Annu. Rev. Ecol. Syst. 1999. 30:201–33

EVOLUTION OF DIVERSITY IN WARNING COLOR AND MIMICRY: Polymorphisms, Shifting Balance, and Speciation

James Mallet[1] and Mathieu Joron[2]
[1]*Galton Laboratory, 4 Stephenson Way, London NW1 2HE, England; e-mail: http://abacus.gene.ucl.ac.uk/jim/; and* [2]*Génétique et Environnement, CC065 ISEM, Université de Montpellier 2, Place Bataillon, F-34095 Montpellier, cedex 5, France*

Key Words aposematism, Batesian mimicry, Müllerian mimicry, defensive coloration, predator behavior

■ **Abstract** Mimicry and warning color are highly paradoxical adaptations. Color patterns in both Müllerian and Batesian mimicry are often determined by relatively few pattern-regulating loci with major effects. Many of these loci are "supergenes," consisting of multiple, tightly linked epistatic elements. On the one hand, strong purifying selection on these genes must explain accurate resemblance (a reduction of morphological diversity between species), as well as monomorphic color patterns within species. On the other hand, mimicry has diversified at every taxonomic level; warning color has evolved from cryptic patterns, and there are mimetic polymorphisms within species, multiple color patterns in different geographic races of the same species, mimetic differences between sister species, and multiple mimicry rings within local communities. These contrasting patterns can be explained, in part, by the shape of a "number-dependent" selection function first modeled by Fritz Müller in 1879: Purifying selection against any warning-colored morph is very strong when that morph is rare, but becomes weak in a broad basin of intermediate frequencies, allowing opportunities for polymorphisms and genetic drift. This Müllerian explanation, however, makes unstated assumptions about predator learning and forgetting which have recently been challenged. Today's "receiver psychology" models predict that classical Müllerian mimicry could be much rarer than believed previously, and that "quasi-Batesian mimicry," a new type of mimicry intermediate between Müllerian and Batesian, could be common. However, the new receiver psychology theory is untested, and indeed it seems to us unlikely; alternative assumptions could easily lead to a more traditional Müllerian/Batesian mimicry divide.

0066-4162/99/1120-0201$08.00

INTRODUCTION

Since their discovery, antipredator mimicry and warning colors have been used as simple and visually appealing examples of natural selection in action. This simplicity is beguiling, and controversy has often raged behind the textbook examples. Warning color and mimicry have been discussed from three different points of view: a traditional insect *natural history* angle, which makes simplistic assumptions about both predator behavior and prey evolution (6, 103, 122, 175); an *evolutionary dynamics* angle, which virtually ignores predator behavior and individual prey/predator interactions (37, 47, 54, 91, 162); and a predator *behavior* (or "*receiver psychology*") angle (59–61, 71, 72, 84a, 115, 147a–150), which tends to be simplistic about evolutionary dynamics. Assumptions are necessary to analyze any mathematical problem, but the sensitivity of mimicry to these different simplifications remains untested.

We believe it will be necessary to combine these disparate views (for example, 111, 133, 181) in order to resolve controversy and explain paradoxical empirical observations about the evolution of mimicry. Mimicry should progressively reduce numbers of color patterns, but the actual situation is in stark contrast: There is a diversity of "mimicry rings" (a mimicry ring is a group of species with a common mimetic pattern) within any single locality; closely related species and even adjacent geographic races often differ in mimetic or warning color pattern; and there are locally stable polymorphisms. The current controversies and problems are not simply niggles with the theory of mimicry, designed to renew flagging interest in a largely solved area of evolutionary enquiry. Recent challenges and critiques cast justifiable doubt on previously unstated assumptions. A further reason for reexamining the evolution of mimicry is that its frequency-dependent selective landscapes are rugged, as in mate choice and hybrid inviability (53), so that mimicry provides a model system for the shifting balance theory (41, 178–180); mimicry may also act as a barrier to isolate species (96). While a number of interesting peculiarities of mimicry arguably have little general importance (57), mimicry excels in providing an intuitively understandable example of multiple stable equilibria and transitions between them (41, 87, 89, 97, 98, 161, 167). Mimicry and warning color are highly variable both geographically within species and also between sister species. In this, they are similar to other visual traits involved in signaling and speciation, such as sexually selected plumage morphology and color in birds (2). Sexual and mimetic coloration may therefore share some explanations.

This article covers only the evolution of diversity in mimicry systems, and we skate quickly over many issues reviewed elsewhere (19–21, 32, 40, 45, 86, 123, 125, 129, 134, 170, 176; see also a list of over 600 references in Reference 90). Our discussion mainly concerns antipredator visual mimicry, though it may apply to other kinds of mimicry and aposematism, for example, warning smells (62, 130) and mimicry of behavioral pattern (18, 152–154). In addition, most of our examples are shamelessly taken from among the insect mimics and their models, usually butterflies, that we know best; careful studies on the genetics or ecology of other systems have rarely been done.

MIMICRY AND WARNING COLOR: THE BASICS

Bates (6) noticed two curious features among a large complex of butterflies of the Amazon. First, color patterns of unrelated species were often closely similar locally; second, these "mimetic" patterns changed radically every few hundred miles, "as if by the touch of an enchanter's wand" (8). Bates argued that very abundant slow-flying Ithomiinae (related to monarch butterflies) were distasteful to predators and that palatable species, particularly dismorphiine pierids (related to cabbage whites), "mimicked" them; that is, natural selection had caused the pattern of the "mimic" to converge on that of the "model" species. This form of mimicry became known as Batesian (122). The term "mimicry" had already been used somewhat vaguely by pre-Darwinian natural philosophers for a variety of analogical resemblances (13), but Bates's discovery was undoubtedly a triumph of evolutionary thinking.

Bates also noticed that rare unpalatable species such as *Heliconius* (Heliconiinae) and *Napeogenes* (Ithomiinae) often mimicked the same common ithomiine models (such as *Melinaea, Oleria*, and *Ithomia*) copied by dismorphiines. He assumed that this "mimetic resemblance was intended" (6, p. 554) because, regardless of its palatability, a rare species should benefit from similarity to a model. However, where both mimic and model were common, as in the similarity of unpalatable *Lycorea* (Danainae) to ithomiines, he felt this was "a curious result of [adaptation to] local [environmental] conditions" (6, p. 517); in other words, convergent evolution unrelated to predation. It was left to Müller (103) to explain clearly the benefits of mimicry in pairs of unpalatable species. If a constant number of unpalatable individuals per unit time must be sacrificed to teach local predators a given color pattern, the fraction dying in each species will be reduced if they share a color pattern, leading to an advantage to mimicry. Thus, mimicry between unpalatable species became known as Müllerian.

Many mimetic species are also warning-colored, but some are not: for example, the larva of a notodontid moth (6) and the pupa of *Dynastor darius* (Brassolidae) (1) both mimic highly poisonous but cryptic pit vipers (Viperidae). The former mimics even the keeled scales of its model; the latter has eyes that mimic the snake's own eyes, even down to the slit-shaped pupils. Many small clearwing ithomiines that Bates studied in the tropical rainforest understory are also very inconspicuous but are clearly mimetic. Mimicry does not require a warning-colored model, only that potential predators develop aversions to the model's appearance. Warning color, or "aposematism" (122), was first developed as an evolutionary hypothesis by Wallace in response to a query from Darwin, four years after Bates' publication on mimicry. Darwin's sexual selection theory (42) explained much bright coloration in animals but could not explain conspicuous black, yellow, and red sphingid caterpillars found by Bates in Brazil because the adult moth could not choose mates on the basis of larval colors. Wallace in 1866 (see 42, 175) suggested that bright colors advertised the unpalatability of the larvae, in the same way that yellow and black banding advertised the defensive sting of a hornet (Vespidae). Warning color in effect must increase the efficiency with which predators learn to avoid

unpalatable prey (see also 59, 61 for excellent discussion of possible advantages of warning color).

NUMBER-DEPENDENT SELECTION ON MIMICRY AND WARNING COLOR

To the natural history viewpoint of early Darwinians (6, 8, 103, 122, 175), it was apparently not clear that explaining aposematism and mimicry as adaptations could be problematic: They had not fully realized that short-term individual benefits and long-term group benefits may conflict. In fact, the selective landscape of mimetic evolution has multiple stability peaks that should often prevent the spread of ultimately beneficial unpalatability, warning color, and some mimicry. To understand why this is so, we must examine the evolutionary dynamics of mimicry.

Müller (103) was the first to formulate the benefits of mimicry explicitly, using mathematical intuition from a natural history perspective (reprinted in 78). He assumed that, while learning to avoid the color pattern of unpalatable species, a predator complex killed a fixed number of individuals per unit time (n_k). Müllerian mimicry is favored, therefore, because the per capita mortality rate decreases when another unpalatable species shares the same pattern. If this traditional naturalist's "number-dependent" (162) view of mimicry is correct, it leads to two interesting predictions, only the first of which Müller himself apparently appreciated. First, although Müllerian mimicry of this kind should always be mutualistic, a rare species ultimately gains far more from mimicry than a common one, in proportion to the square of the ratio of abundances (103). Second, a novel mimetic variant in the rarer species resembling the commoner is always favored because the common species generates greater numerical protection, while a mimetic variant of the commoner species is always disfavored because it loses the strong protection of its own kind and gains only weak protection from the rarer pattern (161). Both these effects will tend to cause rarer unpalatable species to mimic commoner models, rather than the other way around, in spite of the fact that Müllerian mimicry is a mutualism (albeit with unequal benefits) once attained.

Müller's number-dependent selection applies similarly to morphs within a single species (Figure 1). A warning-colored variant within a cryptic but unpalatable prey will suffer a twofold disadvantage: First, it is more conspicuous to predators; second, it does not gain from warning color because predators, not having learned to avoid the pattern, may attack it at higher rate than the cryptic morph. This creates a barrier to initial spread (67), even though, once evolved, warning color is beneficial because by definition it reduces the number of prey eaten during predator learning. In exactly the same way, a novel warning pattern is disfavored within an already warning-colored species, essentially because of intraspecific mimicry (27, 89, 97; also Figure 1). This selection against rarity makes it easy to understand why warning-colored races are normally fixed and sharply separated by narrow overlap zones from other races (27, 50, 87, 93), but in turn makes it hard to understand how geographic races diversified in the first place (6, 8, 87, 89, 98, 137, 167). Sim-

ilarly, if energy is required to synthesize or sequester distasteful compounds, unpalatability itself may be disfavored (49, 63, 67, 160) because unpalatable individuals may sacrifice their lives in teaching predators to avoid other members of their species. Hypotheses to overcome the difficulties with this new, more sophisticated evolutionary dynamic view of aposematism are detailed below.

