of resistance can be temporarily inversed after the introduction (by migration or mutation) of new resistance types (see figure 3). Therefore, the gradient of resistance that we predict is not an asymptotic characteristic of the system but an average distribution over time. This dynamical view may explain the discrepancy between some field experiments and our predictions.

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Second, for certain parameter values, we found a positive correlation between resistance and distance at a small spatial scale, though the resistance level reaches rapidly an asymptote (figure 2*a*). The increase of the resistance gradient across short distances is consistent with experimental data (Parker 1985; Lively 1989; Ebert 1994). The fact that the gradient reaches an asymptote over longer distances, however, could be apparently considered to be in contradiction with several experimental studies. Indeed, Parker (1985) found that the hosts were totally resistant to parasites collected 100 km away and Ebert (1994) found a linear regression between resistance and geographic distance across Europe. One possible explanation could be that the asymptote appears in our results because, in a way analogous to temporal variation, we averaged the probability of resistance over several populations though in at least some cases the resistance gradient increased linearly with distance. A second explanation could be that we considered that a single locus governed the host–parasite interaction, thus restricting the amount of genetic variability and possibly population differentiation. Finally, even though at least in Ebert's study differences in pathogenicity are involved as well as potential differences in infectivity, we did not consider any variation in pathogenicity among strains of parasites.

It is likely that an 'optimal' scale of observation exists. Below that level resistance and virulence patterns would seem to occur at random (i.e. no match between resistance and virulence genes). At the population level, one may find useless (i.e. there are no local parasites to which these alleles confer resistance) alleles for resistance (Burdon & Jarosz 1991, 1992; Jarosz & Burdon 1991). Their presence can actually be explained by metapopulation dynamics, whereby locally useless alleles are maintained in remote populations. Above that level, other evolutionary factors (such as historical background or drift) might be responsible for the observed patterns. For instance, the high virulence of exotic parasites could have several explanations. First, such parasites could be exceptions that have been noted because of their strong effect on their hosts (Ebert 1994) or the low resistance of plants after introduction into an area free of parasites could be interpreted as the loss of useless resistance genes (Olivieri 1984). It should also be noted, that the virulence of exotic parasites may often involve only pathogenicity, which we have not studied. A parasite could be more pathogenic and less infectious at the same time.

Several empirical studies have noted local adaptation of the parasite and led Ebert (1994) to propose the rule that parasites are, with few exceptions, locally adapted to their hosts. Outlayers of this rule could be found for at least three reasons. First, when hosts migrate more than their parasites we would expect no local adaptation of the parasite. Second, non equilibrium dynamics induce both temporal and spatial variance and thus affect local adaptation at a particular place and time. Finally, other factors (e.g. those mentioned in the previous paragraph) could greatly affect our prediction, especially when the distance between host and parasite populations is very large.

Our results suggest that an effective coevolution between a host and a parasite is more likely to occur in a metapopulation than in a single population. As pointed out by Frank (1991) ecological parameters seem to have considerable effects on the outcome of this interaction. Similarly, our study shows that host and parasite migration rates have a strong effect on the ability of the organisms to be locally adapted. Therefore, migration rates are key parameters to understand the coevolutionary process. Because migration rates are also likely to evolve (Olivieri *et al.* 1995) and even to coevolve, it will be necessary to study the parallel evolution of the host and parasite dispersal rates to make long-term predictions about patterns of local adaptation in particular systems.

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REFERENCES

Antonovics, J., Thrall, P., Jarosz, A. & Stratton, D. 1994 Ecological genetics of metapopulations: the *Silene-Ustilago* plant-pathogen system. In *Ecological genetics* (ed. L. A. Real), p. 238. Princeton University Press.

Brown, J. L. 1969 The buffer effect and the productivity in tit populations. *Am. Nat.* **103**, 347–354.

Bulmer, M. 1994 *Theoretical evolutionary ecology*. Sinauer Associates Inc.

Burdon, J. J. 1992 Host population subdivision and the genetic structure of natural pathogen populations. *Adv. Plant Path.* **8**, 81–94.

Burdon, J. J. & Jarosz, A. M. 1991 Host-pathogen interactions in natural populations of *Linum marginale* and *Melampsora lini*. I. Patterns of resistance and racial variation in a large host population. *Evolution* **45**, 205–217.

