Importance of spatial habitat structure on establishment of host defenses against brood parasitism

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We used metapopulation dynamics to develop a mathematical simulation model for brood parasites and their hosts in order to investigate the validity of the "spatial habitat structure hypothesis," which states that a low level of parasite egg rejection in host populations is due to the immigration of acceptor individuals from nonparasitized populations. In our model, we varied dispersal rate and the relative carrying capacity of host individuals in parasitized and unparasitized patches. When both the relative carrying capacity in the parasite-free patch and the dispersal rate increase, the nonparasitized patch will provide more acceptor individuals to the parasite-prone patch. As the relative carrying capacity in the parasite-free patch increases, the equilibrium frequency of rejecters both in the parasite-prone and in the parasite-free patch decreases toward zero for intermediate levels of the dispersal rate. Although the rejecter strategy is more adaptive than the acceptor strategy in the parasite-prone patch, large numbers of acceptors are produced in the parasite-free patch dispersing to the parasitized patch. As the number of individuals in the parasite-free patch increases, parasitism rate can be maintained stable at a high equilibrium level in the parasite-prone patch.

Key words: birds, brood parasite, dispersal, host, metapopulation dynamics, model, rejection behavior, spatial habitat structure. [Behav Ecol 17:700–708 (2006)]

The metapopulation concept focuses on processes among subpopulations of conspecifics occupying separate patches or habitats (Hanski and Gilpin 1991, 1997; Hanski 1999). Therefore, metapopulation studies consider dynamics among subdivided populations as they exchange individuals with different genotypes that may be subject to local extinctions and recolonizations (Hanski and Simberloff 1997). Birds are suitable model organisms for use in a metapopulation approach where the aim is to understand the dynamics of local adaptations (Eser 2000). For instance, ponds and lakes may be regarded as different patches for birds nesting in their edges (Foppen et al. 2000).

Modeling the dynamics for the maintenance of polymorphism in heterogeneous environments (Levene 1953) and effects of restricted migrations (Maynard Smith 1966) has led to the construction of models highlighting the effects of dispersal among patches in a metapopulation complex (Maynard Smith 1966). For instance, many parasites and their hosts coevolve in complex geographic mosaics, provided local selection pressure varies and there is gene flow among populations (Gomulkiewicz et al. 2000). Similarly, coevolution between brood parasites and their hosts may reflect metapopulation processes (Martinez et al. 1999; Røskaft, Moksnes, Stokke, Moskát, and Honza 2002).

Avian brood parasites normally exploit the parental care of their hosts. Successful parasitism normally reduces the host reproductive success dramatically. Hosts of brood parasites are therefore expected to evolve antiparasite defenses. A common defense mechanism is rejection of the parasitic egg shortly after laying (Moksnes et al. 1990; Rothstein 1990; Davies 2000). However, despite prolonged exposure to the costs associated with parasitism, many species accept foreign eggs. Even in host species where all individuals are expected to reject, many individuals accept parasitic eggs (Rothstein 1982). Such acceptance has been explained by a lag in the evolution of rejection behavior because the rejecter gene has either not yet appeared or spread into the population (Rothstein 1982). An alternative explanation is, however, that the evolution has reached an equilibrium between acceptors and rejecters within a population (Rohwer and Spaw 1988; Lotem et al. 1992, 1995). A prerequisite for this hypothesis is that the cost of maintaining rejection behavior is too high for some individuals because they may occasionally reject their own eggs from unparasitized nests. Such rejection of their own eggs is referred to as recognition errors (Lotem et al. 1992; Røskaft, Moksnes, Meilvang, et al. 2002). These errors must not, however, be confused with rejection costs that are costs to their own eggs, for example, breakage, during the host’s ejection of foreign eggs (Røskaft et al. 1993; Røskaft and Moksnes 1998). Mathematical models, with the important prerequisite that recognition errors are costly for the host, have given theoretical support for the equilibrium hypothesis. However, because there is little evidence that recognition errors occur (Røskaft, Moksnes, Meilvang, et al. 2002; Stokke et al. 2002), such errors would probably be rare, and thus their influence on maintaining rejection behavior in many hosts could be questioned. This is supported by the fact that several species have apparently retained rejection traits even after long periods with absence of parasitism (Bolen et al. 2000; Rothstein 2001; Peer and Sealy 2004a; Peer et al. 2005).

