Predator Mixes and the Conspicuousness of Aposematic Signals

John A. Endler[1,*] **and Johanna Mappes**[2,†]

1. Department of Ecology, Evolution, and Marine Biology, University of California, Santa Barbara, California 93106-9610;
2. Department of Biological and Environmental Sciences, University of Jyväskylä, P.O. Box 35, FIN-40014 Jyväskylä, Finland

Submitted May 14, 2003; Accepted October 21, 2003; Electronically published April 19, 2004

Online enhancements: appendix, figures.

ABSTRACT: Conspicuous warning signals of unprofitable prey are a defense against visually hunting predators. They work because predators learn to associate unprofitability with bright coloration and because strong signals are detectable and memorable. However, many species that can be considered defended are not very conspicuous; they have weak warning signals. This phenomenon has previously been ignored in models and experiments. In addition, there is significant within- and among-species variation among predators in their search behavior, in their visual, cognitive, and learning abilities, and in their resistance to defenses. In this article we explore the effects of variable predators on models that combine positive frequency-dependent, frequency-independent, and negative frequency-dependent predation and show that weak signaling of aposematic species can evolve if predators vary in their tendency to attack defended prey.

Keywords: frequency-dependent selection, apostatic selection, aposematism, predation, crypsis.

Many species have evolved defenses against predators (reviews in Cott 1940; Edmunds 1974; Endler 1991*a*). These species often use signals that encourage predators to learn to avoid attacking them; such signals are called aposematic (review in Guilford 1990). In this article we will be concerned with visually aposematic signals, which tend to operate before, or from a longer distance than, olfactory or other signals (Endler 1986; Marples et al. 1994; Sword et al. 2000; Exnerová et al. 2003). Terrestrial aposematic species commonly use red, yellow, and orange, often combined with black, to advertise their unprofitability to visually hunting predators. These colors contrast strongly against green and brown visual backgrounds and make the prey easy to recognize and discriminate from palatable prey (Cott 1940; Sherratt and Beatty 2003). Such strong (conspicuous) signals favor rapid discrimination from edible cryptic prey (Gittleman and Harvey 1980; Sillén-Tullberg 1985; Roper and Wistow 1986; Roper 1990). Stronger signals facilitate more rapid avoidance learning compared to weaker (less visible) signals (Roper and Redston 1987; Alatalo and Mappes 1996; Mappes and Alatalo 1997; Lindström et al. 1999, 2001*a*) and can make the difference between avoidance learning and no avoidance learning. (Schuler and Hesse 1985; Sword et al. 2000). Strong signals are also associated with longer memory retention time (Roper and Redston 1987; Speed 2000) and fewer recognition errors (Guilford 1986). As for other high-efficiency signals, aposematic colors are expected to work best when they have high contrast within the animal and between the animal and its visual background (Endler 1988, 1991*a*, 1991*b*, 1992; Endler and Théry 1996).

In spite of the advantages of highly conspicuous aposematic coloration, there are many unpalatable well-defended or otherwise unprofitable species that are weakly conspicuous, and many can be considered nearly cryptic. For example, larvae of *Dryas julia* butterflies contain pyrazines (Moore et al. 1990), cyanogenic glucosides, linamarin, and lotaustralin (Nahrstedt and Davis 1985), compounds that are known to inhibit predation (e.g., Rothschild et al. 1984; Schappert and Shore 1999). However, although spiny, these larvae are not as conspicuous as "normal" aposematic animals such as *Danaus plexippus* (monarch) larvae—they are dark brown with small white disruptive marks laterally. Spines may actually be a defense against parasitoids that may be immune to the toxins (Dyer 1995). Other protected species verge on being cryptic. Pine sawfly larvae (e.g., *Neodiprion sertifer* and *Diprion pini*) reduce bird and ant predation with active defensive behavior and distasteful chemical compounds (diterpe-

* Corresponding author; e-mail: endler@lifesci.ucsb.edu.

† E-mail: mappes@bytl.jyu.fi.

Am. Nat. 2004. Vol. 163, pp. 532–547.

noids) that they gather from their host plant, the Scots pine (*Pinus sylvestris*; Codella and Raffa 1995, 1996; Larsson et al. 2000). However, some forms of these larvae match their backgrounds well (greenish brownish larvae on the green and brown background) and defend themselves vigorously only if threatened by a predator (Codella and Raffa 1993). Weak visual warning signals can also be found among shield bugs (Acanthosomatidae, Heteroptera): several species discharge noxious secretions from the metathoracic glands when attacked, producing a strong and distinct odor, yet are not very conspicuous visually. In feeding trials, birds avoid bugs after experience attacking them, suggesting that they have associated appearance with the chemical defense (Krall et al. 1999). Olfaction seems only to enhance avoidance learning of visual signals in birds (Jetz et al. 2001; Lindström et al. 2001*b*; and Roper and Marples 1997). *Elasmucha grisea* (Acanthosomatidae) express defensive odors when disturbed but are cryptic (grayish, greenish, brownish) on birch leaves (Chinery 1993). Thus, these species use weak rather than strong visual aposematic signals. The use of weak signals with strong defenses may be a common phenomenon, but it has been largely ignored in previous studies. In fact, weak aposematic signals are usually not even looked for because most current theory does not necessarily lead us to expect it and because the popular examples involve conspicuous coloration. However, the term "aposematic" means only "warning signal" (literally, signaling away) and makes no statement about its conspicuousness. An aposematic signal has to be discriminable and memorable (Guilford 1988, 1990; Endler 1990, 1991*a*), not necessarily conspicuous (Sherratt and Beatty 2003).

What causes some unprofitable species to be conspicuous and others inconspicuous? One possibility is that the visually inconspicuous species are highly visible in the ultraviolet (UV) part of the spectrum (which humans cannot see), as in the larva of *Lithophane ornitopus* (Church et al. 1998; but see Lyytinen et al. 2001), or have strong nonvisual signals; this just reflects our inability to perceive the conspicuous warning signal. However, Lyytinen et al. (2001) found that birds did not learn to associate UV marks with distastefulness even though they learned to associate other colors with it. Another possibility is aggregation; high density may itself be a signal. However, in experiments (Riipi et al. 2001), the conspicuousness of aggregation increased only asymptotically with group size, suggesting limits to aggregation as a warning in itself; grouping may be a weak signal. Grouping is more effective as a defense when combined with a conspicuous signal (Mappes and Alatalo 1997; Riipi et al. 2001), making the occurrence of aggregating species with low conspicuousness puzzling. Still another possibility is that if defenses are inexpensive to produce, then unprofitability is widespread (in the species) and selection for conspicuousness is weak (Leimar et al. 1986; Guilford 1994). But signals may still become more conspicuous if they improve the rate of avoidance learning (Leimar et al. 1986). If discriminability is very important, then an aposematic color pattern can work even if relatively cryptic as long as it is distinct (Sherratt and Beatty 2003). In this article we investigate an entirely different possible reason: variation in predator mixes—combinations of predators with different effects on the prey species.

Different prey species share different predator mixes, and predators differ in their abilities to detect and discriminate among prey signals, their ability to learn to associate the signal with prey defenses, and their abilities to overcome prey defenses (Endler 1988; Sherratt and MacDougall 1995). If predators vary in their ability to ignore distastefulness or avoid toxicity, the relative importance of the different predators can determine whether or not the prey evolves aposematism (Thompson 1984; Endler 1988) and affects whether a polymorphism may occur (Mallet and Singer 1987; Mallet and Joron 1999; Mallet 2001). This may also be an explanation for weak aposematic signals: even if conspicuous to both predator species, if one predator finds a prey distasteful or unprofitable and another predator finds it palatable, it might not be the prey's best strategy to be too conspicuous. In this article we will summarize evidence for variation among predators and then present models that explore the consequences of variation among predators in whether or not they respond to prey as aposematic. Our aim is to show the importance of predator variation in affecting aposematic coloration and to stimulate more work on multiple-predator systems.

Variation among Predators

Predators that can learn to avoid aposematic species, and therefore favor the evolution of aposematic coloration, belong to a variety of taxa including dragonflies (Kauppinen and Mappes 2003), mantids (Berenbaum and Miliczky 1984; Bowdish and Bultman 1993), fish (Tullrot 1998; Tullrot and Sundberg 1991), toads (Brower et al. 1970), lizards (Boyden 1976; Sword et al. 2000), snakes (Terrick et al. 1995), and birds (e.g., herons and egrets: Caldwell and Rubinoff 1983; chickens: Gittleman and Harvey 1980; quail: Marples and Brakefield 1995; jays: Brower et al. 1968; starlings: Schuler and Hesse 1985; robins: Marples et al. 1998; and great tits: Alatalo and Mappes 1988, 1996). These predator taxa differ enormously in their hunting strategies, visual microhabitat, visual systems, learning, and remembering abilities (Alcock 1971; Levine and MacNichol 1979; Lythgoe 1979; Endler 1988, 1991*a*, 1991*b*, 1992; Bednekoff et al. 1997; Biegler et al. 2001;

Briscoe and Chittka 2001). For example, predators may differ in vision or perception; what may be conspicuous for one species may be cryptic to another, or they may forage in sufficiently different microenvironments that the appearance of the same prey signal may be different for each predator species (Endler 1978, 1988, 1991*a*, 1991*b*; Endler and Théry 1996). Within-species variation in foraging microhabitats with different viewing conditions may also yield varying prey signal clarity to each individual predator.

