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Differential Adaptation in Spacially Heterogeneous Environments and Host–Parasite Coevolution

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13.1 Introduction

The terms *adaptive deme formation* and *local adaptation* have been used in the plant–herbivore and host–parasite literature, respectively, to designate one of the following two situations. The first one is when the mean fitness of a population (or deme) is on average larger in the environment this population originated from than in other environments. The second situation is when the mean fitness of a population on its natal environment is on average larger than the mean fitness of populations issued from other environments. We will use the term *local adaptation* to designate the situation when both conditions are satisfied, though this is not always the case. This definition emphasizes the potential differential response of populations with respect to their natal versus nonnatal environments, a phenomenon that should not be restricted to biotic interactions only.

With this definition, the concept of local adaptation implicitly assumes that the relative fitnesses vary in space. This condition is likely to be satisfied in most, if not all, natural situations, because the local environment of each population is composed of biotic and abiotic factors that typically vary in time and space. In this chapter, we attempt to clarify the concept of local adaptation in the context of biotic interactions and, in particular, host–parasite systems. We use the term *host–parasite* in a broad sense, including all interactions involving reciprocal selection and some specificity. A parasite represents all small organisms with a parasitic lifestyle, such as viruses, bacteria, protozoa, fungi, helminths, or small herbivores.

Local adaptation can be relatively easily studied within host–parasite systems, because the local environment of parasites is usually well defined. Each individual
host could be seen as an ephemeral island for the parasite. Moreover, if one assumes sufficient genetic and phenotypic diversity in the host population, each individual host may represent a different type of habitat. In this case, as an analogy of the source–sink concept (Pulliam 1988; Dias 1996), sensitive hosts could be considered as sources, whereas resistant hosts could be considered as sinks for the parasites. However, it should also be noted that adaptation to abiotic environments may be conceptually different from adaptation to biotic environments. The difference arises from the fact that the biotic environment (the host) might evolve in response to the adaptation of the parasite, if the parasite affects host’s fitness. Such a coevolutionary process can greatly affect the predictions concerning adaptation to local environments.

Host–parasite interactions may be formalized using population genetics theory. Population differentiation and local adaptation result mainly from a balance between natural selection and gene flow. We first present the factors and the mechanisms that promote or prevent local adaptation of parasites and then use this theoretical background to propose an experimental design in order to test the local adaptation hypothesis.

13.2 Local Adaptation and Dispersal

13.2.1 Evolution of Dispersal

Dispersal is a life-history trait that has profound demographic and genetic effects on populations. The evolution of dispersal has been studied theoretically by many authors (Hamilton and May 1977; Comins et al. 1980; Levin et al. 1984; Frank 1986; Johnson and Gaines 1990; Denno 1994; Olivieri et al. 1995). Some processes favor an increase in dispersal rates, whereas others act against dispersal. The balance between these opposing forces drives the evolution of dispersal.

In this chapter, we will assume that dispersal reflects gene flow between different habitats. It is known, however, that many herbivorous insects and some parasites do not disperse randomly but preferentially settle on a specific type of habitat (e.g., Thomas and Singer, Chapter 14, this volume; Bernays and Chapman 1994; de Meeûs et al. 1994). We briefly discuss the consequences of such habitat selection mechanisms in a later section.

13.2.2 What Selects against Dispersal?

Two factors select against dispersal. First are costs that are associated with dispersal itself. Dispersing individuals might incur a cost due to either increased mortality or costs during the settling period in the novel environment. Second are costs due to the spatial structure of the environment. In a spatially heterogeneous environment, dispersal will often lead individuals to unsuitable environments. If there is sufficient genetic variability for local adaptation, dispersal is selected against because of negative associations between genes coding for local adaptation and genes increasing the dispersal rate (Balkau and Feldman 1973). Such as-
associations arise because genes increasing the dispersal rate have a higher probability to settle on different environments and hence be, on average, selected against. But even in the absence of genetic variability for local adaptation, dispersal will be selected against in the presence of spatial heterogeneity. This would happen because passive diffusion moves individuals from favorable to less favorable habitats more often than the reverse, since favorable habitats tend to have more individuals (Hastings 1983; Holt 1985).