POPULATION STRUCTURE AND THE EVOLUTION OF WARNING COLOR AND UNPALATABILITY

The Evolution of Unpalatability

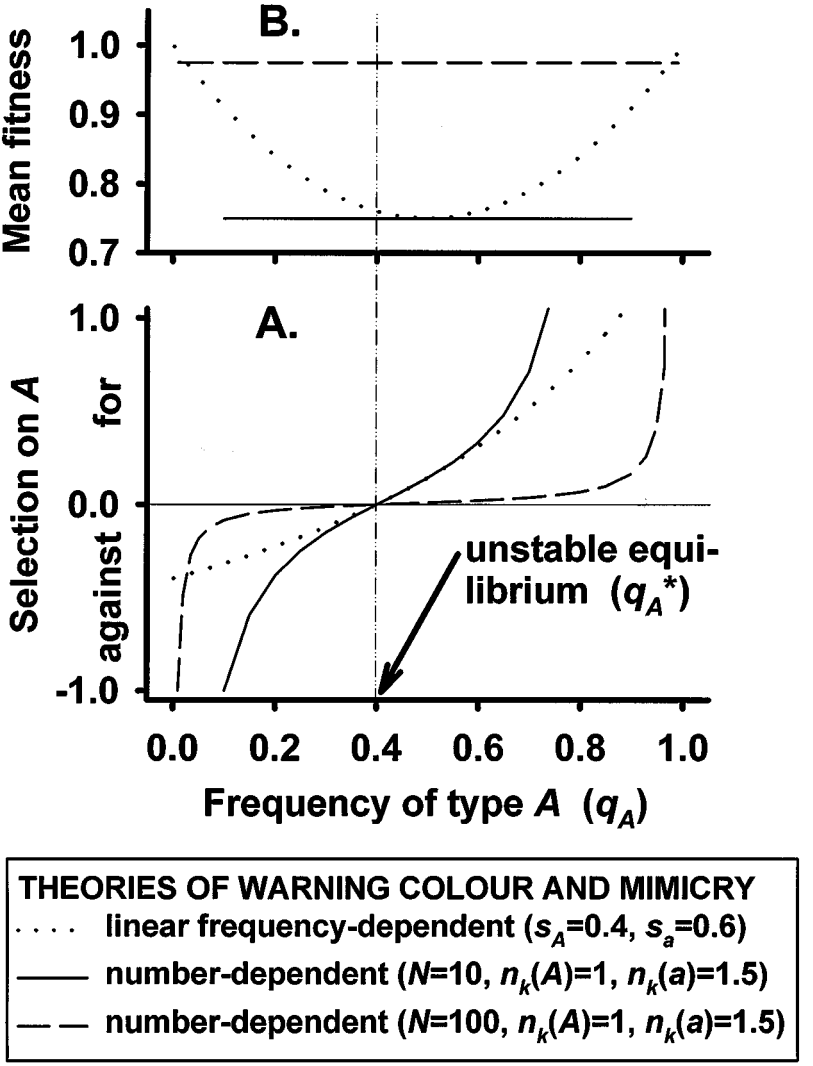
Unpalatability itself is hard to define (20, 51; see also below under *Müllerian Mimicry*), but here we use the term loosely to mean any trait that acts on predators as a punishment, and that causes learning leading to a reduction in attacks. The unpalatable individual may incur costs in synthesis or processing of distasteful chemistry and is often likely to suffer damage during predator sampling, while other members of the population mostly benefit from predator learning. The frequent aposematism of gregarious larvae, often siblings from the same brood, suggests that benefits are shared among kin, and that kin selection could have been responsible for the evolution of unpalatability (49, 63, 67, 160). These authors assumed that altruistic unpalatability was unlikely to evolve unless kin-groups already existed, so explaining the association between gregarious larvae and unpalatability. However, unpalatability may not be very costly. First, although it may be expensive to process distasteful secondary compounds, in some cases the same biochemical machinery is required to exploit available food; for instance, *Zygaena* and *Heliconius*, which both feed on cyanogenic host plants, can also synthesize their own cyanogens (70, 76, 104), presumably using enzyme systems similar to those required in detoxification. Second, because most toxic compounds also taste nasty (arguably, the sense of taste has evolved to protect eaters from toxic chemistry), and because predators taste-test their prey before devouring them, and, finally, because unpalatable insects are often tough and resilient, an unpalatable insect should often gain an individual advantage by sequestering distasteful chemicals. A good example of predator behavior showing this is possible is seen in birds feeding on monarch (*Danaus plexippus*) aggregations at their overwintering sites in Mexico: Birds repeatedly taste-reject butterflies more or less unharmed, until they find a palatable individual, which is then killed and eaten (29).

Another problem with empirical evidence for kin selection is that gregariousness, which reduces per capita detectability of the prey (14, 64, 157, 160), is expected to evolve when there is any tendency toward predator satiation; and one of the best ways of satiating predators is to be distasteful. Thus the association between gregarious larvae and unpalatability can be explained easily because gregariousness will evolve more readily after unpalatability, rather than before it as required under the kin selection hypothesis. This expected pattern of unpalatability first, gregariousness thereafter, is now well supported in Lepidoptera by phylogenetic

analysis (139, 141). In conclusion, the supposed necessity for kin selection in the evolution of unpalatability is now generally disbelieved (59, 97, 139), although kin selection could, of course, help.

Evolution of Novel Warning Colors in Cryptic and Aposematic Defended Prey

Although the realization that aposematic insects may be altruistic came 70 years ago (49), it was finally some 50 years later that the evolution of warning color was explicitly disentangled from the evolution of unpalatability (66, 67). Under



Müller's number-dependent theory, intraspecific Müllerian mimicry acting on a novel warning-colored variant A within a population would strongly favor the commonest wild-type morph *a*; the number-dependence gives rise to frequency-dependent selection, which is purifying, tending to prevent polymorphism (Figure 1*A*). If unpalatable prey often survive attacks, it might be argued that the problem will be surmounted (47, 73, 74, 177). However, fitness will be reduced if attacks are even potentially damaging; the "effective number killed" (n_k) may take fractional or probabilistic values, but the frequency-dependent logic applies in exactly the same way (68, 97).

A critical feature of number-dependence is the great nonlinearity of frequency-dependent selection. Many authors from the evolutionary dynamics tradition have assumed a simpler linear frequency-dependence (dotted line in Figure 1) (47, 54, 87, 91, 121). In fact, the relationship between selection and frequency becomes more sigmoidal as n_k/N decreases. When $n_k \ll N$, there are strong spikes of selection against *A* and *a* when each is rare, but much of the frequency range forms a nearly neutral polymorphic basin (e.g. $N = 100$, Figure 1). Another interesting feature of this model is that the mean fitness surface is flat: Assuming most predators learn the pattern and then avoid it, the mean fitness throughout the frequency spectrum becomes approximately constant at 1- $\{[n_k(A)+n_k(a)]/N\}$ (Figure 1*B*). This is an extreme example of how mean fitness cannot be guaranteed to increase when selection is frequency-dependent (65). [Mimetic and warning color patterns may, of course, vary continuously, rather than as discrete patterns (82); this may also contribute to evolution of warning color and mimicry (see under *Pattern En-*

←

Figure 1 Number- and frequency-dependence in mimicry and aposematism. Müller's number-dependent theory supposes that, while they learn to avoid the pattern of an unpalatable insect, predators kill a constant number, $n_k(i)$ of each morph *i* per unit time in a given area. Assuming the local population has constant size (N) and contains a novel pattern (A) and a wild-type pattern (a), Müller's theory can give the strength of frequency-dependent selection for or against the pattern *A* at different frequencies (q_A) in the population. The fitness of *A* is $W_A = 1 - (n_k(A)/q_A N)$, while that of *a* is $W_a = 1 - [n_k(a)/(1 - q_A)N]$. The measure of frequency-dependent selection acting on *A* relative to *a* used here is $S_A = (W_A/W_a) - 1$; if S_A is positive, *A* is favored, if S_A is negative, *A* is disfavored. The fitnesses are shown in terms of S_A (graph A) and mean fitness (graph B). The dashed and solid lines show frequency-dependent fitnesses for a low total population size ($N = 10$) and a high total population size ($N = 100$), respectively, relative to $n_k(A)$ and $n_k(a)$ (the fractions n_k/N are more important than absolute values of n_k and N). In contrast, linear frequency-dependent selection has been more normally used to study the population genetics of warning color and mimicry (37, 47, 54, 91, 93), for example where $W_A = 1 - s_A(1 - q_A)$, and $W_a = 1 - s_a q_A$. This model gives frequency-dependent fitnesses shown in the dotted curve of the figure. In both number-dependent and frequency-dependent selection, values of *s* and n_k have been chosen to give an unstable equilibrium frequency of $q_A^* = 0.4$, which is the case if *A* has 1.5× greater fitness than *a*.

hancement and Peak-Shift and *Mimetic Polymorphism and Genetic Architecture*, below).]

In nature, not only do warning colors exist, but also novel warning patterns are forever being multiplied in already warning-colored species (see also *Genetic Drift and the Shifting Balance*) in spite of barriers suspected to impede their initial evolution. Various ideas have been proposed:

1. *Novelty and Recognizability* It has been suggested that warning colors are favored because they induce predator neophobia and because they are easier to recognize and learn (59, 84a, 140, 177). Neophobia has some experimental evidence (45, 140), whereas increased memorability is part of the definition of warning color (see above). These factors, coupled with a high survival rate of attacked prey, might seem to allow warning color to increase from low frequency in spite of increased conspicuousness (140, 177). Unfortunately, the problem with fear of novelty is that this survival advantage evaporates after a time, and enhanced learning is useful only if there are enough individuals available to do the teaching. This behavior viewpoint is rarely coupled with much thought about evolutionary dynamics. Thus, an unpalatable and brightly colored sea slug that survives 100% of attacks by fish (158) seems likely to have some risk, or loss of fitness due to fish biting; any such loss of fitness will be progressively diluted as the numbers increase, leading again to frequency-dependent selection against rare morphs. In fact, Müllerian mimicry or monomorphic warning color would be unnecessary if this selection against rarity were not present. An increase of conspicuousness will almost always lead to an initially greater level of attack on the first few individuals with the new pattern, even if the pattern is ultimately advantageous once fixed within the population (59, 81, 97). Essentially, $n_k(A)/1 \leq n_k(a)/N$ for warning color A to spread in a population of size N—the learning advantage of first individual A variant must outweigh the population size advantage of the cryptic wild-type a. With reasonably large prey population sizes, say $N > 10$, for a reasonably unpalatable species, this seems almost impossible; given that A is more conspicuous, the possibility seems even more remote (89, 97). In any case, high rates of beak-marks on the wings of brightly colored unpalatable butterflies attest to a high frequency of potentially lethal attacks (11, 30, 31, 91, 111). Nonetheless, various possibilities allow warning colors to cheat against this apparent selective disadvantage. These are reviewed below.

2. *Preadaptation* This idea is motivated by the fact that many palatable insects, particularly butterflies, are already brightly colored. Cryptic resting postures and rapid, jinking flight allow these insects to expose conspicuous patterns in flight that may be important for intraspecific signaling in mate choice and sexual selection (42) or in territoriality and male-male interactions (138, 169), as deflection markings (45, 128, 176), or in Batesian mimicry. If these species become unpalatable, perhaps as a result of a need to process toxic secondary compounds in food, their conspicuous patterns, already adapted for signaling, could simply be reused in predator education.

3. *Pattern Enhancement and Peak Shift* The representation of a pattern in a predator's memory is likely to be a caricature of the actual pattern. Thus, an exag-

gerated pattern may be avoided by a predator more strongly than the normal pattern on which the predator originally trained, and exaggerated warning patterns will evolve to exploit this predator bias. Training an artificial neural network model can also recreate this kind of perceptual bias for supernormal stimuli (3, 48). Whether perceptual bias is produced in computer models is strongly assumption-dependent (79), but there is good evidence for exaggerated responses to supernormal stimuli in vertebrate perception (156), which seem likely to have been a cause of exaggerated male traits in sexual selection (110, 131). Similar perceptual biases in vertebrates may contribute to the gradual evolution of warning colors (82).

A related idea is "peak shift" whereby, if zones of negative and positive reinforcement are located close together along a perceptual dimension, they may each cause the perceiver to bias their responses further apart (Figure 2). Peak shift is not dissimilar to the old idea that warning colors function by appearing as different as possible from the color patterns of edible prey (49, 59–61, 164). Theory shows that peak shift can produce gradual evolution of warning colors (133, 181), and recent experiments with birds have demonstrated relevant perceptual bias (52, 84).

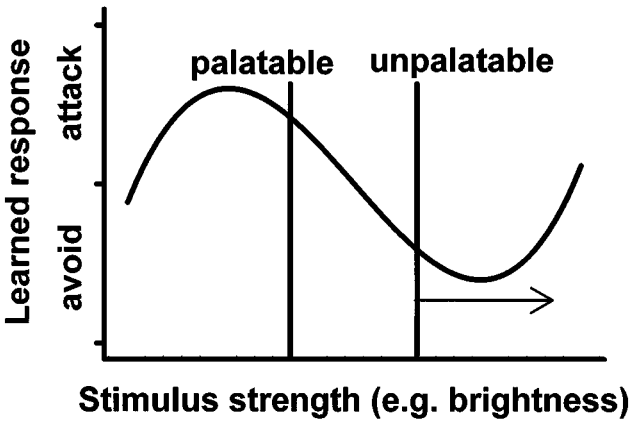


Figure 2 The theory of behavioral "peak shift." If the appearances of palatable and unpalatable species are close to each other along some stimulus dimension, such as conspicuousness, predators may develop a perceptual bias that enhances discrimination, known by behaviorial biologists as peak shift (not to be confused with evolutionary peak shift via the shifting balance). The conflicting pressures on their perceptual/learning system may lead them to avoid patterns brighter than the norm for the unpalatable species more strongly than they avoid the normal pattern; conspicuous unpalatable variants would then have an advantage over the normal pattern, allowing gradual evolution of greater and greater conspicuousness of the unpalatable species (arrow) (61). It is unclear how the palatable species will evolve; it could be selected for mimicry (to the right), or to greater inconspicuousness (to the left) to avoid detection, even though the latter may be costly due to increased predator attack rate once detected.