The 2 above-described hypotheses, lag and equilibrium, ignore the spatial structure of habitats and dispersal among different habitats (but see Soler et al. 1998, 1999; Lindholm 1999; Lindholm and Thomas 2000). Røskaft, Moksnes, Stokke, Moskát, and Honza (2002) and Røskaft, Moksnes,
Stokke, Bicik, and Moskát (2002) argued that the occurrence of rejecters and acceptors within host populations could be explained by the spatial structure of host populations in a metapopulation system. Theoretical analyses have shown that the spatial structure of populations may strongly influence evolutionary processes (Via et al. 1995; Gandon et al. 1996; Grenfell and Harwood 1997; Schlichting and Pigliucci 1998; Gomulkiewicz et al. 2000). The spatial habitat structure (SHS) hypothesis is based on the assumption that most brood parasitizes, like the common cuckoo (Cuculus canorus), use trees as vantage points when searching for host nests. Thus, host populations that breed in areas with trees suffer more from parasitism than populations that breed in open areas without trees (Røskaft, Moksnes, Stokke, Moskát, and Honza 2002).

More generally, this means that when some populations of a host species are parasitized, independent of the cause of parasitism, while others are not, gene flow can occur between host populations exposed to different levels of parasitism. This has been shown to occur in the great spotted cuckoo (Clamator glandarius)–magpie (Pica pica) system (Martinez et al. 1999).

If host defense entails costs due to recognition errors, all individuals in the unparasitized populations should experience selection to accept all eggs in their nests, whereas in parasitized populations there is selection for rejecting parasitic eggs due to the costs of parasitism. In a patchy environment, individuals dispersing from unparasitized populations will carry acceptor genes into parasitized populations, whereas those emigrating from parasitized populations will carry rejecter genes to unparasitized populations. Immigration of acceptor genes will reduce the frequency of rejecters in parasitized populations to a level below that expected in closed populations and vice versa for unparasitized populations.

In theory, therefore, the frequency of parasitism within a population as well as the number of populations with low or no parasitism surrounding the target population should significantly influence the proportion of rejecter individuals in the parasitized population. Heavily parasitized populations will obviously have higher frequencies of rejecter individuals than unparasitized populations, given enough time and a sufficiently low rate of dispersal (Briskie et al. 1992; Brooke et al. 1998; Lindholm and Thomas 2000). Due to the importance of vantage points, their occurrence (mostly the frequency of trees) can be used to predict the parasitism rate as well as the rejection rate among common cuckoo hosts (Røskaft, Moksnes, Stokke, Moskát, and Honza 2002). However, because other physical characteristics (e.g., hole nesting, Rutila et al. 2002) may prevent brood parasites from laying in a host nest, host species with metapopulations varying in such characteristics may be regarded in a similar way as those breeding in habitats with different densities of trees or other vantage points.

More general analyses are required to predict host rejection patterns in different habitats or under different parasitism regimes. To investigate how rejection behavior will spread in a host metapopulation system, we used a mathematical model based on a 2-patch system and extended it to a more general case. Here, hosts in one patch are parasitized by a specialist parasite, whereas conspecifics in the other patch are not parasitized. We showed that the level of rejection in the parasitized population is dependent on both the relative carrying capacities in the patches with and without parasitism and the dispersal between the patches.

**THE MODEL**

All symbols used in the model are explained in Table 1. For simplicity, we consider 2 host subpopulations that occur in neighboring patches and where all individuals are breeding. According to the SHS hypothesis (Røskaft, Moksnes, Stokke, Moskát, and Honza 2002), patch I would be a habitat with trees or where brood parasites have access to host nests (type I habitat). Patch II would be a habitat without trees or where brood parasites have no access to host nests for other reasons (type II habitat). We first focus on local dynamics within patches and then on dispersal between patches.