13.2.3 What Selects for Dispersal?

When the environment is variable, some level of dispersal will be selected for (Levin et al. 1984). An extreme case of temporal variability is the local extinction of populations. Indeed, when extinctions occur, dispersal will be selected for, because each particular deme will eventually become extinct, and only offspring that have emigrated will be able to reproduce (Olivieri et al. 1995). Such temporal variability is likely to occur frequently in many environments either because of environmental stochasticity (abiotic and biotic) or demographic stochasticity. Furthermore, temporal variability will result in spatial variability if the different populations are not perfectly synchronized. For instance, if variability is due to temporal changes in population size, then unless all populations change size at the same time, one would observe populations of various sizes across space at a given time. Finally, the degree of relatedness in each population is also involved in the evolution of dispersal. Dispersal can be adaptive if it reduces competition between close relatives (Frank 1986), and the evolution of dispersal can also be seen as a mechanism for the avoidance of inbreeding depression (Shields 1982).

13.2.4 Local Adaptation and Dispersal

Gene flow is often regarded as a constraining force in evolution, because in a spatially heterogeneous environment, it counteracts selective forces that lead to local adaptation (Slatkin 1987). Therefore, everything else being equal, one should logically expect a negative correlation between dispersal and local adaptation (points 1 and 2 in Fig. 13.1). This prediction has been tested in many host–parasite systems by relating the dispersal ability of the parasite to the presence or absence of local adaptation (Mopper 1996). Contrary to this prediction, parasite mobility does not seem to be strongly related to local adaptation. In some host–parasite systems, no local adaptation was found for sessile parasites (point 3 in Fig. 13.1), whereas in other cases, mobile parasites exhibited local adaptation (point 4 in Fig. 13.1). In the following, we present some arguments that could contribute to the explanation of these findings.

13.2.5 Conventional Wisdom: Negative Correlation between Dispersal and Local Adaptation

This negative correlation can be well explained by the operation of a migration–selection balance in a spatially heterogeneous environment. Natural selection leads
to adaptation to local environmental conditions. When dispersal is low relative to selection, gene flow cannot overcome the effect of selection, and local adaptation occurs (point 1 in Fig. 13.1). Conversely, for larger dispersal rates, immigrants from other populations will frequently introduce genes leading to adaptation to different conditions and will prevent local adaptation (point 2 in Fig. 13.1). In this latter case, because dispersal rates are also likely to evolve, one might ask why the parasite dispersal rate is so large if it prevents local adaptation. This may be explained by the metapopulation dynamics of parasite populations. In particular, some dispersal is adaptive when population extinctions occur (Olivieri et al. 1995). In host–parasite systems, each parasite population is mortal, because each individual host can be seen as an ephemeral habitat. Larger extinction rates can be induced by many factors (e.g., the death of an infected host, the extinction of a host population, or extinction of the parasite population due to its own natural enemies) and, therefore, select for dispersal to colonize new hosts. Peterson and Denno (Chapter 12, this volume) illustrated this point by looking at the effect of host-plant growth form (host persistence–gene flow hypothesis, see their Fig. 9.2) on gene flow. As mentioned before, within-host competition between close relatives (Frank 1986), or avoidance of inbreeding depression (Shields 1982) could also lead to higher migration rates. These mechanisms can be easily understood if the intrademic (within individual host) genetic structure of the parasite is taken into account (Frank 1994; McCauley and Goff, Chapter 9, this volume).
13.2.6 Positive Correlation between Dispersal and Local Adaptation

13.2.6.1 Spatial Heterogeneity as a Prerequisite

Certain transplant experiments with sessile parasites (Rice 1983; Unruh and Luck 1987; Cobb and Whitham 1993) did not reveal local adaptation (point 3 in Fig. 13.1). One possible explanation of such results is the lack of spatial heterogeneity of the environment (the host) at the scale studied. This argument may also explain the lack of local adaptation despite large parasite dispersal rates (point 2 in Fig. 13.1). Indeed, if the environment is not heterogeneous at the spatial scale of the study, transplant experiments will fail to detect local adaptation, even if the parasite has large dispersal rates. This point raises a methodological problem, since, in most studies on local adaptation, the degree of heterogeneity of the environment is only assessed by the relative performances of the parasites. We will further discuss this in section 13.3 (see also Boecklen and Mopper, Chapter 4, this volume; Mopper, Chapter 7, this volume).