It seems likely that at least some warning colors evolved by pattern enhancement. For example, the patterns of conspicuous morphine butterflies *Taenaris* and *Hyantis* are clearly related to those of cryptic morphines and satyrines, such as *Morphopsis* with deflective eyespot patterns similar to many other edible members of the satyrid lineages to which they belong. *Taenaris* and *Hyantis* have evolved unpalatability, perhaps as a result of feeding on toxic Cycadaceae, both as larvae on leaves and as adults on sap and fruits. Compared with the drab *Morphopsis*, color and brightness have been enhanced, eyespot size has been increased, and eyespot number has been reduced. A variety of Batesian mimics from palatable genera such as *Elymnias agondas* (Satyrinae) and females of *Papilio aegeus* (Papilioninae) mimic *Taenaris* and *Hyantis* patterns (117, 118), showing that the latter are unpalatable. Although likely to explain some warning color evolution, it is hard to imagine that all novel warning patterns evolved by enhancement. The color patterns of related species, or even races of *Heliconius* (24, 137, 159), for example, seem so radically divergent as to preclude one being an enhancement of the other. Of course, this is a dubious anthropocentric argument, but the major gene switches in *Heliconius* suggest that radical shifts, rather than gradual enhancement of existing patterns, are responsible for much of the pattern diversity within already warning-colored lineages. If this is the case for switches between warning patterns, then the ubiquity of enhancement and predator perceptual bias, even for the initial switch, seems in doubt.

4. *Müllerian Mimicry* Another way that a newly unpalatable species might become warning-colored is via *Müllerian mimicry*. The constraints on its evolution discussed on p. 204, apply, but the widespread existence of Müllerian mimicry suggests that the idea should work both in the initial evolution of warning color and in its diversification within already unpalatable lineages. Because many species typically join in Müllerian mimicry rings (9, 10, 22–25, 116), it seems likely that, in butterflies, most warning color switches are due to Müllerian mimicry. Only the initial divergence of mimicry rings needs to be explained in some other way (24, 89, 98, 167).

5. *Density- and Apparency-dependent Warning Color* Our formulation so far of number-dependent warning color and Müllerian mimicry (see Figure 1) assumes all individuals are seen by predators, but in fact apparency as well as density per se are important for the ultimate benefits of warning colors. If more prey are killed while predators learn of a warning pattern than would be detected and killed for a cryptic population of the same size, it may pay the prey to remain cryptic. This may explain why many stationary pupae of unpalatable insects, such as *Heliconius*, are brown and resemble dead leaves, while their more apparent and mobile larvae and adults are brightly colored and classically aposematic. Density-dependent color pattern development in *Schistocerca* (desert locusts and their relatives) shows a switch from crypsis at low density to advertisement of food-induced unpalatability at high density, and predation experiments with *Anolis* lizards support this idea (155). If so, pattern enhancement (see point 3 above) of characteristics used by predators for recognition may provide a way in which this

kind of context-dependent warning color evolves (133, 155, 181). Nonetheless, density-dependent facultative warning colors are unlikely in most animals, such as adult butterflies, in which color patterns are largely genetic.

6. *Kin Selection, Kin-founding, and "Green-beard" Selection* Predators attacking kin groups can kill or damage some individuals, but, after doing so, avoid others, who are relatives carrying the same pattern. A superior warning pattern may therefore increase locally under a kind of kin group selection (67). This is somewhat different to classical kin selection because benefits are transferred between individuals of like phenotype, rather than according to degree of relationship (58): The effect has therefore been called family selection (66), or kin-founding (97). Warning color is a concrete and uncheatable green-beard trait (58, 59, 166), a hypothetical type of altruism invented by Dawkins (43), whereby altruists carrying a badge (such as a green beard) recognize other altruists because they also carry the badge. More recently, the general term synergistic selection (61, 81, 82, 99, 139) has been applied to such traits. The synergism can be viewed as a behavioral explanation of the warning color trait, once evolved, but the nature of synergism does not explain its initial evolution because both the fixed absence of the trait and the fixed presence of the trait are evolutionarily stable strategies (99). The population genetic problem of frequency-dependence shown in Figure 1 still arises, and it seems clear that kin-founding could aid the initial increase of novel warning colors (59, 97). Whether kin-founding is important for the initial or subsequent evolution of novel warning colors seems hard to decide (see also under *Genetic Drift and the Shifting Balance*). However, kin-grouping and larval gregariousness in many unpalatable insects does not seem such good evidence now as formerly for kin-founding, for reasons already discussed above under *The Evolution of Unpalatablity*: in most cases, gregariousness seems to have evolved after unpalatability and aposematism (139, 141).

7. *Genetic Drift and the Shifting Balance* Although kin-founding can be looked upon as a purely deterministic model similar to kin-selection (66), it is clear that, like Sewall Wright's "shifting balance" model of evolution (178–180), it requires a small local population size: The phenotypes of a small group of related individuals must dominate the learning and recognition systems of local predators, which is only possible if the total local population is low. The evolution of warning color via kin-founding is in fact a special case of phases I and II of the shifting balance (89, 97, 98, 167). In phase I, genetic drift allows a local population to explore a new adaptive peak; in phase II, local selection causes the population to adapt fully to the new adaptive peak. Although not usually treated in kin-founding models (but see 66), phase III of the shifting balance, i.e. spread of the new adaptive peak to other populations, would clearly be an important final phase in the kin-founding process. This would be equivalent to having local populations with different warning colors competing across narrow bands of polymorphism, as is actually the case in many hybrid zones between geographic races of warning-colored species today; movement of these clines for warning color would be the equivalent of Phase III (87, 98). In warning color, stable and unstable equilibria are peaks

and troughs of relative fitness, but not necessarily of mean fitness. Under purely number-dependent selection (Figure 1), mean fitness is a constant independent of frequency, 1-{[$n_k(A)+n_k(a)$]/N}, and even under linear frequency-dependence, the minimum of mean fitness (at $q_A = 0.5$) is not at the unstable equilibrium ($q_A^* = 0.4$ in Figure 1). If A is more memorable than a, then $n_k(A) < n_k(a)$, but this does not increase the mean fitness when q_A is high, except very close to fixation of A when hardly any a are available to be tasted by predators.

A recent critique of the shifting balance model concluded that chromosomal evolution, warning color evolution, and more general patterns of phenotypic adaptation were almost always better explained by ordinary individual selection (41). For warning color and mimicry, the key problems are that natural selection seems too intense so that drift is unlikely, and, in common with other examples of rugged adaptive surfaces, phase III of the shifting balance seems an inefficient means of spreading better warning patterns. While these problems seem serious, key features of warning color considerably increase the chances of shifting balance occurring. First, although selection for warning color can often be extremely strong, it would be surprising if predator attacks were not sometimes reduced or suspended locally, due to temporary absence of key predators such as flycatchers or jacamars (34, 35, 119, 120). If so, populations can occasionally drift to become polymorphic because of a relaxation of selection. Provided that the prey are abundant compared with their predator, (i.e. $n_k \ll N$), the populations will quickly enter the central basin where selection is weak (e.g. for $N = 100$ in Figure 1). Here drift or mild forms of selection other than that due to warning function may cause a new pattern to rise in frequency above the unstable equilibrium (phase I), whereupon warning selection can fix and refine the new pattern (phase II). An interface between new and old patterns will form, resulting in a cline similar to hybrid zones between races observed today. If one pattern is superior at warning away predators, asymmetries of selection will drive it into the range of the other behind a narrow moving cline, as in phase III (87, 89, 98). Cline movement seems likely; with strong selection in the clines observed in nature (93), fairly rapid movement is predicted (87, 89, 91). The shifting balance proposal is speculative because we know little about the frequency, timing, and depth of episodes of selection relaxation required for phase I, the relative advantages of different warning colors across clinal boundaries required for phase III, and whether population structural constraints will prevent cline movement (4, 69, 89, 98). However, empirical evidence for all phases suggests the shifting balance is likely: (*a*) Polymorphism seems to exist regularly among Müllerian mimics (see below under *Müllerian Mimicry, Polymorphism and the Palatability Spectrum*), showing that although mimicry is sometimes strongly selected (11, 75, 80, 92), at other times, a combination of reduced selection, genetic drift, and nonmimetic selection causes polymorphism in the central basin, and therefore that events triggering phase I seem actually to occur; (*b*) the strong purifying selection that is the problem for phase I promotes phase II; and (*c*) the existence of today's narrow clines and biogeographic evidence for past cline movement and movement in historical times suggest that phase III occurs regularly.

The current disjunct distribution of genetically homologous "postman" patterns of *Heliconius erato* and its Müllerian co-mimic *Heliconius melpomene* in peripheral populations strongly suggests that some such competitive cline movement in favor of central Amazonian "dennis-ray" patterns of this nature has occurred, even if the color patterns have been sometimes restricted to Pleistocene refuges in the past (24, 27, 89, 98, 137, 164). There is some empirical evidence for movement of *Heliconius* clines this century; although slow on a historical scale, the movement of warning color clines could be very fast relative to an evolutionary time scale (89). (*d*) The shifting balance does seem to have a strong potential in explaining geographic divergence within species, the strong differences in warning color and mimicry between sister species, and also the extraordinary diversity and novelty of these patterns (98). If the shifting balance is important for current diversification, there is little reason to doubt that it could also have been important in the murky initial stages of the origins of warning color in aposematic lineages, though evidence has long since been erased by more recent color pattern evolution.

DIMORPHISM AND POLYMORPHISM IN MIMICRY

Sex-Limited Mimicry

In a minority of Batesian mimetic butterflies, females are mimetic, while males, although brightly colored, are not. Such cases can be explained if males are constrained to be nonmimetic by sexual selection, either via female choice (162, 163) or by the requirements of combat or other male-male signaling (138, 169). This topic has been reviewed excellently elsewhere (164; see also 78), so we do not treat it in detail here.

Sexual selection may explain sexually dimorphic mimicry, but there are some peculiarities of female-limited mimicry for which the answers are not known. First, female-limitation seems restricted to putative Batesian mimicry. As far as is known, Müllerian mimics lack strong sexual dimorphism. Presumably, this is explained because Müllerian mimicry is under purifying density-dependent selection: As a mimetic pattern becomes more common, its advantage increases (Figure 1). In contrast, Batesian mimicry becomes less successful as it becomes commoner; thus, sexual selection is more likely to outweigh this weakening mimetic advantage in Batesian mimics (162). Female-limited mimicry also seems virtually confined to butterflies (46, 168), whereas the sexual selection theory should apply to all examples of Batesian mimicry. Here, the explanation may be ecological. Territorial or fighting males of many butterflies fly purposefully, fast, and can escape predators easily. Female butterflies searching for oviposition sites can be particularly vulnerable to predator attacks (111) because they must at times fly slowly, like potential models; thus ecological considerations may explain why butterfly females, but not males, often mimic slow-flying models (111, 169). Ecological constraints on sexually dimorphic mimicry are well demonstrated by cases in which only the

male is mimetic (168), for example, in saturniid moths with nocturnal females but diurnal males (172).

Mimetic Polymorphism and Genetic Architecture

Batesian mimetic butterflies may be polymorphic as well as sexually dimorphic. This phenomenon is best known and studied genetically among female mimetic forms of Papilionidae, particularly *Papilio dardanus* and *P. memnon*, where each morph mimics a different unpalatable model. The maintenance of this polymorphism is easily explained in common Batesian mimics because frequency-dependent selection favors rare mimics. Polymorphisms in Batesian mimics are also well-known in nonbutterfly groups: Good examples exist in hoverflies (170, 171). However, the rarity of accurate polymorphic mimicry of the kind displayed in *Papilio* suggests that special circumstances must be involved. Mimetic polymorphisms in these cases are usually determined at relatively few genomic regions with large effect ("supergenes"), often with almost complete dominance (38, 39, 134, 135). The maintenance of mimetic polymorphisms probably depends rather strongly on supergene inheritance. Without it, nonadaptive intermediates would be produced.

While it is easy to understand the maintenance of polymorphisms at mimetic supergenes, it is far from clear how these supergenes initially evolved. Early Mendelians used these genetic switches as evidence that mutations of major effect were prime movers of adaptation (123). Fisher argued forcefully that most adaptive evolution could be explained via multiple genetic changes of individually small effect being sorted by natural selection (49). Essentially, Fisher proposed that selection rather than mutation was the creative process in adaptation. Goldschmidt (56) then revived mutationist theory in more sophisticated form and proposed that mimics could exploit major ("systemic") mutations that reused the same developmental machinery originally exploited by the model. He felt it unlikely that the same genes were reused by mimics and models, proposing instead that different genes had access to the same developmental pathways. Gradualists were quick to point out cases in which development of mimicry was clearly analogous rather than homologous, such as colored spots on the head and body of models being mimicked by basal wing patches on mimetic *Papilio memnon* (135). Single gene switches in *P. memnon* were demonstrated to consist of tightly linked multiple genetic elements that could be broken apart by recombination or mutation, and it was suggested by gradualists that these "supergenes" had been gradually constructed by a process of linkage tightening to reduce the breakup of adaptive combinations by recombination (38, 39).

