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# EFFECTS OF CHRONIC PESTICIDE STRESS ON WILDLIFE POPULATIONS IN COMPLEX LANDSCAPES: PROCESSES AT MULTIPLE SCALES

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**Abstract**—Populations exposed to pesticides may often be divided into subpopulations, where some subpopulations are exposed to pesticides and others are not. We consider three models for such populations. The first is a simple discrete time model with no density-dependent reproduction. The second is a continuous time model that includes density- dependence for populations not exposed to pesticides. In both models, populations exposed to pesticides are assumed to decline geometrically (exponentially in the continuous time model). Migration between exposed and safe habitat patches is symmetric, so that all individuals leaving safe patches enter exposed patches and vice versa. The rate is assumed to be the same for both kinds of patches. The conditions for persistence of the population in the landscape are the same for both models. Increasing migration rate between patches decreases the ability of the population to persist. Populations that have low rates of increase in safe habitat patches have greater difficulty persisting than those with high rates of increase in safe patches. The toxicity of the pesticide also affects population persistence. More toxic pesticides that result in higher rates of death and/or lower rates of birth in exposed habitat patches lower the ability of the population to persist in the landscape. We consider an additional set of models of a metapopulations that persist because of a balance between colonization and local extinction. In such systems, pesticides may endanger regional persistence by reducing the pool of sites available for colonization. We conclude with an outline of important future directions for theoretical research intended to elucidate the impact of pesticides on populations spatially complex landscapes.

**Keywords**—Metapopulations Landscape ecology Population dynamics Population persistence

## INTRODUCTION

Wildlife populations often exist in landscapes created by fragmentation of natural habitats. Such populations may have dynamics different from those in relatively continuous habitat. The addition of pesticides to a complex landscape may create a system of populations with refuge, or source, habitats—where individuals are not exposed to pesticides and populations tend to persist—interspersed with sink habitats—where individuals are exposed to pesticides and populations are faced with extinction.

Studies of the toxicity of new pesticides may be conducted in either field and laboratory settings. In the laboratory, bioassays are often used to determine the level of toxin exposure that leads to mortality or sublethal effects on physiological functioning. Such studies establish toxicity levels under controlled exposure levels. Field studies are conducted by application of the pesticide, mimicking to a degree normal application conditions, with subsequent monitoring of mortality. Retrieved carcasses are analyzed for toxin levels; live individuals may also be collected to ascertain sublethal effects.

The basic assumption of these studies is that a causal connection can be established between toxicity concentration in tissues of individual animals and the likelihood of their death or impaired reproduction. Furthermore, it is assumed that the average effect of the pesticide on individual organisms translates simply into population mortality and natality rates. Hence, if the effect of the pesticide at a certain dose causes the death rate to exceed the rate of recruitment of new individuals into the population, one predicts the population will go extinct locally; otherwise, the population is assumed to persist.

This approach to assessment of pesticide effects assumes that the population in question exists in a homogeneous environment, and that it is unconnected to populations in areas not affected by the pesticide. Thus, if a pesticide causes a local population decline, that decline is assumed not to affect other populations that are not directly exposed to the pesticide. Wildlife populations in natural and human-dominated landscapes, however, often exist in patches between which considerable migration might occur. For example, suppose a population of frogs occurs in ponds distributed throughout an agricultural landscape. Some ponds may be close to agricultural fields and receive relatively high doses of pesticides through runoff, while others may be located in reserves or wildlife refuges and receive little or no runoff. If individual frogs disperse from pond to pond before breeding, then ponds not exposed to pesticides are connected demographically to those exposed to pesticides. The rate of exchange of individuals among ponds will determine how connected frog populations are in the different ponds. The assessment of pesticide impact needs to be cast at the level of entire landscapes. A landscape is the spatial arena defined by the dispersal biology of the target species.