13.2.6.2 Mobility versus Dispersal

Mobility, or the relative ability of an organism to travel in space, has sometimes been used as a predictor for dispersal rates. However, the apparent paradox of local adaptation despite a potentially high dispersal rate (point 4 in Fig. 13.1) may be explained by the fact that mobility does not always reflect dispersal. Dispersal could be low despite very high mobility either because animals return to breed to their natal sites, or because animals actively choose to breed on specific habitats (and avoid breeding on others). Indeed, if a parasite actively chooses on which host to oviposit, there can be very little gene flow between parasites of different kinds of hosts, despite potentially high mobility capacities. In this context, focusing on parasite mobility rather than quantifying gene flow can be misleading (Thomas and Singer, Chapter 14, this volume).

13.2.6.3 Coevolution and Dispersal

If the environment is temporally variable, a genotype selected for at one point in time may be selected against at a different time in the same site. Such temporal variability can be induced by a coevolutionary process between the parasite and other species (e.g., the host or natural enemies of the parasite). Gandon et al. (1996a) studied theoretically the effects of both host and parasite dispersal rates on local adaptation for a horizontally transmitted parasite that has the same generation time as its host. They used a mathematical model based on Lotka–Volterra equations, and on the assumption that each host genotype can resist one parasite genotype and is susceptible to attack by all other parasites. Therefore, no host genotype is able to resist to all parasite genotypes (i.e., the matching allele model as defined by Frank 1993).

Within this context, the degree of adaptation of hosts to a given parasite population can be defined as the probability of resistance of hosts to this parasite
population. Similarly, the degree of adaptation of parasites to a given host population can be defined as the probability of susceptibility of these hosts to the parasites. Their results indicate that local adaptation of either the host or parasite is very sensitive to the ratio of the host and parasite dispersal rates. When both the host and parasite dispersed at high or equal rates, no local adaptation occurred, because hosts resisted sympatric and allopatric parasites equally (Fig. 13.2, white area). When the host dispersal rate was very low, the parasite exhibited local adaptation as long as the dispersal rate of the parasite was different from zero (Fig. 13.2, vertically shaded area). On the other hand, when the parasite dispersal rate was very low, the host exhibited local adaptation (Fig. 13.2, horizontally shaded area). To illustrate these results, consider the biologically trivial case where hosts do not disperse, while parasites do disperse at some rate. In this case, hosts cannot escape the parasitic attack, because they do not disperse: A given host genotype is unable to escape attack by parasites able to overcome its resistance mechanisms. On the other hand, because parasites do disperse, each parasite genotype has a chance to encounter a host population composed of many hosts susceptible to it. If host genotypes are not uniformly distributed, different host populations will constitute different selective environments for the parasites, and as a consequence, parasites well adapted to their local host population will on average be less adapted to nonlocal host populations. Local adaptation of parasites will arise. By reversing these arguments, one might see why hosts exhibit local adaptation when the host dispersal rate is larger than the parasite dispersal rate.

The previous paragraph referred only to the differential response of host or parasite populations to sympatric and allopatric populations of the species with which they coevolve. Dispersal rates also affect the degree to which each host or parasite population is adapted to its local environment (i.e., the local coevolving population). Gandon et al. (1996a) showed that the proportion of local hosts that parasites were able to attack was maximized by minimizing host and parasite dispersal rates. The previous paragraph explained why very low host dispersal rates are beneficial to the parasites: Hosts are unable to escape parasitic attack in a particular site by dispersing to a site with fewer parasites able to attack them. Low parasite dispersal rates are beneficial to the parasite, because large parasite dispersal rates tend to homogenize parasite populations that can no longer take advantage of the heterogeneous distribution of hosts. In other words, large parasite dispersal rates decrease the degree of adaptation of the parasites to the local hosts by introducing locally maladapted genotypes to each parasite population. This mechanism is exactly the same as that operating in selection–migration balance in population genetics or in source–sink dynamics (Pulliam 1988; Dias 1996). Similar arguments apply to host dispersal rates and adaptation to local environment.

The results of Gandon et al. (1996a) emphasize the importance of the host dispersal rate in determining the level of temporal and spatial variability of the parasitic environment through the distribution of host resistance genes in the metapopulation (Fig. 13.3). For intermediate host dispersal rates, the temporal variability
of the environment is maximized and dispersal can be adaptive, because it enables the parasite to have the right genes at the right time and place (Gandon et al. 1996b). Therefore, if the environment is variable in time, this mechanism leads to the prediction that there should be a positive correlation between dispersal rate and local adaptation (points 3 and 4 in Fig. 13.1).