Takasu et al. (1993) modeled the population dynamics and genetics in a closed system to explore how rejecter alleles, R, which enable host individuals to recognize and reject parasitic eggs, spread in the population (there is now some empirical evidence that host rejection behavior is genetically controlled [Martín-Gálvez et al. 2005]). We assume that local population dynamics and genetics in patches I and II within a breeding season are formulated as

<table>
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<th>Symbols used in the 2-patch model</th>
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\[ P' = x_P P + (1 - e^{-aP}) H_1 e^{-s_1 H_1}, \]
\[ H_I = \frac{k_1}{k_1 + K_I} \left[ s_{II} + W_{Rej}(1 - z_{II}) + W_{Acc} z_{II}^2 \right] H_I, \]  
(1b)

\[ x_I = \frac{s_{II} + x_{II} + s_{II}^2/(4) W_{Rej}}{s_{II} + W_{Rej}(1 - z_{II}) + W_{Acc} z_{II}^2}, \]  
(1c)

\[ y_I = \frac{s_{II} + x_{II} + s_{II}^2/(4) W_{Rej}}{s_{II} + W_{Rej}(1 - z_{II}) + W_{Acc} z_{II}^2}, \]  
(1d)

where \( z \) is the frequency of genotype AA (\( z = 1 - x - y \)). \( s_p \) and \( q_4 \) are survival rates of parasites and hosts, respectively. \( \Gamma \) is the survival rate of an accepted parasitic egg, and \( 1/k_1 \) is the density-dependent factor that limits host density in patch I (Takasu et al. 1993). Similarly, by setting \( P = 0 \) in Equation 1, host density and genotype frequencies at the end of the breeding season in patch II, where no parasitism occurs \( (P = 0) \), are derived as

\[ H_{II} = \frac{k_1}{k_1 + H_{II}} \left[ s_{II} + x_{II} + z_{II}^2/(4) \right], \]  
(2a)

\[ x_{II} = \frac{s_{II} + x_{II} + s_{II}^2/(4) W_{Rej}}{s_{II} + W_{Rej}(1 - z_{II}) + W_{Acc} z_{II}^2}, \]  
(2b)

\[ y_{II} = \frac{s_{II} + x_{II} + s_{II}^2/(4) W_{Rej}}{s_{II} + W_{Rej}(1 - z_{II}) + W_{Acc} z_{II}^2}, \]  
(2c)

using the same notation of parameters as for patch I.

We assume that hosts disperse between patches I and II with a given dispersal rate, \( \mu \), between breeding seasons. The dispersal rate is the proportion of individuals that disperse from one patch to the other \( (0 < \mu < 1) \) and is assumed to be independent of genotypes and patches. The population densities and frequencies of genotypes at the beginning of the next year, \( t + 1 \), after dispersal are then

\[ P_{t+1} = P \cdot \] 

\[ H_{I,t+1} = (1 - \mu) H_I + \mu H_{II}, \] 

\[ x_{I,t+1} = (1 - \mu) x_I + \mu x_{II} H_{II}, \] 

\[ y_{I,t+1} = (1 - \mu) y_I + \mu y_{II} H_{II}, \] 

\[ H_{II,t+1} = \mu H_I + (1 - \mu) H_{II}, \] 

\[ x_{II,t+1} = \mu x_I H_I + (1 - \mu) x_{II} H_{II} \] 

\[ \mu H_I + (1 - \mu) H_{II}, \] 

\[ y_{II,t+1} = \mu y_I H_I + (1 - \mu) y_{II} H_{II} \] 

\[ \mu H_I + (1 - \mu) H_{II}. \] 

These equations constitute the model used for subsequent analyses. Among these variables, \( P, H_I, H_{II}, x_I, y_I, x_{II}, \) and \( y_{II}, \) we are especially interested in the parasitism rate and the rejection rate because these are easily measured in the field and, thus, useful for testing the model.

The parasitism rate in patch I, \( P_{rate1} \), is measured as the product of the probability that a host nest is parasitized and the proportion of pairs for which both individuals are acceptors as follows:

\[ P_{rate1} = (1 - e^{-aP}) z_{II}^2. \]

This parasitism rate corresponds to the proportion of host nests that are found parasitized in the field, provided that host rejection does not occur before researchers find the nest.

The rejection rate, \( R_{rate} \), is measured as the frequency of rejecter pairs (at least one individual in a pair is rejecter) and is different in the 2 populations as follows:

\[ R_{rate} = 1 - z_{II}^2 \] and \[ R_{rate} = 1 - z_{II}^2. \]

This rejection rate can be measured in the field by adding artificial model eggs to the nest and observing the reaction of the nest owners.

Population densities and genotype frequencies are considered at the beginning of the breeding season. We assume that the dispersal of hosts between the 2 patches occurs before the start of breeding.