## POPULATION DYNAMICS IN SPATIALLY COMPLEX ENVIRONMENTS

The purpose of this paper is to examine the dynamics of complex populations made up of source and sink habitats from the perspective of assessing the effects of pesticides on total population persistence. We note that not all instances of pes-

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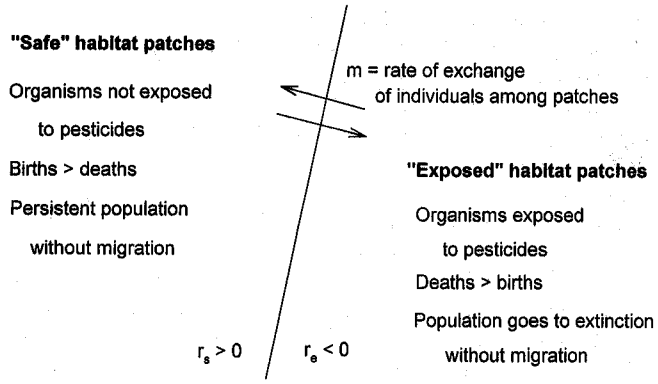


Fig. 1. General structure of population models of the effects of pesticides in a spatially structured population. The landscape is divided between patches where individuals are exposed to pesticides and those where individuals are not exposed. In exposed habitat patches, deaths exceed births so that in the absence of migration, the population would go extinct.

ticide exposure to natural populations will be described by the approaches we consider here. An important underlying assumption that we make in these analyses is that individuals in a population are exposed for a sufficiently long period of time to alter their ability reproduce and survive. We consider relatively simple models that capture some essential features of spatially heterogeneous populations. After presenting results from these models, we discuss the implications of spatial heterogeneity for the persistence of populations exposed to pesticide stress. Our conclusions suggest that regulatory decisions on pesticide use should be guided in part by potential effects of pesticides on populations across large landscapes. Monitoring of pesticide effects may require large spatial and long temporal scale approaches.

Two basic aspects of spatial complexity are important in population dynamics. Each of these need to be considered when assessing the likely impact of a pesticide on population persistence.

First, individual birth or death rates can vary spatially, reflecting underlying pre-existing variation in the environment, or variation arising de novo, for instance, because of the application of a pesticide. The way in which such individual variation in vital statistics influences overall population dynamics is governed by mobility of individual organisms. Two models presented below explore these effects.

Second, subpopulations in different habitat patches may experience semi-independent dynamics, such that extinction is localized within patches. Thus, decline in populations in one patch need not affect directly populations in other patches. The important dynamics in such population systems are in the persistence of populations across the landscape rather than population changes within patches. In an ensemble of patches coupled by dispersal, local colonizations may balance local extinctions, such that the metapopulation as a whole persists, even though each constituent subpopulation inevitably goes extinct. A pesticide may endanger the persistence of the metapopulation by reducing colonization rate or increasing the rate of local extinction, or by rendering totally unsuitable a fraction of the landscape that otherwise would be habitable. The third model we consider highlights the latter effect.

*Effects of pesticides on population vital rates*

The basic structure of the first two models we consider is given in Figure 1. We assume that there is a "metapopulation" composed of subpopulations inhabiting different kinds of patches that are coupled by dispersal. For our purposes, we assume that the major difference between patches is that in some patches, subpopulations are exposed to pesticides. We assume that the lethal and sublethal effects of the pesticide result in a death rate that exceeds the birth rate in these subpopulations. They will go extinct (that is, the subpopulation abundance drops to zero) without any migration from outside. Such subpopulations are often called "sink" populations [1,2]. Subpopulations not exposed to pesticides are assumed to persist in the absence of migration from other subpopulations. These are referred to as "source" populations. We assume that individual organisms can move freely between safe and exposed habitat populations and that their reproductive capacities and probabilities of death are determined only by the patch that they currently reside in. That is, we assume there are no effects of pesticides that are carried over between safe and exposed habitat patches. Clearly, some pesticides that persist for relatively long periods of time can move between patches by physical transport processes or as body burdens. This, in turn, would decrease the suitability of safe patches. If pesticide effects were spread throughout the population in this manner so that all patches were exposed, the persistence of the population would depend on the overall pesticide effect, and it would not be necessary to consider spatially heterogeneous models. Hence, we consider what additional complications arise when pesticide effects are not uniformly distributed among patches. Furthermore, we note that the models we discuss here may be applicable to environmental differences between patches other than exposure to pesticides. For the present, however, we assume that differences between patches are primarily due to presence of pesticides in sufficient quantities to create a source-sink population structure.