13.2.6.4 Natural Enemies of Parasites

Parasites' natural enemies (parasites, parasitoids, predators) can also affect the level of local adaptation. For example, Mopper et al. (1995) found that although leaf miners (Stibosis quadricustatella) were locally adapted to phenotypes of individual host trees (Quercus geminata), natural enemies induced higher levels of mortality on natal hosts than on novel hosts. These results can be interpreted in different ways. First, if one does not assume any specificity in the interaction between the parasite and its natural enemies, this differential mortality rate could
Figure 13.3  (A) Spatial and (B) temporal variability of the environment in relation to the level of host dispersal. The larger the host dispersal rate, the smaller the differentiation among host populations and, therefore, the smaller the spatial heterogeneity of the environment of the parasites (A). In the absence of dispersal, temporal variability is reduced because, at the scale of the population, there is no novelty in the genes that are involved in the coevolutionary interaction. When host dispersal rate is very high, temporal variability is also reduced, because all the genes are in all the populations. The diversity at the scale of the population is very high, and migration always introduces genes that are already present locally. Therefore, the temporal variability of the environment is maximized for intermediate levels of host dispersal (B).
well be explained by the fact that locally adapted parasites are more apparent to
natural enemies. Indeed, locally adapted parasites may be bigger, have larger
populations, or produce more chemical compounds that could increase their con-
spicuousness to natural enemies (Mopper et al. 1995; Mopper 1996).

Second, if one assumes some form of specificity in the interaction between the
parasite and its natural enemies, there would be two interactions at the same time:
one between the host and the parasite, and another between the parasite and its
natural enemies. These two processes would impose opposing forces of selection
on the parasite's life history. In the biological system studied by Mopper et al.
(1995), the host does not represent a highly variable environment in time, because
the host life span is very long compared to that of the parasite, and the host–parasite
interaction should select against parasite dispersal. Conversely, if one assumes
that natural enemies represent a temporally highly variable environment (shorter
generation time and/or higher migration rates than the parasites themselves), the
coevolution between parasites and natural enemies would select for parasite dis-
persal. Therefore, the selected level of dispersal would be a balance between
these two antagonistic selective forces. More generally, a positive correlation be-
tween dispersal and the degree of adaptation to the local environment would arise
whenever offspring fitness is on average worse in the parental environment than
in other environments. The mechanisms mentioned earlier illustrate ways in
which this situation might arise.

13.3 Recombination and Local Adaptation

13.3.1 Recombination and the Red Queen Hypothesis

It has been proposed that antagonistic coevolutionary interactions between hosts
and their harmful parasites may represent a short-term advantage sufficient to
compensate the twofold cost of sexual reproduction (Jaenike 1978; Hamilton
1980; Hamilton et al. 1990; Bell 1982; Ebert and Hamilton 1996). The twofold
cost of sexual reproduction, otherwise termed the "twofold disadvantage of pro-
ducing males" (Maynard Smith 1978), arises from the fact that the proportion of
parthenogenetic females within a population increases twice as fast as that of sex-
ual females (Maynard Smith 1978). Assuming a sex ratio of 1:1, this difference is
due to the fact that in sexual lineages, only half of the offspring are females, the
other half being males, whereas in parthenogenetic lineages all the offspring are
female (extended discussions on this topic can be found in Maynard Smith 1978
and Bell 1982). One of the mechanisms proposed to overcome this twofold cost of
sexual reproduction, and of interest to us here, involves some sort of frequency-
dependent selection (induced by the host–parasite coevolution), which confers
higher fitness to rare host genotypes. This mechanism favors sexual individuals,
because they can produce genetically variable progeny. In the absence of such
spatial and temporal genetic variability (e.g., monocultures), host populations are
more vulnerable to rapidly evolving parasites (Brown 1994). This mechanism was called the Red Queen hypothesis, referring to a term first coined in evolutionary biology by van Valen (1973), who was inspired by the character in L. Carroll’s Through the Looking Glass. According to van Valen’s hypothesis, each evolutionary advance of any species results in the deterioration of the environment of all other species; hence, the environment of all species changes continually, and only species that can evolve fast enough survive. Bell (1982) used the term to refer to the parasite hypothesis of the maintenance of sexual reproduction.