**ANALYSES AND RESULTS**

In the absence of parasites \( (P = 0) \), the equilibrium host population densities are

\[ H_I^* = k_I (f + q_1), \]  
and \[ H_{II}^* = k_I (f + q_1 - 1), \]

provided that the 2 patches are completely separated with no dispersal between them \( (\mu = 0) \). The parameters \( k_I \) and \( k_{II} \) are proportional to the equilibrium host population densities in the absence of parasitism, that is, carrying capacity of patches I and II. Hereafter \( k_I \) and \( k_{II} \) are referred to as the carrying capacity in patches I and II, respectively, because these parameters are related to the upper limit of the population density in a patch and specify the environmental quality of a patch (a patch of large carrying capacity can support a dense population). Without dispersal \( (\mu = 0) \), the local dynamics in patch I converge to 1 of the following 3 outcomes as the carrying capacity \( k_I \) is increased: 1) parasites go extinct, and all hosts are acceptors when \( k_I \) is below a threshold level; 2) parasites and hosts coexist, but all hosts are acceptors when \( k_I \) is intermediate between the first and second threshold; 3) parasites and hosts coexist, and the frequency of rejecters is between 0 and 1 when \( k_I \) exceeds the second threshold (Takasu et al. 1993; Takasu 1998a, 1998b).

Relative to acceptance, rejection is favored by selection in patch I when the parasite density \( P \) is greater than a threshold \( P_s \) at which \( W_{Rej} = W_{Acc} \):

\[ P > P_s = \frac{1}{a} \log \frac{1}{\xi}. \]

In patch II, however, where no parasitism occurs \( (P = 0 < P_s) \), the acceptance allele \( A \) will always be favored over the allele \( R \) due to the occurrence of recognition errors.

We are interested in how the frequency of rejecters changes in patch I when hosts disperse between the 2 patches. In terms of frequency of the rejecter allele \( R \) in patch \( i \) defined as \( r_i = x_i + y_i/2 \) \( (i = I \) or \( II \)), the change of \( r_i \) per year can be expressed as the sum of 2 components:

\[ \Delta r_i = r_{i,t+1} - r_{i,t} = \Delta r_{i,a} + \Delta r_{i,d}, \]

where \( \Delta r_{i,a} \) is the change in patch \( i \) caused by local dynamics (selection) and \( \Delta r_{i,d} \) is the change caused by dispersal (see
Appendix for explicit forms). An equilibrium state, $\Delta n_i = 0$, is possible when rejection is favored in patch I by heavy parasitism, $\Delta n_{i,s} > 0$. However, this equilibrium in I is diluted by immigration of acceptors from patch II, $\Delta n_{i,d} > 0$, whereas the reverse situation holds for patch II. By definition, these dynamics cannot occur in a closed system as modeled by Takasu et al. (1993). Although the model is simple, it becomes analytically intractable; therefore, the following analyses of how rejection behavior will spread in a host metapopulation system are based on numerical simulations. Breeding parameters are adopted from Takasu et al. (1993) and Takasu (1998a). Few data are available to quantitatively estimate the recognition error. Therefore, we assume that rejecter pairs have a reproductive output that is 5% less than acceptor pairs due to the occurrence of recognition errors ($\epsilon = 0.95$).

Case 1: both patches have equal carrying capacity ($k_I = k_{II}$)

Let us begin by comparing the 2 patches when their carrying capacities are equal, $k_I = k_{II}$. We start from an initial state where a small number of parasites invade one host population at a stable equilibrium with a small fraction of rejecter alleles [$P_0 = 0.01, H_{I,0} = k_I(f + s_H - 1)$, $x_i,0 = 0.01, y_i,0 = 0, z_i,0 = 0.99$, for $i = I$ or $II$]. We conducted extensive numerical analyses, and all demonstrated that the dynamics remain qualitatively similar for a wide range of parameter values as long as the closed dynamics modeled by Takasu et al. (1993) have biologically meaningful equilibria (nonnegative equilibria). We first show typical dynamics for dispersal rates ($\mu$) of 0%, 1%, 5%, and 10% over a time span reflecting 200 years (Figure 1). Dispersal rates greater than 10% produce quite similar dynamics corresponding to $\mu = 0.1$. Focusing on patch I, where parasitism occurs, the parasite density increased dramatically over a decade; thereafter, it declined to a low equilibrium level not far above zero (Figure 1A). This occurred because acceptors dominate the initial host population and, thus, parasites reproduce successfully. The parasitism rate, defined as the probability of a host being parasitized times the proportion of acceptor pairs, $P_{rate_I}$, followed a similar pattern as the parasite density $P$, ending at a very low equilibrium level close to zero (Figure 1B). Initially, host density in patch I, $H_I$, decreased due to the increase in parasite density. Thereafter, it fluctuated prior to reaching equilibrium below its original level (Figure 1C). The frequency of rejecter pairs $R_{rate_I}$ increased to an equilibrium level of near 60% (Figure 1E). A characteristic feature of the dynamics in patch I was that the rate of dispersal had only a minor influence on the equilibrium levels of parasite density, parasitism rate, host density, and frequency of rejecters.