In addition to perceptual, cognitive, and hunting differences, there is significant variation among and within species in the level of dietary conservatism, neophobia, and innate avoidance of novel stimuli (data in Coppinger 1970; Schuler and Hesse 1985; Marples et al. 1998; Marples and Kelly 1999; Speed 2000, 2001; Thomas et al. 2003). For example, the hesitation delay of robins (*Erithacus rubecula*) and blackbirds (*Turdus merula*) encountering common prey with novel colors may last up to 3 mo, whereas in great tits (*Parus major*) the hesitation lasts from a few minutes up to 2 h (Marples et al. 1998; Mappes and Alatalo 1997; Thomas et al. 2003). There are significant differences among bird species in the tendency to take familiar aposematic prey in both the field (diet studies) and in the laboratory (Evans and Waldbauer 1982; Exnerová et al. 2003). In experiments, some species treated distasteful firebugs (*Pyrrhocoris apterus*) the same way as tasty mealworms while others avoided them to various degrees, and species varied as to whether they paid attention to the color of the firebugs when artificially altered (Exnerová et al. 2003). These were wild-caught birds, so the differences could be innate or learned, but in any case, what is aposematic to one species may not be to another (Exnerová et al. 2003). In addition, some species may show innate avoidance of some warning colors (Rubinoff and Kropach 1970; Gehlbach 1972; Smith 1975, 1977; Schuler and Hesse 1985). For whatever reasons, among-species variation in their rate of predation will have equivalent effects to differences in diet preferences: if the rate of predation from species that regard the prey as distasteful is a small fraction of the total predation, then we would expect a different outcome than if these species predominated as the prey's predators.

Within-species variation may also be important. There is remarkable within-species variation in the latency to take unpalatable prey (table 1 in Marples and Kelly 1999), and this has a genetic basis in quail (Marples and Brakefield 1995). Although widely ignored in data analysis, there is significant individual variation in learning abilities and responses to distasteful prey. For example, individual scrub jays (*Aphelocoma coerulescens*) vary in whether they use color, color pattern, size, or a combination of these when discriminating between models and mimics (Terhune 1977). More examples are shown in table 1. There is also widespread evidence for within-species variation in predator behavior for tasty prey (Allen and Anderson 1984; Sherratt and MacDougall 1995).

Some among-individual variation may arise from differences in experience, with juveniles more likely to attack unprofitable prey. For example, in an experiment on neophobia, those birds who had previously been given (tasty) butterflies attacked aposematic butterflies more frequently than those with no prior experience with butterflies (Coppinger 1970). Seasonal changes in predator naïveté can strongly affect the evolution of color patterns. For example, there is evidence that the phenology (timing) of appearance of Hymenoptera mimics avoids the flush of naive young predators after the predator breeding season (Waldbauer and Laberge 1985). There may also be age-related changes in the ability to get around prey defenses. Acquisition of new diet items may be a complex behavioral process, and among-individual differences in prior experience may result in very different diet preferences (Marples and Kelly 1999; Thomas et al. 2003), even among siblings if they have had different prey species experiences. Within-species variation is usually treated as noise; the methods sections of published articles on predation or diet preferences frequently contain a phrase such as "animals that did not behave properly were excluded from further analysis." But this variation may be natural and important in the evolution of warning signals.

Even if all other factors are equal, local differences in predation pressure on the predators themselves may reduce their tendency or ability to make decisions about prey (Lima and Dill 1990), and this may further induce variation in the effects of prior experience with prey. This applies to both within- and among-species variation.

There is variation among predator species in how they deal with noxiousness, toxins, or other forms of unprofitability. Predator species vary in their sensitivity to prey toxins; some predators have resistance and can eat toxic prey with no ill effects (Brodie and Brodie 1999; Motychak et al. 1999). For example, bushtits (*Psaltriparus minumus*) can eat monarch butterflies (Calvert et al. 1979; Fink and Brower 1981) that are unprofitable for most other predators. Parasitoids may be insensitive to chemical defenses that work on invertebrate or verterbrate predators and may induce very different selective pressures than predators (Gentry and Dyer 2002). For example, if parasitoids are insensitive to chemical defenses and are the most important source of mortality, and if conspicuous coloration would attract their attention, then selection would favor crypsis even if other predators favor bright colors. Some predators have adapted behaviors to overcome unpalatability (Yosef and Whitman 1992). For example, Scott's orioles (*Icterus parisorum*), black-capped orioles (*Icterus*

Table 1: Examples of variation in the probability of eating unpalatable prey (avoidance of aposematically colored food items) by great tits (*Parus major*)

Bird rank	Experiment 1[a] (signal strong, unpalatability strong)	Experiment 2[a] (signal weak, unpalatability strong)	Experiment 3[b] (signal moderate, unpalatability moderate)	Experiment 4[c] (signal moderate, unpalatability strong)
1	.00	.27	.00	.00 (2)
2	.07	.33	.04	.17
3	.20	.40	.08	.20 (4)
4	.20	.40	.08	.25 (3)
5	.33	.47	.16	.27
6	.40	.47	.20	.29
7	.53	.53	.32	.33 (4)
8	.60	.53	.36	.38 (3)
9	…	…	…	.40 (2)
10	…	…	…	.50 (2)
11	…	…	…	.60
Mean ± SD	.29 ± .21	.43 ± .09	.16 ± .13	.32 ± .15
CV (SD/mean)	.72	.21	.81	.46

Note: Columns are independent experiments with different birds; individuals in each column are arranged in rank tendency to eat the unpalatable prey. In experiments 1 and 2 (Riipi et al. 2001), birds were presented 24 palatable and 24 distasteful (quinine) artificial prey. Distasteful items had either strong or weak aposematic visual signals. The birds were allowed to eat 15 prey items in each of five consecutive trials. Mortality risk of conspicuous prey (number of conspicuous prey taken/conspicuous prey available) was calculated for the last trial. The tendencies to take prey were significantly different among birds in both experiments: $F = 7.88$, df $= 7, 32$, $P < .0001$; $F = 2.99$, df $= 7, 32$, $P = .016$. These tests were done on the residuals of the mean number of aposematic prey taken for each trial (correcting for learning in sequential trials). In experiment 3 (J. Mappes, R. V. Alatalo, L. Lindström, and A. Lyytinen, unpublished), birds were presented 200 prey items in which 100 were moderately unpalatable and the aposematic signal was moderately conspicuous. The birds were allowed to eat 50 prey items in each of two trials, 1 wk apart. Mortality risk was calculated for the last trial. In experiment 4 (Mappes and Alatalo 1997), birds were presented 12 unpalatable prey sequentially. Each trial lasted 30 min, and risks are calculated from the number of aposematic prey eaten.

[a] Riipi et al. 2001.

[b] J. Mappes, R. V. Alatalo, L. Lindström, and A. Lyytinen, unpublished.

[c] Mappes and Alatalo 1997. Numbers in parentheses are numbers of tied ranks (24 birds total in this experiment).

abeillei), and black-headed grosbeaks (*Pheuctius melanocephalus*) kill monarch butterflies and eat only the body parts with less toxic compounds (Calvert et al. 1979; Fink and Brower 1981). Loggerhead shrikes (*Lanius ludovicianus*) stick unpalatable locusts on twigs and leave them to dry for a few days. Drying diminishes unpalatability, and birds judge by the change in color when the stored insects can be eaten (Yosef and Whitman 1992). Various species of cuckoos and bronze cuckoos regularly eat hairy, spiny, or noxious prey, and some go to extensive trouble to separate the noxious from the edible parts (Schodde and Tidemann 1990; Hughes 1997, 2001; Davies 2000). Sympatric cuckoo species spend different amounts of time preparing noxious food (Bender 1961; Hughes 2001), while other genera would ignore the food entirely. Within-species variation in dealing with unpalatability and resistance to toxicity is probable but has rarely been investigated (Bowers and Farley 1990). Both within- and among-species differences in the handling of distasteful or toxic prey ensure that the efficacy of antipredator defenses will vary depending on what kinds of predator individuals and species are present.

It is clear that most stages of predation (Endler 1986) show among-individual and among-species variation. If among-predator variation represents significant variation in nature, then it might be important in affecting the evolution of aposematic coloration. We will test this with models.

General Modeling Approach

We will investigate the consequences of variation in predator treatment of distasteful prey by means of a single-locus genetic model that attempts to capture the essence of the system. The population will be polymorphic for two morphs (phenotypes) of differing visibility and will be subject to two different kinds of predators in various proportions. The two predators will differ in their responses to prey. We will combine different predation modes to address the main question: can the less visible form pre-

dominate or can a polymorphism with more and less visible forms be maintained when the species is defended against some but not all predators?

Consider a large population of a diploid prey species that is polymorphic at a single autosomal Mendelian locus A, with two alleles **A** and **a**. Let allele **A** be dominant with a frequency p; $q = 1 - p$. Let there be two kinds of predators, a fraction d that treat the prey as distasteful (or otherwise unprofitable) and a fraction $(1 - d)$ that do not, for whatever reason. Our models assume that "tasty" means neither distasteful nor toxic and that "distasteful" means both distasteful (or noxious) and toxic or otherwise unprofitable. We are making no assumptions about the identity of the two kinds of predators; they can be different species or different individuals within a species, and the differences could arise from predator genetics, ontogeny, or experience. Predators do not evolve; this makes our models conservative because coevolution between predator and prey can lead to polymorphism (Gavrilets and Hastings 1998).

The two phenotypes (A- and aa, henceforth abbreviated *A* and *a*) will differ in their relative visibility to each predator, but we will assume no variation in vision or perception among predators. Let v represent the visibility of *A* relative to *a* ($0 \le v \le 1$). Except in models E and 4 (details below), the two morphs (*A* and *a*) are equally cryptic when $v = 1/2$; for models E and 4, the morphs are equally visible when $v = 1$. In the models, v is an effect that could also arise from nonvisual effects on fitness, but we will interpret it as effects of relative visibility in this article. The results could apply to other sensory modes if v is a measure of sound level, odor concentration, or other signal strength. Visibility can also affect fitnesses in other ways, but our aim is to keep the models as simple as possible. Our models investigate the consequences of differences in conspicuousness of one morph relative to that of another and make no explicit assumptions about the degree of absolute visibility. For example, both theoretical and experimental studies suggest that crypsis may not be perfect on any one background if the species is found by predators on several different visual backgrounds; the degree of crypsis evolved is a compromise between the best phenotypes in the different visual backgrounds (Endler 1984; Merilaita et al. 1999, 2001). Nevertheless, one phenotype that has a lower relative visibility (v) than another phenotype, averaged over all backgrounds, should increase. In fact, the compromise strategy for different visual backgrounds discussed by Merilaita et al. (1999, 2001) resembles the effects of variation in predator modes that we will demonstrate in models 1–4 below; color patterns evolve as a compromise among various selective factors.

