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COMMUNITY GENETICS: EXPANDING THE SYNTHESIS OF ECOLOGY AND GENETICS

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Abstract. Community genetics synthesizes community ecology and population genetics and yields fresh insights into the interplay between evolutionary and ecological processes. A community genetics framework proves especially valuable when strong selection on traits results from or impinges on interspecific interactions, an increasingly common phenomenon as more communities are subject to direct management or anthropogenic disturbances. We draw illustrations of this perspective from our ongoing studies of three representative communities, two managed and one natural, that have recently undergone large perturbations. The studied communities are: (1) insect pests of crop plants genetically engineered to produce insecticidal toxins; (2) insect-pollinated plants in habitats severely fragmented by agriculture and urbanization; and (3) a pathogen and its crop host now grown extensively outside their native ranges. We demonstrate the value of integrating genetic and ecological processes to gain a full understanding of community dynamics, particularly in nonequilibrium systems that are subject to strong selection.

Key words: *anthropogenic disturbance;* Bt *maize; community genetics;* Echinacea angustifolia*; evolution of resistance; genetic engineering; habitat fragmentation; nonequilibrium dynamics; plant–insect interactions; plant–pathogen interactions;* Ustilago maydis.

INTRODUCTION

Janis Antonovics (1992) articulated a vision for a new field of inquiry, ''community genetics'' (a term suggested by Dr. J. P. Collins, Arizona State University), to investigate the ''role of genetic variation in influencing species interactions and determining community structure.'' Community genetics is a synthesis of community ecology and evolutionary genetics; it directly assesses the interplay between genetic variation and community dynamics to develop a mechanistic understanding of the evolution of organisms in the context of the communities that they occupy.

Our community concept is that developed by Gleason (1917, 1926, 1927), demonstrated by Whittaker (1956), and supported by the work of Davis (1981) on community assembly. In this concept, species assemble in communities according to their individualistic attributes. We superimpose on this concept a contemporary understanding of the ubiquity of genetic variation. Thus, a community is the multispecies assemblage of genetically variable populations that together occupy a given place. The strength of interaction between members of a community varies. Strong interactions can arise even for species that have associated only recently. For example, in the case of species invasions, strong interactions may be apparent from the time that a species arrives at a given location (Pritchard and Schluter 2001). Assembly of novel communities may have evolutionary, as well as ecological, consequences within few generations (Reznick et al. 1997, 2001, Davis and Shaw 2001). When, in addition, ecological interactions strongly influence the genetic composition of populations, the conceptual framework of community genetics becomes valuable.

Community genetics addresses questions about the evolution of interactions among organisms in a broader context than that of the more stringent framework of coevolution where ''an evolutionary change in a trait . . . in one population in response to . . . a second population, [is] followed by an evolutionary response by the second population to the change in the first'' (Janzen 1980). A situation in which the ecological success of one species depends on the genotypes of a second species would not necessarily be considered coevolution by Janzen's stringent criteria, but would fit well into the framework of community genetics, whether or not the genetic composition of populations of the first species is affected by the ecological interaction. For example, invasion of a novel pathogen could dramatically affect the population genetic structure of a

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host, without reciprocal genetic effects on the pathogen population (Alexander et al. 1996).

Community genetics focuses on feedbacks between ecological interactions within and among species and genetic variability within species. It acknowledges that these feedbacks can operate on the scale of a few generations. As conceived by Levin and Udovic (1977) for a pair of species, the community genetics approach allows for rapid evolutionary change contingent on community composition. It does not view a species as a taxonomic unit as in purely ecological studies (e.g., Tilman et al. 1997), but rather as a collection of populations for each of which the genetic composition may depend on the community context and population history. Thus, the genetic composition of populations within a species may differ substantially from one community to another. We argue that a community genetics framework is particularly valuable when (1) an abrupt change in community structure, e.g., with anthropogenic disturbances, or (2) historical process of community assembly generate nonequilibrium communities and impose strong selection on members of a community.

Community genetics emerged from ecological genetics by the inclusion of a community context. Collins (1986) traces ecological genetics back to the beginning of the 20th century; some of its roots also trace back to Fisher (1927) and Dobzhansky (1947). Collins points out that during the initial development of the field of ecology, evolutionary thinking was pervasive, quite soon, however, ecological and evolutionary studies diverged as ecological studies increasingly focused on identifying proximate causes of distributions and the abundance of organisms.

Ecological genetics emphasizes the importance of considering both ecological conditions and genetic composition in studying population and community dynamics. Early studies in ecological genetics sought to explain genetic differences among populations in relation to the biotic and abiotic environment (Dobzhansky 1941, Birch 1960, Ford 1964). Classic examples include studies of melanic forms of moths in relation to air pollution (Kettlewell 1955), of tolerance of soil contamination by heavy metals (Bradshaw 1952, Antonovics et al. 1971), of mimicry in lepidoptera (van Zandt Brower 1958), and of banding patterns of snail shells (Cain and Sheppard 1954).

Observing that ecological and evolutionary processes commonly occur on the same temporal scale and that evolutionary change is detectable in a single generation, Antonovics (1976) advanced an integrated view of evolution and ecology for studies of species abundance and diversity. Antonovics and Levin (1980) further pointed out that the spatial scale of ecological processes may be smaller or larger than that of evolutionary processes, and that both may differ from the spatial scale of selection. As a consequence, without an understanding of the underlying genetic structure of populations and their evolutionary histories, it may be difficult to understand and impossible to predict ecological patterns, even in simplified, experimental communities. A number of coevolutionary studies using laboratory experiments, empirical studies of natural populations, and theoretical approaches have demonstrated that the outcome of genetic and ecological interplay may not be readily predictable at the outset of an experiment. For example, in experiments with *Escheria coli* infected by the virus T5, the susceptible genotype was driven to extinction following the origin and spread of a resistant genotype. However, when susceptible *E. coli* were infected by the virus T4, a resistant host type emerged that coexisted with the susceptible host type. A cost of resistance at low viral titers might have been the mechanism for coexistence (Lenski and Levin 1985). These two experiments demonstrated that communities may evolve along different paths to different equilibria. More importantly, such experimental community approaches illustrate that when genetic parameters are included, predictive models can be developed for conditions under which alternative outcomes might be expected. Community genetics provides a framework for such studies.

