Diversity in Müllerian mimicry: The optimal predator sampling strategy explains both local and regional polymorphism in prey

Thomas G. Aubier\(^1,2,3\) and Thomas N. Sherratt\(^4\)

\(^1\)UMR 5175, Centre d’Ecologie Fonctionnelle et Evolutive, 1919 route de Mende, 34090 Montpellier, France
\(^2\)UMR 7205, Muséum National d’Histoire Naturelle, CP50, 45 rue Buffon, 75005 Paris, France
\(^3\)E-mail: thomas.aubier@normalesup.org
\(^4\)Department of Biology, Carleton University, Ottawa, Ontario K1S 5B6, Canada

Received May 19, 2015
Accepted September 24, 2015

The convergent evolution of warning signals in unpalatable species, known as Müllerian mimicry, has been observed in a wide variety of taxonomic groups. This form of mimicry is generally thought to have arisen as a consequence of local frequency-dependent selection imposed by sampling predators. However, despite clear evidence for local selection against rare warning signals, there appears an almost embarrassing amount of polymorphism in natural warning colors, both within and among populations. Because the model of predator cognition widely invoked to explain Müllerian mimicry (Müller’s “fixed \(n_k\)” model) is highly simplified and has not been empirically supported; here, we explore the dynamical consequences of the optimal strategy for sampling unfamiliar prey. This strategy, based on a classical exploration–exploitation trade-off, not only allows for a variable number of prey sampled, but also accounts for predator neophobia under some conditions. In contrast to Müller’s “fixed \(n_k\)” sampling rule, the optimal sampling strategy is capable of generating a variety of dynamical outcomes, including mimicry but also regional and local polymorphism. Moreover, the heterogeneity of predator behavior across space and time that a more nuanced foraging strategy allows, can even further facilitate the emergence of both local and regional polymorphism in prey warning color.

KEY WORDS: Apostatic selection, dynamic programming, exploration–exploitation model, neophobia, predator cognition, shifting balance.

The convergent evolution of warning signals among unpalatable species, known as Müllerian mimicry (Müller 1879), provides one of the most celebrated examples of the power of natural selection. In short, Müllerian mimicry between species is thought to arise as a consequence of selection to adopt a common phenotype, thereby reducing the cost of educating predators (Mallet and Joron 1999). Indeed, selection for monomorphism on warning color has been extensively shown using various empirical approaches, including direct observations of the behavior of avian predators (Chai 1986 1996; Langham 2004), population genetic studies in hybrid zones (Mallet et al. 1990), and mark-recapture experiments (Mallet and Barton 1989; Kapan 2001). However, despite this positive frequency dependence (strength in numbers), species involved in Müllerian mimicry complexes are frequently not monomorphic but instead polymorphic. Thus, many Müllerian mimics exhibit geographic races or spatial mosaics of phenotypes, including moths (Sbordoni et al. 1979), butterflies (Sheppard et al. 1985; Brower 1996), birds (Dumbacher and Fleischer 2001), frogs (Symula et al. 2001), bumblebees (Williams 2007), millipedes (Marek and Bond 2009), and velvet ants (Wilson et al. 2012). Even more paradoxically, different warning colors can be observed in the same locality in mimetic species, such as neotropical ithomiine butterflies (Beccaloni 1997), some heliconii butterflies like *Heliconius numata* (Brown and Benson 1974; Joron 1996), birds (Dumbacher and Fleischer 2001), frogs (Chouteau and Angers 2012).

Despite the fact that Müllerian mimicry has been studied for centuries, we still do not fully understand how this diversity of
warning colors is generated and maintained. At a regional scale, the formation of a spatial mosaic of various mimetic forms is thought to arise as a result of a combination of stochastic effects and localized frequency-dependent selection for Müllerian mimicry (Mallet and Singer 1987; Sherratt 2006; Mallet 2010; Choueau and Angers 2011). Nevertheless, simulations involving genetic drift and localized frequency dependence conducted by Sherratt (2006) only rarely led to the formation of a mosaic of morphs when there was only one phenotype at the initial state. He also showed that boundaries between patches of the mosaic are unstable leading to the collapse of the smallest patches. At a local scale, heterogeneity in predator microhabitat use could theoretically explain local polymorphism (Gompert et al. 2011). This process would function in a similar manner to the situation modeled by Sherratt (2006), except that the segregation is ecological instead of geographical. Spatially heterogeneous selection from different Müllerian models coupled with a suitable genetic architecture could also permit polymorphism (Joron et al. 1999). Other genetic factors such as dominance among alleles responsible for warning colors could favor the persistence of polymorphism (Llaurens et al. 2013).

Although a variety of models of predator cognition have been used to understand Müllerian mimicry (e.g., Speed 1993; Mallet 1999; Speed and Turner 1999), it is possible that the difficulty of explaining polymorphism is due to the simplified representation of predator sampling behavior that has been typically assumed. In particular, Müller (1879) proposed that a predator will sample a fixed number of unpalatable prey exhibiting a phenotype \( k \) before it finally learns to avoid them. This “fixed \( n_k \)” rule has been used in most of the models dealing with aposematic prey as it represents a simple and parsimonious assumption that generates selection for monomorphism. However, there are many ways to generate selection for monomorphism and the simple foraging rule assumed by Müller has not been supported empirically (Sherratt 2008). Indeed two features of predator cognition and behavior are necessarily overlooked when theoreticians implement the “fixed \( n_k \)” rule to study Müllerian mimicry. First, whenever investigated the number of prey attacked has always been positively correlated to the number of unpalatable prey presented to the predator (Greenwood et al. 1989; Lindström et al. 2001b; Beatty et al. 2004; Rowland et al. 2010a b). Second, predators can sometimes display neophobia—a short- or long-term (also known as “dietary conservatism” in Marples and Kelly 1999) tendency for predators to avoid attacking novel prey especially when rare (Shuttleworth 1972; Mappes and Alatalo 1997; Marples et al. 1998 2007; Greenberg 2003; Thomas et al. 2004; Franks and Oxford 2009). We can explain these two features simultaneously if we consider the sampling strategy for unfamiliar prey as an exploration—exploitation trade-off (Sherratt 2011). When a predator encounters a prey with an unfamiliar phenotype, it must decide whether to take a risk and attack it or avoid such prey altogether. Attacking a prey would permit it to gain a potential meal but also a valuable information about the level of palatability associated with the phenotype (exploration). At some point however, the predator may use the currently available information and reject the phenotype if it is largely unpalatable (exploitation). The optimal sampling strategy was quantitatively elucidated by Sherratt (2011) who used a simple dynamic programming algorithm (Jones 1978; Clark and Mangel 2000) coupled with a Bayesian learning model. If all prey with a given phenotype are unpalatable, then the number of individuals sampled by the predator before complete rejection will depend on the size of this prey population. For example, if the unfamiliar phenotype is common in the prey community, then there is more to lose if some of those prey turn out to be palatable, and the predator should consequently sample more of them. Conversely a predator should not be expected to sample individuals with a rare color pattern—that is, they should show neophobia—in some conditions (if the cost due to toxicity is high enough, for instance).