The simplest possible model of population dynamics in a system of subpopulations like that just described is for a population with a discrete breeding season changing in a density independent manner. In such a model, birth and death rates are constant over time within each patch. In particular, these rates do not depend on the number of individuals currently in the patch. In such a population, there are only two kinds of population behavior: either the population increases without limit or it goes extinct. Thus, understanding population persistence amounts to finding those conditions under which population change is positive.

The model described above can be stated more precisely with a pair of coupled difference equations. Let $N_s(t)$ be the number of organisms in safe habitat patches in generation $t$ and $N_e(t)$ be the number of organisms in habitats exposed to pesticides. Then the population sizes in each habitat type through time can be described as follows:

$$N_s(t) = N_s(t-1) + r_s N_s(t-1) - mN_s(t-1) + mN_e(t-1)$$
$$N_e(t) = N_e(t-1) + r_e N_e(t-1) + mN_s(t-1) - mN_e(t-1) \quad (1)$$

Here, $r_s$ and $r_e$ are the finite per capita birth rates minus per capita death rates in each subpopulation. In the absence of migration, the proportional change of the population in exposed patches is $\lambda_e = 1 + r_e$. Note that in exposed patches, because the per capita death rate exceeds the per capita birth rate, $-1 < r_e < 0$, so that the proportion of the population left is less than unity, so the population will decline. Likewise, the proportional change of the population in safe patches is $\lambda_s$

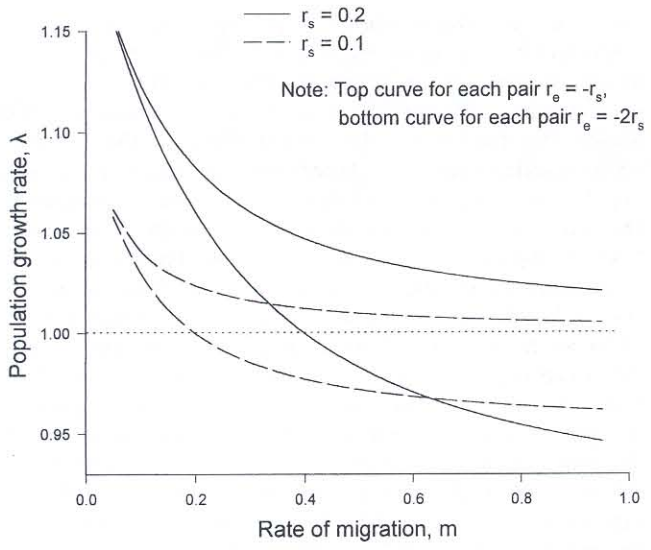


Fig. 2. Growth rate in a discrete-time, density-independent population as a function of rate of migration between source and sink populations. The $r_s$ is the rate of growth in source populations and $r_e$ is the rate of decline of populations exposed to pesticides.

$= 1 + r_s$. Because births exceed deaths in these patches, $r_s > 0$ and there is a proportional increase in these populations. The parameter $m$ represents the rate of migration each generation, which is the fraction of individuals moving from one patch type to the other each generation. Because there is free exchange of individuals among patches, the rate of migration is symmetrical: the proportion of individuals that leave safe patches and go to exposed ones is the same as those that leave exposed patches and go to safe ones (this basically assumes that safe and exposed areas are roughly the same size).

The dynamic behavior of the difference equations in Equation 1 can be examined by considering the rate of change of the entire population. That is, if $N(t) = N_s(t) + N_e(t)$ is the total population size in all patches, then $N(t)/N(t-1) = \lambda$ is the growth rate of the population when the population achieves a stable patch distribution where a defined fraction of the population is in each kind of patch and this fraction does not change over time [3]. This growth rate is a function of the parameters $r_s$, $r_e$, and $m$. The exact relationship is obtained by writing Equation 1 as a matrix equation and finding the largest eigenvalue of the transition matrix (see Caswell [4] for technical details).