More recently, opinion has swung back (but only part way) toward mutationism. It has been realized that it would be hard to construct supergenes by means of natural selection alone. Separate elements of a supergene must have been tightly linked initially in order that a sufficiently high correlation between favorable traits was available for selection for tighter linkage. Thus polymorphic mimicry must to

some extent have depended on the pre-existence of gene clusters (36, 37). If so, this could explain why Müllerian mimics and models such as *Heliconius* often themselves show major gene inheritance. Müllerian mimics are not expected to have polymorphisms, and usually they do not (but see below under *Müllerian Mimicry*); thus they are not expected to require supergene inheritance of their color patterns under the gradualist hypothesis. *Heliconius* patterns are inherited at multiple loci; this was interpreted as confirming a gradualist expectation for polygenic inheritance of mimicry (113, 137, 164). However, a closer look at *Heliconius* shows that many of the pattern switches are indeed major, have major fitness effects, and can also in some cases be broken down into tightly linked component parts by recombination or mutation (87a, 93, 137), again suggesting mimetic "supergenes." For example, in both Müllerian mimics *H. erato* and *H. melpomene*, a large forewing orange patch known as "dennis," and orange hindwing "ray" patterns are very tightly linked but are separable via recombination or mutation that shows up only in rare individuals from hybrid zones (87a). Probably, mutations with major effect are required even in Müllerian mimicry because, during adaptation, a Müllerian mimic loses its current warning pattern while approaching that of a model. There is thus a phenotypic fitness trough between the old pattern and the new pattern. Only if a mutation produces instant protection by the new pattern can the gene be favored, unless the two patterns are already extremely close. After approximate mimicry has been achieved by mutation, multilocus "modifiers" can improve the resemblance in the normal way (37, 105, 161, 164). This hybrid view of Müllerian mimicry, known as the Nicholson "two-step" theory, combines what is arguably a mutationist argument with a gradualist hypothesis to explain the perfection of resemblances.

This explanation fits major gene adaptations in Müllerian mimicry, especially as it is now realized that Fisher's argument for adaptation via small mutations has serious flaws (112), even without the frequency-dependent stability peaks of mimicry (Figure 1). However, two-step theory cannot explain why genes for forewing and hindwing patterns should be tightly linked in both model and mimic in *Heliconius*. Why should *H. erato* and *H. melpomene* (the former is almost certainly the model driving the divergence—see 55, 89) both diverge geographically using probable supergenes of major genetic effect? One possibility is that genetic architecture for color pattern change in *Heliconius* simply has limited flexibility (87a). We now know that there is widespread reuse of homeotic gene families throughout the animal kingdom, including some involvement in color pattern development in butterflies (16, 33). It would be very surprising if mimicry gene families were not also reused similarly (106–108, 164) in the lineages leading to *H. erato* and *H. melpomene*. This argument is similar to Goldschmidt's (56), but in one sense more extreme, since Goldschmidt thought it likely only that the same patterning control would be reused, rather than the very same genes. Others argue from similar data that the evidence is in favor of analogous rather than homologous developmental pathways and gene action (88), but a true test will be possible only when mimicry genes are characterized at the molecular level in both lineages (51, 95).

In conclusion, current opinion based on nearly a century of genetic studies and mathematical population genetic theory suggests how mimetic as well as other adaptations may often require mutations of major effect, at least initially, both because of the ruggedness of the selective landscape, and probably also because of constraints imposed by pattern genetics. Perfection of these adaptations then involves effects generated at multiple genes of increasingly small effect. The genetic architectures required, especially for polymorphic mimicry, may be rare. This would explain why some lineages involved in mimicry, such as the Papilionidae, are able to colonize multiple mimicry rings and become polymorphic (164), while others are rarely mimetic. Disruptive mimetic selection is perhaps as likely to be an agent causing an alternative, speciation, as it is to be a common cause of polymorphism (see *Mimicry and Speciation*, below).

Müllerian Mimicry, Polymorphism, and the Palatability Spectrum

Müllerian mimicry and warning color are standard textbook examples of frequency-dependent selection within species (e.g. 99, 126). Polymorphisms should be rare due to high rates of attack on rare variants (Figure 1;) (27, 47, 67, 87, 89). In general, workers in the field of mimicry assert that this is so (89, 97, 98, 161, 164, 167), but there are some very embarrassing exceptions to the rule among even the best known Müllerian mimics. The most famous case is *Danaus chrysippus* and its Müllerian mimics *Acraea encedon*, *A. encedana*, together with their Batesian mimic *Hypolimnas misippus*. While distinct color patterns are virtually fixed in the peripheries of their respective ranges, these species are highly polymorphic over an area of Central and Eastern Africa larger than Europe (57, 146). Similarly embarrassing widespread polymorphisms are found in two-spot ladybirds (15, 85) and in *Laparus doris* (Heliconiinae) (151, 159). Arguably, mimicry in many of these cases is weak: Non- or poorly mimetic morphs are common (85, 114, 143, 144, 159). However, there are equally problematic examples in which mimicry is very accurate. For instance, *Heliconius cydno* is mostly monomorphic in Central America (94, 142), but becomes polymorphic throughout much of the Andes of Colombia and Western Ecuador (80, 83); each morph can be clearly identified as an accurate mimic of other *Heliconius*, particularly *H. sapho* and *H. eleuchia*. The pinnacle of Müllerian mimetic polymorphism is found in *Heliconius numata*. This species is polymorphic throughout virtually its whole range, and some populations of the Amazon basin near the slopes of the Eastern Andes may have up to seven different morphs, each an accurate mimic of a separate species of *Melinaea* or *Mechaniti* (Ithomiinae) (23, 26). Three explanations have been proposed, and we here add a further hypothesis that may contribute to the persistence of polymorphisms once they have been established.

1. *Batesian Overload and Coevolutionary Chase* If an unpalatable species has many Batesian mimics, it may suffer from Batesian overload. According to this hypothesis, the deleterious effects of mimics may force the model to diverge from

its normal pattern to escape mimicry, leading to a coevolutionary chase of model by mimic. This idea has generated some controversy (54, 71, 72, 109, 165) but has been well reviewed recently (164, 165), and we merely summarize: It does not seem likely that coevolutionary chase or Batesian overload can explain polymorphisms in unpalatable models. Frequency-dependent purifying selection on the models must almost always be stronger than the diversifying selection due to mimetic load (57, 78, 109, 165).

2. *The Palatability Spectrum* Unpalatability cannot be absolute; there must be variation in unpalatability, which could lead to some interesting evolutionary effects. Müllerian and Batesian mimicry are differentiated by means of palatabilities. Models and Müllerian mimics are negatively reinforcing, while Batesian mimics positively reinforce predator attacks. Hence, the straightforward view that Batesian mimics are parasitic and hurt their models, while Müllerian mimics are mutualistic and benefit their models (103). However, a second equally straightforward idea apparently conflicts with this view: If two Müllerian mimics are not equally unpalatable, the presence of the more palatable could increase the rate of attack on the less palatable, so that weakly unpalatable mimics may harm stronger models or co-mimics, leading to a parasitic form of Müllerian mimicry. A series of behavioral modelers since the 1960s have suggested that parasitic Müllerian mimicry may explain some of the embarrassing examples of polymorphism in aposematic species. Because benefits and costs become decoupled from the Müllerian/Batesian palatability divide in this latter prediction, a new terminology must be developed. An appropriate name for the new parasitic form of Müllerian mimicry is "quasi-Batesian" (148). [There is also a category of palatability-defined Batesian mimicry that is beneficial to the model as well as the mimic—"quasi-Müllerian" mimicry (84a, 147a, 151, 165). This is possible if seeing a palatable mimic "jogs" the memory, reminding predators of unpleasant experiences with the model, thus leading to greater avoidance of the model than if there were no mimic. Quasi-Müllerian mimicry seems unlikely (151); anyway, it should not lead to polymorphism and is not discussed further.] In quasi-Batesian mimicry, the more palatable mimic may suffer increasing attacks as its numbers increase relative to the model's, even though its effect while alone would be to reduce its predation progressively as density increases (Figure 3*B*,*C*) (71, 72, 115, 148–151). It has been suggested that this leads to the evolution of polymorphism in Müllerian mimicry systems (71, 72, 147a–151).

The behavioral assumptions that lead to quasi-Batesian mimicry pose a severe threat to traditional natural history and evolutionary dynamical views of mimicry, possibly "the end of traditional Müllerian mimicry" (148). This problem never arose until behavioral biologists attempted to model memory realistically. It is apparent that Müller and subsequent naturalists and evolutionists made an unstated assumption: that the sum of learning and forgetting over all predators would cause an approximately constant number (n_k) of unpalatable individuals of each phenotype to be killed (or damaged) per unit time (Figure 1). Purifying frequency-dependent selection results from Müller's assumption because

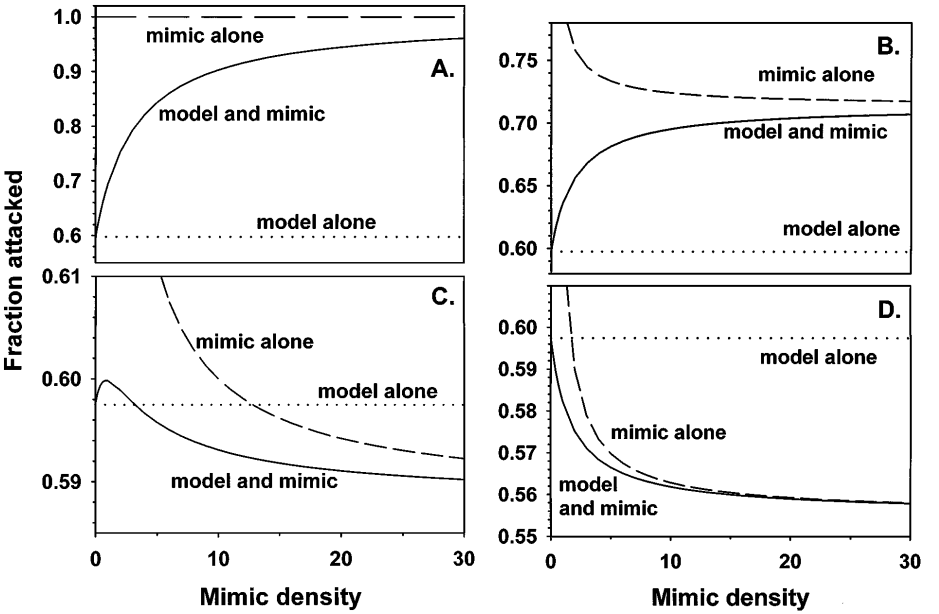


Figure 3 Mimicry and the palatability spectrum. The effect of assumptions about learning and forgetting on fitnesses of model and mimic are shown in this figure. Equilibrated attack rates at varying mimic densities are shown for model alone, mimic alone, and model-mimic pair. Comparisons of attack rate clearly demonstrate whether mimic, model, or both benefit from the association (115) (the mimic is here considered by convention to be the more palatable species). In all panels, the model density is a constant set at 1.6 (115). These assumptions (71, 115, 148, 149, 151, 166) can reproduce classical parasitic Batesian mimicry (*A*; $\lambda_{Mo} = 0.2$, $\lambda_{Mi} = 1$, $\alpha_{Mo} = 0.3$, $\alpha_{Mi} = 0.0$) and mutualistic Müllerian mimicry (*D*; $\lambda_{Mo} = 0.2$, $\lambda_{Mi} = 0.2$, $\alpha_{Mo} = 0.3$, $\alpha_{Mi} = 0.3$), but they also produce intermediate types of mimicry, including parasitic quasi-Batesian mimicry between pairs of unpalatable species (*B*; $\lambda_{Mo} = 0.2$, $\lambda_{Mi} = 0.6$, $\alpha_{Mo} = 0.3$, $\alpha_{Mi} = 0.5$), and a cusped quasi-Batesian/Müllerian combination (*C*; $\lambda_{Mo} = 0.2$, $\lambda_{Mi} = 0.3$, $\alpha_{Mo} = 0.3$, $\alpha_{Mi} = 0.5$). The curves were generated from a general equation for attack rate equilibrium at particular density (115, 149). Note that Owen & Owen's own figures are sketches only, and contain some incorrect features (149).

the average attack fraction n_k/N decreases as the total number of individuals, N, increases. The existence of quasi-Batesian mimicry, in contrast, requires that the attack fraction on a Müllerian mimic increases as N increases, implying that n_k can be a rising function of N rather than a constant. We here follow the development of these ideas and discuss why we feel the assumptions that lead to quasi-Batesian mimicry may not be met in most real situations.