Intriguingly, the optimal sampling strategy is capable of generating completely different selection pressures on the prey community dependent on the underlying conditions—from familiar purifying selection against the rare morph to diversifying apostatic selection through neophobia (Clarke 1962). Neophobia has already been recognized as an important generator of polymorphism in conspicuous prey (Thomas et al. 2004; Franks and Oxford 2009). However, given that the two opposing selective forces can act in the same population dependent on conditions, it is hard to gain a clear understanding of when the behavior can promote Müllerian mimicry and/or polymorphism using verbal reasoning alone. Here, we quantitatively evaluate the impact of the optimal sampling model at the population level, asking whether it can promote both local and regional diversity by implementing it in a single-cell and spatial individual-based model. We show that optimal sampling rules can select for a wide range of evolutionary outcomes from Müllerian mimicry to color diversification. Moreover, given that the optimal sampling strategy is influenced by a range of factors including prey density and the costs of consuming unpalatable prey, we also characterize the role of heterogeneity in environmental conditions in shaping the evolutionary dynamics.

### Methods

#### OPTIMAL PREDATOR SAMPLING STRATEGY

As in Sherratt (2011), we use Bayesian learning and dynamic programming algorithms to determine the optimal predator sampling strategy for unfamiliar prey. This model allows us to evaluate the number \( n_k \) of unpalatable prey with an unfamiliar phenotype a predator should sample before complete rejection. Contrary to
Müller’s “fixed $n_k$” model this number depends, among other factors, on the size of the prey community sharing this particular phenotype.

**Bayesian learning**

If a predator encounters successively prey with the same unfamiliar phenotype, it has to estimate the likelihood ($P$) of individuals of this type being chemically defended or not. For $P = 1$, all the prey sharing the warning signal are defended (this is the situation modeled in this article) and for $P = 0$ they are all undefended. We assume that the predator would incur a cost $c$ on attack if the prey happens to be defended, but a benefit $b$ if the prey is undefended. For $P > \frac{b}{b+c}$ there is a net overall cost on attack and the predator should not attack prey with this given phenotype. However, the true value of $P$ is not known by the predator and (should it deem it worthwhile) it should sample to estimate $P$ (exploration), incurring costs and benefits in the process. If prey are predominantly defended then at some point it should reject such prey altogether on the basis of its estimation (exploitation).

As prey items are either defend or undefended, therefore the distribution of the number of defended prey from those sampled follows a binomial distribution. The conjugate distribution for a binomial is a Beta distribution, so specifying the prior probability distribution (belief of the predator before its next sampling event) as a Beta distribution allows us to retain the Beta distribution in the posterior (beliefs after sampling). Specifically, if we assume the prior for $P$ follows a Beta distribution with shape parameters $\alpha_p$ and $\beta_p$ (Beta($\alpha_p, \beta_p$)), then after sampling $n$ prey with $r$ individuals that happen to be defended (toxic), the posterior distribution will follow: Beta($\alpha_p + r, \beta_p + n$). As in any Beta distribution, the expectation of $P$ is therefore:

$$
\pi_p(r, n) = \frac{\alpha_p + r}{\alpha_p + \beta_p + n}.
$$

(1)

For simplicity, we assume a uniform prior probability distribution in which the predator initially believes all values of $P$ are equally likely by implementing $\alpha_p = \beta_p = 1$, generating an expectation in naïve predators that an unfamiliar prey item is defended of 0.5.

**Dynamic programming algorithm**

By using dynamic programming theory (Clark and Mangel 2000) we can determine what is the optimal decision of a predator—either deferring or attacking—at any trial with a given knowledge state based on its previous experience.

If the predator sampled $n$ prey of a given phenotype with $r$ individuals among them, which happen to be toxic ($r \leq n$) the knowledge state of the predator is described by the state variables $r$ and $n$. Using Bayesian learning theory, we calculate the predator expectation $\pi_p(r, n)$ that an individual prey will be chemically defended on the next attack (see above). To determine what is the optimal behavior of a predator, which encounters a prey with the same phenotype, we compare the expected long-term future pay-off $S_D(r, n)$ (if it defers) and $S_A(r, n)$ (if it attacks) for each possible behavior. If $S_A(r, n) > S_D(r, n)$, then attacking is the optimal behavior because it either increases the information concerning this prey type and/or provides an immediate reward. The maximal pay-off $S(r, n)$ at this trial correspond to the pay-off associated with the optimal behavior:

$$
S(r, n) = \max\{S_D(r, n), S_A(r, n)\}.
$$

(2)

We calculate $S_D(r, n)$ and $S_A(r, n)$ as follows:

$$
S_D(r, n) = 0,
$$

(3)

$$
S_A(r, n) = \pi_p(r, n) [S(r + 1, n + 1) - c]
+ (1 - \pi_p(r, n)) [S(r, n + 1) + b].
$$

(4)

As the calculation of the expected pay-off $S_A(r, n)$ depends on the expected pay-off at the next trial—terms $S(r + 1, n + 1)$ and $S(r, n + 1)$—we have to work backwards from the maximal number of trials $N$ (number of prey with this given unfamiliar phenotype encountered) by setting $S(r, N) = 0$ for all $0 \leq r \leq N$.

We only consider here communities of unpalatable prey ($P = 1$). As we assume that $N$ is known by the predator, it will sample a certain number $n_p$ of prey before deciding to decline all individuals with this particular phenotype. This assumption leads to the same features of sampling behavior than if $N$ was unknown by the predator (Sherratt 2011), and it decreases dramatically the runtime of the model. Indeed there is no stochasticity in the sampling algorithm, thanks to these assumptions and we can deduce directly this optimal number $n_p$ of prey sampled with a given phenotype before rejection from the dynamic programming algorithm without forward iteration. Contrary to Müller’s “fixed $n_k$” model, this number $n_p$ depends on the ratio $c/b$ (cost due to toxicity on benefit due to palatability; see also Mallet’s 1999 dose-dependent sampling argument) and on the number $N$ of individuals sharing the phenotype. For example, if the unfamiliar phenotype is common (high $N$) and if the cost due to toxicity is lower than the benefit provided by palatable prey (low $c/b$), there is more to lose if some of those prey turn out to be palatable. Mathematically, the second term in the equation (4) would be higher than the first term, even if $\pi_p(r, n)$ is close to 1, and the predator should consequently sample more of them ($S_A(r, n) > S_D(r, n)$ during more trials). Conversely, if the unfamiliar phenotype is rare (low $N$) and if the cost due to toxicity is high enough (high $c/b$), we get $S_A(0, 0) < S_D(0, 0)$ for $\pi_p(0, 0) = 0.5$ at the first trial, which means that the predator should not be expected to sample individuals with this rare color pattern—that is, it should show neophobia.
Having calculated $n_i$ under a range of conditions, we can evaluate its demographic consequences on prey populations using the individual-based model described below.