There is a large empirical and theoretical literature on how both vertebrate and invertebrate predators treat polymorphic prey (Allen and Clarke 1968; Clarke 1969; Greenwood 1984; Allen 1988; Endler 1991*a*; Sherratt and Harvey 1993; Sherratt and MacDougall 1995; Gavrilets and Hastings 1998), and we will be concerned with three simplified modes of predation: (1) if the prey are neither distasteful nor unprofitable (tasty), then a morph's fitness is a constant that depends on the degree of visibility; (2) if the prey are distasteful, then a morph's fitness is positively frequency dependent and fitness increases with frequency (Leimar et al. 1986; Mallet and Barton 1989); (3) a tasty prey has negative frequency-dependent or apostatic fitness and fitness decreases with frequency (Allen and Clarke 1968; Greenwood 1984; Allen 1988; Endler 1991*a*; Gavrilets and Hastings 1995). Different natural systems may differ in the mix of predators using these three modes of selection, so we will model several combinations of them.

Fitness is also likely to change with density because it is the absolute rate of encounter that trains predators not to eat distasteful prey or favors the formation of a search image for cryptic prey, and density effects can sometimes overcome frequency effects (Clarke 1972; Leimar et al. 1986). For example, at very low population density, the slope of the frequency dependence may be lower than at high population density. This in turn can affect the tendency of the system to result in monomorphism (positive) or polymorphism (negative frequency dependence). By working with relative frequency-dependent effects rather than absolute frequency-dependent effects (as in many previous models) or density effects (as in the unusually realistic and detailed models of Leimar et al. 1986), we capture the basic fact that fitness changes with encounter rates (frequency or density) in known ways, while keeping the models manageable. However, this means that these models may not apply well at very low, extremely high, or strongly fluctuating prey densities.

We followed the modeling method described in Lewontin (1958); we found roots (equilibria) and tested their stability. For trivial roots ($p = 0$ or $p = 1$) and those outside [0, 1], we examined the sign of Δp for p just above 0 or just below 1 (Lewontin 1958; $\Delta p = 0$ at a trivial equilibria). This confirmed the stability or instability of the nontrivial ($0 < p < 1$) equilibrium (p^*) in each model.

In some models the nontrivial equilibrium p^* was unstable, and therefore the starting allele frequency p_o is important. The unstable p^* can be regarded as a threshold of fixation because a population with $p_o > p^*$ will become fixed for **A** ($p = 1$), whereas **A** will be lost if $p_o < p^*$. As p^* decreases, there are more and more possible different p_o such that $p_o > p^*$. We can measure the relative size of this zone of fixation ($p_o > p^*$) as $\phi = 1 - p^*$ (see fig. A1 in the online edition of the *American Naturalist*), and we will call ϕ the fixation tendency. If p_o are uniformly distributed among populations, the probability that **A** will be

Table 2: Summary of base models

	Model A, type C	Model B, type +FD	Model C, type −FD	Model D, types −FD and C	Model E, types −FD and C′
W_{AA}	$1-v$	$1 + v\ (p^2 + 2pq)$	$1-v\ (p^2 + 2pq)$	$1-r\ (p^2 + 2pq)-v/2$	$1-r\ (p^2 + 2pq)$
W_{Aa}	$1-v$	$1 + v\ (p^2 + 2pq)$	$1-v\ (p^2 + 2pq)$	$1-r\ (p^2 + 2pq)-v/2$	$1-r\ (p^2 + 2pq)$
W_{aa}	v	$1 + (1-v)q^2$	$1-(1-v)q^2$	$1-rq^2-(1-v)/2$	$1-rq^2-(1-v)/2$
Outcome	Fix or loss	Unstable p^*: $p^* = 1 - \sqrt{v}$; fix or loss	Stable p^*: $p^* = 1 - \sqrt{v}$	Stable p^* for $r = 1/2$; $p^* = 1 - \sqrt{v}$	Stable p^* for $r = 1/2$; $p^* = 1 - \sqrt{v/2}$

Note: C = constant fitness; FD = positive (+) or negtive (−) frequency dependence; C′ = asymmetrical constant fitness. See the appendix in the online edition of the *American Naturalist* for details.

fixed over all populations is $\phi = 1 - p^*$. If p_o are normally distributed, then more of their p_o will be above p^*, so fixation is more probable as ϕ increases. This will be true for many other population distributions, although the relationship may not be monotonic. Consequently, we will use ϕ as a rough measure of the tendency for allele **A** to be fixed, noting only that the larger the ϕ, the more possible p_o will be above the threshold of fixation p^*. This is only reasonable on average; $p_o < p^*$ may occur in some ancestral populations, and the less conspicuous morph would be fixed in those populations (Leimar et al. 1986). It could be argued that p_o will always start very low (mutation as the source), so ϕ has little to do with the probability of fixation. However, our models are based on situations with varying kinds of predators, and these can change in both ecological and evolutionary timescales. If predator communities change erratically in time (switching from avoiding and taking the prey), then the population's p_o could take any value, and populations would not necessarily be clustered near $p_o = 0$. In addition, we are concerned with what happens to morphs once they are present within the species distribution (intermediate p_o) rather than their origin (low p_o). The quantity ϕ is a rough measure of the tendency for populations to become fixed but is always negatively related to the threshold of fixation, p^*.

Solutions were obtained with Mathematica version 4.1, and results (especially regions with imaginary roots) were checked by simulation with MATLAB version 6.1 (R13). The nb and m files are available on request. Before presenting the combined models (1–4), we will review the properties of each basic component (base models A–E). We will then use the base models as modules to build the main models (1–4 below) in order to explore the effects of multiple predators; the two predator types prey according to different modules.

Base Models

We constructed five base models to capture the basic properties of known single-predator responses to polymorphic prey. The base models vary in how fitness is related to genotype frequency and visibility to predators (table 2): model A: frequency-independent effects of visibility; model B: fitness increases with frequency at a rate dependent on visibility (aposematic); model C: fitness decreases with frequency at a rate dependent on visibility (apostatic); model D: fitness of each phenotype decreases with frequency at the same rate plus visibility-related differences in frequency-independent fitnesses; model E: as for model D, but frequency-independent fitnesses are asymmetrical. Because the base models are needed to understand the main models but do not contain any new theoretical results, the details of the base models are presented in the appendix in the online edition of the *American Naturalist*. We will justify their design and summarize their results here. Readers not interested in the justifications can skip to the summary at the end of this section.

Model A: Visibility Affects Fitness Independent of Phenotype Frequency

In this model the dominant phenotype's (A) fitness decreases with visibility (v) while the recessive phenotype's (a) fitness increases with v (table 2). Allele **A** will be fixed ($p \to 1$) if $v < 1/2$, and **A** will be lost ($p \to 0$) if $v > 1/2$, with selective neutrality for $v = 1/2$. In other words, the more cryptic morph will always be fixed. This is the usual explanation for crypsis of palatable species. See the appendix for details.

Model B: Positive Frequency Dependence

Here the fitness of each phenotype increases with its frequency at a rate dependent on the phenotype's visibility. This is the classical relationship for aposematic coloration and also the relationship for tasty prey at very high density. This relationship has also been used in other models (e.g., Mallet 1986; Mallet and Barton 1989). It is a simplification of the rich dynamics resulting from variation in distastefulness, unprofitability, learning, memory, encounter rate,

relatedness, and other factors (Leimar et al. 1986; Guilford 1988, 1990, 1994; Lindström et al. 2001*c*; Servedio 2000), but it captures the essence of the process. Interestingly enough, positive frequency dependence can arise even in the absence of distastefulness if there is individual variation among predators and high prey density (Sherratt and MacDougall 1995). It can also happen without distastefulness with high density alone when the visual background may be other members of the prey species (Allen 1972; Allen and Anderson 1984; Greenwood 1984), as in the case of aggregating larvae or schooling fish.

The initial stages of evolution of aposematic coloration are problematic because the initial density of distasteful morphs may be too low to train the predators and because poorly protected but conspicuous prey may have a significant mortality (Guilford 1990). There have been a number of suggested mechanisms that minimize this problem, including aggregation and kin selection (Guilford 1990), predators testing prey before eating them (Guilford 1994), general dietary conservatism (Thomas et al. 2003), and plasticity in warning signals (Sword 2002). All of these affect the details of the form and slope of the frequency dependence and the initial fate of new aposematic forms. For simplicity we will assume that both morphs (with different effects of v) are already present in the population (intermediate p) and ask what happens to them once the positive frequency dependence is established.

Model B is a great simplification of the rich dynamics of aposematic coloration; under some conditions brighter coloration may not be favored (Leimar et al. 1986; Endler 1988). This means that we are being conservative about our enquiry into the origin of less conspicuous aposematic signals: more complexity (reducing the conditions favoring greater conspicuousness) would make the main models more likely to favor less conspicuous aposematic coloration.

Because most empirical studies indicate that learning is faster and retention is longer for greater visibility, let the rate of increase of the fitness of each phenotype be dependent on its relative visibility v (table 2). The result is an unstable nontrivial equilibrium at $p^* = 1 - \sqrt{v}$; allele **A** will be either fixed or lost, depending on whether the starting frequency p_o is above or below p^*. In model B, $p^* = 1 - \sqrt{v}$, hence $\phi = 1 - p^* = \sqrt{v}$; the tendency to fix allele **A** increases with its visibility. This is the usual explanation for warning coloration being highly conspicuous, but note that fixation is possible for low v, depending on p_o. It is also one reason for discussions of the difficulty of evolving conspicuous aposematic coloration; if ancestors are cryptic, then p_o is low, $p_o < p^*$ is probable, and fixation of cryptic genotypes (*a*) should occur (Leimar et al. 1986). Getting above p^* is difficult unless ϕ is high or mutation has a large effect on v. See the appendix for details and a plot of p^* and ϕ versus v.