Consideration of the potential for rapid evolutionary change and how this may differ among populations has been extended to the case of climate change by Etterson (2000) and Etterson and Shaw (2001) in a study of three populations of an annual legume, *Chamaecrista fasciculata*, drawn from a range of 1000 km. Treating this species' extensive latitudinal and climatic range as a proxy for the rapid warming projected over the coming decades, Etterson documented both strong selection on a Minnesota population when grown in Kansas and Oklahoma, and considerable genetic variation in traits under selection. However, this study also demonstrated that antagonistic genetic correlations between traits are likely to impede evolutionary response, causing it to lag behind the predicted change in climate.

Thompson's (1994, 1997) concept of a geographic selection mosaic further frees the study of evolutionary interactions from the constraints of strict coevolutionary definitions (Janzen 1980). By focusing on ecological interactions of organisms in metapopulations, the geographic selection mosaic accounts for spatial variation of selection across different communities. Interactions between organisms vary qualitatively across a species' range, such that in some communities, strong interactions occur (hot spots), whereas with a different community composition, the same organisms affect each other's fitness only weakly (cold spots). Spatial differences in selection due to intraspecific genetic variation can both affect and be affected by the community composition.

An attractive feature of the geographic mosaic hypothesis is that it yields the following testable predictions (Thompson 1999). (1) Traits may vary among populations; in particular, they will be similar in some

communities but not in others. (2) There is potential for different outcomes of interspecific interactions due to genetic differences among populations and to differences in the abiotic or biotic environment. (3) Where the range of a species extends over a mosaic community such that interactions and, consequently, selection vary, coevolved traits may not occur throughout the range. Long-term studies, such as the *Silene–Mycobotrium* system (Antonovics 1992, Alexander et al. 1996), the *Greya–Lithophragma* system (Thompson 1999), and the *Linum–Melampsora* system (Burdon and Thrall 1999) provide evidence for the geographic selection mosaic.

The Conceptual Framework

The community genetics framework promotes new understanding when selection alters genetic composition on the same time scale as that on which numerical abundances change. Such concordant change is expected, with strong selection acting on traits mediating interspecific interactions. In these cases, ecological and evolutionary processes can no longer be treated separately. Although evolutionary genetics addresses strong selection and the potential for rapid evolution (Dobzhansky 1941), for instance, insecticide resistance (Georghiou 1986, Lenormand et al. 1999, Raymond et al. 2001), drug resistance (Garrett 1994), and the evolution of competitors (Pimentel and Al-Hafidh 1965, Pimentel et al. 1965), the potential for evolutionary change over a few generations remains underappreciated. Darwin (1859), despite acknowledging rapid evolution in artificial selection, regarded natural selection as a weak force apparent only over the geological time scale. Moreover, analysis of the consequences of strong selection poses significant theoretical challenges; although these have not been insurmountable (e.g., for quantitative genetics, Turelli and Barton [1994]), theoretical work has concentrated more heavily on the more tractable case of weak selection (e.g., Kimura 1964, Kingman 1982, Neuhauser 1999). Thus, the frequently mentioned distinction between evolutionary and ecological time scales is reflected in a modeling framework in which theoretical analyses assume weak selection on the order of the inverse of the population size, and imply that the effects of selection must be manifested on a time scale on the order of the population size. In many population genetic models (e.g., Kimura 1985), noticeable changes take thousands to millions of generations when the system is not in equilibrium. In contrast, in standard ecological models such as logistic growth or Lotka-Volterra competition models, noticeable changes occur from one generation to the next when the system is not in equilibrium. Treated in this way, weak selection has only subtle effects on population genetic dynamics over a few generations, whereas ecological changes may be considerable, thus resulting in a separation of time scales.

Both theoretical and empirical studies that simultaneously address ecological and evolutionary factors face challenges. Theoretical studies face the challenge of multidimensionality. Even in the simplest framework of ordinary differential equations, two equations are needed to model the ecological interactions of two species. Adding genetic variation to one or both species quickly increases the number of equations beyond that which is analytically tractable. However, multidimensional models permit complex behavior (multiple stable equilibria, limit cycles, and chaos) and explicit consideration of spatial dynamics, such as in Thompson's (1999) geographic selection mosaic. Empirical studies can be similarly challenged by multidimensionality. Experiments to assess effects of multiple factors and their interactions require many treatment combinations and, with replications, become very large. If the outcome of interactions varies in space and time, only long-term studies over large spatial scales reveal the full range of realized outcomes. Moreover, with variation over space and time, comparison among data sets is compromised and outcomes may appear unpredictable. Multidimensionality can also result in a proliferation of model parameters that cannot be adequately estimated in empirical studies.

We are optimistic that an integrated approach will ultimately result in a general framework that can accommodate the complexities that arise from considering ecological and evolutionary processes simultaneously. We argue that the value of an integrated, community genetics approach is particularly great under three conditions: nonequilibrium, genetic variation within species, and strong selection. We claim that the co-occurrence of these three conditions is not rare.

We present empirical studies together with mathematical models for which the framework of community genetics is particularly useful. These studies are characterized by strong selection following a large perturbation. The theoretical models are informed by empirical studies and include both ecological and evolutionary dynamics; leaving out one or the other aspect weakens the model predictions.

The first two studies investigate the interplay of ecological and evolutionary dynamics in the evolution of resistance to transgenic crops (*Bt* maize). Evolution of resistance is often modeled neglecting much of the ecological context. We demonstrate that adding ecological interactions (such as population regulation or density-dependent predation rates) can alter the predictions of simpler evolutionary models. The large-scale introduction of a transgenic crop, which represents a selection episode at an unprecedented scale, has motivated research to develop management strategies informed by the framework of community genetics.

The first study demonstrates that adding population regulation to a genetic model of spatially varying selection alters the prediction of the rate of evolution of resistance in the European corn borer (*Ostrinia nubi-*

lalis) to *Bt* maize. The second study focuses on the effect of natural enemies on the evolution of resistance in the same system. Differential egg mortality rates on resistant and susceptible genotypes of corn borers are mediated by differences in oviposition rates in *Bt* and non-*Bt* maize fields caused by restricted movement of females and by density-dependent responses of predators. This has the potential to affect the rate at which resistance evolves.

The third study addresses the effects of habitat fragmentation on the persistence of previously extensive populations of long-lived, self-incompatible plants (*Echinacea angustifolia*), as found in the North American tallgrass prairie. Habitat fragmentation is often modeled in a purely ecological framework (e.g., Nee and May 1992). We demonstrate that explicit consideration of the genetic basis of the plant mating system (Bataillon and Kirkpatrick 2000, Glemen et al. 2001) more fully reveals the consequences of fragmentation and suggests approaches to promoting persistence of the remnant populations.