Let us next focus on the dynamics in patch II where no parasitism occurs and, thus, rejection is not selected for. The host density $H_{II}$ declined from the original level but
stabilized shortly at equilibrium (Figure 1D). The frequency of rejecter pairs $R_{rateII}$ will increase to a certain level, provided that rejecters immigrated from patch I (Figure 1F). The equilibrium levels in patch II (host density and frequency of rejecter pairs) were quite sensitive to the rate of dispersal. The greater the dispersal rate, the closer the equilibrium levels in patch II will be to those in patch I. This is because the 2 populations are well mixed as the rate of dispersal is increased.

Case 2: the patches have unequal carrying capacities ($k_I \neq k_{II}$)

As the carrying capacity in patch II increased relative to that of patch I, quite different equilibrium levels were reached. This can occur when the frequency of allele R increases in patch I due to heavy parasitism. Thereafter, its frequency is reduced due to dilution by dispersal of acceptors from the parasite-free patch II. This leads to a lower frequency of rejecters in the parasitized population compared with that in a closed system without dispersal. Especially when the carrying capacity in patch II relative to patch I is large enough, it is possible that the increase of allele R driven by parasitism is canceled out by the immigration of acceptors, leading to a situation where the frequency of allele R continues to decrease to zero. In such cases, patch II acts as a source from which acceptors disperse to a sink patch I where the acceptors from the source population support the parasite population in the sink population.

We numerically calculated the equilibrium levels of the frequency of rejecter pairs in patches I and II, $R_{rateI}$ and $R_{rateII}$, and the parasitism rate in patch I, $P_{rateI}$, as a function of the carrying capacity in patch II. We made these calculations for various rates of dispersal (Figure 2) in order to investigate whether the relative carrying capacity in patch II influences the frequency of rejecters in patch I. In all cases, the equilibrium state was reached within a few hundred years.

Figure 2A depicts the dependence of the equilibrium frequency of rejecter pairs in patch I on the carrying capacity in patch II, $k_{II}$. As the relative carrying capacity in patch II increases, it provides more acceptors to patch I. When the relative carrying capacity in patch II becomes large, the dispersal from patch II contributes to lower the equilibrium frequency of rejecter pairs in patch I. The rate of dispersal is nonmonotonously related to the equilibrium frequency of rejecter pairs in patch II for $k_{II}$ large enough, which shows the highest decline at an intermediate level of dispersal rate. When dispersal is low (low $\mu$), patch I is effectively isolated (closed) so that $k_{II}$ will not affect the equilibrium frequency of rejecter pairs. By contrast, when dispersal is high, the 2 populations are well mixed, and rejecter pairs are less effectively diluted by dispersal from patch II even when $k_{II}$ greatly exceeds $k_I$.

Figure 2B shows the dependency of the equilibrium frequency of rejecter pairs in patch II. Except for an extremely low carrying capacity in patch II, the equilibrium frequency of rejecter pairs increases as dispersal from patch I increases. Because no parasitism occurs in patch II, the fitness of rejecters is always less than that of acceptors in II, but rejecters can persist in patch II due to dispersal from patch I.

At equilibrium, the parasitism rate increased nonmonotonously provided there was dispersal to patch I ($\mu > 0$) (Figure 2C). For $\mu = 0.05$, severe parasitism (nearly 100%) was maintained in I, provided that the carrying capacity in patch II was extremely high. Under these conditions, patch I formed a complete sink where few hosts could reproduce successfully, but dispersal of acceptors from patch II sustains parasites in I. The dependency of $P_{rateI}$ on $\mu$, however, is nonmonotonous (see below). Without dispersal ($\mu = 0$), the parasitism rate at equilibrium in patch I should be quite low, in the order of $1 - z$, where $z$ is the cost of host defense due to recognition error (Takasu et al. 1993) and this cost would be nearly negligible (Marchetti 1992; Roskaft et al. 1993). Dispersal between the 2 patches allowed parasitism to stabilize at a high level (Figure 2C).