For any given combination of the growth parameters in source ($r_s$) and sink ($r_e$) populations, the total population growth rate, λ, is

$$\lambda = \frac{2 + r_s + r_e - 2m + \sqrt{(r_s - r_e)^2 + 4m^2}}{2} \qquad (2)$$

λ is a decreasing function of migration rate (Fig. 2). If $|r_e| \leq r_s$, then the population growth rate never falls below one, so the population persists for all possible rates of migration. However, if $|r_e| > r_s$, that is, the per capita rate of decline in patches exposed to pesticides is greater than the rate of increase in the patches, then increasing migration rate will eventually lead to $\lambda < 1$, so that the population will go extinct if migration rates exceed a certain value. This threshold value for migration rate is

$$m = \frac{r_s r_e}{r_s + r_e} \qquad (3)$$

We will explore this condition more completely in a moment. For now, note that for the simple density-independent model, migration rate can lead to extinction of the total population if conditions in patches exposed to pesticides lead to declines that exceed the rate of increase in safe patches. Hence, a sufficiently toxic pesticide may not only cause extinction of those subpopulations directly exposed to it but may also cause extinction of the species across an entire landscape, if there is free migration between subpopulations.

It is important to ask if the conclusions drawn from the density-independent model are a consequence of its simple, discrete-time structure, or whether it is a more general property of metapopulations distributed across a heterogeneous landscape. Here we examine a model of continuous-time dynamics for a species with density-dependent mortality and reproduction [1]. Assume that in safe habitat patches, birth and death are density-dependent so that per capita death rate exceeds per capita birth rate when abundance exceeds $K_s$. The parameter $K_s$ is sometimes referred to as the "carrying capacity" in studies of wildlife populations. In patches exposed to pesticides, we assume that carrying capacity is zero (i.e., $K_e = 0$). That is, for all abundances, death rate exceeds birth rate. We model the population dynamics in exposed patches as an exponential decline at rate $r_e$. Using logistic demographics in the safe patches, we can write a pair of differential equations to describe the dynamics for the total system as

$$\frac{dN_s}{dt} = r_s N_s\left(1 - \frac{N_s}{K_s}\right) - mN_s + mN_e$$

$$\frac{dN_e}{dt} = r_e N_e + mN_s - mN_e \qquad (4)$$

The parameters have similar interpretations to those in the density-independent model, with the following two exceptions: (1) They represent instantaneous rates of change and (2) $r_s$ is the maximum rate of per capita rate of growth achieved when densities are very low ($N_s \to 0$). Note that $r_e < 0$ (with no lower bound, unlike the discrete-time model). These equations have a stable equilibrium for total population size ($N = N_s + N_e$), given by Holt [1] as

$$N = K_s\left(1 + \frac{r_e m}{r_s(m - r_e)}\right)\left(1 + \frac{m}{m - r_e}\right) \qquad (5)$$

The equilibrium total population size is usually less than the carrying capacity for safe habitat patches because the product of two terms in parentheses will be less than one except for very small migration rates and for $r_s$ near zero [1]. When the proportion of carrying capacity in safe patches represented by the equilibrium population ($N/K_s$) is plotted against migration rate, it declines with increasing rate of migration (Fig. 3). Similar to the conditions in the density-independent model, when the rate of decline in exposed patches is less than or equal to the maximum rate of increase in the safe patches, the population will persist in the landscape, that is, equilibrium population size will be greater than zero for all possible migration rates. However, when $|r_e| > r_s$, then increasing migration rate will eventually lead to extinction of the metapopulation in the landscape (Fig. 3). The migration rate above which the metapopulation ceases to persist is identical to that given in Equation 4 for the density-independent model. That is, density dependence does not affect the conditions for metapopulation persistence, only the size of the population given that it will persist. The basic reason is that when populations are