The fourth study concerns the evolutionary interaction between a host plant (maize) and its pathogen (corn smut, *Ustilago maydis*) after a massive range expansion of the host species following domestication. This system exhibits remarkably durable resistance, contrary to the commonly observed accelerated evolution of virulence in agricultural monocultures. Such an evolutionary ''cold spot'' might be caused by historical biogeographic and contemporary genetic factors.

Nonequilibrium Dynamics

Evolution of resistance to transgenic Bt *crops*

Transgenic *Bt* crops have been developed by inserting a gene that codes for a crystalline protein (Cry toxin), which is selectively toxic to some insects, from the soil bacterium *Bacillus thuringiensis* (*Bt*) into the genome of a crop. Presently, potato, cotton, maize, broccoli, cabbage, canola, eggplant, poplar, soybean, rice, tobacco, and tomato have been transformed to express a Cry toxin, but only *Bt* cotton and *Bt* maize are now commercially grown in the United States. At least nine different types of Cry toxins have been used in crops that have been commercialized in the United States, each with its own unique toxicity spectrum and pattern of expression. Many of these *Bt* crops are very effective at killing target pests, in many cases allowing $<$0.1% survival in field conditions (Andow 2001).

Transgenic crops have stirred passions both for and against them, drawing attention to scientific investigations into their potential environmental and human health risks (NRC 2002). One of the significant environmental risks associated with these plants is that the target pests will evolve resistance to the Cry toxins, rendering the *Bt* crops ineffective (Tabashnik 1994, Gould 1998).

Table 1. Frequencies and relative fitness of the genotypes in the two patch types. *S* is a susceptible allele, *R* is a resistant allele, and *p* is the frequency of the *R* allele.

Measurement	Genotype		
	SS	*RS*	*RR*
Frequency (patch 1, toxic)	$(1 - p)^2$	$2p(1 - p)$	p^2
Frequency (patch 2, refuge)	$(1 - p)^2$	$2p(1 - p)$	p^2
Relative fitness (patch 1)	k	$Lh + k(1 - h)$	L
Relative fitness (patch 2)	1	$\kappa\eta + (1 - \eta)$	κ

Notes: Genotype frequencies are for a randomly mating population. Relative fitness in the toxic field is related to the ability of the genotypes to survive the toxin, and generally, $k \ll L$. Here h determines dominance of resistance, with $h = 1$ for completely dominant resistance and $h = 0$ for completely recessive resistance. Relative fitness in the refuge is related to the cost of resistance, with κ the cost to the *RR* homozygote and η the dominance of the cost.

The study of the evolutionary dynamics of resistance evolution began with Melander (1914), but it was Comins (1977) who developed a mathematical model incorporating selection and population dynamics. Since that time, additional mathematical (May and Dobson 1986; Ives and Andow submitted) and simulation (e.g., Tabashnik 1994, Alstad and Andow 1995, Gould 1998, Hillier and Birch 2002) models have added to our understanding of resistance evolution (for a concise review, see Rausher [2001]). All of these models are patch models, with a toxin selecting for resistance in one (or more) patches and no selection (or counterselection) in the other patch (or patches). Although the results of these theoretical investigations are complex, the evolutionary dynamics depend largely on the genetic parameters (selection coefficient, dominance) and interpatch dispersal.

Here we focus on a simpler problem. To understand resistance evolution, it is necessary to have some kind of underlying genetic model, whether population genetic or quantitative genetic. For example, Levene's (1953) two-patch, niche-selection population model foreshadows Comins (1977), but does not treat population dynamics explicitly. It remains unclear what, if anything, a population dynamic component to the model adds to our understanding of resistance evolution and, more generally, of spatially varying directional selection.

A standard migration–selection population genetic model is described in Crow and Kimura (1970:267). This model has no population dynamics and has been used to understand how migration and selection can maintain genetic polymorphism. To clarify generally the role of population dynamics in directional selection problems, we use the Comins (1977) model with and without population regulation. Here patch 1 is exposed to the toxin and patch 2 is not exposed to the toxin; the relative fitness of the genotypes is given in Table 1. Complete mixing of genes among patches occurs between generations. Population regulation in this

model is determined by simple density dependence (Hassell 1975).

To reveal an effect of population regulation, we concentrate on a special case of resistance evolution, the high-dose plus refuge strategy, which now is used to delay resistance evolution in *Bt* crops in the United States (Alstad and Andow 1995). Under this model, the genetics of resistance are restricted to the case of recessive resistance and no cost of resistance. In addition, to simplify analysis, we assume that movement is random and that all individuals are redistributed at each time step.

The genetic parameters for the model are described in Table 1. One can show that if resistance is not overdominant ($0 \leq h \leq 1$, where h is dominance), then the equilibria are the same for the model with and without population regulation. The resistant allele will go to fixation if the relative fitness of the resistant homozygote, L, exceeds the relative fitness of the susceptible homozygote, k; the susceptible allele will go to fixation when the reverse holds. In these simple directional selection models, there are no novel equilibria when population regulation is added to a population genetic model. From an ecological perspective, however, the equilibria are only a part of the story. Indeed, for highly forced systems (Palumbi 2001*b*), they may be only a minor part of the story. It is through the nonequilibrium dynamics that the evolutionary process will interact most strongly with the surrounding ecological community, and it is to these nonequilibrium dynamics that we turn.

We denote the frequency of resistant alleles in the population by p. For rare, recessive resistance (p and h very small), the evolutionary dynamic of the Comins (1977) model can be approximated by a Taylor expansion. For the Comins model, lacking population regulation, a second-order Taylor expansion around $p = 0$ and $h = 0$ gives

$$\Delta p = \frac{(L - k)F_1(1 - Q)}{F_1k(1 - Q) + F_2Q}p(p + h) \qquad (1)$$

where F_1 and F_2 are the fecundities in the *Bt* field and non-*Bt* field, respectively, and Q is the proportion of the refuge. This second-order approximation fits the full model until $p > 0.2$ and $h > 0.1$. However, when $p > 0.2$, resistance rapidly reaches fixation in the population, so for all practical purposes, the approximation is excellent. This approximation shows that resistance evolves at a faster rate with higher dominance (h) and resistance allele frequency (p). Recessive resistance (low h) evolves much more slowly than dominant resistance (higher h). The fraction in Eq. 1 is the reproductive advantage of the *RR* genotype (resistant homozygote) over the *SS* genotype (susceptible homozygote) in the toxic patch, relative to the reproductive rate of the *SS* genotype in both patches. Population density does not enter into the equation, and evolution does not depend on the population dynamics.