**POPULATION MODEL**

We construct here an individual-based model to understand how the optimal predator sampling strategy would affect the prey community composition over the long term. We considered both a single-cell model and a spatial model involving multiple cells. For convenience, we assume individuals are haploid and generations do not overlap.

**Single-cell model**

We consider two defended species (1 and 2). There is no interspecific competition and we assume they are characterized by the same carrying capacity $K_1 = K_2 = K$. These species are polymorphic and each individual $i$ exhibits a color represented by a discrete number $m_i$. There are $N_m$ distinct morphs possible such that $m_i \in \{1, 2, \ldots, N_m\}$. Therefore, individuals from different species can share the same morph (and thereby resemble one another) or look distinct. Both a reproduction and a predation event occur in random order at each generation.

During the reproduction phase, each individual is assumed to give birth to $g$ offspring and the entire population is reconstituted from offspring. Mutation affecting coloration can occur and each offspring can exhibit a different morph than its parent with probability $\epsilon$. We use the Beverton–Holt equation—derived from the discrete version of the logistic growth—to calculate the survival probability $v_j$ of one of these offspring belonging to species $j$:

$$v_j = \frac{1}{1 + (g - 1)N_j/K},$$

(5)

with $N_j$ the number of individuals of species $j$ and $K_j$ the carrying capacity of species $j$.

During the predation phase, predators are present in each cell at a given time step with probability $P_{pred}$. Each one of the $N_p$ predators present in the cell will sample $n_i$ individuals of each phenotype before rejection. This number $n_i$ can be chosen and constant ($n_1 = n_2$)—known as the Müller’s rule (Mallet 1999; Sherratt 2006). It can also be determined by the optimal predator strategy described above.

**Spatial model**

We consider here the same system with two defended polymorphic species. However they are distributed within a regular $G \times G$ lattice.

Processes—reproduction and predation—happen in a random order in each cell in a similar manner than in the single-cell model. We also implement here a migration phase at the end of each time step. Each individual can migrate with a probability $\mu$ to one of the eight surrounding cells (King’s move). The borders of the lattice are assumed to be reflective so edge cells have fewer neighboring cells. Cells can vary in two ways. First, their underlying quality can depend on abiotic and biotic factors that vary across space and time (Lenormand et al. 2009). To represent this variation across space, the carrying capacity of each species in any given cell is drawn from a normal distribution with mean $K$ and SD $\sigma_k$. Second, we assume that the availability of alternative prey varies across space, which will have implications for the energetic state and the risk tolerance of predators. If alternative prey are abundant, for example, then predators should be sated and therefore less likely to sample rare unfamiliar prey. To represent this variation across space, the value of $c/b$ in each cell is drawn from a normal distribution with mean $c/b$ and SD $\sigma_c$. Cells with low values of $c/b$ are therefore characterized by low energetic state (hungry) predators. For simplicity, variation in $K$ and $c/b$ across time is assumed to happen periodically every $T_{K,c}$ generations at which time we redraw all those parameters in their respective normal distributions.

To account for spatial autocorrelation, we conducted additional simulations in which carrying capacities were autocorrelated across landscapes. The autocorrelation was implemented using a multivariate normal distribution $X \sim \text{MVN}(0, \Sigma)$, where the covariance matrix $\Sigma$ incorporates the spatial association. A function $D$ representing the decay in correlation between pairs of points with distance is used to compute $\Sigma$. We chose the exponential form that models similarity in carrying capacity between sites as an exponential decay with distance. If $h_{ij}$ represents the Euclidean distance between points $i$ and $j$, then $D(h_{ij}) = e^{-\phi h_{ij}}$, where $\phi$ is the parameter describing how rapidly the correlation declines with distance (low $\phi$ generating strong autocorrelation).

**Simulations and statistics**

The model was implemented in C++. We initialized the model by considering cells started with each species assigned at their carrying capacity. Given that Müllerian mimicry readily arises (see below) our interest in diversity generation, all individuals from species 1 and 2 were set to be the same morph at the initial state in most simulations. When we carried out analysis on the spatial model, we chose the smallest grid size possible ($G = 20$) to decrease the runtime. The edge effect would have favored polymorphism in an important way with a grid smaller than $20 \times 20$, as we observed higher prey diversity when the borders are reflective than in the case when the opposite edges are assumed linked as a torus (Fig. S1). For $G \geq 20$, we can assume that the edge effect is negligible. Each simulation was run for 500,000 generations, allowing sufficient time for stable states to be reached (Fig. S2). The predator community was set to $N_p = 2$ predators per cell. Under the optimal sampling strategy, prey polymorphism in the spatial model is enhanced when the number of predators is lower.
or higher (Fig. S3). Therefore, we implemented \( N_p \) in a way that it makes the emergence of prey diversity difficult. Genetic mutation rate per locus per generation is estimated at about \( 10^{-5} \) in eukaryotes (Drake et al. 1998). However, in our model, the phenotypic mutation rates could be an order of magnitude higher because the genetic basis of coloration, often involving polymorphic “supergene” architecture, can involve multiple large-effect “switch” loci (Jiggins and McMillan 1997; Kronforst et al. 2012), which are developmentally dependent (Joron et al. 2006). We therefore implemented a phenotypic mutation rate of \( \epsilon = 10^{-3} \), but we have also undertaken a sensitivity analysis to explore the role of this parameter (Figs. S4 and S5).

We kept track of the density of each morph for each species in any given cell. The dominant morph of each species corresponds to the morph with the highest frequency in the cell. Under certain conditions, switches of the dominant morph occurred over time and we measured the frequency of those switches.