The original idea for what is now called quasi-Batesian mimicry was proposed by Huheey (71 and earlier). After a single trial experience with an unpalatable individual, the predator was imagined to learn to avoid it totally; thereafter, the

predator would forget after seeing, but not attacking, a fixed number of individuals with the same pattern. In this formulation, unpalatability affected only the rate of memory loss, rather than its acquisition; very unpalatable species caused slower forgetting than mildly unpalatable species. Increasing the density of less nasty mimics caused a rise in the average forgetting rate and led to an increasing fraction of models attacked. Thus, if two unpalatable species differed in palatability, only one benefited, while the other suffered, though the more palatable species on its own was still unpalatable in the sense that predators are negatively reinforced. Mimicry, even when at the point of equal palatability, was neutral; increases in density of either co-mimic caused a faster rate of both learning and forgetting, rather than a reduction in fraction attacked. The predicted absence of mutualistic mimicry in Huheey's theory was strongly attacked (12, 115, 136, 151). The problem appeared to be the event-triggered forgetting model, in which avoidance lapsed after a certain number of prey were avoided. This meant that the total number of prey in the population had no effect on the evolution; selection was assumed to depend only on relative frequency of mimics and models.

To avoid this pathology of Huheey's formulation, it was proposed that forgetting should be time-dependent (12, 115, 136, 166), rather than depending on the number of avoidances; forgetting should cause the attack fractions to decline or rise exponentially with rates α_{Mi} and α_{Mo} (for mimic and model, respectively) toward the "naive attack rate" asymptote, i.e. a naive attack fraction (115, 149). At the same time, a more flexible system of learning was proposed, in which unpalatability was represented as an asymptotic fraction of prey attacked, λ_{Mi} and λ_{Mo}; these asymptotes were again approached exponentially, with learning rates forming another set of parameters (84a, 115, 147a, 148, 151). These theories could reproduce the full spectrum of mimicry from Batesian mimicry (Figure 3*A*) to Müllerian mimicry (Figure 3*D*), including quasi-Batesian mimicry (Figure 3*B*), and also a curious form of biphasic mimicry, which is quasi-Batesian at low mimic density, but traditionally Müllerian at higher mimic densities (Figure 3*C*).

The behavior of these models is easy to explain. Learning and forgetting each result in an exponential approach to a different asymptote of attack fraction, so the combination of the two processes will itself lead to a stationary resultant attack fraction independent of density for either model or mimic on their own. The joint attack fraction on model and mimic together (assuming models and mimics are visually indistinguishable) is simply an average between the curves for model and mimic asymptotic attack fractions. When mimic density is very low, the joint response is very like that of the model; when mimic density is high, the effect of the mimic dominates, and the joint response increasingly obeys the mimic's asymptote. Because the averaging process is of the form of a harmonic mean (115) rather than an arithmetic mean, curious peaks in the density response can occur, the "Owen & Owen effect" (149) (Figure 3*C*), implying a quasi-Batesian/Müllerian transition across a density threshold. Speed & Turner (151, 167a) recently examined the behavior of a number of different formulations and combinations of these basic memory assumptions. They concluded that (*a*) many of the assumptions

produce quasi-Batesian responses like that of Figure 3*B-C* and (*b*) behavioral experiments on mimicry and warning color are not usually set up to test for density responses and therefore cannot easily be used to test whether mimicry falls into quasi-Batesian categories. Well-known polymorphic Müllerian mimics often have intermediate levels of acceptance in tests both with caged and wild birds (20, 34, 35, 77, 119, 120, 132), showing that many supposedly unpalatable species may often be attacked. Therefore, the known biology of predation on unpalatable species as well as theory mesh with the possibility of a palatability spectrum that could lead to quasi-Batesian mimicry.

However, if theories like those in Figure 3 are correct, the whole basis for traditional number-dependent and frequency-dependent mimicry of Figure 1 is suspect. Our own belief is that new and incorrect assumptions lurking in the behavioral models are to blame for the conflict. Our objections, which are more fundamental than those raised in an earlier critique (84a), are as follows:

(*a*) We think it unlikely that attack rates on unpalatable species will reach an asymptotic fraction independent of density, unless that fraction is zero. To understand this, imagine that forgetting is switched off, so that all learning is perfect (see also 84a). Under this assumption, the new theories (115, 148) predict that learning should asymptote at constant frequency; number-dependence enters into memory dynamics only through time-based forgetting. With no forgetting, there is then no number-dependent selection, and mutualistic Müllerian mimicry becomes impossible (149–151). Intuitively, it seems odd that perfect memory does not lead to extremely successful Müllerian mimicry, and we here attempt to show why this intuition is correct. With no forgetting, the absence of Müllerian mimicry is due to a density-independent asymptotic attack fraction. In other words, as the density of an unpalatable mimic in Figure 3 rises, the predator is supposed to stuff itself with more and more unpalatable prey in order to maintain a constant asymptotic fraction of prey attacked. Learning to avoid prey is more likely to depend on dose received by the predator per unit time, rather than dose per individual prey. This will lead to a fraction attacked that declines with density rather than a constant asymptotic fraction. Note that this argument does not depend on "hunger levels," because unpalatable prey are unlikely to form a large component of the diet (166). The new theories in effect have the same problem in their learning module (i.e. not being time-based) as did Huheey's much- criticized forgetting module (12, 115, 136). It seems much more likely to us that for "unpalatable" prey, an asymptotic number of prey attacked per unit time would be required for learning, leading to strongly number-dependent and frequency-dependent selection like that of Figure 1, and a resultant attack fraction that declines to zero as prey densities increase.

(*b*) It is hard to justify the term "unpalatability" unless the effect is density-independent; predators should reject and increasingly avoid unpalatable prey whenever they encounter them at whatever density. However, the new theories see a species as unpalatable if it has a learning asymptote lower than the "naive attack fraction," and as palatable if it has a learning asymptote higher than the

naive attack fraction (149, 151). But only when the asymptotic attack fraction is zero do we produce avoidance, whatever the attack fraction prior to experience; this was the case, for example, in an original simulation model designed to disprove Huheey's assertions, and which recovered only Batesian and Müllerian mimicry, with a sharp transition between them (166). If our argument is correct, the whole of the palatability spectrum above an asymptotic attack fraction of zero is then "palatable," and quasi-Batesian mimicry simply becomes Batesian, parasitic mimicry. The "palatability spectrum" represented by $0 <$ asymptotic attack fraction ≤ 1 is just that, a spectrum of palatability rather than of unpalatability. Under this view, levels of unpalatability may differ, but they cause changes only in rates of learning and forgetting, rather than in level of the learning asymptote itself, which must be zero.

(*c*) Another problem is that, strictly speaking, "attack fraction" is not "palatability" at all, but a transformation of palatability onto a behavioral response axis. What we mean by "unpalatability" is easiest to interpret as a simple linear, or perhaps logarithmic, function of noxious compound dosage, which can vary from zero to infinity. The behavioral effect of these compounds may be to produce an asymptotic attack fraction of 0%, 100%, or somewhere in between (Figure 3). However, the behavioral response will certainly be a sigmoidal function of dose; the majority of dosages will yield approximately 100% (palatable) or 0% (unpalatable) asymptotic attack, with only a relatively narrow intervening band of dosages giving rise to intermediate levels of attack. Thus, the behavioral "palatability spectrum" as modeled by attack fraction is a highly distorted view of the underlying palatability, or dosage, of noxious chemistry; in fact, most of the dosage spectrum is not considered by these attack rate spectrum models (115, 147a–151). In reality, intermediate asymptotic attack fractions, even if they do exist, are likely to form a small part of the palatability (dosage) spectrum.

Empirical data from caged and wild birds showing intermediate levels of attack on models are of great interest, but they do not necessarily conflict with the points made above. Attack fractions in the laboratory or in nature tell us neither how they vary with prey density (167a) nor how they asymptote. The behavioral, "receiver psychology" view, which leads to possibly novel forms of mimicry, suggests that attack fraction will reach a nonzero asymptote as density is increased; the number-dependent (natural history) view predicts that attack fractions on unpalatable insects will always decline with increasing density. Unfortunately, experiments have not clearly distinguished between these alternatives because they were designed with other ends in mind (167a). It does not seem impossible to design more appropriate experiments, however.

In conclusion, theories of palatability from a receiver psychology angle have led to a potentially major upset in traditional views of mimicry. To decide which view is correct, we need to understand memory dynamics of actual predators, and, given that many of the controversial theories are supposedly based on a standard Pavlovian learning theory (124, 148), understanding the evolutionary results of memory on mimicry could lead to advances in memory theory in general. Even if

quasi-Batesian mimicry turns out, as we believe, to be unlikely, the threat posed by these new theories demonstrates the naiveté of the original natural history assumption that memory is a black box producing number-dependence.

3. *Spatial and Temporal Variation in Mimetic Selection* Geographic variation in mimetic color patterns within a mimic can obviously be maintained by geographic divergence of models. If mimicry is Müllerian, then divergence becomes self-reinforcing. Patches of habitat with different Müllerian mimetic patterns will be separated by zones of polymorphism; the width of the polymorphic region will be proportional to average dispersal distance and inversely proportional to the square root of the strength of selection (92), as for clines in general (5). Thus, if selection is weak and dispersal extensive, bands of polymorphism may be wide compared to areas of monomorphism. This situation undoubtedly pertains in many species; for example, it is often not realized how common this is within *Heliconius*. The hybrid zones between races of *Heliconius erato* or *H. melpomene* are renowned for their narrowness (e.g. 87, 93); however, zones of polymorphism between weakly differentiated races, for instance in the Amazon basin, are much broader, so that polymorphism is almost the norm (see maps in 24, 27; many other maps of *Heliconius* races oversimplify the actual distributions).

A similar situation may exist for wide bands of polymorphism in the unpalatable *Acraea encedon, A. encedana, Danaus chrysippus*, and their Batesian mimic *Hypolimnas misippus* in Central and Eastern Africa: Peripheral populations of these species are nearly monomorphic (146). Similarly, spatially varying mimetic and other selection pressures, rather than quasi-Batesian mimicry (151), may explain the polymorphisms of ladybirds such as *Adalia bipunctata* (15, 85) and butterflies such as *Laparus doris* (159).

There may also be temporal as well as spatial variation in mimetic selection. The diverse polymorphism of *Heliconius numata* may be selected because the models (ithomiines in the genus *Melinaea*) vary greatly in abundance over time and space (26). However, it would be hard to explain how polymorphism is maintained via temporal variation unless the color pattern loci have, on average, a net heterozygote advantage. Given that the supergenes affecting mimicry in *H. numata* are visually dominant (26), any heterozygous advantage must usually be nonvisual. Another example of polymorphism in a Müllerian mimic with multiple models is *Heliconius cydno*. There are strong differences across W. Ecuador in the frequency of models *Heliconius sapho* and *H. eleuchia*, causing divergent patterns of natural selection (80). In conclusion, the observed polymorphisms of many Müllerian mimics can be explained without quasi-Batesian mimicry, via spatial and possibly temporal variation in model abundance.

4. *The Shape of Frequency-Dependence* The maintenance of polymorphism in unpalatable species will be considerably aided by the shape of frequency-dependence, given number-dependent selection (Figure 1). When population sizes of prey (N) are large relative to the numbers sacrificed during predator learning, the fraction n_k/N will be small, say $1/100$ or less, and there will be little selection in the central polymorphic basin.

Although we do not know the values of n_k/N typical in the wild, a variety of experiments (11, 75, 80, 92) indicate that selection can be strong, i.e. $n_k/N \geq 1/10$. On the other hand, it seems likely that many predators will require few learning trials to avoid an aposematic insect. Models and common Müllerian mimics will often outnumber their predators considerably, and, furthermore, predators live much longer and will often be able to generalize between prey generations. Experiments by Kapan on *H. cydno* in W. Ecuador showed that selection against polymorphism was much weaker where *H. cydno* was abundant than where it was rare (80). Thus, it seems not unlikely that $n_k/N \leq 1/100$, at least some of the time.

Drift can explain the origin but not the maintenance of polymorphism in the central basin. However, polymorphisms, once attained, should be removed only slowly via mimetic selection. Second-order selective forces such as nonvisual selection (for instance, thermal selection in ladybirds), arbitrary mate choice, or other factors (15, 85,144–147) may become important and contribute to nonadaptedness of mimetic polymorphisms. Strong selection at some times and places ($n_k/N = 1/10$ or greater) is clearly required to produce near-perfect resemblance and narrow hybrid zones between races. But if a Müllerian mimic or model, perhaps by an ecological fluke, becomes abundant relative to its predators ($n_k/N \leq 1/100$), it could then be relatively free to experiment with nonadaptive and polymorphic color patterns. In short, the shape of frequency-dependence, together with varying selection for mimicry and mild selection of other types can explain Müllerian polymorphism without the need for quasi-Batesian mimicry.