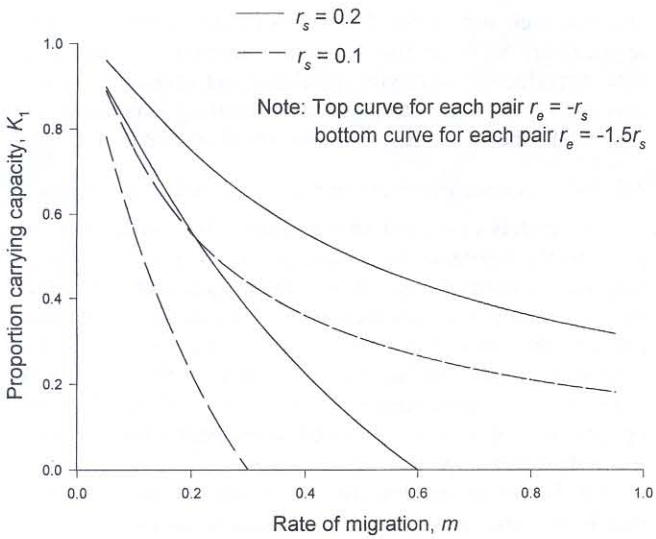


Fig. 3. The proportion of carrying capacity in source populations that the total population achieves as a function of rate of migration between populations for a continuous-time, density-dependent population. Note that density dependence applies only to source populations because carrying capacity in sink populations is zero. $r_s$ is the maximum rate of growth in source populations and $r_e$ is the rate of decline in sink populations.

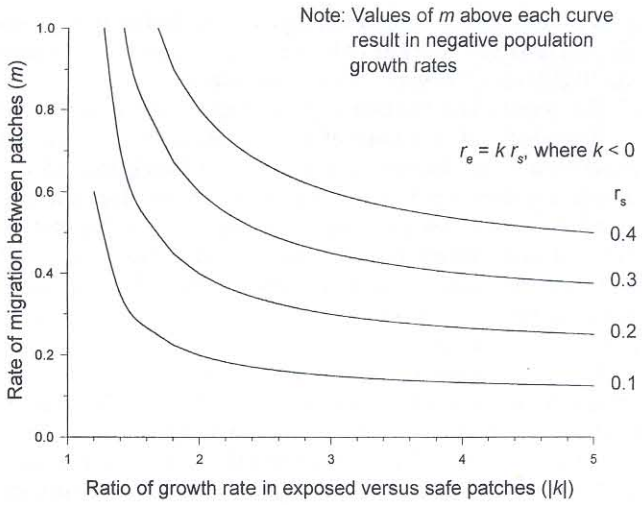


Fig. 4. Conditions for population persistence in both models considered in the text. For any given combination of growth rates in source and sink populations ($r_s$ and $r_e$, respectively), values for the rate of migration that exceed the value on the specified curve will cause the entire metapopulation to go extinct.

at risk of extinction, population densities are likely to be low, and hence the effects of density dependence are likely to be negligible.

We conclude that for the density-dependent model in continuous time, migration rate can lead to extinction of the total population if conditions in those patches exposed to pesticides lead to declines that exceed the maximum rate of increases in safe patches. As with the previous model, a sufficiently toxic pesticide will not only cause extinction of those subpopulations directly exposed to it but will also cause extinction of the species across the entire landscape if there is free, regular migration between subpopulations. This conclusion seems to be a general phenomenon arising in metapopulations with subpopulations that freely exchange individuals.

We now consider in more detail the conditions for metapopulation persistence given in Equation 3, which characterize both models (with the appropriate interpretations given to population growth parameters $r_s$ and $r_e$). Our aim is to draw some qualitative conclusions regarding what kinds of processes that affect the persistence of a metapopulation in a landscape where some of its populations are declining due to pesticide exposure.

Note that for both models, increasing the connectedness among patches increases the likelihood that the metapopulation will go extinct, because in both models, the criterion for persistence becomes more stringent with increasing migration rate (Figs. 2, 3). Hence, when a pesticide is toxic enough to drive exposed populations to extinction, persistence of the metapopulation is more likely to be compromised if patches are tightly connected.

As the maximum growth rate in patches not exposed to pesticides decreases, there is an increased likelihood of metapopulation extinction (Fig. 4). High maximum growth rate in protected areas allows a metapopulation to offset the negative effects of migration on persistence as subpopulations near extinction are able to recover more rapidly. Species with high maximal growth rates (e.g., many insect pests) are thus better able to persist in the face of localized pesticide applications than, say, large-bodied species with low fecundities (e.g., many endangered vertebrate species).