To compare the species’ phenotypic compositions in any given cell, we measure their similarity using the cosine similarity index \( I_S \):

\[
I_S = \frac{\langle \vec{x}, \vec{y} \rangle}{\|\vec{x}\|\|\vec{y}\|} = \frac{\sum_{i=1}^{n} x_i y_i}{\sqrt{\sum_{i=1}^{n} x_i^2} \sqrt{\sum_{i=1}^{n} y_i^2}},
\]

\( \vec{x} = (x_i) \) and \( \vec{y} = (y_i) \) correspond to the phenotypic compositions of species 1 and 2, respectively, with \( x_i \) and \( y_i \) the number of individuals sharing the phenotype \( i \) \((i \in [1, N_m])\) for each species in a given location. If the vectors \( \vec{x} = \vec{y} \), then \( I_S = 1 \). If \( \vec{x} \) and \( \vec{y} \) are completely different, then \( I_S = 0 \). However, a high value of \( I_S \) does not necessarily mean there has been high selection for mimicry or even matching mimicry rings between the two species, because if all morphs are equally represented by chance drift in both species, then such populations will still have high \( I_S \). To address this limitation, we compare the actual similarity index \( I_S \) to the mean similarity index \( I^* \). Between randomized vectors \( \vec{x} \) and \( \vec{y} \). These vectors are obtained by unsorting the vectors \( \vec{x} \) and \( \vec{y} \)—that is, values of \( x_i \) and \( y_i \) are allocated to random phenotypes. The normalized similarity index is calculated as follows: \( I_{S_k} = I_S - I^* \) \((I_{S_k} \in [-1, 1])\). If \( I_{S_k} > 0 \), mimicry is observed more often than by chance and we can conclude that there is selection for mimicry. If \( I_{S_k} = 0 \), there is no selection for mimicry. If \( I_{S_k} < 0 \), mimicry is observed less often than by chance and we can conclude that mimicry is counter-selected.

Phenotypic diversity within any species in the spatial model were analyzed using standard estimators of \( \alpha \) (within cell), \( \beta \) (between cell), and \( \gamma \) (all grid) diversity indexes (Whittaker and Whittaker 1972). To estimate \( \alpha \) or \( \gamma \) diversities, we used the classical Shannon–Wiener indexes \( H_\alpha \) and \( H_\gamma \) (Shannon 1948), which are based on the proportional abundances:

\[
H_{\alpha/\gamma} = -\sum_{i=1}^{n} p_i \ln p_i,
\]

with \( n \) the number of morphs observed, and \( p_i \) the proportional abundance within cell \( (H_\alpha) \) or in the entire grid \( (H_\gamma) \) of the \( i \)th morph. If there is only one morph at 100% of the population the \( p_i \) are either equal to 0 or 1 and then \( H_{\alpha/\gamma} = 0 \). Conversely we get \( H_{\alpha/\gamma} > 0 \) when there is no morph at 100% of the population \( (0 < p_i < 1) \). We estimated \( \beta \) diversity using Shannon’s formula:

\[
H_\beta = H_\gamma - H_\alpha.
\]

When the overall diversity is higher than the local diversity \( (H_\gamma > H_\alpha) \) it means that there is variation of the phenotype composition between cells \( (H_\beta > 0) \).

See Table 1 for a summary of the notations which includes the default values implemented in this study. We ran 20 replicates for each parameter combination tested.

## Results

### SELECTION GENERATED BY MÜLLER’S “fixed n_k” MODEL

Müller’s “fixed \( n_k \)” model leads to nonlinear frequency-dependent selection on color pattern (Fig. 1). Thus, a new color pattern in low frequency would be selected against compared to the wild-type color pattern. Indeed, implementing a constant \( n_k \) during the predation phase leads to constant monomorphism in the single-cell model. For all values of carrying capacity \( K \) or \( n_k \) implemented, the dynamics of prey phenotypic composition is characterized by the presence of a dominant morph—the initial morph—which does not change and which is always correlated with the dominant morph of the other species (Fig. 2A). Individuals exhibiting a rare morph are indeed strongly counter-selected even if \( n_k \) happens to be very low \((n_k = 1 \text{ and } N_p = 2 \text{ in Fig. 3A})\). As the phenotypic mutation rate is low \((\epsilon = 10^{-3} \text{ in all simulations})\) and population sizes are in the order of 100s, phenotypic mutation into the same new morph of more than one individual is a rare event in a given generation. The individual with a novel phenotype is therefore often eaten by the predators before it can reproduce. This observation readily explains why we did not observe the emergence of local \( (\alpha \text{ diversity}) \) or regional polymorphism \( (\beta \text{ diversity}) \) in the spatial model (Figs. 4A and 5A) even when we implemented spatial heterogeneity in the carrying capacity landscape \((\sigma_K > 0)\) or when we implemented stochasticity in predator presence/absence per cell per generation (even for \( P_{pred} = 0.5 \text{ in Fig. S6A})\). The strong selection for mimicry and monomorphism does not depend on the initial condition we chose. In particular, we obtained qualitatively identical results when starting simulations with species exhibiting distinct phenotypes, because mimicry rapidly evolves (Figs. S7A and S8A). After implementing such heterogeneous
Table 1. Notation and numerical values.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Definition</th>
<th>Default value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Müller’s “fixed n_k” theory</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>n_k</td>
<td>Number of toxic prey with the same phenotype k sampled before rejection</td>
<td>{1, 2,… 20}</td>
</tr>
<tr>
<td><strong>Optimal predator sampling strategy</strong></td>
<td>Ratio of cost to benefit of attacking chemically defended versus undefended prey</td>
<td>[1, 10]</td>
</tr>
<tr>
<td>c/b</td>
<td></td>
<td></td>
</tr>
<tr>
<td>(α_p, β_p)</td>
<td>Shape parameters of the β distribution followed by the prior belief</td>
<td>(1, 1)</td>
</tr>
<tr>
<td><strong>Population model</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ε</td>
<td>Phenotypic mutation rate affecting the color pattern per individual per generation</td>
<td>10⁻³</td>
</tr>
<tr>
<td>g</td>
<td>Number of offspring per individual</td>
<td>10</td>
</tr>
<tr>
<td>K</td>
<td>Mean carrying capacity for each species in any given cell</td>
<td>[50, 400]</td>
</tr>
<tr>
<td>μ</td>
<td>Migration rate</td>
<td>10⁻³</td>
</tr>
<tr>
<td>N_m</td>
<td>Number of distinct morphs possible</td>
<td>9</td>
</tr>
<tr>
<td>N_p</td>
<td>Number of predators in each cell</td>
<td>2</td>
</tr>
<tr>
<td><strong>In spatial model only:</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>G</td>
<td>Size of the grid G × G in the spatial model</td>
<td>20</td>
</tr>
<tr>
<td>P_pred</td>
<td>Probability that predators are present in each cell at any given time</td>
<td>1</td>
</tr>
<tr>
<td>T_{K,c}</td>
<td>Period of redraw of all K and c/b values across space</td>
<td>[10,000,∞]</td>
</tr>
<tr>
<td>σ_c</td>
<td>SD of c/b in any given cell</td>
<td>[0, 2]</td>
</tr>
<tr>
<td>σ_K</td>
<td>SD of the carrying capacity K for each species in any given cell</td>
<td>[0, 100]</td>
</tr>
</tbody>
</table>

Square brackets and curly brackets, respectively, refer to continuous and discrete ranges analyzed.

initial state, it is possible to get a spatial mosaic, but it is only composed of the two initial morphs and there is no emergence of further diversity (Fig. S9A). In sum, the use of this simplified model of predator cognition (“fixed n_k” rule) does not readily explain the emergence of phenotypic diversity observed in mimetic clades.