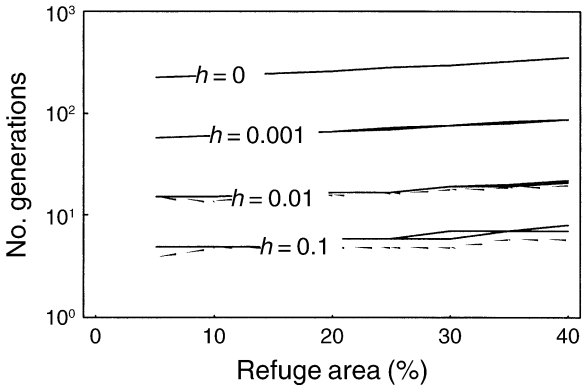

FIG. 1. Number of generations until control failure (i.e., resistance allele frequency = 50%) from the full Comins (1977) model and the second-order Taylor approximation (Eq. 2) vs. percentage of area planted as refuge for levels of heterozygote expression $h = 0$, 0.001, 0.01, and 0.1, with random movement and high *SS* mortality ($k = 10^{-3}$). For each value of h, simulations were performed for different levels of ''effective'' reproduction in nontoxic fields; the lines are often coincident. Dashed lines give the predictions from the approximation.

The Comins (1977) model with population regulation yields different rates of evolution. The second-order Taylor approximation ($p = 0$ and $h = 0$) is derived by Ives and Andow (submitted) and is

$$\Delta p \approx (L - k)\left(\frac{x_1'}{x_1' + x_2'}\right)p(p + h) \qquad (2)$$

where x' is evaluated at $p = 0$; i.e., the various x' values are the population sizes of *SS* genotypes in the toxic and refuge patches. The second-order approximation fits the full population genetic model until $p > 0.2$ and $h > 0.1$ (Fig. 1). This is similar to Eq. 1, with the same dependence on h and p. However, when population regulation is added to the model, the proportion of the *SS* population that occurs in the toxic field also influences the rate of evolution. Compared to the model without population regulation, simple density-dependent population dynamics will alter the rate of evolution of resistance. We conclude that it often will be necessary to consider both the population dynamics and the genetic dynamics when investigating the rates of evolutionary change.

The directional selection model is one of the simplest models of evolutionary biology. The results we present here suggest that evolutionary rates will be different when simple density-dependent population dynamics are explicitly considered compared to a model without an explicit population dynamic. Hence, nonequilibrium dynamics may persist for longer or shorter periods of times and perturbations from equilibria may occur more readily or less readily compared to predictions from pure population genetic models. The model that we discuss here is a simplified directional selection model, with random movement and random mating and a rare

recessive allele. It is likely that more complex directional selection models will show even richer behaviors. If these results do generalize to more complex evolutionary models, ecological interactions among species within communities may have considerable influence on the pace of evolutionary change.

Natural enemies and the evolution of resistance

Because resistance traits have strong effects on herbivore mortality, development, and behavior, it follows that the herbivore's natural enemies will be indirectly (or directly) affected by these traits (Boethel and Eikenbary 1986). Reciprocally, members of the third trophic level, including predators, parasitoids, and pathogens, also affect the outcome of plant–insect interactions, including the rate of resistance evolution by herbivores to antiherbivore defenses of plants. A community genetics perspective helps us to understand how natural enemies may affect the rate of resistance evolution of herbivores to plant defenses because of the role that natural enemies can play in biasing the mortality of resistant vs. susceptible herbivore genotypes. Gould et al. (1991) suggested that natural enemies could lead to either an increase or a decrease in the rate of resistance evolution in herbivores, depending on the details of the herbivore–natural enemy interaction. The evolution of herbivore resistance will be accelerated if the attack on susceptible genotypes is disproportionately greater than the attack on resistant genotypes; conversely, the rate of resistance evolution will be reduced by disproportionate attacks on resistant genotypes (Gould et al. 1991, Gould 1994).

In the case of strong resistance (e.g., plants expressing high doses of *Bt* toxin), differential attack of resistant and susceptible herbivores will be mediated primarily by herbivore density (Gould 1994). The density of feeding-stage herbivores on *Bt* plants will initially be orders of magnitude lower than the density of feeding-stage herbivores on non-*Bt* plants. However, resistant individuals will form a much higher fraction of the herbivores feeding on the *Bt* plants than on the non-*Bt* plants. Thus, we expect a negative relationship between herbivore density and the frequency of resistant genotypes among feeding-stage herbivores. The question therefore becomes: are low-density herbivores at higher or lower risk of natural enemy attack than herbivores at high densities (Gould 1994; J. White, *unpublished data*)? A positively density-dependent attack rate implies lower risk at low densities, which would allow resistant genotypes to 'escape' from attacks by natural enemies in the *Bt* crop. The result would be accelerated resistance evolution. An inversely density-dependent attack rate, on the other hand, would imply increased risk at lower herbivore densities and a delay in the evolution of resistance, especially if the attack rate were inversely density dependent over a range of particularly low prey densities. Thus, the effects of natural enemies attacking feeding-stage herbivores on the rate of resistance evolution will be inversely proportional to their relative impact on herbivores on toxic vs. nontoxic plants. If attack rates are greater on toxic plants, the rate of resistance evolution will be slowed, and vice versa.

The effects of egg mortality on resistance evolution are less transparent because the egg stage precedes selection based upon plant genotype. Indeed, Gould et al. (1991) suggested that the presence or magnitude of egg mortality should have no effect on the rate of resistance evolution of herbivores to plant toxins when herbivore oviposition rates are equal on toxic and nontoxic plants. But should we expect equal oviposition rates? At least two scenarios could lead to lower herbivore oviposition rates on toxic plants: (1) female choice, and (2) lower densities of ovipositing females in plantings of toxic plants. Female avoidance of plants engineered to express transgenic insecticidal toxins has not yet been documented. Lower densities of ovipositing females in plantings of toxic plants are likely, however, if these plantings are large and if female movement is at all restricted. In *Bt* maize, a key herbivore is *Ostrinia nubilalis* (Hübner) [Lepidoptera: Crambidae], the European corn borer. Because *Bt* fields generate far fewer adults of this species during its second-generation flight period than do non-*Bt* fields or refuges, the density of *Ostrinia* egg masses is lower in *Bt* maize (D. A. Andow and D. N. Alstad, *unpublished data*). It is therefore likely that *Ostrinia* egg masses will be present at lower densities in *Bt* vs. non-*Bt* maize and that this difference will be increasingly large as the size of *Bt* plantings increases. Under conditions of unequal oviposition rates in *Bt* vs. non-*Bt* maize, density-dependent egg mortality rates have an opportunity to influence the attack rates on eggs in the two habitats.