The major assumptions of the Red Queen hypothesis for the maintenance of sex are that (1) there is genetic variation in the parasites for virulence and/or infectivity; (2) there is genetic variation in the host population for resistance to specific strains of parasites; (3) infection by parasites reduces the fitness of individual hosts; and (4) parasites constantly adapt to host genotypes. The first three points are necessary for coevolution to occur (genetic variation and antagonistic selection). The fourth point, pertaining to local adaptation, is the subject of this chapter.

Given these assumptions, the prediction is that asexual hosts should be more prone to infection by parasites than sexual hosts. In other words, the Red Queen hypothesis predicts that parasites should be more locally adapted on asexual individuals than on sexual ones. This prediction is supported by several field studies on very different types of organisms (Lively 1987, 1992; Lively et al. 1990; Morritz et al. 1991; Burt and Bell 1991; see Ladle 1992 for a review), which revealed a strong correlation between asexual reproduction and some parasite fitness traits (e.g., parasite load, prevalence). A large monoculture of short-living hosts might represent the same temporal stability as a single long-living host (e.g., a tree). Heavy parasite loads on asexual host lines can therefore be conceptually interpreted in the same way as local adaptation of parasites to long-living, sexually reproducing hosts.

13.3.2 Recombination and Dispersal

The Red Queen hypothesis postulates that recombination allows the host to prevent local adaptation of the parasites (Hamilton 1980). Gandon et al. (1996a) showed that large host dispersal rates could be another way to counteract adaptation of the parasites. We now discuss the interaction of these two traits.

Ladle et al. (1993) used Hamilton’s model (Hamilton et al. 1990) to study the evolution of sexual reproduction in a metapopulation. This allowed the authors to study the effect of both the host and the parasite dispersal rates on the evolution of sex. They found that host recombination was selected only for intermediate or large dispersal rates of both the host and the parasite (cf. Fig. 13.4). These results suggest that sex could be unnecessary to release hosts from pathogens when at least one of the dispersal rates, that of parasites or hosts or both, is small. Their results could be explained in the light of two other studies. First, Gandon et al. (1996a) demonstrated that the host was locally adapted when the hosts migrate
Figure 13.4  Recombination and dispersal. Schematic representation of the effect of both the host and the parasite dispersal rates ($migH$ and $migP$, respectively; linear scale) on the selection of sexual recombination in the host metapopulation. The vertical shading represents the area where sexual reproduction succeeds (Sex) over asexual reproduction (modified from Ladle et al. 1993). In the unshaded area, the asexual strain succeeds (Asex). We included in the shaded area the cases where both the host and the parasite have large dispersal rates because of the results obtained by Ladle et al. 1993, page 157.

more than the parasites. Although the two models are not identical, this result suggests that sex may not be selected for when host dispersal rates are larger than parasite dispersal rates, because dispersal provides the host population with sufficient genetic diversity. Indeed, when the parasites are not locally adapted, recombination is unnecessary to counteract the effect of parasites (fourth assumption of the Red Queen hypothesis), because their average deleterious impact is very low. Second, when the host dispersal rate is very small for small to moderate population sizes, one would expect the genetic diversity of the host to be very low at the population level (Judson 1995). This could greatly affect the efficiency of recombination to produce genetically variable progeny (second assumption of the Red Queen hypothesis) and, therefore, to prevent parasites from being locally adapted.

There are different ways to prevent parasite local adaptation: sexual reproduction and dispersal. Furthermore, the study of the interaction between dispersal and recombination leads to interesting predictions. When the host dispersal rate is very large, sexual reproduction is unnecessary to allow the host to become locally
adapted, and, because of the twofold cost of producing males, recombination is
not selected for. Conversely, when the host dispersal rate is too small, recombin-
ation is inefficient in preventing parasites from becoming locally adapted (cf. Figs.
13.2 and 13.4). One has to keep in mind that these results are not sufficient to
make long-term predictions about patterns of local adaptation in host–parasite
systems. Other life-history traits such as host and parasite migration rates, and
parasite recombination rate, that are not studied by these models are also likely to
evolve and consequently affect the predictions.