Figure 3 shows how the rate of dispersal ($\mu$) affects the equilibrium frequencies of rejecter pairs, $R_{rateI}$, and parasitism rate, $P_{rateI}$. When the carrying capacity in the 2 patches was equal, the dispersal rate had an impact only on $R_{rateII}$ (Figure 3A). When the carrying capacity in patch II was very high, however, both patches were highly sensitive to variations...
in the dispersal rate (Figure 3B). Parasitism was maximized at an intermediate level of dispersal. In both cases, the rejecter frequencies in patches I and II became similar as $\mu$ increased because the 2 populations are well mixed.

**Case 3: a general case**

We have shown that in the simple 2-patch model the spread of rejecters is critically affected by the relative carrying capacity in the parasitism-free patch. When the carrying capacity in the nonparasitized patch is relatively high, the rejecters will not increase in frequency even in the parasitized patch. The 2-patch model can, however, easily be extended to consider more realistic cases with more patches. Let us assume a 2-dimensional array of $N \times N$ patches where the population dynamics and genetics of Equations 1a–d are applied on each patch where parasitism occurs. Equations 2a–c are applied on parasitism-free patches. Host individuals disperse between adjacent patches before breeding season with a dispersal rate $\mu$; hosts disperse to 4 patches surrounding a focal patch inside, but if a focal patch is located at the periphery, birds can disperse to 3 adjacent patches and to 2 patches if the focal patch is at a corner. The mathematical expression of this model is very complex. On the other hand, a 2-dimensional patch model seems to have biological relevance to the SHS hypothesis suggested empirically by Røskaft, Moksnes, Stokke, Moskát, and Honza (2002).

Figure 4 shows the population densities of parasites and hosts, the frequencies of rejecter pairs, and the parasitism rate at the equilibrium states when the model is run on a 4 x 4 patch system where the carrying capacities of the 16 patches are set equal. When only one patch is parasitized (Figure 4A),

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**Figure 3**
The equilibrium frequency of rejecter pairs $(1 - \frac{z}{2})$ in patch I and patch II, and parasitism rate $[(1 - e^{-\mu})z]^{2}$ as a function of the dispersal rate $\mu$ for carrying capacities $k_{I} = 100$, $k_{II} = 100$ (A) and $k_{I} = 100$, $k_{II} = 1000$ (B).

**Figure 4**
The parasite density $P$, the parasitism rate $(1 - e^{-\mu})z^{2}$, the host density $H_{P}$ and the frequency of rejecter pairs $1 - \frac{z}{2}$ at equilibrium in $4 \times 4$ patch arrays ($i, j = 1, 2, 3, 4$) where hosts disperse to adjacent patches with a dispersal rate $\mu = 0.05$. All the patches have the same carrying capacities ($k_{0} = 100$). The number of populations that are parasitized varies: only one local patch is parasitized (A), 2 patches are parasitized (B), 4 patches are parasitized (C), and all patches are parasitized (D). The other parameters used are the same as in Figure 1. The x and y axes refer to the positions within the patches.
the frequency of rejecters in the parasitized patch will be close to zero due to the immigration of acceptors from the 4 surrounding patches where no parasitism occurs. This low frequency of rejection is achieved even at relatively high parasitism rates. When the number of parasitized patches is increased to 2 (Figure 4B), the immigration of acceptors can no longer swamp the 2 parasitized patches and rejecters instead pervade into neighboring patches where no parasitism occurs and rejecters are selected against. This tendency is further amplified in the case where the central 4 patches are parasitized (Figure 4C). When all 16 patches are parasitized, the model is equivalent to the closed system analyzed by Takasu et al. (1993). Both the parasite density and the parasitism rate remain low at equilibrium (Figure 4D). The difference from the simple 2-patch model is that a disproportionate carrying capacity in parasitism-free patches is not necessary for acceptors to swamp rejecters who are always selected for in the parasitized patches. Sink populations are easily realized for the 2-dimensional spatial structure.