MIMICRY AND SPECIATION

Bates, Wallace, and Darwin were all of the opinion that strong natural selection, which must occur sometimes to explain mimicry, could lead to speciation. The continuum between forms, races, and species of diversely patterned tropical butterflies led to this idea in the first place (6, 7, 173, 174). This view has since faded into the background, probably because of a postwar concentration on reproductive characteristics ("reproductive isolating mechanisms") thought important in speciation under the "biological species concept" (100). However, mimicry causes strong selection against nonmimetic hybrids or intermediates and should therefore contribute strongly to speciation and species maintenance, by acting as a form of ecologically mediated postmating isolation. Together with the evolution of assortative mating, mimetic shifts may have led to speciation in butterflies such as *H. himera* and *H. erato* (96, 101).

If mimicry contributes to speciation, mimetic shifts should often be associated with speciation within phylogenies. Mimicry-related speciation would explain the curious pattern of "adaptive radiation" in *Heliconius*: Müllerian co-mimics are usually unrelated, while closely related species almost always belong to different mimicry rings (164). Mimetic pattern has been switched between eight of nine pairs of terminal sister taxa in a mtDNA phylogeny of *Heliconius* (17, 95). Many

sister taxa that have switched mimicry are known from other groups as well. For example, among butterflies, the viceroy (*Limenitis archippus*) mimics queen and monarch butterflies (*Danaus* spp.), while its close relative, the red-spotted purple (*Limenitis arthemis astyanax*) mimics an unpalatable papilionid, *Battus philenor*. The two *Limenitis* are very closely related and hybridize occasionally in the wild (127). Similar examples exist in the Papilionidae. Mimetic lineages do not seem to speciate more rapidly than nonmimetic lineages in the genus *Papilio* (F Sperling, in litt.); however, closely related species do often differ in their mimicry ring.

While we believe mimicry contributes to speciation, this section must remain somewhat speculative. We cannot point to any convincing case in which mimicry has been the major or only cause of speciation. But then perhaps speciation is almost always caused by multiple, rather than single, episodes of disruptive selection.

EVOLUTION AND MAINTENANCE OF MULTIPLE MIMICRY RINGS

A naive view of Müllerian mimicry would suggest that all similarly sized species should converge locally onto a single color pattern. In fact, there are often ten or more mimicry rings among ithomiine and heliconiine butterflies of the Amazon basin (6, 9, 102, 137). The reason for the lack of a single uniform mimicry ring among similarly sized butterflies is currently disputed and parallels, at an interspecific level, the debate on Müllerian polymorphisms.

Papageorgis (116) provided data from Peru showing that different heliconiine and ithomiine mimicry rings fly at different heights in the forest canopy. She suggested that dual selection for camouflage and mimicry might explain these patterns. In other words, particular mimicry rings are better camouflaged in the lighting conditions pertaining at their favored flight heights. However, heliconiine flight heights are now well-documented to overlap far more extensively than appeared from Papageorgis' data, although weak mimicry associations do exist for habitat and nocturnal roosting height (25, 28, 94). It is unclear how dual selection would work, and it is anyway hard to imagine that the garish reds, yellows, blacks, and iridescent blues of heliconiines are ever very cryptic against subdued forest backdrops.

Nonetheless, recent studies of ithomiines do demonstrate some patterning of mimicry rings in flight height as well as in horizontal (habitat-related) distribution (10, 25, 44, 102). A possible explanation for these community patterns is that different guilds of predators are found preferentially in the different habitats or microhabitats, so that, within each habitat, mimicry is tuned to local predator knowledge (10, 94). There must be some selection pressure of this sort to explain the microhabitat associations; however, it would be hard to imagine birds ignoring butterflies a meter or two higher or lower than their normal flight height in the forest understory, and it seems highly unlikely that the proposed subcommunities of predators and particular mimicry rings are very discrete. The overlap between mimicry rings is rather more noticeable to the naturalist than the somewhat statistical differences

in average heights or microhabitats (10, 25, 44, 94, 102). Instead, statistical differences may exist because newly invading unpalatable species are most likely to join mimicry rings already most prevalent in their habitats. Major mimicry rings that overlap substantially may be unlikely to join together as species accumulate in each ring for the same reason that intraspecific polymorphisms have a nearly neutral central basin (Figure 1); the selection for convergence of two abundant mimicry rings will simply not be that strong.

CONCLUSIONS: MIMETIC DIVERSITY AND THE FORM OF FREQUENCY DEPENDENCE

We have shown that the shape of number-dependent selection on the color patterns of unpalatable species can help explain many mutually conflicting data of mimicry and mimetic diversity. When the attack fraction is high because of a high predator/prey ratio, selection on mimicry can clearly be extremely strong and has been measured to be so in a handful of field studies. But when predator/prey ratios are low ($n_k/N \leq 1/100$), there is a wide central basin of near-neutrality where only weak purifying selection acts on polymorphisms. Therefore, once an unpalatable butterfly becomes abundant relative to predators, n_k/N decreases hyperbolically, and its morphology becomes less constrained by selection. A temporary relaxation of selection may then result in polymorphisms, which become relatively impervious to further bouts of selection. The weakness of purifying selection in polymorphic populations can help explain why puzzling polymorphisms persist in some Müllerian mimics. Such polymorphisms enable populations to explore the selective landscape, which can increase the chances of shifting balance, one of the few ways to explain the empirical observation that utterly novel color patterns evolve continually in warning-colored and mimetic butterflies. Similarly, weak selection against multiple rings may be partially responsible for the diversity of mimicry in any one area.

But these arguments will fail if predator memory and perception do not produce number-dependent selection. If predators behave according to some current theories of "receiver psychology," these conclusions based on extensions of traditional, number-dependent Müllerian theory are in jeopardy. We do not think that this is the case; however, appropriate experimental studies are urgently required to test between these conflicting models of memory and forgetting.

ACKNOWLEDGMENTS

We are very grateful to George Beccaloni, Chris Jiggins, Gerardo Lamas, Russ Naisbit, Mike Speed, Felix Sperling, Maria Servedio, Greg Sword, John Turner, Dick Vane-Wright, Dave Williams for critiques, conversations, and comments, and to NERC, BBSRC, the British Council, and the Ministries of Higher Education and Research and of Foreign Affairs, France, for financial support.

LITERATURE CITED

1. Aiello A, Silberglied RE. 1978. Life history of *Dynastor darius* (Lepidoptera: Nymphalidae: Brassolinae) in Panama. *Psyche* 85: 331–45
2. Andersson M. 1994. *Sexual Selection*. Princeton, NJ: Princeton Univ. Press
3. Arak A, Enquist M. 1993. Hidden preferences and the evolution of signals. *Philos. Trans. R. Soc. London Ser. B* 340:207–14
4. Barton NH. 1979. The dynamics of hybrid zones. *Heredity* 43:341–59
5. Barton NH, Gale KS. 1993. Genetic analysis of hybrid zones. In *Hybrid Zones and the Evolutionary Process*, ed. RG Harrison, pp. 13–45. New York: Oxford Univ. Press
6. Bates HW. 1862. Contributions to an insect fauna of the Amazon valley. Lepidoptera: Heliconidae. *Trans. Linn. Soc. London* 23:495–566
7. Bates HW. 1863. *A Naturalist on the River Amazons*. London: John Murray
8. Bates HW. 1879. [commentary on Müller's paper]. *Trans. Entomol. Soc. London* 1879:xxviii–ix
9. Beccaloni GW. 1997. Ecology, natural history and behaviour of ithomiine butterflies and their mimics in Ecuador (Lepidoptera: Nymphalidae: Ithomiinae). *Trop. Lepid.* 8: 103–24
10. Beccaloni G. 1997. Vertical stratification of ithomiine butterfly (Nymphalidae: Ithomiinae) mimicry complexes: the relationship between adult flight height and larval hostplant height. *Biol. J. Linn. Soc.* 62:313–41
11. Benson WW. 1972. Natural selection for Müllerian mimicry in *Heliconius erato* in Costa Rica. *Science* 176:936–39
12. Benson WW. 1977. On the supposed spectrum between Batesian and Müllerian mimicry. *Evolution* 31:454–55
13. Blaisdell M. 1982. Natural theology and Nature's disguises. *J. Hist. Biol.* 15:163–89
14. Bradbury JW. 1981. The evolution of leks. In *Natural Selection and Social Behavior: Recent Research and New Theory*, ed. RD Alexander, DW Tinkle, pp. 138–69. New York: Chiron
15. Brakefield PM. 1985. Polymorphic Müllerian mimicry and interactions with thermal melanism in ladybirds and a soldier beetle: a hypothesis. *Biol. J. Linn. Soc.* 26: 243–67
16. Brakefield PM, Gates J, Keys D, Kesbeke F, Wijngaarden PJ, et al. 1996. Development, plasticity and evolution of butterfly eyespot patterns. *Nature* 384:236–42
17. Brower AVZ. 1994. Phylogeny of *Heliconius* butterflies inferred from mitochondrial DNA sequences (Lepidoptera: Nymphalinae). *Mol. Phylogenet. Evol.* 3: 159–74
18. Brower AVZ. 1995. Locomotor mimicry in butterflies? A critical review of the evidence. *Philos. Trans. R. Soc. London Ser. B* 347:413–25
19. Brower AVZ. 1996. Parallel race formation and the evolution of mimicry in *Heliconius* butterflies: a phylogenetic hypothesis from mitochondrial DNA sequences. *Evolution* 50:195–221
20. Brower LP. 1984. Chemical defence in butterflies. In *The Biology of Butterflies*, ed. RI Vane-Wright, PR Ackery, pp. 109–34. London: Academic
21. Brower LP. 1988. Preface. *Am. Nat.* 131(Suppl.):S1–S3
22. Brown KS. 1973. *A Portfolio of Neotropical Lepidopterology*. Rio de Janeiro, Brazil: Privately published. 28 pp.
23. Brown KS. 1976. An illustrated key to the silvaniform *Heliconius* (Lepidoptera: Nymphalidae) with descriptions of new subspecies. *Trans. Am. Entomol. Soc.* 102: 373–484
24. Brown KS. 1979. *Ecologia geográfica e*

evolução nas florestas neotropicais. Livre de Docencia. Campinas, Brazil: Univ. Estadual de Campinas
25. Brown KS. 1988. Mimicry, aposematism and crypsis in neotropical Lepidoptera: the importance of dual signals. *Bull. Soc. Zool. France* 113:83–101
26. Brown KS, Benson WW. 1974. Adaptive polymorphism associated with multiple Müllerian mimicry in *Heliconius numata* (Lepid.: Nymph.). *Biotropica* 6: 205–28
27. Brown KS, Sheppard PM, Turner JRG. 1974. Quaternary refugia in tropical America: evidence from race formation in *Heliconius* butterflies. *Proc. R. Soc. London Ser. B* 187:369–78
28. Burd M. 1994. Butterfly wing colour patterns and flying heights in the seasonally wet forest of Barro Colorado Island, Panama. *J. Trop. Ecol.* 10:601–10
29. Calvert WH, Hedrick LE, Brower LP. 1979. Mortality of the monarch butterfly (*Danaus plexippus* L.): avian predation at five overwintering sites in Mexico. *Science* 204:847–51
30. Carpenter GDH. 1939. Birds as enemies of butterflies, with special reference to mimicry. *Proc. VII Int. Kongr. Entomol., Berlin* 1938:1061–74
31. Carpenter GDH. 1941. The relative frequency of beakmarks on butterflies of different edibility to birds. *Proc. Zool. Soc. London Ser. A* 3:223–31
32. Carpenter GDH, Ford EB. 1933. *Mimicry*. London: Methuen
33. Carroll SB, Gates J, Keys DN, Paddock SW, Panganiban GEF, et al. 1994. Pattern formation and eyespot determination in butterfly wings. *Science* 265:109–14
34. Chai P. 1986. Field observations and feeding experiments on the responses of rufous-tailed jacamars (*Galbula ruficauda*) to free-flying butterflies in a tropical rainforest. *Biol. J. Linn. Soc.* 29:166–89
35. Chai P. 1996. Butterfly visual characteristics and ontogeny of responses to butterflies by a specialized bird. *Biol. J. Linn. Soc.* 59:37–67
36. Charlesworth B. 1994. The genetics of adaptation: lessons from mimicry. *Am. Nat.* 144:839–47
37. Charlesworth D, Charlesworth B. 1975. Theoretical genetics of Batesian mimicry. II. Evolution of supergenes. *J. Theor. Biol.* 55:305–24
38. Clarke CA, Sheppard PM. 1971. Further studies on the genetics of the mimetic butterfly *Papilio memnon*. *Philos. Trans. R. Soc. London Ser. B* 263:35–70
39. Clarke CA, Sheppard PM, Thornton IWB. 1968. The genetics of the mimetic butterfly *Papilio memnon*. *Philos. Trans. R. Soc. London Ser. B* 254:37–89
40. Cott HB. 1940. *Adaptive Coloration in Animals*. London: Methuen
41. Coyne JA, Barton NH, Turelli M. 1997. Perspective: a critique of Sewall Wright's shifting balance theory of evolution. *Evolution* 51:643–71
42. Darwin C. 1871. *The Descent of Man, and Selection in Relation to Sex*. London: John Murray. 2nd ed.
43. Dawkins R. 1976. *The Selfish Gene*. Oxford, UK: Oxford Univ. Press
44. DeVries PJ, Lande R. 1999. Associations of co-mimetic ithomiine butterflies on small spatial and temporal scales in a neotropical rainforest. *Biol. J. Linn. Soc.* In press
45. Edmunds M. 1974. *Defence in Animals*. Harlow, Essex: Longmans
46. Edmunds M, Golding YC. 1999. Diversity in mimicry. *Trends Ecol. Evol.* 14:150
47. Endler JA. 1988. Frequency-dependent predation, crypsis, and aposematic coloration. *Philos. Trans. R. Soc. London Ser. B* 319:459–72
48. Enquist M, Arak A. 1993. Selection of exaggerated male traits by female aesthetic senses. *Nature* 361:446–48
49. Fisher RA. 1930. *The Genetical Theory of Natural Selection*. Oxford: Clarendon