We constructed a population genetic model that allowed for positive density-dependent and inverse density-dependent mortality to act upon the eggs of an herbivore that is affected by a high-dose plant toxin in the larval stage. The model includes a nontoxic refuge and allows a fraction of females to oviposit in the same field in which they developed. Males are assumed to mate at random with females in both toxic and refuge fields. Simulations of the model show that if egg predators or parasitoids show a positively density-dependent attack rate, they impose disproportionately higher mortality in non-*Bt* fields, which accelerates the evolution of resistance to *Bt* maize in *Ostrinia* (Fig. 2). In effect, the *Bt* plants constitute a refuge from natural enemy attack and the reduced egg mortality exposes disproportionately more larvae to selection in the *Bt* fields (i.e., more resistant, but not susceptible, larvae would be allowed to develop). Alternatively, with inversely density-dependent egg mortality, the rate of resistance evolution declines (Fig. 2). Here, the refuge from natural enemy attack is in the non-*Bt* maize, where both resistant and susceptible larvae benefit. The model also shows that the level of egg mortality itself can

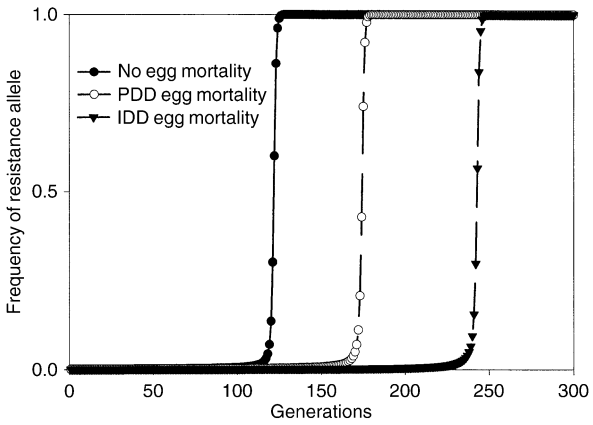

FIG. 2. Results of simulations illustrating the effects of different forms of egg mortality on the evolution of resistance of herbivores to high-dose insecticidal toxins in plants. Single-locus recessive resistance is assumed with an initial frequency of a resistance allele equal to 0.001. The area planted to toxic fields is four times the area planted to nontoxic fields, resulting in a 20% nontoxic refuge. One-fifth of the females oviposit in the same field where they developed as larvae, and the remaining females are distributed evenly across toxic and nontoxic fields. Egg mortality occurs prior to selection in the model and is simulated in three ways: (1) no egg mortality; (2) an egg mortality rate that increases with herbivore density by $N(i)/[k + N(i)]$, where k is a constant and $N(i)$ is the density of toxic or nontoxic plants (positive density-dependent egg mortality, PDD); and (3) an egg mortality rate that decreases with herbivore density by $1 - N(i)/[k + N(i)]$ (inversely density-dependent egg mortality, IDD). The constant k was set to equalize overall egg mortality rates in the PDD and IDD runs prior to fixation of the resistance allele. Herbivore fecundity was set at 100, and larvae that survive selection (or feed on nontoxic plants) are subject to density-dependent intraspecific competition.

influence the rate of evolution resistance, with higher rates of mortality slowing down the evolution of resistance.

We now turn to the natural enemies of *Ostrinia* eggs to evaluate if any are likely to cause density-dependent mortality. *Ostrinia* eggs are eaten by a number of egg predators that can collectively impose mortality rates of up to (and in some cases exceeding) 50% (Andow and Risch 1985, Andow 1990, 1992). The most important *Ostrinia* egg predators are the native lady beetle *Coleomegilla maculata*, lacewing larvae, and various predatory bugs (Andow 1990, 1992). The recently introduced multicolored Asian lady beetle, *Harmonia axyridis*, also feeds on *Ostrinia* eggs (Hoogendoorn and Heimpel 2002). *Ostrinia* eggs are subject to parasitism, but naturally occurring egg parasitism impacts <1% of *Ostrinia* eggs over most of the species' North American range (Andow 1992). Augmentative releases of native and introduced *Trichogramma* spp. can cause substantial egg mortality, but these releases, for the most part, have not been considered economically feasible, in part because multiple releases have to be made each year (Smith 1996, Andow 1997). Recent work with an egg parasitoid recently imported from northeastern China (*T. ostriniae*) suggests, however, that only a single release is needed early in the season, making it more likely that releases may become economically viable (Wright et al. 2001, 2002).

Do egg predators or parasitoids show density-dependent responses (either positive or negative) to *Ostrinia* egg density? Predators of European corn borer eggs are generalist feeders that typically also utilize aphids, other arthropods, and corn pollen in addition to European corn borer eggs (Andow 1996). Predation on *Ostrinia* eggs depends on the community composition of alternative prey, so that the response to egg density is indirect and complex (J. Harmon, *unpublished data*). Parasitism of *Ostrinia* eggs by *T. ostriniae* appears to be density independent at low host densities and inversely density dependent at higher egg densities (Wang and Ferro 1998). Like other parasitoids, *T. ostriniae* presumably can become limited by the number of eggs that they carry or by handling time if host densities get high enough (Getz and Mills 1996, Rosenheim 1996, Heimpel and Rosenheim 1998). If *Ostrinia* egg densities are high enough to induce egg or handling-time limitation in non-*Bt* maize and low enough for these factors not to come into play in *Bt* maize, then egg parasitism could indeed be higher in *Bt* maize, with a concomitant reduction in the rate of evolution resistance in *Ostrinia*.

Habitat fragmentation

Habitat destruction and resulting fragmentation are major causes of species extinctions. Investigators concerned about the persistence of remnant plant populations have documented ecological and genetic effects of habitat fragmentation (Leach and Givnish 1996, Young et al. 1996, Newman and Pilson 1997). To obtain a community genetics perspective on the interplay of these effects, we have incorporated the empirical results of Wagenius (2000) on *Echinacea angustifolia* (Asteraceae), the narrow-leaved purple coneflower, into an individual-based, spatially explicit, stochastic simulation model. *Echinacea angustifolia* is native to the North American tallgrass prairie, which has been reduced to isolated fragments in a matrix of agriculture during the past 150 yr. Contrary to the results of a purely ecological model, we find that the genetic properties of our study system exacerbate the risk of extirpation.