**SELECTION GENERATED BY THE OPTIMAL SAMPLING STRATEGY**

As with Müller’s “fixed n_k” model, the optimal sampling strategy can lead to nonlinear frequency-dependent selection on a color morph (with c/b = 1, Fig. 1). Predators following such optimal sampling strategy would strongly favor the commonest wild-type morph in prey. Interestingly, however, in certain conditions (with c/b = 2 for instance, Fig. 1), the optimal sampling strategy can also place positive selection pressure on a novel morph when it is very rare. This is due to neophobia (Sherratt 2011)—a predator is not expected to sample individuals with a rare color pattern if its energetic state is high because the overall expected reward is too low. Therefore, the optimal sampling strategy can generate either antiapostatic selection (positive frequency-dependent selection) or apostatic selection (negative frequency-dependent selection) depending on the combination of parameters.

**Single-cell model**

When we implemented the predator optimal sampling strategy in the single-cell model, we found that the initial morph can be replaced by another morph depending on the combination of parameters, namely the carrying capacity K and the ratio c/b used in the dynamic programming algorithm (Fig. 2B). Depending on the values of those parameters, four qualitative forms of prey phenotype dynamics can be discerned. When defended prey are not very costly to attack, the optimal sampling strategy generates positive frequency-dependent selection (c/b = 1, Fig. 1), which leads to purifying selection against rare morphs. The optimal sampling strategy can also be characterized by neophobia in other conditions (c/b = 2, prey rare, Fig. 1). Such predator behavior would favor prey with rare appearances, at least initially. Despite this advantage, these protected phenotypes display only limited growth in the population because they are strongly attacked if they become more represented in the population, which would favor the maintenance of the initial dominant morph. Therefore, if there is purifying selection or if the predators display neophobia only for very low frequencies, the initial dominant morphs of both species are stable as in the simulations with Müller’s “fixed n_k” (zone 1 in Figs. 2B and 3B).
Figure 1. Warning color selection on a new color pattern \( k \) among a wild-type pattern \( p \). Here, we assume the prey population has a constant size \( N = 400 \) and we consider different frequencies \( q_k \) of the phenotype \( k \) in the population. The fitness of \( k \) is \( W_k = 1 - n_k(q_k)/(q_kN) \), while that of \( p \) is \( W_p = 1 - n_p(1-q_k)/(1-q_k)N \). The number of individual sampled \( n_k(q_k) \) is constant in Müller’s “fixed \( n_k \)” model (with \( n_k = 1 \) and \( N_p = 2 \)) while it depends on the number of prey sharing the same color pattern in the optimal sampling strategy (with \( c/b = 1 \) and \( N_p = 2 \)). The measure of frequency-dependent selection acting on \( k \) relative to \( p \) used here is \( s_k = W_k/W_p - 1 \). If \( s_k \) is positive, the color pattern \( k \) is favored compared to \( p \). For \( q_k > 0.5 \), individuals sharing the pattern \( p \) are selected in a similar way than \( k \) in the present graph. Only with the optimal sampling strategy do we see active selection for rare morphs by virtue of the neophobia they generate under certain conditions (when \( c/b = 2 \) here).

But three other dynamics of prey phenotypic composition characterized by the emergence of phenotypic diversity exist when \( K \) is low enough (prey generally uncommon) or when \( c/b \) is high enough—either because of a high cost caused by toxicity (high \( c \)) or because of a high predator energetic state (low \( b \)). The dynamics of the phenotypic compositions of the two species can be characterized by mimetic species with dominant morphs which can switch stochastically (zone 2) by local polymorphism without mimicry (zone 3) and by phenotypic drifts of dominant morphs without mimicry (zone 4). In zone 2, the optimal sampling strategy is characterized by a neophobic response toward the rare morph. Even if individuals with rare phenotypes are frequently attacked if they become more represented in the population, a number of their offspring can occasionally survive by chance alone and be protected by positive frequency-dependent selection. Under these conditions, a previously rare morph can replace the dominant morph leading to a switch of the dominant phenotype. Here, the growth rate \( g \) has an effect on the frequency of switches with a medium value maximizing the frequency of switches—if \( g \) is too small, there is not enough offspring with a novel morph to attain the stage in which they are protected by the “strength in numbers” and if \( g \) is too high, the probability of switch is low as drift occurs easily in small populations (Fig. S10). In zone 3, predators display neophobia even if there is a large population of individuals sharing the same phenotype and there is a strong selection pressure for local polymorphism to avoid predation. In that case there is no more Müllerian mimicry between both species. Finally, in zone 4, when neophobia even happens for larger prey populations compared to their carrying capacity, predation is rare and a dynamics of phenotypic drifting occurs. These four prey composition dynamics also exist if we consider an initial state with two monomorphic species with distinct phenotypes (Figs. S7B and S8B). Lower phenotypic mutation rate \( \epsilon \) decreases the frequency of switches and favors the maintenance of monomorphism (Fig. S4). For instance, when \( \epsilon = 10^{-5} \), there is still generation of diversity through stochastic switches, however, species are not polymorphic in zone 3 because the phenotypic mutation rate is too low compared to the rate of frequency change due to drift to maintain local diversity. Interestingly, there is still no selection for mimicry as the predation rate is highly relaxed under these conditions. The sensitivity of the model to the carrying capacity and to the phenotypic mutation rate clearly highlights that the switches of the dominant morphs are stochastic and are driven by phenotypic drift. In sum, by implementing the predator optimal sampling strategy, we show here that prey phenotypic diversity can emerge depending on the growth rate \( g \), the carrying capacity of the prey population \( K \), and the ratio \( c/b \).