The difference in growth rates between patches also affects the ability of a metapopulation to persist. As the rate of decline in exposed patches increases relative to rate of increase in safe patches, the rate of migration in the metapopulation that leads to its extinction declines (Fig. 4). That is, when subpopulations in exposed patches go extinct very rapidly, it takes a smaller rate of migration to drive the metapopulation extinct than when subpopulations in exposed patches go extinct slowly.

One difference between the discrete-time and continuous-time models that has implications for real populations is that, in general, persistence is more likely with discrete generations. For a species with discrete generations, the lower bound on the rate of population decline in exposed habitats is $\lambda_e = 0$, or $r_e = -1$. Moreover, the maximal rate of movement is $m = 1$ (which would correspond to regular migration between habitat types). Given these limiting, worst-case parameter values, $r_s > 1$ will always ensure persistence of the metapopulation.

By contrast, with continuous-time dynamics and movement, for any given $r_s$, if $|r_e| > r_s$, the population faces extinction at a sufficiently large $m$. The reason for this difference in the two models is that with discrete generations, one assumes that individuals in safe habitats remain protected between successive generations, with no exposure to pesticide-laden habitats—in effect, a kind of refuge. In the continuous-time model, individuals continually flow back and forth between safe and exposed habitats; no individual remains protected from the pesticide very long. Hence, for species that have life cycles leading to discrete generations, effects of migration on the demographic consequences of pesticide stress may be somewhat less than for species without discrete generations.

To summarize, three major demographic processes affect the ability of a metapopulation to persist when some populations decline due to pesticide effects and all populations are coupled by regular movements of individuals. First, relatively high migration rates increase the likelihood of metapopulation extinction. Second, low rates of increase in safe habitat patches prevent the metapopulation from recovering from the negative

effects of migration. And third, high rate of decline in exposed patches relative to the rate of increase in safe patches decreases the likelihood of metapopulation persistence.

Our results have methodological implications. Assume that a "snapshot" of mortality effects is taken shortly after an experimental application of a pesticide. Finding that only a modest fraction of individuals has been exposed does not necessarily imply a low extinction risk: individuals may not be exposed immediately because they are in "safe areas," yet with time they may move and become exposed. The parameter $m$ in our models scales the rate of such exposure of initially protected individuals.

These conclusions are based on deterministic models with relatively simple demographic structure. We have not considered the effects of environmental or demographic stochasticity nor of age structure. These demographic processes may alter our conclusions somewhat. We expect, however, that the conditions outlined above are "best case" scenarios. That is, when we consider more complex models, we expect that the conditions for metapopulation persistence will become more stringent. Holt [5] considered a stochastic version of a discrete version of the logistic growth equation (analogous to the model described in Equation 4). Unlike the deterministic models considered here, when stochastic effects are included in a model, metapopulation size (i.e., the number of distinct subpopulations) becomes important to persistence because small populations are more likely to go extinct due to chance events than large ones [6–8]. Holt [5] found that metapopulation persistence (assessed by the frequency of population excursions to low densities) increased with migration when rates of migration were very low, but above a relatively small rate of migration, metapopulation persistence declined with increasing migration rate.

Several additional effects should be briefly mentioned. First, the population growth rate $\lambda$ describes the long-term rate of change of a spatially distributed population once it has settled into its stable patch distribution (sensu Holt [3]). Immediately following the application of a pesticide, there will be a period of transient dynamics, such that the realized $\lambda$ varies over time, often quite considerably from the long-term value it will eventually reach. A population that has a long-term $\lambda > 1$ may, for a brief period, decline substantially, endangering its persistence (due, say, to effects of demographic stochasticity). Conversely, immediately after pesticide application, the realized $\lambda$ may exceed unity, even though it will eventually be less than one. Thus, an experimenter might measure no statistically significant decline in the population after pesticide application when the ultimate fate of the population is extinction.

Second, we have assumed that demographic rates are determined by the *habitat* (viz., with versus without pesticide). But the mechanistic basis of pesticide effects on populations stems from individual pesticide loadings: an individual that just moved into a habitat with pesticides is assumed to have different vital rates than one that has resided there for a while. A full analysis of this effect would require state-dependent models, so that individuals were categorized by pesticide loadings as well as locations.