Echinacea angustifolia shares five key features with many of the plants that formerly dominated unbroken prairie: (1) long life (*Echinacea* is a long-lived perennial and plants rarely flower before their third year); (2) reproduction strictly by seed (*Echinacea* does not spread vegetatively, so regeneration of populations depends exclusively on seed production); (3) self-incompatibility (seed set from each floret depends on receipt of pollen from another plant; McGregor [1968], Leuszler et al. [1996], Franke et al. [1997]); (4) pollination by generalist insects (service by nonspecialized pol-

linators is likely to limit flights for pollen or nectar to short distances; Kunin [1992]); (5) no specialized mechanisms of seed dispersal (*Echinacea* seeds are unlikely to colonize distant habitat patches). These features of *Echinacea* make it a suitable model for the conservation of many native perennial plants.

In a 6400-ha study area of western Minnesota farmland, Wagenius (2000) mapped >2000 *Echinacea* plants in 48 remnant populations varying in size from one to several thousand flowering individuals. He found that pollen limitation increases with the isolation of individual plants and that pollen limitation reduces seed yield. He also found that plants grown from seeds collected in small populations are less vigorous than those from large ones. Each of these findings could result from ecological or genetic processes.

Ongoing research tests ecological and genetic hypotheses about the causes of pollen limitation and reduced vigor. Two non-exclusive processes could influence pollen limitation. An ecological hypothesis holds that isolated plants receive fewer visits and, consequently, less pollen from pollinating insects than non-isolated plants because of restrictions on pollinator flight. According to a genetic hypothesis, plants receive similar amounts of pollen regardless of their isolation, but isolated plants receive a greater proportion of incompatible pollen from related plants that share self-incompatibility alleles than do non-isolated plants. Preliminary results do not support the ecological hypothesis. However, there is evidence that the receipt of incompatible pollen varies with isolation; mating incompatibility rates are now being assessed in small and large fragments. Similarly, two factors might influence plant vigor: under an ecological hypothesis, aspects of the abiotic environment (such as the fire regime) or biotic environment (such as abundance of exotic plants) differ in small and large remnants. Under a genetic hypothesis, genetic composition differs between small and large remnant populations. Not surprisingly, plant vigor varies with both ecological and genetic factors.

To assess how the interplay of the ecological and genetic processes affects population persistence, we developed an individually based, spatially explicit, stochastic simulation model. The model builds on previous models of habitat fragmentation and incorporates additional processes that affect population growth and persistence: density-dependent mortality, seedling recruitment as a function of the fire regime, and dispersal of pollen and seeds, as well as heterozygosity, as functions of local *Echinacea* abundance.

The simulation model demonstrates that a purely ecological framework is insufficient to predict the consequences of habitat fragmentation in our system. The purely ecological framework is based on Levins' (1969) metapopulation model, which describes the fraction of occupied patches, $u(t)$, in an infinite patch model with global dispersal:

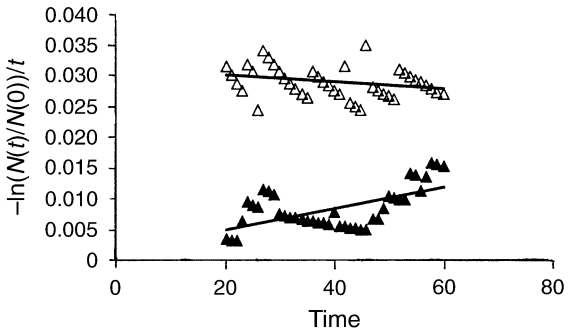

FIG. 3. Results of simulations on patch mortality as a function of time. The regression line for self-incompatible plants (solid triangles) shows a positive slope, indicating an increasing extinction rate due to reduced seed production caused by reduced availability of compatible pollen. The regression line for self-compatible plants (open triangles) shows a slope close to 0, indicating that patch mortality is constant over time.

$$\frac{du}{dt} = cu(1 - u) - eu. \tag{3}$$

Here, the parameter c denotes the colonization rate and e denotes the extinction rate of each patch. If a fraction D of the habitat is permanently destroyed, the model yields the following (Nee and May 1992):

$$\frac{du}{dt} = cu(1 - D - u) - eu. \tag{4}$$

It follows that the fraction of occupied patches in equilibrium is given by

$$\hat{u} = 1 - D - \frac{e}{c} \tag{5}$$

implying that the population dies out if the fraction of destroyed habitat D exceeds $1 - e/c$.

The model in Eq. 4 assumes that both the colonization rate and the extinction rate of a patch remain unchanged after habitat destruction. (Similar assumptions are made in the theory of biogeography.) Neither assumption holds: colonization depends on reproductive output, which decreases with fragmentation due to reduced availability of compatible (unrelated) plants. Likewise, persistence depends on reproductive output and individual survival, both of which are compromised when individual fitness declines with inbreeding in small remnants (Newman and Pilson 1997). For these reasons, we include genetic components in our model, varying the number of self-incompatibility alleles and the rate of inbreeding, to predict the fate of the remnant populations. In the simulation, we initially run the model without habitat fragmentation until an equilibrium is reached. A fixed fraction of the habitat is then destroyed and the fate of the patches is followed. Considering self-incompatibility alone, we find that the probability of extirpation increases over time (Fig. 3). Standard ecological models assume a constant patch

extinction rate and thus cannot predict this trend. Therefore, standard ecological models overestimate the persistence of remnant populations. Ongoing work is exploring the further consequences of inbreeding in the context of habitat fragmentation.

Habitat fragmentation poses threats from mechanisms as diverse as increased mating system incompatibility and reduced fire frequency. The community genetics perspective considers the joint consequences of these aspects of fragmentation, promoting understanding of how ecological and evolutionary processes together affect population persistence.

Domestication as invasion

Beginning <150 yr ago, vast acreages of temperate forests and grasslands in North America were converted to agricultural production, and in that process, novel crop plant genotypes were introduced (Smith 1989). In North America, the conversion to modern maize arguably represents one of the largest plant range expansions within human history. Although the impact of agricultural conversion on the landscape is readily apparent, the impact on plant pathogens and the coevolution of plant and pathogen is less apparent, but no less important. We focus on possible effects of the rapid geographic expansion of maize and its associated fungal pathogen, *Ustilago maydis*, corn smut.