Negative Frequency Dependence or Apostatic Selection

Palatable species are often subject to predators who preferentially prey on the more common form. This fitness relationship is also known as apostatic or pro-apostatic selection (Greenwood 1984). It can arise from a search image, a predator with a functional response, or foraging patterns (Endler 1991*a*). There are two different ways this can be modeled if we wish to account for varying visibility: the visibility of a morph can affect either the rate of frequency dependence (as in model B but with opposite sign) or its fitness can be independent of frequency (as in model A).

Model C: Negative Frequency Dependence, Rate Proportional to Visibility

In this model the fitness of each phenotype declines with its frequency at a rate dependent on the phenotype's visibility (table 2). It is the same as model B but with a negative instead of a positive relationship. This is the classical relationship in apostatic selection. The result is a stable nontrivial equilibrium at $p^* = 1 - \sqrt{v}$; the population will remain polymorphic with the less conspicuous morph more common (details in appendix). Note that in some published apostatic selection models, oscillations and chaotic behavior may occur rather than stability (Gavrilets and Hastings 1995); those conditions are roughly equivalent to our models with v very much >1 (we use $0 \leq v \leq 1$). In other models combining negative frequency dependence and other forms of selection (Wright 1969; Clarke 1972; and the main models below), the polymorphism is not always stable. Although negative frequency dependence maintains a polymorphism, the more visible morph will be at a lower frequency that declines with its relative visibility. This is expected for palatable species, which are in fact frequently polymorphic (Greenwood 1984).

Although models B and C have the same equilibrium, their consequences are opposite because their slopes at the same equilibria are opposite; fixation tendency $\phi = \sqrt{v}$ for model B and a stable equilibrium at $p^* = 1 - \sqrt{v}$ for model C. For details, see appendix.

Model D: Negative Frequency Dependence and Frequency-Independent Visibility Effects

In model C the visibility v affected the relative rates of decline of fitness with frequency, as it would if the visibility of a morph affected the predator's learning or forgetting

rate for that morph's color pattern. It is also possible that learning and forgetting rates are similar for each morph. This may happen if, for example, both morphs had a strong chemical defense that operated after detection, reinforcing postdetection avoidance behavior (as in Roper and Marples 1997; Lindström et al. 2001*b*). In this case visibility would only affect encounter and detection, which would be a function of the visual contrast with the background and not the morph's frequency (unless at very high density; see "Model B: Positive Frequency Dependence"). The effects are modified in this way in model D.

Let both phenotypes have the same frequency-dependent relationship (r, $0 < r < 1$) and differ by v in their frequency-independent fitness component (table 2). This is equivalent to avoidance learning independent of v but detection dependent on v. There are many other ways this system could be modeled (Clarke 1964, 1972; Clarke and O'Donald 1964; Wright 1969; Thompson 1984), but their qualitative results are similar (e.g., Thompson 1984, who also treats positive frequency dependence). Model D was chosen because it yields relatively simple solutions in combination with the other components in the main models. It results in either a stable equilibrium or fixation or loss depending on v and r. The population will be polymorphic when $r \geq 1/2$. For $r < 1/2$ (weaker frequency dependence), the population will be polymorphic for intermediate v (similar phenotypic visibilities), the range of v declining with decreasing r. Nontrivial p^* declines with v for all r. Smaller r results in a faster decline of p^* with v and larger r results in a slower decline and a smaller range of p^*. In the main models using model D, we will set $r = 1/2$. This yields a stable nontrivial equilibrium $p^* = 1 - \sqrt{v}$, the same as model C. Basically, under model D, the less conspicuous morph will be more common. For details, see appendix.

Model E: Negative Frequency Dependence and Asymmetrical Frequency-Independent Visibility

In model D both phenotypes have negative frequency dependence, and both morphs were also subject to frequency-independent fitnesses of equal and opposite effects. Because this may be unrealistic, in model E we will make the frequency-independent fitnesses asymmetrical but retain the same frequency-dependent effects. This model is similar to model D except that the effect of visibility is greater on morph a than morph A (table 2). Results are qualitatively similar to models C and D; the less conspicuous morph will be more common. For $r = 1/2$, the stable equilibrium is $p^* = 1 - \sqrt{v/2}$, and qualitative results are similar for other r. As for model D, we will set $r = 1/2$ when combining this model with others in the main models. For details, see the appendix.

Summary of Base Models (A–E)

The base models contain no surprises, matching our intuition and both empirical and theoretical results in the literature. Frequency-independent fitness leads to fixation of the less visible (more cryptic) morph (model A). Positive frequency-dependent fitness (model B) leads to fixation of the more visible morph with a fixation tendency (ϕ) positively related to its visibility (the classical aposematic model). Negative frequency-dependent fitness (apostatic selection) leads to a stable polymorphism with the less visible morph more common (models C–E), but it can also lead to the fixation of the less visible morph if the strength of the frequency dependence is low and/or the visibility of A is either very high or very low (models D and E).

Main Models

The base models (A–E) were designed to summarize the effects of single-predator modes for predators that either ignore or pay attention to the antipredator defenses of the prey. In order to explore the effects of variation in predators, we will now combine the base models to make the main models. In each case they will be combined by assuming that a fraction of predation by predators that regard the prey as distasteful (model B) is d and the fraction of predation by predators that regard the prey as palatable (models A, C–E) is $(1 - d)$. Therefore, the fitness components will combine in the general form

$$\begin{aligned}&\text{fitness of a given phenotype} =\\ &\quad d\ (\text{positive FD or model B})\\ &+ (1 - d)\ (\text{constant or negative FD or model A, C, D, or E}),\end{aligned} \tag{1}$$

where FD indicates frequency-dependent fitness.

Model 1: Positive Frequency Dependence and Frequency Independence

A fraction d of the predators regard the prey as aposematic (model B), and a fraction $(1 - d)$ regard the prey as palatable, with relative visibility v (model A). The fitnesses are

$$\begin{aligned}W_{AA} &= 1 + dv(p^2 + 2pq) - (1 - d)v,\\ W_{Aa} &= 1 + dv(p^2 + 2pq) - (1 - d)v,\\ W_{aa} &= 1 + d(1 - v)q^2 - (1 - d)(1 - v).\end{aligned} \tag{2}$$

Calculating p', the **A** allele frequency in the next genera-

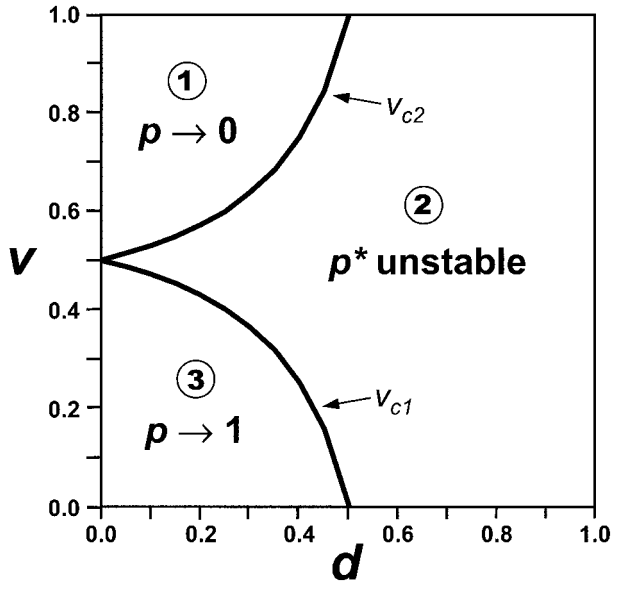

Figure 1: Model 1, positive frequency dependence and frequency independence for various combinations of visibility (v) and proportion of defense-sensitive predators (d). The two morphs are equally visible when $v = 1/2$, and the two predation modes are equally common when $d = 1/2$. In zone 1 ($v > v_{c2}$), the more visible phenotype A (genotypes *AA*, *Aa*) is always lost ($p \to 0$), and in zone 3 ($v < v_{c1}$), the other more visible phenotype a (*aa*) is always lost ($p \to 1$). In zone 2 ($v_{c1} < v < v_{c2}$), there is an unstable equilibrium p^*, and allele **A** will be fixed or lost depending on its initial frequency p_o: fixed if $p_o > p^*$ and lost if $p_o < p^*$. Note how the unstable zone increases (greater distance between v_{c1} and v_{c2}) with increasing d. Fixation tendencies ($\phi = 1 - p^*$) are shown in figure 2.

tion, and subtracting p (as in the appendix) yields the change in p per generation ($p' - p \equiv \Delta p$):

$$\Delta p = [p(1-p)^2(1 - 2d - 2v + 3dv + 2dp - dp^2)]/ [2d + v - 2dv + 2p(1 - 3d - 2v + 4dv) - p^2(1 - 7d - 2v + 4dv) - 4p^3d + p^4d]. \quad (3)$$

Setting $\Delta p = 0$ and solving for p (Lewontin 1958), we obtain four trivial (0, 1, 1, and >1) and one nontrivial ($0 < p < 1$) root, indicating a polymorphic equilibrium at

$$p^* = 1 - \sqrt{\frac{1 - d - 2v + 3dv}{d}}. \quad (4)$$

The slope of Δp at p^* is

$$\frac{2(1 - d - 2v + 3dv)^{3/2}(-\sqrt{d} + \sqrt{1 - d - 2v + 3dv})}{d(2v - 1 - 3dv - 2v^2 + 3dv^2)}. \quad (5)$$

For $d > 1/2$, p^* declines with v, and for $d < 1/2$, p^* increases with v. However, there are no stable nontrivial ($0 < p^* < 1$) equilibria. The slope (eq. [5]) is positive for $d > 1/2$, indicating that all p^* are unstable (Lewontin 1958), as in base model B. For $d < 1/2$, there are three zones, separated by the critical visibility lines $v_{c1} = (1 - 2d)/(2 - 3d)$ and $v_{c2} = (d - 1)/(3d - 2)$; see figure 1. The outer two zones (*1* and *3* in fig. 1) have trivial equilibria; $p \to 0$ for zone 1 ($v > v_{c2}$) and $p \to 1$ for zone 3 ($v < v_{c1}$). The middle zone 2 ($v_{c1} < v < v_{c2}$) has nontrivial equilibria (p^*), but the slope in this region is positive, indicating that p^* in this zone are unstable. When $d = 0$, the middle zone disappears, and allele **A** is either lost or fixed, as in base model A. This model reduces to base models A and B when $d = 0$ and $d = 1$, respectively.