Spatial model

Despite the uniform initial morph distribution across the lattice, we found that implementing the predator optimal sampling strategy in the spatial model can favor the emergence of a spatial mosaic of morphs (Figs. 4 and 5) just as it facilitated polymorphism in the nonspatial model. Even if there is no variability of \( K \) and \( c/b \) across space (\( \sigma_K = 0, \sigma_c = 0 \), respectively), the intracellular (\( \alpha \)) and between-cell (\( \beta \)) diversities are positive with the optimal sampling strategy implemented, whereas they are equal to zero with Müller’s “fixed \( n_k \)” model. However, as noted above, the carrying capacity of the prey population \( K \) and the ratio \( c/b \) depends on biotic and abiotic factors that are unlikely to be constant across space and time. High variability of \( K \) and \( c/b \) across space (high \( \sigma_K \) and \( \sigma_c \), respectively) clearly favors the emergence of \( \alpha \) and \( \beta \) diversities, leading to an overall high diversity.
Figure 2. Effects of the two models of predator cognition on the composition of the prey community in the single-cell model. (A) Effect of the carrying capacity \( K \) and of the number of toxic prey sampled per phenotype \( n_k \) when Müller’s "fixed \( n_k \)" model is implemented. (B) Effect of the carrying capacity \( K \) and of the ratio of expected cost to expected benefit \( c/b \) when the optimal sampling strategy is implemented. The frequencies of the species’ dominant morph were recorded to measure the level of phenotypic polymorphism. A switch corresponds to a change of the dominant morph. To quantify selection for Müllerian mimicry, the similarity between species’ phenotypic compositions is compared to the similarity between randomized prey communities, that is, the relative frequencies of each phenotype are reassigned at random. A normalized similarity index approaching 1 means that mimicry is observed much often than by chance, whereas a normalized similarity index close to 0 means that there is no selection for mimicry. In the case with the optimal sampling strategy implemented, black lines—arbitrary drawn—delimit four zones in the parameter space, which are characterized by distinct prey composition dynamics. A logarithmic scale is used to display the number of switches per 10,000 generations. We assume that species are monomorphic and share the same phenotype at the initial state. Predation did not lead to prey extinction in any of these simulations. For each cognition model, 20 × 20 parameter combinations are tested. Parameter values: see Table 1.

(diversity \( \gamma \)). In some cells, the prey community would be under the optimal conditions to get switches of the dominant morph (zone 2 described in Fig. 2) or local polymorphism (zone 3), leading to the emergence of the spatial mosaic. Interestingly, variation of carrying capacity and/or cost/benefit ratio across time would also favor the emergence of diversity. High frequency of redraw of the carrying capacity and of the predator energetic state (low \( T_{K,c} \)) would permit each cell to be at some point in a state favoring switches or polymorphism. Even if some cells are generating prey diversity through heterogeneity of predator behavior, we find that there is still a high selection pressure for monomorphism (frequency of the dominant morph close to 1) and for mimicry between species (normalized similarity index > 0) throughout the lattice, which is consistent with the empirical data. However, if predators have strong neophobic responses in a given location, mimicry between the dominant morphs can be counter-selected (normalized similarity index < 0 when the frequency of the dominant morph is below 0.5 in Fig. S11), yet conditions leading to such extreme behavior are very rare with the parameters implemented. We also observe that there is only selection for mimicry between the dominant morphs because the normalized similarity index drops to 0 if we compare the phenotypic compositions without the dominant morphs (Fig. S11). In cells with the conditions leading to a predator neophobic behavior, phenotypes with low frequencies are not selected for mimicry. Similarly, the normalized similarity index is lower when \( \sigma_c \) increases (Fig. 5): here predators have stronger neophobic behavior in more cells, leading to a lower normalized similarity index due to the higher proportion of nonmimetic phenotypes with low frequencies. We have also shown that the effect of these variations across space on
DIVERSITY IN MÜLLERIAN MIMICRY

Figure 3. Examples of simulations illustrating the prey composition dynamics in the single-cell model when (A) Müller’s “fixed $n_k$” model (with $n_k = 1$) or (B) the optimal sampling strategy is implemented. In this last case, the four prey composition dynamics characterized in Figure 2B are represented. The graphs at the top represent the predation rate per capita on the prey community obtained from the both models of predator cognition, the dotted line corresponds to the carrying capacity per species, $K = 200$, and the dashed line to the combined carrying capacity, $2 \times K$. At the bottom, we plot the time series of the density of each morph (lines with different colors) for both species. We assume that species are monomorphic and share the same phenotype (red) at the initial state. To illustrate the prey composition dynamics for each zone of the parameter space in Figure 2B, we chose $c/b = 1, 4, 6, 9$ from left to right (when the optimal sampling strategy is implemented). Parameter values: see Table 1.

Polymorphism is even stronger when the landscape is autocorrelated—that is, cells close to each other are more likely to share the same properties (Fig. S12). Increasing the number of predators, $N_p$, favors the emergence of regional and local polymorphism when the optimal sampling strategy is implemented (Fig. S3). Under these conditions, positive selection for rare morphs is enhanced as the common morph is more predated, whereas rare morph still do not suffer from predation. As the number of individuals with the rare morph increases, so does the probability of switching. Also, in contrast to the simulations with Müller’s “fixed $n_k$” model, implementing stochasticity in predator presence/absence per cell per generation enhances the generation of diversity (Fig. S6B).

Our sensitivity analysis also shows that the generation of polymorphism depends on the values of the migration rate $\mu$ and of the phenotypic mutation rate $\epsilon$. Thus, the generation of polymorphism in the spatial model is possible for lower phenotypic mutation rates if the migration rate is lower (Fig. S5). The migrants can be seen as “biased-mutants” that carry the dominant morph of the surrounding cell. Therefore, if the migration rate is too high compared to the phenotypic mutation rate, the generation of the mosaic is not possible as the migrants would lead to switches to the initial morph. The phenotypic mutation rate must be in the same order of magnitude than the migration rate or higher, to observe the emergence of polymorphism. That is why, there was no generation of mosaics when we implemented a phenotypic mutation rate equal to $10^{-5}$, for all values of the migration rate explored ($\mu \geq 10^{-4}$). As the definition of a cell is arbitrary here, we can expect that migration rates would be lower if each cell corresponds to larger geographic ranges (because the perimeter/area ratio declines as the range increases). In other words, if the phenotypic mutation rate is low, there would be generation of polymorphism, but the patches of the resulting mosaic would be larger.
Examples of prey community composition in the spatial model has been used to help explain the evolution of diversity indexes obtained are like the dominant phenotype if there is local monomorphism (these maps correspond to the superposition of the ones represented in Fig. S13). The morph composition is shown for both species. At the initial state all individuals were set to be the red morph. The Shannon's diversity indexes obtained are $H_a = 0.52$, $H_\bar{a} = 1.64$, and $H_p = 2.15$ for the simulation with the optimal sampling strategy implemented. They are all equal to 0 for the simulation with Müller’s “fixed $n_k$” model. Parameter values: see Table 1.