Finally, pesticides themselves exhibit patterns of mobility, moving by physical transport processes away from their initial site of application. Furthermore, sedentary predator or consumer species that do not tend to move between patches may become exposed to pesticides via the mobility of their prey species, even if they reside in sites not exposed to pesticide application. All these effects—transient population dynamics, state-dependency, overlying pesticide and organismal spatial processes, and accounting for interspecific interactions—are ripe for further empirical and theoretical scrutiny.

*Effects of pesticides on colonization-extinction dynamics*

The models examined above assume that certain sites are persistently habitable by a species, whereas other sites are persistently uninhabitable due to the presence of a pesticide. Because regular movement couples subpopulations in different patches, the actual (versus potential) extinction of a subpopulation is inseparable from the extinction of the entire population. Because movement tends to expose individuals to suboptimal environments, increased movement rates indirectly jeopardize metapopulation persistence.

Yet, for some species, movement among patches clearly may be essential for regional persistence. Local extinction may arise to local causes (e.g., disturbances, succession, etc.), unsynchronized across space. The ability to colonize empty, suitable patches may permit the long-term persistence of a species in a landscape.

The standard model for analyzing metapopulation dynamics driven by coupled colonization and extinction stems from Levins [9] (see Holt [5] and Hanski [10] for reviews of recent literature). Rather than monitoring the number of individuals in each patch (as in above models), Levins chose to track the fraction of patches occupied in the landscape, denoted by $p$. The dynamics of the metapopulation are described by

$$\frac{dp}{dt} = cp(1 - p) - ep \tag{6}$$

where $e$ is the rate of extinction and $c(1 - p)$ is the rate of colonization of empty patches per occupied patch. The quantity $(1 - p)$ is the fraction of patches that is currently empty. At equilibrium, the fraction of patches occupied is $\hat{p} = 1 - e/c$. Metapopulation persistence requires that $c > e$.

Now, assume that the application of a pesticide makes a fraction $k$ of the patches permanently uninhabitable. This deterioration of the landscape quality can be modeled using Equation 6 by discounting the proportion of patches available for colonization by $k$, so

$$\frac{dp}{dt} = cp(1 - p - k) - ep \tag{7}$$

(see Nee and May [11]). The equilibrial occupancy rate is reduced to $p^* = 1 - e/c - k$ or $p^* = \hat{p} - k$. In this case, the metapopulation goes extinct when $k > \hat{p}$.

This leads to a simple rule-of-thumb for predicting when pesticide application is likely to endanger persistence of the entire population in the landscape. Given that a species persists because of a regional balance between local colonizations and extinctions, the fraction of the landscape currently occupied is the maximum fraction of the habitat that can be made unsuitable by the pesticide that will allow continued persistence of the species. Rare species (those with low intrinsic abilities to colonize new habitat patches and relatively high likelihood of going extinct in a patch) are particularly at risk. That risk is increased even if the pesticide is applied to patches that do not currently contain the species since this reduces the proportion of patches available for colonization. Although such a pattern of pesticide application may seem appropriate as a short-term tactic, it ignores the long-term consequence of re-

ducing the pool of suitable, empty patches available for colonization.

This message supports our contention that short-term assessment of pesticide impacts underestimates extinction risks, because they do not account for dynamical consequences of individual movements among habitat patches played out across many generations.

## DISCUSSION AND CONCLUSIONS

Our results have important implications for the assessment of pesticide effects on wildlife populations in spatially complex landscapes. We offer these comments more as a guide to the kinds of issues that should be addressed when examining pesticide effects rather than a set of rules that might determine what must be done in specific cases. Our point is that these issues are complicated by the presence of spatial heterogeneity in populations. Models that include the effects of spatial heterogeneity are more realistic than conventional population models. The implications of such models can be used to refine expectations of the likely effects of pesticides on wildlife populations.