The maize–smut interaction is ideal for the study of coevolutionary dynamics under nonequilibrium conditions because genotypes of both species can be manipulated and the population history of maize is well understood. The transition from small, genetically variable teosinte populations of Central America to the large, monotypic maize plantings of North American agriculture is expected to accelerate the evolution of virulence in associated maize pathogens. *U. maydis* is a naturally occurring pathogen on both teosinte and maize (Duran 1987) and has tracked maize from domestication to present-day plantings. The history of maize domestication and spread are well documented (Galinat 1992), as is the molecular genetic basis of its evolution from teosinte (e.g., Doebley 1992, Hilton and Gaut 1998). Virtually every maize plant grown in North America has a recorded pedigree, but much less is known about *U. maydis* evolution. Historical records show that maize breeding programs for smut resistance of the early 1900s were successful; surprisingly, the pathogen has not evolved to overcome smut resistance traits developed at that time (Christensen 1963), and smut resistance in maize has proven durable over 50 yr. Our long-term goal is to account for the difference between the observations of low levels of smut infection on widely planted corn varieties and an expectation for rapid evolution of virulence in agricultural monocultures. More immediately, we ask how maize domestication and geographic range expansion have affected the population genetic structure of *U. maydis*.

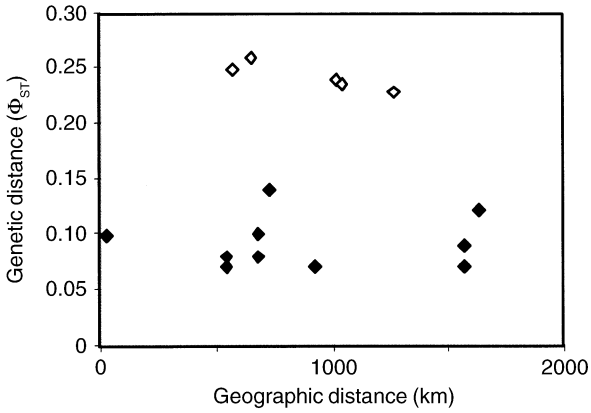

FIG. 4. Correlation of genetic (as Φ_{ST} values) and geographic distances in pairwise comparisons of North American populations of *Ustilago maydis*. Open symbols show pairwise comparisons between Ohio and other North American populations. The Φ_{ST} levels from pairwise comparisons show no correlation with distance, using a Mantel test ($rY = -0.006$, $P = 0.35$; Schneider et al. 1997).

Seven North American populations of *U. maydis* were sampled from field corn and sweet corn, and genetic relatedness of the populations was assessed using 11 genomic probes for restriction fragment length polymorphisms (RFLPs). Using Φ_{ST}, a multilocus analog (Schneider et al. 1997) to Wright's inbreeding coefficient, F_{ST}, genetic variation within and between North American populations was estimated. Values for Φ_{ST} varied from 0.07 to 0.26 across pairwise comparisons of these populations. Overall levels of heterozygosity are quite high (~0.4) and estimated levels of inbreeding within one population are low ($F_{IS} \sim 0.06$), despite the fact that sib matings could occur. Using an Index of Association test (Agapow and Burt 1999), analysis of linkage disequilibrium revealed that the alleles demonstrating significant association between loci within populations were not the same in all sampled populations. In contrast to the population genetic structure observed for many crop pathogen species (e.g., Boeger et al. 1993), we did not observe isolation by distance (Slatkin 1993), as one might expect with wind-borne dispersal of spores and high migration rates (see Fig. 4). Together, these data demonstrate that *U. maydis* populations are sexually reproducing and maintain high levels of heterozygosity, distributed unevenly among genetically variable populations across the major geographic regions in which maize is grown (J. Garton and C. Ramos, *unpublished manuscript*).

We developed two hypotheses to explain our observations. (1) The variation that we observe across smut populations reflects historical founder events as maize was brought into North America about 1000 yr ago. These populations have not come to equilibrium by genetic drift and migration. (2) The genetic variation among smut populations is the result of host or regional environmental selection effects (e.g., Ahmed et al.

1996). These are not exclusive hypotheses. To generate expectations for historical sampling effects alone, in collaboration with Frank Shaw, we modeled the process of establishing North American descendant populations. All observed variation was placed in an ''ancestral urn'' of Central America and alleles were drawn to found new descendant populations, without migration among descendants. The model is comparable to assuming that smut was brought in with the introduction of the crop, but that little migration between populations subsequently occurred. We used data for the self-incompatibility locus of the fungus, *b*, for which 18 alleles are described and occur in approximately equal frequencies due to balancing selection (Zambino et al. 1997). Thus, each allele would have an approximately equal chance of being sampled from the ancestral population and, upon arrival in a new population, would be less likely to be lost due to drift. The draws were independent of each other and we assumed that the source population was large enough so that sampling with replacement was a good approximation (binomial sampling). Using this model, we constructed a statistical test based on comparing the empirical observations for the occurrence of *b* alleles in subpopulations (Zambino et al. 1997) with those obtained from the simulation model. The simulations were carried out with 5, 20, and 100 descendant populations and with the number of alleles drawn varied at 3, 5, 10, 20, and 30. Each combination of parameters was repeated 1000 times. Our simulation results do not allow rejection of the null hypothesis that the distribution of *b* mating-type alleles in current populations were determined by historical, random draws from an ancestral population. However, the statistical power is low in this chi-square test.

The second selection hypothesis is suggested by the data for the Ohio population, which displays pairwise Φ_{ST} values ($\sim$0.24) twice those for other pairwise comparisons in North America (see Fig. 4). In the mid-1900s, breeding for smut resistance was conducted at the research station from which collections were made. Further, pairwise comparison of the North American populations with one Uruguay collection suggests that selection due to host genotype or other regional environmental factors could have a large impact over a short time. We observe high Φ_{ST} ($\sim$0.4), values typically associated with differences between species, yet the Uruguay and North American populations can only have been separated by a period of 4000–5000 yr. Altogether, our data suggest that relatively large or diverse populations of smut arrived in North America with maize, and that insufficient time since the introduction ($\leq$1000 yr) has elapsed to distribute that variation evenly across the widespread maize plantings.