**Discussion**

**PREDATOR OPTIMAL SAMPLING STRATEGY CAN SELECT FOR POLYMORPHISM**

Diversity in Müllerian mimicry has long been seen as a paradox (Joron and Mallet 1998; Borer et al. 2010) because localized homogenizing selection for uniformity in warning colors has been extensively demonstrated (Chai 1986 1996; Mallet and Barton 1989; Mallet et al. 1990; Kapan 2001; Langham 2004; Borer et al. 2010; Chouteau and Angers 2011) Indeed, Mallet (2010, p. 100) remarked that “we see an almost embarrassing amount of polymorphism in natural warning colour.” Many Müllerian mimics show spatial variation in form (Sbordoni et al. 1979; Sheppard et al. 1985; Brower 1996; Dumbacher and Fleischer 2001; Symula et al. 2001; Williams 2007; Marek and Bond 2009; Wilson et al. 2012) and different warning colors can even be observed in the same locality in some mimetic species (Brown and Benson 1974; Beccaloni 1997; Joron and Mallet 1998; Joron et al. 1999; Borer et al. 2010). On a theoretical side, Müller’s “fixed $n_k$” model has been used to help explain the evolution of Müllerian mimicry for decades (Müller 1879) as it represents a simple and parsimonious assumption that generates selection for uniformity. However, the predator behavior originally assumed by Müller—a fixed number of prey is sampled whatever the density of the prey—has no empirical support (Sherratt 2008). Selection for uniformity does not necessarily mean that $n_k$ is always fixed—indeed, animal cognition would rarely be expected to be so simple and it is demonstrably nonoptimal. Moreover, as we have shown, Müller’s model does not readily explain the generation of phenotypic diversity either within a location or between locations.

As discussed in Sherratt (2011), the predator optimal sampling strategy obtained from the dynamic programming algorithm can generate important features of predator cognition such as neophobia—a tendency for predators to avoid attacking novel prey (Shettleworth 1972; Greenberg 2003; Marples et al. 2007)—and a correlation between the number of unfamiliar prey available and the number attacked before sampling ceases (Greenwood et al. 1989; Lindström et al. 2001b; Beatty et al. 2004; Rowland et al. 2010a b). Given its flexibility, the optimal sampling strategy can lead to various selection regimes on the prey community from purifying selection to apostatic selection (Allen and Clarke 1968; Greenwood 1984; Allen 1988). As might be expected, the increase of $c/b$—ratio of cost to benefit of attacking chemically defended versus undefended prey—leads not only to a decreased predation rate, but also to a neophobic response. Depending on the values of the prey carrying capacity and of $c/b$, the predator sampling strategy can lead to a variety of dynamics of the prey phenotypic composition across species from monomorphic mimicry to phenotypic drift. Thus, the use of a more realistic model of predator cognition compared to Müller “fixed $n_k$” model can readily explain the emergence of color diversity in the unpalatable prey community. The tendency for predators to avoid attacking rare prey in some conditions will generate apostatic selection (Hubbard et al. 1982) and can be an important driving force behind polymorphism, not just of unpalatable prey (Thomas et al. 2003; Marples et al. 2005; Sherratt 2011), but also of palatable prey (Franks and Oxford 2009). However, despite generating local polymorphism under some conditions, the nondominant polymorphic forms in the two species do not evolve to match one another in frequency.
Figure 5. Effects of the two models of predator cognition on the composition of the prey community after 500,000 generations in the spatial model with (A) Müller’s “fixed $n_k$” model or (B) the optimal sampling strategy ($c/b = 3$) implemented. When Müller’s “fixed $n_k$” is implemented, we show the effects of the fixed number of toxic prey sampled per phenotype ($n_k$) and of the variation across space of the carrying capacity ($\sigma_K$) on the prey phenotype diversity. When the optimal sampling strategy is implemented, we consider the variation across space of the ratio $c/b$ ($\sigma_c$), the variation across space of the carrying capacity $K$ ($\sigma_K$), and the period of redraws of all $K$ and $c/b$ values ($T_{K,c}$). We assume the carrying capacity in any given cell is drawn in a normal distribution with mean $K = 200$. Shannon’s indexes $H_\alpha$ and $H_\beta$ are used to estimate $\alpha$ (within-cell) and $\beta$ (between-cell) diversities, respectively. The frequencies of the species’ dominant morph were recorded to measure the level of phenotypic polymorphism. To quantify selection for Müllerian mimicry, the similarity between species’ phenotypic compositions is compared to the similarity between randomized prey communities—that is, the relative frequencies of each phenotype are reassigned at random. Data distribution is represented by box-and-whiskers plot. The central line, the box upper/lower limits, and the line upper/lower limits correspond to the median, the upper/lower quartile, and the maximum/minimum, respectively. Parameter values: see Table 1.
so the model does not explain the maintenance of various mimicry rings that can coexist in nature (Beccaloni 1997; Joron and Mallet 1998; Chouette and Angers 2012). This is perhaps not surprising, given that the apostatic selection driven by neophobia promotes rare phenotypes overall, independent of species.

The generation of polymorphism depends greatly on the migration rate and the phenotypic mutation rate. It suggests that the ecology and the genetic basis of coloration of prey could explain certain features of the spatial mosaic of phenotypes, such as the size of the patches, or the speed of the boundaries movement (not studied here, but see Sherratt 2006). Our model would predict that organisms that are characterized by low dispersal abilities would be more likely to be distributed into a mosaic with a high patchiness. Such correlations could be tested using data on multiple organisms exhibiting Müllerian mimicry. In a similar way, organisms with high phenotypic mutation rates should also show high level of polymorphism—such as H. numata due to its “supergene” architecture (Joron et al. 2011) for instance.

NEOPHOBIA AS A FACTOR IN THE “SHIFTING BALANCE” THEORY

Wright’s “shifting balance” theory (Wright 1932) has been suggested to explain geographic variation in Müllerian mimicry complexes (Mallet 1986 2010). In this theory, random variation of allele frequencies by genetic drift of some subpopulations would be a decisive factor in finding adaptive opportunities—that is, in moving across an adaptive valley to the base of a higher adaptive peak in the fitness landscape. In the case of the evolution of diversity in warning color, the processes at the origin of the drift in color pattern are not completely identified. For instance, an invasion phase—which may not be a common scenario—can favor the emergence of diversity because a prey with a novel phenotype can colonize by chance a new locality (Sherratt 2006). Also, Chouette and Angers (2012) showed empirically that predation was relaxed in localities characterized by local polymorphism of poison-dart frogs. As even palatable prey were under-predated, they assumed a lower density of predators. They concluded that relaxed predation could initiate the emergence and the colonization of new morphs and claimed that their study was the first empirical demonstration of Wright’s “shifting balance” theory in a natural system. Nevertheless, our simulations indicate that the absence of predators clearly favors the phase of stochastic drift in Wright’s “shifting balance” theory, the emergence of diversity in warning color does not appear that paradoxical anymore.