50. Fox RM. 1955. On subspecies. *Syst. Zool.* 4:93–95
51. French V. 1997. Pattern formation in colour on butterfly wings. *Curr. Opin. Genet. Dev.* 7:524–29
52. Gamberale G, Sillén–Tullberg B. 1996. Evidence for a peak-shift in predator generalization among aposematic prey. *Proc. R. Soc. London Ser. B* 263:1329–34
53. Gavrilets S. 1997. Evolution and speciation on holey adaptive landscapes. *Trends Ecol. Evol.* 12:307–12
54. Gavrilets S, Hastings A. 1998. Coevolutionary chase in two-species systems with applications to mimicry. *J. Theor. Biol.* 415–27
55. Gilbert LE. 1983. Coevolution and mimicry. In *Coevolution*, ed. DJ Futuyma, M Slatkin, p. 263–81. Sunderland, MA: Sinauer
56. Goldschmidt RB. 1945. Mimetic polymorphism, a controversial chapter of Darwinism. *Q. Rev. Biol.* 20:147–64; 205–30
57. Gordon IJ, Smith DAS. 1999. Diversity in mimicry. *Trends Ecol. Evol.* 14:150–51
58. Guilford T. 1985. Is kin selection involved in the evolution of warning coloration? *Oikos* 45:31–36
59. Guilford T. 1990. The evolution of aposematism. In *Insect Defenses. Adaptive Mechanisms and Strategies of Prey and Predators*, ed. DL Evans, JO Schmidt, pp. 23–61. New York: State Univ. New York Press
60. Guilford T, Dawkins MS. 1991. Receiver psychology and the evolution of animal signals. *Anim. Behav.* 42:1–14
61. Guilford T, Dawkins MS. 1993. Receiver psychology and the design of animal signals. *Trends Neurosci.* 16:430–36
62. Guilford T, Nicol C, Rothschild M, Moore B. 1987. The biological roles of pyrazines: evidence for a warning odour function. *Biol. J. Linn. Soc.* 31:113–28
63. Hamilton WD. 1964. The genetical evolution of social behaviour. *Theor. Biol.* 7:1–52
64. Hamilton WD. 1971. Geometry for the selfish herd. *J. Theor. Biol.* 31:295–311
65. Hartl DL, Clark AG. 1989. *Principles of Population Genetics*. Sunderland, MA: Sinauer. 2nd ed.
66. Harvey PH, Bull JJ, Pemberton M, Paxton RJ. 1982. The evolution of aposematic coloration in distasteful prey: a family model. *Am. Nat.* 119:710–19
67. Harvey PH, Greenwood PJ. 1978. Anti-predator defence strategies: some evolutionary problems. In *Behavioural Ecology*, ed. JR Krebs, NB Davies, pp. 129–51. Oxford: Blackwell Sci.
68. Harvey PH, Paxton RJ. 1981. The evolution of aposematic coloration. *Oikos* 37: 391–93
69. Hewitt GM. 1988. Hybrid zones—natural laboratories for evolutionary studies. *Trends Ecol. Evol.* 3:158–67
70. Holzkamp G, Nahrstedt A. 1994. Biosynthesis of cyanogenic glycosides in the Lepidoptera—incorporation of [U-C-14]-2-methylpropanealdoxime, 2S-[U-C-14]-methylbutanealdoxime and D,L-[U-C-14]-N-hydroxyisoleucine into linamarin and lotaustralin by the larvae of *Zygaena trifolii*. *Insect Biochem. Mol. Biol.* 24: 161–68
71. Huheey JE. 1976. Studies in warning coloration and mimicry. VII. Evolutionary consequences of Batesian–Müllerian spectrum: a model for Müllerian mimicry. *Evolution* 30:86–93
72. Huheey JE. 1988. Mathematical models of mimicry. *Am. Nat.* 131(Suppl.):S22–41
73. Järvi T, Sillén-Tullberg B, Wiklund C. 1981. The cost of being aposematic. An experimental study of predation on larvae of *Papilio machaon* by the great tit *Parus major*. *Oikos* 36:267–72
74. Jävi T, Sillén-Tullberg B, Wiklund C. 1981. Individual versus kin selection for aposematic coloration. A reply to Harvey and Paxton. *Oikos* 37:393–95
75. Jeffords MR, Sternberg JG, Waldbauer GP. 1979. Batesian mimicry: field demonstra-

tion of the survival value of pipevine swallowtail and monarch color patterns. *Evolution* 33:275–86
76. Jones DA, Parsons J, Rothschild M. 1962. Release of hydrocyanic acid from crushed tissues of all stages in the life cycle of species of the Zygaeninae (Lepidoptera). *Nature* 193:52–63
77. Jones FM. 1932. Insect coloration and the relative acceptability of insects to birds. *Trans. R. Entomol. Soc. London* 80:345–85
78. Joron M, Mallet J. 1998. Diversity in mimicry: paradox or paradigm. *Trends Ecol. Evol.* 13:461–66
79. Kamo M, Kubo T, Iwasa Y. 1998. Neural network for female mate preference, trained by a genetic algorithm. *Philos. Trans. R. Soc. London Ser. B.* In press
80. Kapan D. 1998. *Divergent natural selection and Müllerian mimicry in polymorphic* Heliconius cydno (*Lepidoptera: Nymphalidae*). PhD diss. Univ. B-C
81. Leimar O, Enquist M, Sillén-Tullberg B. 1986. Evolutionary stability of aposematic coloration and prey unprofitability: a theoretical analysis. *Am. Nat.* 128:469–90
82. Leimar O, Tuomi J. 1998. Synergistic selection and graded traits. *Evol. Ecol.* 12: 59–71
83. Linares M. 1997. Origin of neotropical mimetic diversity from a three-way hybrid zone of *Heliconius cydno* butterflies. In *Tropical Diversity and Systematics*, ed. H Ulrich, pp. 93–108. *Proc. Int. Symp. Biodiversity Syst. Trop. Ecosyst.* Bonn, 1994. Bonn: Zool. Forsch. inst. Mus. Alex. Koenig
84. Lindström L, Alatalo RV, Mappes J, Riipi M, Vertainen L. 1999. Can aposematic signals evolve by gradual change? *Nature* 397:249–51
84a. MacDougall A, Dawkins MS. 1998. Predator discrimination error and the benefits of Müllerian mimicry. *Anim. Behav.* 55:1281–88
85. Majerus MEN. 1998. *Melanism. Evolution in Action.* Oxford: Oxford Univ. Press. xiii + 338 pp.
86. Malcolm SB. 1990. Mimicry: status of a classical evolutionary paradigm. *Trends Ecol. Evol.* 5:57–62
87. Mallet J. 1986. Hybrid zones in *Heliconius* butterflies in Panama, and the stability and movement of warning colour clines. *Heredity* 56:191–202
87a. Mallet J. 1989. The genetics of warning colour in Peruvian hybrid zones of *Heliconius erato* and *H. melpomene*. *Proc. R. Soc. London Ser. B* 236:163–85
88. Mallet J. 1991. Variations on a theme? *Nature* 354:368 (Review of HF Nijhout. 1991. *The Development and Evolution of Butterfly Wing Patterns*. Washington, DC: Smithsonian Inst.
89. Mallet J. 1993. Speciation, raciation, and color pattern evolution in *Heliconius* butterflies: evidence from hybrid zones. In *Hybrid Zones and the Evolutionary Process*, ed. RG Harrison, pp. 226–60. New York: Oxford Univ. Press
90. Mallet J. 1999. Mimicry references. http://abacus.gene.ucl.ac.uk/jim/Mim/mimicry.htm.
91. Mallet J, Barton N. 1989. Inference from clines stabilized by frequency-dependent selection. *Genetics* 122:967–76
92. Mallet J, Barton NH. 1989. Strong natural selection in a warning color hybrid zone. *Evolution* 43:421–31
93. Mallet J, Barton N, Lamas G, Santisteban J, Muedas M, Eeley H. 1990. Estimates of selection and gene flow from measures of cline width and linkage disequilibrium in *Heliconius* hybrid zones. *Genetics* 124:921–36
94. Mallet J, Gilbert LE. 1995. Why are there so many mimicry rings? Correlations between habitat, behaviour and mimicry in *Heliconius* butterflies. *Biol. J. Linn. Soc.* 55:159–80
95. Mallet J, Jiggins CD, McMillan WO.

1996. Mimicry meets the mitochondrion. *Curr. Biol.* 6:937–40
96. Mallet J, McMillan WO, Jiggins CD. 1998. Mimicry and warning color at the boundary between races and species. In *Endless Forms: Species and Speciation*, ed. S Berlocher, D Howard, pp. 390–403. New York: Oxford Univ. Press
97. Mallet J, Singer MC. 1987. Individual selection, kin selection, and the shifting balance in the evolution of warning colours: the evidence from butterflies. *Biol. J. Linn. Soc.* 32:337–50
98. Mallet JLB, Turner JRG. 1998. Biotic drift or the shifting balance—Did forest islands drive the diversity of warningly coloured butterflies? In *Evolution on Islands*, ed. PR Grant, pp. 262–80. Oxford: Oxford Univ. Press
99. Maynard Smith J. 1998. *Evolutionary Genetics*. Oxford, UK: Oxford Univ. Press
100. Mayr E. 1963. *Animal Species and Evolution*. Cambridge, MA: Harvard Univ. Press
101. McMillan WO, Jiggins CD, Mallet J. 1997. What initiates speciation in passion-vine butterflies? *Proc. Natl. Acad. Sci. USA* 94:8628–33
102. Medina MC, Robbins RK, Lamas G. 1996. Vertical stratification of flight by ithomiine butterflies (Lepidoptera: Nymphalidae) at Pakitza, Manu National Park, Perú. In *Manu. The Biodiversity of Southeastern Peru*, ed. DE Wilson, A Sandoval, pp. 211–16. Washington, DC: Smithsonian Inst.
103. Müller F. 1879. *Ituna* and *Thyridia*; a remarkable case of mimicry in butterflies. *Trans. Entomol. Soc. London* 1879:xx–xxix
104. Nahrstedt A, Davis RH. 1983. Occurrence, variation and biosynthesis of the cyanogenic glucosides linamarin and lotaustralin in species of the Heliconiini (Insecta: Lepidoptera). *Comp. Biochem. Physiol.* 75B:65–73
105. Nicholson AJ. 1927. A new theory of mimicry in insects. *Aust. Zool.* 5:10–104
106. Nijhout HF. 1991. *The Development and Evolution of Butterfly Wing Patterns*. Washington, DC: Smithsonian Inst.
107. Nijhout HF, Wray GA. 1988. Homologies in the colour patterns of the genus *Heliconius* (Lepidoptera: Nymphalidae). *Biol. J. Linn. Soc.* 33:345–65
108. Nijhout HF, Wray GA, Gilbert LE. 1990. An analysis of the phenotypic effects of certain colour pattern genes in *Heliconius* (Lepidoptera: Nymphalidae). *Biol. J. Linn. Soc.* 40:357–72
109. Nur U. 1970. Evolutionary rates of models and mimics in Batesian mimicry. *Am. Nat.* 104:477–86
110. O'Donald P. 1980. *Genetic Models of Sexual Selection*. Cambridge, UK: Cambridge Univ. Press
111. Ohsaki N. 1995. Preferential predation of female butterflies and the evolution of Batesian mimicry. *Nature* 378:173–75
112. Orr HA. 1998. The population genetics of adaptation: the distribution of factors fixed during adaptive evolution. *Evolution* 52:935–49
113. Orr HA, Coyne JA. 1992. The genetics of adaptation: a reassessment. *Am. Nat.* 140: 725–42
114. Owen DF, Smith DAS, Gordon IJ, Owiny AM. 1994. Polymorphic Müllerian mimicry in a group of African butterflies: a reassessment of the relationship between *Danaus chrysippus*, *Acraea encedon* and *Acraea encedana* (Lepidoptera: Nymphalidae). *J. Zool.* 232:93–108
115. Owen RE, Owen ARG. 1984. Mathematical paradigms for mimicry: recurrent sampling. *J. Theor. Biol.* 109:217–47
116. Papageorgis C. 1975. Mimicry in neotropical butterflies. *Am. Sci.* 63:522–32
117. Parsons M. 1984. Life histories of *Taenaris* (Nymphalidae) from Papua New Guinea. *J. Lepid. Soc.* 38:69–84
118. Parsons M. 1991. Butterflies of the