There are many ways metapopulation structure may arise in a wildlife population. Most easily envisioned is when human activities break previously contiguous habitat up into fragments of different sizes, harboring populations within each fragment that are partially separated from one another. However, other kinds of spatial structure may characterize even populations in relatively continuous habitat. Suppose, for example, that a population consists of individuals occupying territories within a homogeneous landscape. Suppose that a certain fraction of those individuals have territories that are exposed to a pesticide, and they experience lethal and sublethal effects. Because exposed and nonexposed territories may be adjacent to one another, movement between them may be relatively unrestricted. Hence, across generations, there may be a high rate of migration from the exposed portion of the population to the unexposed portion. Such a spatial structure might lead to population decline even if, at any given time, a sizeable portion of the territories were not exposed to the pesticide. Conventional methods of detecting pesticide effects might find that a sizeable number of individuals in the population were not exposed to the pesticide and conclude that the pesticide had little or no effect. However, the metapopulation structure imposed on the population by spatial variation in pesticide exposure, coupled with individual motility, might compromise its ability to persist.

If the mechanisms of pesticide effects involve changes in vital rates within a population, then the first set of models we considered implies that at least three demographic processes can affect metapopulation persistence. First, recall that relatively free exchange of individuals among subpopulations decreases the ability of a metapopulation to persist. As argued in the previous paragraph, uniformity of habitat is not necessarily synonymous with lack of metapopulation structure. In evaluating pesticide effects on a population, it is extremely important to identify the spatial structure of the population. In particular, it is crucial to know something about dispersal of individuals in the landscape. This will provide two pieces of information usually missing from studies of pesticide effects. First, it will give some indication of the size of the area actually affected by the pesticide. Even though pesticide residues may not be found in some places, if there is sufficient exchange of individuals across boundaries within which pesticides are actually released into the environment, the actual area affected by those pesticides could be much larger than implied by the boundaries. Second, the rate of dispersal of individuals across the landscape will provide information on the degree to which exposed and unexposed portions of the population are independent. Ideally, the demographic effects of the pesticide should be isolated only to those populations actually exposed. It is critical to evaluate whether this is the case for any specific population.

Second, recall that metapopulation persistence in a landscape depends on the rate of decline in sink habitat patches. When subpopulations are exposed to a highly toxic pesticide, the chances of the metapopulation persisting are small, even if a relatively large portion of individuals is not exposed to the pesticide. Recall that the persistence of a metapopulation in our models did not depend directly on population size. Thus, if a pesticide is toxic enough, even if a small portion of the population is exposed to it, there may nevertheless be a significant decline in the population over time. Note that an extinction in such a population will not be instantaneous but will occur over relatively long periods of time. Initial toxicity coupled with information on dispersal may provide information on the long-term effects of a pesticide on a population, even if it is difficult to establish direct effects of the pesticide on the population.

Finally, the rate of increase in the proportion of the population not exposed to a pesticide is also very important in determining the persistence of the total population. Species that have slow population growth rates will more likely suffer the negative effects of a pesticide than one that has a relatively high growth rate, particularly if individuals in such a species are able to migrate successful among habitat patches. At a larger spatial scale, the region in a species' geographic range where it is exposed to pesticide stress may determine how it reacts to pesticide exposure. At the edge of its boundary, individuals of a species often find fewer appropriate resources and, in general, may have lower population growth rates [12,13]. Hence, near the edge of a species' geographic range, all else being equal, pesticide effects are likely to increase the likelihood of extinction of a metapopulation.

Because metapopulation persistence in some species may be more a function of dispersal dynamics than of variation in population vital rates, it may sometimes be incorrect to assume that because no detectable lethal or sublethal effects of a pesticide can be documented, then this will ensure population persistence in the face of pesticide application. This is particularly true if the effects of the pesticide are indirect and remove a portion of available habitat patches through loss of some important habitat component like a key food species. By doing so, the fraction of the landscape open to colonization may be reduced or made uninhabitable, and this in turn may increase the likelihood of extinction across the entire landscape.

We conclude that the dynamics of populations in complex landscapes have important consequences for assessment of the demographic consequences of pesticide exposure. In particular, evidence gathered to determine the effects of a pesticide on a population may be inappropriate if the population being considered has spatial structure. We suggest that future work done on pesticide effects collects information on the spatial structure of the population and on the dispersal capabilities of the species. Furthermore, the geographic context of the population should be included in the study. Long-term monitoring of tar-

get wildlife populations will be critical in detecting the demographic consequences of exposure to pesticides.

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