As noted above, neophobic responses have been demonstrated empirically in many laboratory experiments. However, predators are likely to respond differently in the wild. Therefore, to understand the true role of neophobia in natural systems, the frequency of neophobic responses should be investigated further in the field. Coppinger (1969 1970) has already found that wild-caught avian predators often avoid novel insect prey. Götmank (1994 1996) also recorded a lower predation rate on European blackbirds whose wings had been painted with brightly colored patches. Marples et al. (1998) likewise showed that some individual wild birds may reject novel prey consistently on the basis of unfamiliarity alone. Observing neophobia in the field is challenging because the experimenter needs to present only few individuals, leading to a lower statistical power. Indeed testing a high number of replicates with the same phenotype in the same locality can modify the predator behavior and can prevent neophobia from being recorded. Testing few replicates in many localities may permit to measure some neophobic behaviors (if any) in the field.

POLYMORPHISM CAN EASILY EMERGE THROUGH INTRINSIC HETEROGENEITY ACROSS SPACE AND TIME

The prey environment is known to fluctuate in space and time (Lenormand et al. 2009) especially over broad scales. Such fluctuations would affect the prey density and the energetic state of predators, both of which alter the optimal sampling strategy of predators. Indeed if there are few unfamiliar prey, the possible benefit of sampling them would be low even if they all happen to be palatable, so that predators would attack few if any of them. The availability of alternative prey has been shown to affect the mortality rates of Batesian mimics (Hetz and Slobodchikoff 1988). In the same way, the number of defended prey sampled depends on the number of alternative undefended prey presented (Nonacs 1985; Lindström et al. 2001a 2004) and the attack rate on defended prey increases if alternative prey are concealed (Carle and Rowe 2014). In contrast to Müller’s “fixed n_k” rule, a predator would be expected to behave differently depending on its energetic state by strategically trading off the costs and the benefits of consuming unfamiliar prey to make decision (Sherratt 2003; Sherratt et al. 2004; Barnett et al. 2007). Müller’s “fixed n_k” rule is inherently a rigid rule, so the only parameter that we can possibly vary in space is n_k itself (which might depend, e.g., on
the toxicity of the phenotype in question). The lack of a strategic flexibility in the sampling rule renders it largely insensitive to environmental variation. Indeed, as we have shown, even variation in \( n_k \) among cells itself would not promote diversity.

It has been shown empirically that some predators species are bolder than others in attacking chemically defended species (Exnerová et al. 2003; Nokelainen et al. 2014). Therefore, spatial heterogeneity in predator community composition could also generate a geographical mosaic of selection and could favor the emergence of polymorphism, in a manner entirely analogous to the polymorphism generated when \( c/b \) vary. Whatever the source of heterogeneity that drives variation in \( c/b \), given the plasticity in predator behavior that the optimal sampling model predicts, and its role in the generation of polymorphism, further empirical investigations of such link between the abundance of prey—both unpalatable and alternative palatable prey— and the nature of the predator sampling strategy should now be carried out in the field.

Conclusion and Perspectives

Our study highlights the role of predator cognition—especially neophobia—in shaping diversity in Mullerian mimicry. Through a more realistic predator behavior model than the “fixed \( n_k \)” rule, we are able to explain the seemingly paradoxical generation of diversity in Mullerian mimicry observed in nature. To our knowledge, this study is the first theoretical work explicitly demonstrating the emergence of warning color diversity with a uniform initial state.

As we suggested, even if the different sampling strategies generated by the optimal sampling strategy theory have already strong empirical support in laboratory, this theory should now be tested in nature. In particular, we need to begin to quantify the spatial heterogeneity of predator behavior in the field to elucidate the selection pressures that shape the mimetic prey communities over broad ranges. In addition, the frequency of neophobic behavior in nature should be investigated, because it can drive the emergence of diversity through stochastic switches. On a theoretical side, the effect of Batesian mimics on the spatial mosaic generated has not been studied. It could increase the frequency of phenotypic switches of the Mullerian mimics and it could favor the formation of the mosaic even with low phenotypic mutation rates. Moreover, even if the optimal strategy can generate local polymorphism through neophobia, it does not explain the existence of the coexisting mimicry rings we observe in nature. More work should be done to understand how they can be generated and maintained.

Naturally, polymorphisms can be generated by a variety of different mechanisms, including hybridization and mimicry of different sympatric models. However, our work suggests that heterogeneity of predator sampling behavior can play an important and so far unrecognized role in diversity generation in Mullerian mimicry complexes. Ironically, the same sampling behavior that leads to Mullerian mimicry, recognized by Müller over a century ago, can also help explain the generation and maintenance of polymorphisms through neophobia.

ACKNOWLEDGMENTS

We thank D. W. Kikuchi for comments on the manuscript. We are very grateful to M. Servedio, D. Roze, and two anonymous reviewers for comments and suggestions that improved our manuscript. This work was supported by a bursary from the French Ministry of Higher Education and Research to TGA. We are also grateful to NSERC (TNS) and FCRF (TGA) for funding.

LITERATURE CITED


Carle, T., and C. Rowe. 2014. Avian predators change their foraging strategy on defended prey when undefended prey are hard to find. Anim. Behav. 93:97–103.


Supporting Information
Additional Supporting Information may be found in the online version of this article at the publisher’s website:

Figure S1. Parameter analysis of the spatial model with the optimal sampling strategy implemented. Effects of the grid size.
Figure S2. Time series with the spatial model and the optimal sampling strategy implemented for different values of σ_c.
Figure S3. Parameter analysis of the spatial model with the optimal sampling strategy implemented. Effects of the number of predators.
Figure S4. Effects of the two models of predator cognition on the composition of the prey community in the single-cell model. Effects of the phenotypic mutation rate.
Figure S5. Parameter analysis of the spatial model with the optimal sampling strategy implemented. Combined effects of the phenotypic mutation rate and the migration rate.
Figure S6. Parameter analysis of the spatial model with the optimal sampling strategy implemented. Effects of the probability of predators presence per cell per generation.
Figure S7. Effects of the two models of predator cognition on the composition of the prey community in the single-cell model when species are monomorphic and exhibit distinct phenotypes at the initial state.
Figure S8. Examples of simulations illustrating the prey composition dynamics in the single-cell model when species are monomorphic and exhibit distinct phenotypes at the initial state.
Figure S9. Examples of prey community composition in the spatial model when species are monomorphic and exhibit distinct phenotypes at the initial state.
Figure S10. Effect of the growth rate on the composition of the prey community in the single-cell model when the optimal sampling strategy is implemented.
Figure S11. Complementary analysis in the spatial model. Normalized similarity index I_s values within cell in comparison with the frequency of the dominant morph on the left graph.
Figure S12. Effect of the autocorrelation parameter on the composition of the prey community in the spatial model with the optimal sampling strategy implemented.
Figure S13. Other representation of prey community composition in the spatial model.