**DISCUSSION**

In a simple metapopulation system composed of 2 patches where hosts are parasitized (patch I) or not (patch II), the frequency of rejecter alleles can vary from 0% to 100%, dependent on dispersal between patches. This occurs because the frequency of rejecter alleles R in patch I is highly sensitive to the relative carrying capacities of each patch and the level of dispersal between them. The rejection rate can be as low as zero in the parasite-prone patch because of immigration of acceptors from the parasitism-free patch. Previous theoretical models for avian brood parasitism (May and Robinson 1985; Kelly 1987; Takasu et al. 1993) have focused only on closed host–parasite systems where all hosts suffered the same risk of parasitism and where homogeneous spatial structure was implicitly assumed. However, as we have demonstrated, the spatial organization of local patches (the SHS) can affect the development and/or maintenance of host defenses among populations. As Røskaft, Moksnes, Stokke, Moskát, and Honza (2002) suggested, the level of host defense might be largely affected by how local patches of different habitats are distributed. We suggest that researchers should pay more attention not only to local habitats but also to the broader scale of habitats where host individuals can disperse.

We have assumed that the allele R is dominant over A and that parasitism is accepted only when both male and female of a breeding pair are acceptors. Changing this assumption, making R recessive or allowing only one sex to be responsible for rejection, does not change the outcome of the model qualitatively. In the analyses, we have furthermore assumed that the rejection of a parasitic egg entails recognition errors, that is, the reproductive output of rejecter pairs, \( W_{\text{rec}} \), is always less than that of acceptor pairs, \( W_{\text{acc}} \), when \( P = 0 \). However, if there are no defense costs (no recognition error, \( e = 1 \), as suggested for several taxa (Bolen et al. 2000; Rothstein 2001; Peer and Sealy 2004b; Peer et al. 2005)), \( W_{\text{rec}} \) will always be greater than \( W_{\text{acc}} \) for \( P > 0 \) and the parasites will go extinct as the rejecters increase in frequency. The frequency of rejecters, however, will never reach 100% because the parasitic pressure that selects for rejecters will decline as the parasite decreases in density. When the parasite is extinct, the allele R will be neither advantageous nor disadvantageous, given no recognition errors, and it could be fixed or lost in the population due to genetic drift. Such an effect has been ignored in our deterministic model.

We have shown that the equilibrium frequency of rejecters in patch I declines as the carrying capacity in patch II increases relative to that of patch I. This result can be reasonably understood by realizing the balance of emigration and immigration of rejecters between patches I and II. Increasing the dispersal rate, \( \mu \), promotes emigration of rejecters from patch I to patch II. However, when \( \mu \) is large, rejecters will return to patch I from patch II before they are selected against in patch II where no parasitism occurs, resulting in the nonmonotonic relationship between the equilibrium state and the dispersal rate.

We investigated a simple 2-patch model and then extended it to a more realistic 2-dimensional array model to explore how habitat structure can affect the spread of a rejecter allele R. In the 2-dimensional array model, we have shown that even when the carrying capacities in local patches are equal, rejecters will not necessarily spread if the number of parasitized patches is low. This highlights the importance of the spatial configuration of the 2 types of patches.

It is quite likely that hosts face nonuniform risks of being parasitized depending on nesting site and habitat structure. Thus, SHS most likely affects the spread of rejection of parasitic eggs. Previous models, however, have ignored spatial structure. By contrast, our model suggests that the frequency of rejecters can be far lower than that of a closed system. A relatively high parasitism rate can be sustained depending on the carrying capacities of patches with and without parasitism. We suggest that the combination of prolonged low levels of rejection and a frequent parasitism found among several cuckoo hosts can be explained by the differences potentially associated with the habitat structure as shown above.

The SHS hypothesis might provide a better explanation for the maintenance of intermediate levels of rejection among European hosts (Sæther et al. 1995; Røskaft, Moksnes, Stokke, and Honza 2002), which the previous hypotheses ignoring spatial structure of habitats do not adequately explain. For instance, the dunnock (Prunella modularis) does accept nonmimetic cuckoo eggs (Davies 1992), and this absence of egg rejection in this species has been explained by a lag in the evolution of host defenses or by quite a slow spread of the rejecter allele R (Davies and Brooke 1989). However, as judged from ancient literature (see Davies 1992), the dunnock has been parasitized for at least 600 years in Great Britain, but there are few records of parasitism elsewhere (A Moksnes and E Røskaft, unpublished data). Thus, gene flow from unparasitized populations may have contributed to prevent a response in parasitized populations. The reed warbler (Acrocephalus scirpaceus) is another European species that shows a variability of response to cuckoo parasitism (Lindholm and Thomas 2000). Such a variation in rejection behavior has been explained by phenotypic plasticity and conditional responses. Our model emphasizes the need to consider gene flow and spatial structure of habitats as explanations. Undoubtedly, some reed warbler populations are parasitized, whereas others are not (Lindholm 1999; Lindholm and Thomas 2000; BG Stokke, E Røskaft, and A Moksnes, unpublished data), setting the stage for such an explanation.