13.4 Local Adaptation and Experimental Design

13.4.1 Transplant Experiments

The most common way to test for the local adaptation phenomenon is to experi-
mentally manipulate parasites and compare their performances on native and
novel hosts. For herbivorous insects, a novel environment can be an individual
plant from the same population, a different population, or even a separate host
species (Mopper et al. 1995). This type of experiment allows the researcher to test
whether parasites are locally adapted, examine the spatial scale in which local
adaptation occurs and, provided many populations are sampled, determine
whether local adaptation is correlated with distance (Parker 1985; Ebert 1994;
Lively and Jokela 1996). As noted before, however, a lack of differential re-
sponses of parasites could simply mean that hosts are not heterogeneous at the
spatial scale examined.

There are two ways to circumvent this problem. One is to compare patterns of
local adaptation at multiple scales. Another is to first measure differentiation
among host populations. Ideally, differentiation among host populations should
be evaluated on traits relevant to the host–parasite interaction. But, because such
traits are often difficult to measure or identify, neutral markers may be employed
to quantify genetic differentiation of host populations. It should be clear, though,
that differentiation measured by neutral markers may not reflect the potential dif-
ferentiation of traits relevant to the interaction, especially for sexually reproduc-
ing species.

Moreover, hosts should be chosen with care. In particular, the presence or ab-
sence of sexual reproduction on the differential success of parasites could greatly
affect the results (Burt and Bell 1991). Furthermore, age differences between par-
asitized hosts could bias the experiment, and if the age of an individual host is
correlated with the age of its parasite population, parasites may be more adapted
on older hosts (Burt and Bell 1991; Cobb and Whitham, Chapter 3, this volume).
This pattern could arise because, under the previous assumption, the age of a host
would be correlated with the period during which selection acts among parasites
within a single host. Younger hosts could be colonized by parasites that are not
necessarily adapted to them, but within-host competition in the parasite popula-
tion could eliminate such locally maladapted parasites on older hosts. Moreover, the level of resistance of an individual host may vary with its age. It can either decrease because of host senescence (Miller 1996) or increase (as in acquired immunity). For example, while studying developmental changes in resistance to herbivory, Kearsley and Whitham (1989) found that a single plant could change very rapidly in its resistance traits. Therefore, host individuals should derive from the same reproductive regime (sexual or asexual) and be similar in age.

Furthermore, as noted by Karban (1989), potential conditioning effects during parasite development on individual hosts may bias the interpretation of transplant experiments: what looks like genetic differentiation and local adaptation could emerge because of such maternal effects and not due to genetic adaptation. To avoid this, it has been suggested that parasites and hosts used in transplant experiments should be kept separated for at least one generation (Karban 1989; Ebert 1994).

There is evidence from some host–parasite systems that there is a significant effect of the infectious dose (the number of parasites inoculated) on the outcome of the inoculation. Ebert (1995) studied the interaction between Daphnia magna and a microsporidian parasite, and found that high inoculation doses increase the sporeload, but that the relation between infectious dose and sporeload is not always monotonic. Hochberg (1991) found that higher doses of the granulosis virus of Pieris brassicaceae resulted in substantial reductions in production of pathogen progeny because of intrahost competition. Therefore, the number of infections initiated per host should be carefully controlled (M. Hochberg personal communication).

13.4.2 What Should Be Measured?

Because the aim of local adaptation experiments is to study the adaptive ability of the parasites, it is essential to measure parasite traits that affect parasites’ fitness. In particular, measuring only host damage (parasite virulence/levels of herbivory) may be inadequate, because such damage may not reflect parasite fitness (Levin and Svanborg Eden 1990). A frequently used variable is the parasite infectivity (Parker 1985; Lively 1989; Lively and Jokela 1996), which measures the proportion of successful attacks on a given host population. A parasite would be locally adapted if its infectivity is higher on the host population from which it derived than on a remote host population (Parker 1985; Lively 1989; Lively and Jokela 1996). Infectivity could also be used at the individual-host scale by comparing the proportion of successful attack on the ancient (natal) host compared to novel hosts (e.g., Edmunds and Alstad 1978; Mopper et al. 1995; see also Alstad, Chapter 1, this volume). Infectivity alone, however, might reveal local adaptation only at large spatial scales, whereas quantitative measures of parasite fitness might reveal local adaptation at finer scales. This was most likely the case in the interaction of a microsporidian and its Daphnia host populations (Ebert 1994). Because host resistance can also be quantitative, parasite fitness could also be measured by
variables such as parasite mortality rate (Mopper et al. 1995) or reproductive output (Ebert 1995).