Because there are no nontrivial stable equilibria, we can use a plot of $\phi = (1 - p^*)$ versus v and d to indicate (as in fig. A1) the tendency of populations to become fixed for **A** given a random initial **A** allele frequency p_o under various d and v. This is shown in figure 2.

For small values of d (most predators ignore the unpalatability), and $d < 1/2$, the more visible morph will be lost if $v > v_{c2}$ or $v < v_{c1}$, or if $v_{c2} > v > v_{c1}$, then it will be lost on average with a tendency $\phi = 1 - p^*$ (see fig. 1). For $v < 1/2$, it is the other morph (a) that is more conspicuous, and it has a greater tendency to be fixed. Thus, for $d < 1/2$, the model behaves like base model A in that the effects of visibility overwhelm the effects of positive frequency dependence. The difference between model 1 and base model A is the presence of the middle zone in

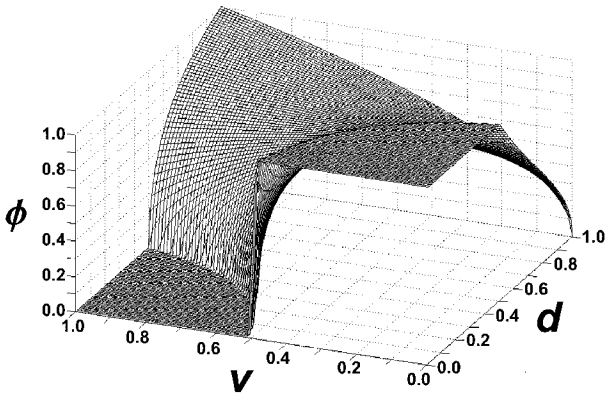

Figure 2: Model 1, fixation tendency ($\phi = 1 - p^*$) of allele **A** as a joint function of v and d (the nontrivial equilibria p^* are always unstable). Origin at lower right. For $d < 1/2$ (front half of surface), fixation is more probable for less visible phenotypes (smaller v) and is more probable for more visible phenotypes for $d > 1/2$ (rear half of surface). The differences are greater as d diverges from 1/2. The extremes ($d = 0$ and 1) are the same as base models A and B, respectively (compare with fig. A2 in the online edition of the *American Naturalist* for base model B).

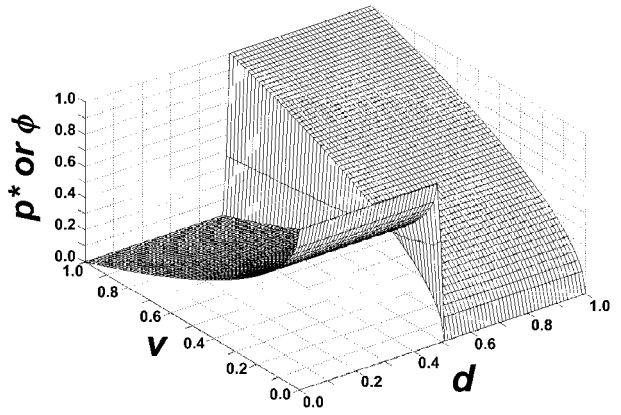

Figure 3: Models 2 and 3, positive frequency dependence and negative frequency dependence (the p^* of these two models are identical). Origin at bottom center. For $d < 1/2$, there is a stable equilibrium p^* independent of d that declines with v, as in base models C and D. For $d > 1/2$, there is an unstable equilibrium p^* that results in a fixation tendency of $\phi = 1 - p^*$. This is independent of predation style (d) and increases with visibility v, as in base model B. p^* is shown for $d < 1/2$, and ϕ is shown for $d \geq 1/2$.

which ϕ depends on starting frequency rather than the p_o-independent threshold at $v = 1/2$ in model A; see figure 2.

For larger values of d ($d > 1/2$, most predators learn to avoid eating the prey), the more visible morph ($v > 1/2$) is more likely to be fixed, as in base model B (fig. A1). The rate of change of the fixation tendency (ϕ) with v is faster as d gets closer to 1 (fig. 2).

The general result is that the more different d is from 1/2, in either direction, the more rapid the change in ϕ with visibility. At $d = 1/2$, the effects of positive frequency dependence and frequency-independent crypsis are roughly equal, but one predominates as d diverges from 1/2. The more visible morph is likely to be lost when $d < 1/2$ and fixed when $d > 1/2$, as expected from the relative contributions of the two selective modes. It is clear that a mixture of these two factors means that for a large part of the parameter space (fig. 1, *zone 2*), we cannot be certain for any one population whether or not the more conspicuous morph will be fixed. Weak signals can be fixed.

Model 2: Positive and Negative Frequency Dependence

A fraction d of the predators regard the prey as aposematic (base model B), inducing positive frequency-dependent fitness. A fraction $(1 - d)$ regard the prey as tasty and induce negative frequency-dependent (apostatic) fitness (base model C). The rates of frequency dependence for both predators is proportional to relative visibility v. The fitnesses are

$$\begin{aligned} W_{AA} &= 1 + dv(p^2 + 2pq) - (1 - d)v(p^2 + 2pq), \\ W_{Aa} &= 1 + dv(p^2 + 2pq) - (1 - d)v(p^2 + 2pq), \\ W_{aa} &= 1 + d(1 - v)q^2 - (1 - d)(1 - v)q^2. \end{aligned} \tag{6}$$

The change in p per generation is

$$\begin{aligned} \Delta p = [p(1 - p)^2(1 - 2d)(1 - v - 2p + p^2)]/[2d + v - 2dv \\ + 4p(1 - 2d - v + 2dv) - 2p^2(3 - 6d - v + 2dv) \\ + 4p^3(1 - 2d) - p^4(1 - 2d)]. \end{aligned} \tag{7}$$

Setting $\Delta p = 0$ and solving for p, we obtain four trivial (0, 1, 1, and >1) and one nontrivial ($0 < p < 1$) root, indicating a polymorphic equilibrium at

$$p^* = 1 - \sqrt{v}. \tag{8}$$

Although the equilibrium is independent of d, the slope of Δp at p^* is

$$\frac{2(1 - 2d)(1 - \sqrt{v})v^{3/2}}{v - 1 - 2dv - v^2 + 2dv^2}. \tag{9}$$

This is negative for $d < 1/2$ and positive for $d > 1/2$, indicating stability for $d < 1/2$ and instability for $d > 1/2$. For $d < 1/2$, there is a stable equilibrium p^* that declines with increasing v, as in base model C. The negative slope is > -1, indicating smooth approach to equilibrium with no cycles (Lewontin 1958). For $d > 1/2$ the equilibrium is unstable, so the fixation tendency ($\phi = \sqrt{v}$) increases with v, as in base model B; see figure 3. As in model 1, the more visible morph is more likely to be fixed if $d > 1/2$, but unlike model 1, the system will be polymorphic for $d < 1/2$. However, like model 1, for $d < 1/2$, the frequency declines with visibility. Model 2 reduces to base model C for $d = 0$ and to base model B for $d = 1$.

Model 3: Positive and Negative Frequency Dependence with Symmetrical Frequency Independence

A fraction d of the predators regard the prey as aposematic (base model B), including positive frequency-dependent fitness at a rate v. A fraction $(1 - d)$ regard the prey as tasty and induce negative frequency-dependent (apostatic) fitness at a rate r (base model C). Unlike model 2, visibility does not affect the rate of negative frequency dependence but does have a frequency-independent effect, as in base model D. The fitnesses are

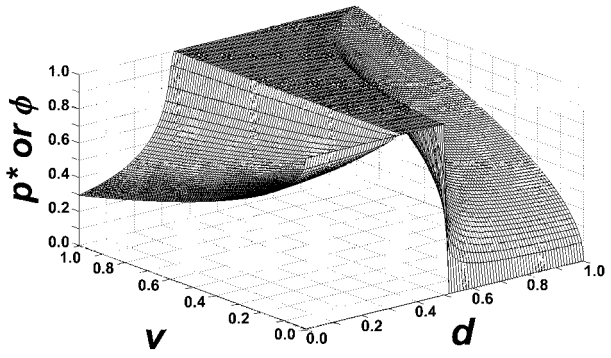

Figure 4: Model 4, positive frequency dependence, negative frequency dependence, and asymmetrical frequency independent fitness (see text). Origin at bottom center. For $d < 1/3$, there is a stable equilibrium p^* that declines jointly with increasing v and decreasing d. For $d > 1/3$, allele **A** will be fixed, or, if $v < v_{c3}$ (see text), there is an unstable nontrivial equilibrium. The result is an increasing fixation tendency (ϕ) for increasing v and decreasing d. The conditions for $d = 0$ and 1 are the same as base models E and B, respectively. p^* is shown for $d < 1/3$, and ϕ is shown for $d \geq 1/3$.

$$\begin{aligned} W_{AA} &= 1 + dv(p^2 + 2pq) - (1 - d)r(p^2 + 2pq) - (1 - d)v/2, \\ W_{Aa} &= 1 + dv(p^2 + 2pq) - (1 - d)r(p^2 + 2pq) - (1 - d)v/2, \\ W_{aa} &= 1 + d(1 - v)q^2 - (1 - d)rq^2 - (1 - d)(1 - v)/2. \end{aligned} \tag{10}$$

For simplicity we will set $r = 1/2$ (see fig. A2).