Remarkably, then, the current populations of smut in North America might be the ''footprints'' of early introductions rather than the result of high migration rates that we often associate with agricultural pathogens (Burdon and Silk 1997). To now resolve the relative importance of historical founder events and strong selection in establishing the observed patterns of genetic variation in *U. maydis* populations, we will combine experimental population approaches, models, and sampling in geographically diverse locations, especially Central America. Our results to date suggest that the pathogen population in North America is primarily outcrossing and sexual, rather than inbreeding or clonally reproducing. Such a pathogen reproductive system, combined with the quantitative resistance in maize (e.g., Lubberstedt et al. 1998), may contribute to the strikingly slow evolution of virulence in this pathogen's populations. Lack of correspondence between pathogen virulence and host resistance structure has previously been attributed to the patchy distribution of genetically variable host plants and environment (Burdon et al. 1989, Clarke et al. 1990, Jarosz and Burdon 1991, Burdon and Silk 1997) and to the cost of resistance (Bergelson and Purrington 1996). If additional studies reinforce our view of the evolutionary interaction of maize and its very common pathogen, corn smut, we might add a third factor. Could history and the genetic structure of the maize–smut interaction conspire to make an evolutionary ''cold spot,'' analogous, but opposite in outcome, to Thompson's (1999) evolutionary ''hot spots''?

Discussion

Community genetics jointly considers genetic and numerical dynamics of interacting populations to yield fresh insight into evolution within the context of communities. We demonstrated the value of a community genetics approach for studies characterized by nonequilibrium dynamics in which strong interactions act on genetic variation. Nonequilibrium situations are common now; anthropogenic disturbances impose strong selection regimes on many natural and managed communities. Indeed, unmanaged natural communities are increasingly scarce and management itself can be a large disturbance. In addition to the fundamental insights that community genetics offers, its approaches become more compelling as the human impact on the landscape mounts. Principles that we derive from such studies will be valuable in sustainable management of resources, conservation genetics, and urban planning.

The idea that evolutionary change can occur on relatively short time scales is not new and can be traced back to work by Dobzhansky and others in the 1940s. For instance, Timofeef-Ressovsky (1940) and Dobzhansky (1943) demonstrated rapid evolutionary changes coinciding with seasonal cycles. Rapid evolutionary changes can also occur in a static abiotic environment as a result of dynamic interactions among organisms within a community, as predicted in the Red Queen model and its derivatives (Van Valen 1973).

A community genetics perspective can bring novel insight not only to nonequilibrium situations such as

we discuss here, but also to the case of equilibrium dynamics with strong balancing selection. For instance, Antonovics (1992) investigated a model of host–pathogen interactions, in which he demonstrated that the observed coexistence of susceptible and resistant plant genotypes was only consistent with a model that combined ecological and genetic dynamics. We focused on nonequilibrium situations because they are more likely to produce pronounced effects of interactions. The four studies in this paper demonstrate (1) that an ecological or an evolutionary framework by itself is insufficient to understand or predict outcomes of organismal interactions during the transient phase following perturbation, and (2) that novel predictions about community change can emerge from mathematical models that incorporate both ecological and genetic processes. Only when we consider both factors in concert can we understand community dynamics following a large perturbation that imposes strong selection on the community. In each of the studies that we discussed, strong selection occurs naturally; it is characterized by an abrupt and large change that imposes strong selection on the community. The transient phase to equilibrium may be long lasting and, with increased human impact, an equilibrium may never be reached.

We believe that our examples are illustrative of the common phenomenon of nonequilibrium conditions in contemporary communities. Agricultural and forested areas, which cover over half of the earth's terrestrial area, are subjected to massive ecological and evolutionary disturbances, generating nonequilibrium dynamics within those habitats, as illustrated in our first example of resistance evolution. These same activities generate nonequilibrium dynamics in native habitats by fragmenting and isolating these habitats, which we illustrated in our example of patch dynamics in prairies.

We recognize that many of our examples involve only two-species ''communities.'' We have chosen to highlight these relatively simple interactions to lay bare the structure of community genetics and illustrate some of the necessary conditions under which it can truly matter. We suggest, however, that this focus does not limit the generality of our analysis. Indirect interactions in ecological communities appear to be common (Holt and Lawton [1994], Wooton [1994]; see also the companion paper by Whitham et al. [2003]). Hence, evolutionary change in two-species interactions has the potential to affect other members of the community through indirect species interactions. For instance, genetic variation in resistance to herbivores in plants can affect the herbivores' natural enemies (Price et al. 1980). Plant defenses reduce the fitness of predators and parasitoids of herbivores feeding on them (Campbell and Duffey 1979, Duffey et al. 1986, Obrycki 1986). Thaler (1999) also recently showed that the production of plant defenses is correlated with the production of volatiles that attract parasitoids of the herbivores feeding on the defended plants. In this case and some others, the attraction of an herbivore's natural enemies appears to be a component of the plant's defensive reaction (DeMoraes et al. 1998, Bradburne and Mithen 2000). In examples such as these, one can envision evolution within entire multispecies complexes.

The importance of taking a combined ecological and evolutionary approach to understand the effects of large-scale perturbations was recently emphasized by Palumbi (2001*a*) in a *Science* article entitled ''Humans as the World's Greatest Evolutionary Force'' (see also Palumbi 2001*b*). The rapid growth of the human population has led to unprecedented alterations of natural ecosystems and widespread introduction of novel organisms. Consequences of human impact are felt in all areas, including epidemiology (emerging diseases, resistance to antibiotics), pest management (evolution of resistance to pesticides), species invasions (globalization, homogenization), species extinctions (habitat fragmentation, climate change), and expansion of agricultural land (to destroy natural habitats).

The magnitude and spatial extent of disturbances are staggering; no habitat seems to be fully protected. Coastal marine environments around the world have been massively perturbed by dredging, pollution, impoundments, and an astounding number of invasive species (Carlton 1999). Riparian habitats in remote mountains have been massively perturbed by removal of beaver, damming of rivers, recovery of beaver, and biological species invasions. In highly perturbed systems, ecological and evolutionary forces are equally important.

A community genetics perspective relying on a theoretical framework not only leads to a more complete understanding of the consequences of human-induced selection pressure, but also provides a sound basis for the development of management strategies. Developing a unified theoretical framework is of paramount importance because our actions induce both ecological and evolutionary change. Without such a comprehensive framework, our understanding of the complexity by which communities and ecosystems respond to our actions will be severely compromised, and we will never be able to develop general management strategies to address these responses.

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