Burdon, J. J. & Jarosz, A. M. 1992 Temporal variation in the racial structure of flax rust (*Melampsora lini*) populations growing on natural stands of wild flax (*Linum marginale*): local versus metapopulation dynamics. *Plant Path.* **41**, 165–179.

Burdon, J. J., Jarosz, A. M. & Kirby, G. C. 1989 Pattern and patchiness in plant-pathogen interactions – causes and consequences. *A. Rev. Ecol. Syst.* **20**, 119–136.

Burdon, J. J., Brown, A. H. D. & Jarosz, A. M. 1990 The spatial scale of genetic interactions in host-pathogen coevolved systems. In *Pests, pathogens and plant communities* (ed. J. J. Burdon & S. R. Leather), pp. 233–247. Oxford: Blackwell Scientific Publications.

Ebert, D. 1994 Virulence and local adaptation of a horizontally transmitted parasite. *Science, Wash.* **265**, 1084–86.

Frank, S. A. 1991 Ecological and genetic models of host-pathogen coevolution. *Heredity* **67**, 73–83.

Frank, S. A. 1993*a* Coevolutionary genetics of plants and pathogens. *Evol. Ecol.* **7**, 45–75.

Frank, S. A. 1993*b* Evolution of host-parasite diversity. *Evolution* **47**, 1721–1732.

Frank, S. A. 1993*c* Specificity versus detectable polymorph-

ism in host–parasite genetics. *Proc. R. Soc. Lond.* B **254**, 191–197.
Frank, S. A. 1994 Recognition and polymorphism in host–parasite genetics. *Phil. Trans. Soc. Lond.* B **346**, 283–293.
Hassell, M. R., Comins, H. N. & May, R. M. 1991 Spatial structure and chaos in insect population dynamics. *Nature, Lond.* **353**, 255–258.
Jarosz, A. M. & Burdon, J. J. 1991 Host-pathogen interactions in natural populations of *Linum marginale* and *Melampsora lini*. II. Local and regional variation in patterns of resistance and racial structure. *Evolution* **45**, 1618–1627.
Judson, O. P. 1995 Preserving genes: a model of the maintenance of genetic variation in a metapopulation under frequency-dependent selection. *Genet. Res., Camb.* **65**, 175–191.
Lively, C. M. 1989 Adaptation by a parasitic trematode to local populations of its snail host. *Evolution* **43**, 1663–1671.
Morand, S., Manning, S. D. & Woolhouse, M. E. J. 1996 Parasite–host coevolution and geographic patterns of parasite infectivity and host susceptibility. *Proc. R. Soc. Lond.* B **263**, 119–128.
Olivieri, I. 1984 Effect of *Puccinia cardui-pycnocephali* on Slender thistles (*Carduus pycnocephalus* and *C. tenuiflorus*). *Weed Sci.* **32**, 508–510.
Olivieri, I., Michalakis, Y. & Gouyon, P. H. 1995 Metapopulation genetics and the evolution of dispersal. *Am. Nat.* **146**, 202–228.
Parker, M. A. 1985 Local population differentiation for compatibility in an annual legume and its host-specific fungal-pathogen. *Evolution* **39**, 713–723.
Parker, M. A. 1993 Constraints on the evolution of disease resistance in an annual legume. *Heredity* **71**, 290–294.
Thompson, J. N. & Burdon, J. J. 1992 Gene-for-gene coevolution between plants and parasites. *Nature, Lond.* **360**, 121–126.
Thrall, P. H., Biere, A. & Uyenoyama, M. K. 1995 Frequency-dependent disease transmission and the dynamics of the *Silene-Ustilago* host-pathogen system. *Am. Nat.* **145**, 43–62.

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APPENDIX

Local stability analysis

In the absence of any match between the host and the parasite types local population dynamics can be described by the following equations:

$$\Delta H = H\left[r_h\left(1-\frac{H}{K}\right)-dP\right],$$

$$\Delta P = P[-s+r_p H],$$

where H and P are the abundance of the host and the parasite populations.

To study the local stability at the equilibrium point of the above system we calculated the jacobian matrix (Bulmer 1994). We found that a necessary and sufficient condition for local stability is:

$$Kr_p - s < 1.$$

Therefore, the equilibrium point is locally stable as long as this condition is verified.

When this is not the case (i.e. when K and/or r_p are high), the equilibrium point is locally unstable. Our simulation study showed that the interaction can be globally stable but, when the level of $Kr_p - s$ was very high, the interaction led to oscillations of increasing amplitude and to parasite extinction.