The change in p per generation is

$$\begin{aligned} \Delta p = {} & 2p(1 - p)^2(1 - 2d)(1 - v - 2p + p^2) \\ & \div [4d + v + 2p(3 - 2v - 7d + 6dv) \\ & - p^2(11 - 23d + 6dv - 2v) \\ & + 8p^3(1 - 2d) - 2p^4(1 - 2d)]. \end{aligned} \tag{11}$$

Setting $\Delta p = 0$ and solving for p, we obtain four trivial (0, 1, 1, and >1) and one nontrivial ($0 < p < 1$) root, indicating a polymorphic equilibrium at

$$p^* = 1 - \sqrt{v}, \tag{12}$$

as in model 2. Although the equilibrium is independent of d, the slope of Δp at p^* is

$$\frac{4(1 - 2d)(1 - \sqrt{v})v^{3/2}}{-1 - d - 2dv + 2dv^2}. \tag{13}$$

This is negative for $d < 1/2$ and positive for $d > 1/2$, indicating stability for $d < 1/2$ and instability for $d > 1/2$. The negative slope is > -1, indicating a smooth approach to equilibrium. The results are the same as in model 2 (fig. 3) except for the magnitude of Δp and the slope magnitude at p^* (cf. eqq. [7]–[9] and [11]–[13]).

Model 4: Positive and Negative Frequency Dependence with Asymmetrical Frequency Independence

This is similar to model 3 but assumes that the frequency-independent predation affects one phenotype more than the other (as in model E) and depends on visibility v. The fitnesses are

$$\begin{aligned} W_{AA} &= 1 + dv(p^2 + 2pq) - (1 - d)r(p^2 + 2pq), \\ W_{Aa} &= 1 + dv(p^2 + 2pq) - (1 - d)r(p^2 + 2pq), \\ W_{aa} &= 1 + d(1 - v)q^2 - (1 - d)rq^2 - (1 - d)(1 - v)/2. \end{aligned} \tag{14}$$

For simplicity, as in model 3, we will set $r = 1/2$ (see also fig. A2).

The change in p per generation is

$$\begin{aligned} \Delta p = {} & \{p(1 - p)^2[2 - 4d - v + 3dv - 4p(1 - 2d) \\ & + 2p^2(1 - 2d)]\}/[4d + v - 3dv \\ & + p(5 - 2v - 14d + 10dv) \\ & - p^2(11 - 23d + 5dv) + (8p^3 - 2p^4)(1 - 2d)]. \end{aligned} \tag{15}$$

Setting $\Delta p = 0$ and solving for p, we obtain four trivial (0, 1, 1, and >1) and two nontrivial ($0 < p^* < 1$) roots, indicating two possible polymorphic equilibria at

$$p^* = 1 \pm \frac{\sqrt{v(1 - 5d + 6d^2)}}{\sqrt{2}(2d - 1)}. \tag{16}$$

This model has more complex behavior because both roots can be nontrivial ($0 < p^* < 1$), depending on d and v. The slopes for the two roots are

$$\frac{(3d - 1)\sqrt{2(1 - 5d + 6d^2)}[\mp 2 \pm 4d + \sqrt{2v(1 - 5d + 6d^2)}]v^{3/2}}{(2d - 1)[2 + v - 2d(1 + v^2) + d^2(-4 - 5v + 6v^2)]}, \tag{17}$$

where the $\mp$ and $\pm$ indicate that the first root $p_1^* = 1 + k$ goes with the slope terms $-2 + 4d$ and the second root $p_2 = 1 - k$ goes with the slope terms $2 - 4d$. An examination of the behavior of equations (16) and (17) as a function of d and v reveals that there is a stable (slope < 0) nontrivial equilibrium when $d < 1/3$. As in models 2 and 3, the slope is > -1, indicating a smooth approach to equilibrium. Allele **A** is fixed ($p = 1$) when $1/3 \leq d \leq 1/2$. For $d > 1/2$, allele **A** is fixed if $v > v_{c3}$, where $v_{c3} = 2(2d - 1)/(3d - 1)$. If $d > 1/2$ and $v < v_{c3}$, then p_2^* is nontrivial and unstable, giving a fixation tendency of $\phi = 1 - p_2^*$. Figure 4 shows the general results of model

4. Like models 2 and 3, there is a stable polymorphism for smaller d, although now stability requires $d < 1/3$ rather than $d < 1/2$. Like models 2 and 3, the polymorphism has a lower frequency of the more visible morph for increasing visibility (v) and decreasing d. Like models 1–3, for $d > 1/2$, ϕ increases with v. However, ϕ decreases for increasing d, unlike model 1 (where it increases with d), or models 2 and 3 (where it is independent of d).

Summary and Conclusions

In spite of the different assumptions about predator behavior in the four models, their qualitative predictions are very similar. When the fraction of predators that notice and learn to avoid noxious prey (d) is less than 1/2 (1/3 in model 4), crypsis or low visibility (v) is favored. This may occur with (models 2–4) or without (model 1) a stable polymorphism for visibility. If there is a polymorphism, the equilibrium declines with v. When $d > 1/2$, higher visibility or conspicuous aposematism is favored. When the mixture of predators is roughly even (d in the vicinity of 1/2), almost anything can happen, especially when the two color morphs are similar in visibility ($v \approx 1/2$); fixation of the less conspicuous morph is possible, or a population can have a polymorphism with varyingly visible prey. When some predators ignore the distastefulness, we do not necessarily expect the population to evolve high visibility.

For $d > 1/2$, fixation of the most conspicuous morph is probabilistic rather than guaranteed because the population state is based on an unstable equilibrium p^* rather than a constant sign of Δp for all p. This is also a property of pure aposematic models ($d = 1$ or model B). An unstable equilibrium means that the starting population frequency p_0 determines the outcome, and with randomly distributed p_0 the fixation tendency for a population is $\phi = (1 - p^*)$. Although as visibility (v) increases, ϕ increases (figs. 2–4), populations can be above and below p^*. If p_o is uniformly distributed, otherwise identical populations will vary in fixation tendency with variance $\phi(1 - \phi)$; for other p_o distributions, the variance of ϕ will tend to be larger for intermediate ϕ. For all models this variance is highest at intermediate v and d, again indicating that distasteful populations may not necessarily evolve high visibility. This assumes infinite population size; the variance of outcome will be even greater for small populations. In addition, given an ancestral population with low v and low p_o, a mutation that causes a large increase in v is more likely to be fixed in a small population than another mutation causing a small change in v because a small random change in gene frequency (Δp) at high visibility (v) is more likely to bring p above the unstable equilibrium p^* than the same Δp at low v, leading to fixation of the more conspicuous allele (see fig. A1).

Intermediate d is the transition between conditions favoring crypsis and conditions favoring conspicuous coloration. This is the point at which the effects of positive frequency-dependent selection yield to apostatic or frequency-independent selection. In many cases the transition can be sudden (models 1–3); small changes in d can make the difference between fixing the less or more conspicuous allele (model 1) or maintaining a polymorphism in a distasteful species. Note that d is the fraction of individual predators treating prey as unprofitable; more than two predator species may be involved in the two groups d and $1 - d$.

In nature the proportion of predators finding the prey distasteful (d) is not constant but will fluctuate from place to place and from year to year. If d varies geographically, then the models predict geographic variation in visibility, possibly with sharp geographic transitions. Sharp transitions can occur because small changes in d at intermediate d result in large changes in p^*, or changes from polymorphism to fixation. Temporal fluctuations can result if d varies with experience and the age structure changes in time. Predators could also vary genetically and spatially in d. If d varies around an intermediate mean, then conditions may allow the less conspicuous morph to persist or even result in its fixation. This depends on the range of variation in d and v (figs. 2–4). Temporal fluctuations in d will blur the boundaries between high- and low-d areas.

Given the variety of predators in nature and our results, one may well wonder why some genera and even families are all conspicuously aposematic. These taxa may have developed sufficient defenses that no predators can kill them ($d = 1$). In Dendrobatid frogs there is a correlation between noxiousness and conspicuousness (Summers and Clough 2001). Such a correlation may arise from different visual backgrounds, different evolutionary experience (times) with sensitive predators or differing predator mixes. More studies of aposematic taxa and their complete predatory communities would be valuable. It would be interesting to investigate what part of the parameter space of figures 2–4 and A2 is most densely populated in nature, and if uneven, what predisposes populations to certain combinations of predators (d) and visibility (v).

In summary, when $d < 1/2$, polymorphism is probable if apostatic (negative frequency-dependent) selection is present (models 2–4; figs. 3, 4), while fixation of either morph will occur depending on v and p_o in the absence of apostatic selection (model 1; figs. 1, 2). In model 1 fixation of the less conspicuous morph is probable if the ancestral population is cryptic because $p_o < p^*$. If $d > 1/2$, conditions favor monomorphism in all the models, but this depends on the degree of crypsis; fixation of the less

cryptic morph is still possible. We conclude that conditions favoring weak signals in protected species are generous if predators vary in whether they treat the prey as distasteful. It would be interesting to know whether weakly signaling aposematic species are more often subject to a diversity of predators (varying *d*) than strongly signaling species. In any case it is important to know the complete suite of predators of a prey species, their relative risks to the prey, and what fraction of them treat the prey as distasteful or unprofitable. Because selection on visibility depends on the visual backgrounds and the types and abilities of the predators, predicting the response to selection by a single predator is unlikely to succeed in many cases. We cannot assume that because a species is distasteful or unprofitable to some predators that it will necessarily signal strongly to all predators.