It is well known that the fitness of a given organism is dependent on many biotic and abiotic factors (Hunter and Price 1992), which could affect the level of local adaptation of an organism. Examining the sources of insect mortality in *Sitisbosis quadricustatella* populations, Mopper et al. (1995) found that the host plant (*Quercus geminata*) was not the sole source of insect mortality. Leaf-miner natural enemies could significantly affect fitness and possibly counteract insect local adaptation to host-plant traits. Therefore, when transplant experiments are conducted in the wild, selective pressures independent from the host–parasite interaction should be included.

Such selective pressures, however, will affect local adaptation only if there is a significant interaction with the host plant. For instance, if the rate of parasitoid attack on a given insect species is the same on all host plants the insect parasitizes, it is unlikely that parasitoid attack may explain differential responses of insects to host plants, even if parasitoid-induced mortality is very large. Stiling and Rossi report such a case in Chapter 2 of this volume. These authors transplanted clones of the plant *Borrichia frutescens* among four islands to test whether populations of the gall midge *Asphondylia borrichiae* were adapted to specific host clones. They found that midges were locally adapted, for which host-related differences in midge fecundity was the most likely explanation. Even though parasitism by four wasp species accounted for approximately 40% of midge mortality, parasitoid attack rates were equivalent on natal and transplanted host clones.

**13.5 Conclusion**

Conventional wisdom predicts that gene flow should prevent local adaptation. However, a major assumption of this prediction is the absence of temporal variability in the environment. Several factors might cause the environment to be variable over time. Depending on the factors acting and the potential of organisms to respond to them, we distinguish three forms of temporal variability. The first is when the habitat quality is temporally variable in a way that the organism cannot adapt to the new environment. Such variability could be induced by environmental or demographic stochasticity, an extreme case being the extinctions of populations. Theoretical studies have shown that in such temporally variable environments, some level of dispersal is adaptive (McPeek and Holt 1992; Olivieri et al. 1995).

A second form of variability occurs when habitat quality is temporally variable such that the organism living in it may track the new environment (i.e., may become adapted to it). For example, such variability can be induced by abiotic factors such as temperature, nutrients, or humidity variations, which may vary over time in a given location. In this case, a given organism may become adapted to the local environment by incorporating mutations advantageous in the new environmental conditions (for an experimental test of such adaptive evolution see
Bennet et al. 1992). The incorporation of such beneficial mutations in a temporarily variable environment can be enhanced if the organism evolves in one (or a combination) of the three following ways: increasing its recombination rate (Maynard Smith 1980), its mutation rate (Ishii et al. 1989), or its migration rate.

The third type of temporal variability occurs when there is reciprocal selection between the habitat and the organism (coevolution). Such variability typically occurs in host–parasite systems when there is a certain level of specificity involved in the interaction, and when host and parasite generation times do not differ by too many orders of magnitude. This might also enhance the evolution of traits that facilitate local adaptation. Hamilton showed that in such a heterogeneous environment, sexual recombination can evolve (Hamilton 1980; Hamilton et al. 1990). We believe this argument could also be applied for dispersal abilities. Indeed, Gandon et al. (1996a) found that migration is necessary for local adaptation, and Ladle et al. (1993) found a significant interaction between migration and host recombination. All these results suggest that migration and sexual reproduction are important life-history traits that allow a given organism to catch the Red Queen (to be locally adapted in a temporally variable environment) by possessing good genes at the right time and place (Gandon et al. 1996b).

All three types of temporal variability are involved in host–parasite interactions and could lead to counterintuitive predictions concerning the correlation between the level of local adaptation and the level of gene flow. For example, parasite dispersal may be adaptive if the hosts are ephemeral (the death of an infected host leads to the extinction of a parasite population). If temporal variability is induced by variable, antagonistic selection pressures between the parasite and its biotic environment (including the host and the natural enemies), dispersal might also be adaptive because, contrary to the conventional wisdom, it may lead to local adaptation. However, dispersal may also lead to less suitable hosts and have costs in terms of reduced fitness. Therefore, selection for the optimal dispersal rate is constrained by colonization of new hosts, encountering unsuitable hosts, and avoidance of natural enemies.

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13.6 References