Bololo–Wau Valley. *Handb. Wau Ecol. Insti.* 12. Honolulu: Bishop Mus.
119. Pinheiro CEG. 1996. Palatability and escaping ability in neotropical butterflies: tests with wild kingbirds (*Tyrannus melancholicus*). *Biol. J. Linn. Soc.* 59: 351–65
120. Pinheiro CEG. 1997. *Unpalatability, mimicry and escaping ability in neotropical butterflies: experiments with wild predators*. DPhil. thesis. Univ. Oxford
121. Plowright RC, Owen RE. 1980. The evolutionary significance of bumble bee color patterns: a mimetic interpretation. *Evolution* 34:622–37
122. Poulton EB. 1890. *The Colours of Animals*. London: Trübner
123. Punnett RC. 1915. *Mimicry in Butterflies*. Cambridge, UK: Camb. Univ. Press
124. Rescorla RA, Wagner AR. 1972. A theory of Pavlovian conditioning: variations in the effectiveness of reinforcement and non-reinforcement. In *Classical Conditioning II: Current Research and Theory*, ed. A Black, WF Prokasy, pp. 64–99. New York: Appleton-Century-Crofts
125. Rettenmeyer CW. 1970. Insect mimicry. *Annu. Rev. Entomol.* 15:43–74
126. Ridley M. 1996. *Evolution*. Oxford, UK: Blackwell Sci.
127. Ritland DB. 1990. Localized interspecific hybridization between mimetic *Limenitis* butterflies (Nymphalidae) in Florida. *J. Lepid. Soc.* 44:163–73
128. Robbins RK. 1980. The lycaenid "false head" hypothesis: historical review and quantitative analysis. *J. Lepid. Soc.* 34: 194–208
129. Rothschild M. 1985. British aposematic Lepidoptera. In *The Moths and Butterflies of Great Britain and Ireland*, ed. J Heath, AM Emmet, 2. Cossidae–Heliodinidae, pp. 9–62. Great Horkesley, Essex: Harley Books
130. Rothschild M, Moore BP, Brown WV. 1984. Pyrazines as warning odour components in the monarch butterfly, *Danaus plexippus*, and in moths of the genus *Zygaena* and *Amata* (Lepidoptera). *Biol. J. Linn. Soc.* 23:375–80
131. Ryan MJ, Rand AS. 1993. Sexual selection and signal evolution: the ghost of biases past. *Philos. Trans. R. Soc. London Ser. B* 340:187–96
132. Sargent TD. 1995. On the relative acceptabilities of local butterflies and moths to local birds. *J. Lepid. Soc.* 49:148–62
133. Servedio MR. 1998. *Preferences, signals, and evolution: theoretical studies of mate choice copying, reinforcement, and aposematic coloration*. PhD. diss. Univ. Texas at Austin
134. Sheppard PM. 1958. *Natural Selection and Heredity*. London: Hutchinson
135. Sheppard PM. 1959. The evolution of mimicry: a problem in ecology and genetics. *Cold Spring Harbor Symp. Quant. Biol.* 24:131–40
136. Sheppard PM, Turner JRG. 1977. The existence of Müllerian mimicry. *Evolution* 31:452–53
137. Sheppard PM, Turner JRG, Brown KS, Benson WW, Singer MC. 1985. Genetics and the evolution of muellerian mimicry in *Heliconius* butterflies. *Philos. Trans. R. Soc. London Ser. B* 308:433–613
138. Silberglied RE. 1984. Visual communication and sexual selection among butterflies. In *The Biology of Butterflies*, ed. RI Vane-Wright, PR Ackery, pp. 207–23. London: Academic
139. Sillén-Tullberg B. 1988. Evolution of gregariousness in aposematic butterfly larvae: a phylogenetic analysis. *Evolution* 42:293–305
140. Sillén-Tullberg B, Bryant EH. 1983. The evolution of aposematic coloration in distateful prey: an individual selection model. *Evolution* 37:993–1000
141. Sillén-Tullberg B, Hunter AF. 1996. Evolution of larval gregariousness in relation to repellant defences and warning coloration in tree–feeding Macrolepidoptera: a phylogenetic analysis based on

independent contrasts. *Biol. J. Linn. Soc.* 57:253–76
142. Smiley JT. 1978. *The host plant ecology of* Heliconius *butterflies in Northeastern Costa Rica*. PhD diss. Univ. Texas at Austin
143. Smith DAS. 1976. Phenotypic diversity, mimicry and natural selection in the African butterfly *Hypolimnas misippus* L. (Lepidoptera: Nymphalidae). *Biol. J. Linn. Soc.* 8:183–204
144. Smith DAS. 1980. Heterosis, epistasis and linkage disequilibrium in a wild population of the polymorphic butterfly *Danaus chrysippus*. *Zool. J. Linn. Soc.* 69:87–110
145. Smith DAS. 1981. Heterozygous advantage expressed through sexual selection in a polymorphic African butterfly. *Nature* 289:174–75
146. Smith DAS, Gordon IJ, Depew LA, Owen DF. 1998. Genetics of the butterfly *Danaus plexippus* (L.) in a broad hybrid zone, with special reference to sex ratio, polymorphism and intragenomic conflict. *Biol. J. Linn. Soc.* 65:1–40
147. Smith DAS, Owen DF, Gordon IJ, Lowis NK. 1997. The butterfly *Danaus chrysippus* (L.) in East Africa: polymorphism and morph–ratio clines within a complex, extensive and dynamic hybrid zone. *Zool. J. Linn. Soc.* 120:51–78
147a. Speed, MP. 1993. *Mimicry and the psychology of predation*. PhD thesis. Univ. Leeds
148. Speed MP. 1993. Muellerian mimicry and the psychology of predation. *Anim. Behav.* 45:571–80
149. Speed MP. 1999. Robot predators in virtual ecologies: the importance of memory in mimicry studies. *Anim. Behav.* 57: 203–13
150. Speed MP. 1999. Robot predators, receiver psychology, and doubts about Mullerian mimicry: comments on MacDougall and Dawkins. *Anim. Behav.* In press
151. Speed MP, Turner JRG. 1999. Learning and memory in mimicry: II. Do we understand the mimicry spectrum? *Biol. J. Linn. Soc.* In press
152. Srygley RB. 1994. Locomotor mimicry in butterflies? The associations of positions of centres of mass among groups of mimetic, unprofitable prey. *Phil. Trans. Roy. Soc. London Ser. B* 343:145–55
153. Srygley RB. 1998. Locomotor mimicry in Heliconius butterflies: contrast analyses of flight morphology and kinematics. *Philos. Trans. R. Soc. London Ser. B* 353:1–13
154. Srygley RB, Chai P. 1990. Flight morphology of neotropical butterflies: palatability and the distribution of mass to the thorax and abdomen. *Oecologia (Berlin)* 84:491–99
155. Sword GA. 1999. Density-dependent warning coloration. *Nature* 397:217
156. Tinbergen N. 1951. *The Study of Instinct*. Oxford: Oxford Univ. Press
157. Treisman M. 1975. Predation and the evolution of gregariousness. I. Models for concealment and evasion. *Anim. Behav.* 23:779–800
158. Tullrot A, Sundberg P. 1991. The conspicuous nudibranch *Polycera quadrilineata*: aposematic coloration and individual selection. *Anim. Behav.* 41:175–76
159. Turner JRG. 1971. Studies of Müllerian mimicry and its evolution in burnet moths and heliconid butterflies. In *Ecological Genetics and Evolution*, ed. ER Creed, pp. 224–60. Oxford, UK: Blackwell Sci.
160. Turner JRG. 1975. Communal roosting in relation to warning colour in two heliconiine butterflies (Nymphalidae). *J. Lepid. Soc.* 29:221–26
161. Turner JRG. 1977. Butterfly mimicry–the genetical evolution of an adaptation. *Evol. Biol* 10:163–206
162. Turner JRG. 1978. Why male butterflies are non-mimetic: natural selection, sex-

ual selection, group selection, modification and sieving. *Biol. J. Linn. Soc.* 10: 385–432
163. Turner JRG. 1979. Oscillation of gene frequencies in Batesian mimics: a correction. *Biol J. Linn. Soc.* 11:397–98
164. Turner JRG. 1984. Mimicry: the palatability spectrum and its consequences. In *The Biology of Butterflies*, ed. RI Vane-Wright, PR Ackery, pp. 141–61. London: Academic
165. Turner JRG. 1995. Mimicry as a model for coevolution. In *Biodiversity and Evolution*, ed. R Arai, M Kato, Y Doi, pp. 131–50. Tokyo: Natl. Sci. Mus. Found.
166. Turner JRG, Kearney EP, Exton LS. 1984. Mimicry and the Monte Carlo predator: the palatability spectrum and the origins of mimicry. *Biol. J. Linn. Soc.* 23:247–68
167. Turner JRG, Mallet JLB. 1996. Did forest islands drive the diversity of warningly coloured butterflies? Biotic drift and the shifting balance. *Philos. Trans. R. Soc. London Ser. B* 351:835–45
167a. Turner JRG, Speed MP. 1996. Learning and memory in mimicry. I. Simulations of laboratory experiments. *Philos. Trans. R. Soc. London. Ser. B* 351:1157–70
168. Vane-Wright RI. 1976. A unified classification of mimetic resemblances. *Biol. J. Linn. Soc.* 8:25–56
169. Vane-Wright RI. 1984. The role of pseudosexual selection in the evolution of butterfly colour patterns. In *The Biology of Butterflies*, ed. RI Vane-Wright, PR Ackery, pp. 251–53. London: Academic
170. Waldbauer GP. 1988. Aposematism and batesian mimicry. *Evol. Biol.* 22:227–59
171. Waldbauer GP, Sheldon JK. 1971. Phenological relationships of some aculeate Hymenoptera, their dipteran mimics, and insectivorous birds. *Evolution* 25: 371–82
172. Waldbauer GP, Sternburg JG. 1975. Saturniid moths as mimics: an alternative interpretation of attempts to demonstrate mimetic advantage in nature. *Evolution* 29:650–58
173. Wallace AR. 1854. On the habits of the butterflies of the Amazon Valley. *Trans. Entomol. Soc. London* 2:253–64
174. Wallace AR. 1865. On the phenomena of variation and geographical distribution as illustrated by the Papilionidae of the Malayan region. *Trans. Linn. Soc. Lund* 25:1–71
175. Wallace AR. 1878. *Tropical Nature and Other Essays*. London: MacMillan
176. Wickler W. 1968. *Mimicry in Plants and Animals*. New York: McGraw Hill
177. Wiklund C, Järvi T. 1982. Survival of distasteful insects after being attacked by naïve birds: a reappraisal of the theory of aposematic coloration evolving through individual selection. *Evolution* 36:998–1002
178. Wright S. 1932. The roles of mutation, inbreeding, crossbreeding and selection in evolution. *Proc. 11th, Int. Congr. Genet., The Hague* 1:356–66
179. Wright S. 1982. Character change, speciation, and the higher taxa. *Evolution* 36:427–43
180. Wright S. 1982. The shifting balance theory and macroevolution. *Annu. Rev. Genet.* 16:1–19
181. Yachi S, Higashi M. 1998. How can warning signals evolve in the first place? *Nature* 394:882–84