Acknowledgments

Many thanks to J. Allen, M. Blows, B. Clarke, L. Day, N. Doerr, M. Jaroensutasinee, H. Kokko, L. Lindström, J. Mallet, S. Merilaita, K. Prudic, and G. Tully for useful discussions and comments on the manuscript. We would especially like to thank the two anonymous reviewers who made some particularly helpful suggestions about the manuscript. The Academy of Finland provided funding for J.M. during her visit to University of California, Santa Barbara, to work with J.A.E.

Literature Cited

Alatalo, R. V., and J. Mappes. 1988. Frequency-dependent selection by predators. Philosophical Transactions of the Royal Society of London B 319:485–503.

———. 1996. Tracking the evolution of warning signals. Nature 382:708–710.

Alcock, J. 1971. Interspecific differences in avian feeding behaviour and the evolution of Batesian mimicry. Behaviour 40:1–9.

Allen, J. A. 1972. Evidence for stabilizing and apostatic selection by wild passerines. Nature 237:348–349.

———. 1988. Frequency dependent selection by predators. Philosophical Transactions of the Royal Society of London B 319:485–503.

Allen, J. A., and K. P. Anderson. 1984. Selection by passerine birds is anti-apostatic at high prey density. Biological Journal of the Linnean Society 23:237–246.

Allen, J. A., and B. C. Clarke. 1968. Evidence for apostatic selection by wild passerines. Nature 220:501–502.

Bednekoff, P. A., R. P. Balda, A. C. Kamil, and A. G. Hile. 1997. Long-term spatial memory in four seed-caching corvid species. Animal Behaviour 53:335–341.

Bender, R. O. 1961. Food competition among closely related sympatric species. Wilson Bulletin 73:214.

Berenbaum, M. R., and E. Miliczky. 1984. Mantids and milkweed bugs: efficacy and aposematic coloration against invertebrate predators. American Midland Naturalist 111:64–68.

Biegler, R., A. McGregor, J. R. Krebs, and S. D. Healy. 2001. A larger hippocampus is associated with longer-lasting spatial memory. Proceedings of the National Academy of Sciences of the USA 98:6941–6944.

Bowdish, T. I., and T. L. Bultman. 1993. Visual cues used by mantids in learning aversion to aposematically colored prey. American Midland Naturalist 129:215–222.

Bowers, M. D., and S. Farley. 1990. The behavior of grey jays, *Perisoreus canadensis*, towards palatable and unpalatable Lepidoptera. Animal Behaviour 39:699–705.

Boyden, T. C. 1976. Butterfly palatability and mimicry: experiments with *Ameiva* lizards. Evolution 30:73–81.

Briscoe, A. D., and L. Chittka. 2001. The evolution of color vision in insects. Annual Review of Entomology 46:471–510.

Brodie, E. D., III, and E. D. Brodie, Jr. 1999. Arms-race between predators and prey. BioScience 49:557–568.

Brower, L. P., W. N. Ryerson, L. L. Coppinger, and S. C. Glazier. 1968. Ecological chemistry and the palatability spectrum. Science 161:1349–1351.

Brower, L. P., F. H. Pough, and H. R. Meck. 1970. Theoretical investigations of automimicry. I. Single trial learning. Proceedings of the National Academy of Sciences of the USA 66:1059–1066.

Caldwell, G. S., and R. W. Rubinoff. 1983. Avoidance of venomous sea snakes by naive herons and egrets. Auk 100:195–198.

Calvert, W. H., L. E. Hedrik, and L. P. Brower. 1979. Mortality of the monarch butterfly (*Danaus plexippus* L.): avian predation experiments at five overwintering sites in Mexico. Science 204:847–851.

Chinery, M. 1993. Collins guide to insects of Britain and Western Europe. 3d ed. Harper-Collins, London.

Church, S. C., A. T. D. Bennett, I. C. Cuthill, S. Hunt, N. S. Hart, and J. C. Patridge. 1998. Does Lepidopteran larvel crypsis extend into the ultraviolet? Naturwissenschaften 85:189–192.

Clarke, B. 1964. Frequency-dependent selection for the dominance of rare polymorphic alleles. Evolution 18: 364–369.

———. 1969. The evidence for apostatic selection. Heredity 24:347–352.

———. 1972. Density-dependent selection. American Naturalist 106:1–13.

Clarke, B. C., and P. O'Donald. 1964. Frequency-dependent selection. Heredity 19:201–206.

Codella, S. G., and K. F. Raffa. 1993. Defense strategies of folivorous sawflies. Pages 261–294 *in* M. R. Wagner and

K. F. Raffa, eds. Sawfly life history adaptations to woody plants. Academic Press, San Diego, Calif.

———. 1995. Host plant influence on chemical defense in conifer sawflies (Hymenoptera: Diprionidae). Oecologia (Berlin) 104:1–11.

———. 1996. Individual and social components of wood ant response to conifer sawfly defense (Hymenoptera: Formicidae, Diprionidae). Animal Behaviour 52:801–811.

Coppinger, R. P. 1970. The effect of experience and novelty on avian feeding behavior with reference to the evolution of warning coloration in butterflies. II. Reactions of naive birds to novel insects. American Naturalist 104: 323–335.

Cott, H. B. 1940. Adaptive coloration in animals. Methuen, London.

Davies, N. B. 2000. Cuckoos, cowbirds and other cheats. Poyser, London.

Dyer, L. A. 1995. Effectiveness of caterpillar defenses against three species of invertebrate predators. Journal of Research on the Lepidoptera 34:58–68.

Edmunds, M. 1974. Defense in animals: a survey of anti-predator defenses. Longman, London.

Endler, J. A. 1978. A predator's view of animal color patterns. Evolutionary Biology 11:319–364.

———. 1984. Progressive background matching in moths, and a quantitative measure of crypsis. Biological Journal of the Linnean Society 22:187–231.

———. 1986. Defense against predation. Pages 109–134 *in* M. E. Feder and G. E. Lauder, eds. Predator-prey relationships, perspectives and approaches from the study of lower vertebrates. University of Chicago Press, Chicago.

———. 1988. Frequency-dependent predation, crypsis and aposematic coloration. Philosophical Transactions of the Royal Society of London B 319:505–523.

———. 1990. On the measurement and classification of colour in studies of animal colour patterns. Biological Journal of the Linnean Society 41:315–352.

———. 1991*a*. Interactions between predators and prey. Pages 169–196 *in* J. R. Krebs and N. B. Davies, eds. Behavioral ecology. 3d ed. Blackwell Scientific, Oxford.

———. 1991*b*. Variation in the appearance of guppy color patterns to guppies and their predators under different visual conditions. Vision Research 31:587–608.

———. 1992. Signals, signal conditions, and the direction of evolution. American Naturalist 139(suppl.):S125–S153.

Endler, J. A., and M. Théry. 1996. Interacting effects of lek placement, display behavior, ambient light, and color patterns in three Neotropical forest-dwelling birds. American Naturalist 148:421–452.

Evans, D. L., and G. P. Waldbauer. 1982. Behavior of adult and naive birds when presented with a bumble bee and its mimic. Zeitschrift für Tierpsychologie 59:247–259.

Exnerová, A., E. Landová, P. Štys, R. Fuchs, M. Prokopová, and P. Cehláriková. 2003. Reactions of passerine birds to aposematic and non-aposematic firebugs (*Pyrrhocoris apterus;* Heteroptera). Biological Journal of the Linnean Society 78:517–525.

Fink, L. S., and L. P. Brower. 1981. Birds can overcome the cardenolic defense of monarch butterflies in Mexico. Nature 291:67–70.

Gavrilets, S., and A. Hastings. 1995. Intermittency and transient chaos from simple frequency-dependent selection. Proceedings of the Royal Society of London B 261:233–238.

———. 1998. Coevolutionary chase in two-species systems with applications to mimicry. Journal of Theoretical Biology 191:415–427.

Gehlbach, F. R. 1972. Coral snake mimicry reconsidered: the strategy of self-mimicry. Forma et Functio 5:311–320.

Gentry, G. L., and L. A. Dyer. 2002. On the conditional nature of Neotropical caterpillar defenses against their natural enemies. Ecology 83:3108–3119.

Gittleman, J. L., and P. H. Harvey. 1980. Why are distasteful prey not cryptic? Nature 286:149–150.

Greenwood, J. J. D. 1984. The functional basis of frequency-dependent food selection. Biological Journal of the Linnean Society 23:177–199.

Guilford, T. 1986. How do "warning colours" work? conspicuousness may reduce recognition errors in experienced predators. Animal Behaviour 34:286–288.

———. 1988. The evolution of conspicuous coloration. American Naturalist 131(suppl.):S7–S21.

———. 1990. The evolution of aposematism. Pages 23–62 *in* D. L. Evans and J. O. Schmidt, eds. Insect defenses: adaptive mechanisms and strategies of prey and predators. State University of New York Press, Albany.

———. 1994. "Go-slow" signaling and the problem of automimicry. Journal of Theoretical Biology 170:311–316.

Hughes, J. M. 1997. Mangrove cuckoo (*Coccyzus minor*). Pages 1–20 *in* A. Poole and F. Gill, eds. Birds of North America no. 299. Academy of Natural Sciences, Philadelphia.

———. 2001. Black-billed cuckoo (*Coccyzus erythrophalmus*). Pages 1–24 *in* A. Poole and F. Gill, eds. Birds of North America no. 587. Academy of Natural Sciences and Birds of North America, Philadelphia.

Jetz, W., C. Rowe, and T. Guilford. 2001. Non-warning odors trigger innate color aversions—as long as they are novel. Behavioral Ecology 12:134–139.

Kauppinen, J., and J. Mappes. 2003. Why are wasps so intimidating: field experiments on hunting dragonflies

(Odonata: *Aeshna grandis*). Animal Behaviour 66:505–511.

Krall, B. S., R. J. Bartelt, C. J. Lewis, and D. W. Whitman. 1999. Chemical defense in the stink bug *Cosmopepla bimaculata.* Journal of Chemical Ecology 25:2477–2494.