We constructed our model to represent a specialist parasite within a host metapopulation structure, and therefore, it is not directly applicable to a generalist parasite system. However, the model is easy to extend to a system of host metapopulations utilized by a generalist parasite. This could, for instance, be the case for the hosts of the 3 host generalist Melodrus cowbirds: the bronzed cowbird (M. aeneus), the brown-headed cowbird (M. ater), and the shiny cowbird (M. bonariensis). They have a shorter evolutionary history as brood parasites than cuckoos (Rothstein et al. 2002) and are generalists that use many different host species (Friedmann 1929; Rothstein et al. 2002; for metapopulation dynamics of cowbird hosts, see Fauth et al. 2000; Lloyd et al. 2005). However, cowbirds have expanded their breeding ranges dramatically.
over the past 150 years. The brown-headed cowbird’s original range was restricted to the central plains where it associated with the bison (Bison bison) (Mayfield 1965), and in general, the most vulnerable hosts were therefore those breeding in the open habitats of the plains. Human activities, like clearing of land for agricultural production of livestock and grains, have led to a significant expansion of the cowbird’s breeding range (Mayfield 1965; see also Winfree 2004). Because brown-headed cowbirds also use vantage points such as trees when searching for host nests (Clofteter 1998), the SHS hypothesis may explain why some of the oldest cowbird hosts in prairie landscapes still accept parasitic eggs (Sealy 1999; Peer et al. 2000; Peer and Sealy 2004a). An extension of a closed system of generalist parasites as modeled by Takasu (1998b) could provide a major contribution to fully explore how SHS affects the establishment of host defense in generalist parasite–host interactions. Although taking into account all these factors will increase the dimensionality of model and make analysis difficult, further studies are needed to analyze this scenario in more detail.

Although we used a brood parasite–host system as a base for the analysis of our model, it could be useful for many general purposes. The present model can generally be used to predict the pattern of how genotypes will spread between populations, given that the potential abundance of patches, as well as the dispersal rate between them, is known. The model can also be applied in conservation biology in relation to small and patchy environments where dispersal occurs between patches.

**APPENDIX**

In the 2-patch model, the change in the allele frequencies of R in patches I and II reflect the local dynamics (selection) and dispersal. The allele frequencies were calculated as follows:

\[ \Delta n_{I,s} = \frac{n_{I,s}^2}{n_{I,s}^2 + 1} \frac{s f - e^{-ad}}{1 + \frac{e^{-ad}}{s f}} \]

\[ \Delta n_{I,d} = \frac{\mu H_{I}}{(1 - \mu)H_{I} + \mu H_{II}} (r_{II} - r_I) \]

\[ \Delta n_{II,s} = \frac{n_{II,s}^2 (s - 1)}{n_{II,s}^2 + 1} \frac{f}{(1 - \frac{1}{3} f_s)} \]

\[ \Delta n_{II,d} = \frac{\mu H_{II}}{\mu H_{I} + (1 - \mu)H_{II}} (r_I - r_{II}) \]

These equations show that the frequency of allele R increases in patch I when the parasite density is greater than the threshold (\( \Delta n_{I,s} > 0 \) when \( P > P_I \)). However, the frequency of R decreased due to dispersal from patch II where no parasitism occurs, and thus, the frequency of R there would remain low (\( \Delta n_{II,s} < 0 \)). At equilibrium, \( \Delta n_I = 0 \) and \( \Delta n_{II,s} = 0 \), the following conditions must hold: \( \Delta n_{II,s} > 0 \), \( \Delta n_{s,s} < 0 \) and \( \Delta n_{II,s} < 0 \), \( \Delta n_{II,s} > 0 \). Thus, with \( \mu > 0 \), the parasite density in patch I at equilibrium is larger than the threshold \( P_I \).

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