Larsson, S., B. Ekbom, and C. Björkman. 2000. Influence of plant quality on pine sawfly population dynamics. Oikos 89:440–450.

Leimar, O., M. Enquist, and B. Sillén-Tullberg. 1986. Evolutionary stability of aposematic coloration and prey unprofitability: a theoretical analysis. American Naturalist 128:469–490.

Levine, J. S., and E. F. MacNichol, Jr. 1979. Visual pigments in teleost fishes: effects of habitat, microhabitat, and behavior on visual system evolution. Sensory Processes 3:95–131.

Lewontin, R. C. 1958. A general method for investigating the equilibrium of gene frequency in a population. Genetics 43:419–434.

Lima, S. L., and L. M. Dill. 1990. Behavioral decisions made under the risk of predation: a review and prospectus. Canadian Journal of Zoology 68:619–640.

Lindström, L., R. V. Alatalo, J. Mappes, M. Riipi, and L. Vertainen. 1999. Can aposematic signals evolve by gradual change? Nature 397:249–251.

Lindström, L., R. V. Alatalo, A. Lyytinen, and J. Mappes. 2001*a*. Predator experience on cryptic prey affects the survival of conspicuous aposematic prey. Proceedings of the Royal Society of London B 268:357–361.

Lindström, L., C. Rowe, and T. Guilford. 2001*b*. Pyrazine odour makes visually conspicuous prey aversive. Proceedings of the Royal Society of London B 268:159–162.

Lindström, L., R. V. Alatalo, A. Lyytinen, and J. Mappes. 2001*c*. Strong antiapostatic selection against novel rare aposematic prey. Proceedings of the National Academy of Sciences of the USA 98:9181–9184.

Lythgoe, J. N. 1979. The ecology of vision. Oxford University Press, Clarendon, Oxford.

Lyytinen, A., R. V. Alatalo, L. Lindström, and J. Mappes. 2001. Can ultraviolet cues function as aposematic signals? Behavioral Ecology 12:65–70.

Mallet, J. 1986. Hybrid zones of *Heliconius* butterflies in Panama and the stability and movement of warning colour clines. Heredity 56:191–202.

———. 2001. Causes and consequences of a lack of coevolution in Müllerian mimicry. Evolutionary Ecology 13:777–806.

Mallet, J., and N. Barton. 1989. Inference from clines stabilized by frequency-dependent selection. Genetics 122: 967–976.

Mallet, J., and M. Joron. 1999. Evolution of diversity in warning color and mimicry: polymorphisms, shifting balance and speciation. Annual Review of Ecology and Systematics 30:201–233.

Mallet, J., and M. Singer. 1987. Individual selection, kin selection, and the shifting balance in the evolution of warning colors: the evidence from butterflies. Biological Journal of the Linnean Society 32:337–350.

Mappes, J., and R. V. Alatalo. 1997. Effects of novelty and gregariousness in survival of aposematic prey. Behavioral Ecology 8:174–177.

Marples, N. M., and P. M. Brakefield. 1995. Genetic variation for the rate of recruitment of novel insect prey into the diet of a bird. Biological Journal of the Linnean Society 55:17–27.

Marples, N. M., and D. J. Kelly. 1999. Neophobia and dietary conservatism: two distinct processes? Evolutionary Ecology 13:641–653.

Marples, N. M., W. Van Veelen, and P. M. Brakefield. 1994. The relative importance of colour, taste and smell in the protection of an aposematic insect, *Coccinella septempunctata.* Animal Behaviour 48:967–974.

Marples, N. M., T. J. Roper, and D. G. C. Harper. 1998. Response of wild birds to novel prey: evidence of dietary conservatism. Oikos 83:161–165.

Merilaita, S., J. Tuomi, and V. Jormalainen. 1999. Optimization of cryptic coloration in heterogeneous habitats. Biological Journal of the Linnean Society 67:151–161.

Merilaita, S., A. Lyytinen, and J. Mappes. 2001. Selection for cryptic coloration in a visually heterogeneous habitat. Proceedings of the Royal Society of London B 268: 1925–1928.

Moore, B. P., W. V. Brown, and M. Rothschild. 1990. Methylalkylpyrazines in aposematic insects, their host plants and mimics. Chemoecology 1:43–51.

Motychak, J. E., E. D. Brodie, Jr., and E. D. Brodie III. 1999. Evolutionary response of predators to dangerous prey: preadaptation and the evolution of tetrodotoxin resistance in garter snakes. Evolution 53:1528–1535.

Nahrstedt, A., and R. H. Davis. 1985. Biosynthesis and quantitative relationships of the cyanogenic glucosides, linamarin and lotaustralin, in genera of the Heliconiini (Insecta: Lepidoptera). Comparative Biochemistry and Physiology B 82:745–749.

Riipi, M., R. V. Alatalo, L. Lindström, and J. Mappes. 2001. Multiple benefits of gregariousness cover detectability costs in aposematic aggregations. Nature 413:512–514.

Roper, T. J. 1990. Responses of domestic chicks to artificially coloured insect prey: effects of previous experience and background colour. Animal Behaviour 39:466–473.

Roper, T. J., and N. M. Marples. 1997. Odour and colour as cues for taste-avoidance learning in domestic chicks. Animal Behaviour 53:1241–1250.

Roper, T. J., and S. Redston. 1987. Conspicuousness of

distasteful prey affects the strength and durability of one-trial avoidance learning. Animal Behaviour 35:739–747.

Roper, T. J., and R. Wistow. 1986. Aposematic coloration and avoidance learning in chicks. Quarterly Journal of Experimental Psychology 38:141–149.

Rothschild, M., B. P. Moore, and W. V. Brown. 1984. Pyrazines as warning odour components in the monarch butterfly, *Danaus plexippus* and in moths of the genera *Zygaena* and *Amata* (Lepidoptera). Biological Journal of the Linnean Society 23:375–380.

Rubinoff, I., and C. Kropach. 1970. Differential reactions of Atlantic and Pacific predators to sea snakes. Nature 228:1288–1290.

Schappert, P. J., and J. S. Shore. 1999. Effects of cyanogenesis polymorphism in *Turnera ulmifolia* on *Euptoieta hegesia* and potential *Anolis* predators. Journal of Chemical Ecology 25:1455–1460.

Schodde, R., and S. C. Tidemann. 1990. Reader's Digest complete book of Australian birds. Reader's Digest, Sydney.

Schuler, W., and E. Hesse. 1985. On the function of warning coloration: a black and yellow pattern inhibits prey-attack by naive domestic chicks. Behavioral Ecology and Sociobiology 16:249–255.

Servedio, M. R. 2000. The effects of predator learning, forgetting, and recognition errors on the evolution of warning coloration. Evolution 54:751–763.

Sherratt, T. N., and C. D. Beatty. 2003. The evolution of warning signals as reliable indicators of prey defense. American Naturalist 162:377–389.

Sherratt, T. N., and I. F. Harvey. 1993. Frequency-dependent food selection by arthropods: a review. Biological Journal of the Linnean Society 48:167–186.

Sherratt, T. N., and A. D. MacDougall. 1995. Some population consequences of variation in preference among individual predators. Biological Journal of the Linnean Society 55:93–107.

Sillén-Tullberg, B. 1985. Higher survival of an aposematic than of a cryptic form of a distasteful bug. Oecologia (Berlin) 67:411–415.

Smith, S. M. 1975. Innate recognition of coral snake pattern by a possible avian predator. Science 187:759–760.

———. 1977. Coral-snake pattern recognition and stimulus generalisation by naive great kiskadees (Aves: Tyrannidae). Nature 265:535–536.

Speed, M. P. 2000. Warning signals, receiver psychology and predator memory. Animal Behaviour 60:269–278.

———. 2001. Can receiver psychology explain the evolution of aposematism? Animal Behaviour 61:205–216.

Summers, K., and M. E. Clough. 2001. The evolution of coloration and toxicity in the poison frog family (Dendrobatidae). Proceedings of the National Academy of Sciences of the USA 98:6227–6232.

Sword, G. A. 2002. A rule for phenotypic plasticity in the evolution of aposematism. Proceedings of the Royal Society of London B 269:1639–1644.

Sword, G. A., S. J. Simpson, O. T. M. El Hadi, and H. Wilps. 2000. Density-dependent aposematism in the desert locust. Proceedings of the Royal Society of London B 267:63–68.

Terhune, E. C. 1977. Components of a visual stimulus used by scrub jays to discriminate a Batesian model. American Naturalist 111:435–451.

Terrick, T. D., R. L. Mumme, and G. D. Burghardt. 1995. Aposematic coloration enhances chemosensory recognition of noxious prey in the garter snake *Thamnophis radix*. Animal Behaviour 49:857–866.

Thomas, R. J., N. M. Marples, I. C. Cuthill, M. Takahashi, and E. A. Gibson. 2003. Dietary conservatism may facilitate the initial evolution of aposematism. Oikos 101: 458–466.

Thompson, V. 1984. Polymorphism under apostatic and aposematic selection. Heredity 53:677–686.

Tullrot, A. 1998. Evolution of warning coloration in the nudibranch *Polycera quadrilineata*. Ph.D. thesis. Göteborg University, Göteborg.

Tullrot, A., and P. Sundberg. 1991. The conspicuous nudibranch *Polycera quadrilineata*: aposematic coloration and individual selection. Animal Behaviour 41:175–176.

Waldbauer, G. P., and W. E. Laberge. 1985. Phenological relationships of wasps, bumblebees, their mimics and insectivorous birds in northern Michigan, USA. Ecological Entomology 10:99–110.

Wright, S. 1969. Evolution and the genetics of populations. Vol. 2. The theory of gene frequencies. University of Chicago Press, Chicago.

Yosef, R., and D. W. Whitman. 1992. Predator exaptations and defensive adaptations in evolutionary balance: no defense is perfect. Evolutionary Ecology 6:527–536.

Associate Editor: Sergey Gavrilets