14. *Mimicry: The Palatability Spectrum and its Consequences*

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This paper is dedicated also to the memory of Professor Ford's most distinguished graduate student, Philip Sheppard, who not only inspired most of the work recounted here, but did much of it as well.

Why Do Some Things Taste Nasty?

'Palatability spectrum' is ecojargon for the fact that all things are not equally nice to eat. The phenomenon is the product of the coevolution which occurs between all organisms, dictating their food preferences as a result of variations in nutrience, availability, competition, ease of capture and so on. The aspect of the palatability spectrum of most interest to the student of butterfly mimicry is outright nastiness: the production of some substance unpleasant or even poisonous to predators which would otherwise find the prey perfectly acceptable. As the work of Rothschild, Brower and many others (e.g. Brower & Brower 1964, Rothschild 1972*b*) has shown, the story generally starts with the production of chemical defences by plants (but see also Ch. 12), to deter either herbivores or phytophagous insects. Sooner or later some insect overcomes the plant defence by detoxifying or sequestering the chemical. Provided the plant is an evolutionary success the insect has a new set of niches available, multiplying in evolutionary time, so that a whole group of insects, largely untroubled by competition, may diversify on the radiating plant group. Thus we find danaines on Asclepiadaceae, heliconiines on Passifloraceae (Benson *et al.* 1976) and ithomiines on Solanaceae.

But whether a success is in terms of multiplication of species or not, the insect has the additional option of using the defence substance of the plant for its own protection. Whether they simply use the plant chemicals direct (*Danaus plexippus*), or convert them into related substances, or have developed the ability to synthesize compounds independently of any input from the plant (*Zygaena*), the result is that some insects are not nearly no nice, or so safe, to eat as others. The stage is set for the evolution, first, of warning coloration, and then of mimicry.

Why Are Warning Colours Bright?

Some species develop chemical defence and leave it at that: the Buff-tip moth (*Phalera bucephala*) is distasteful, but like the majority of palatable species, it is cryptically coloured, being an excellent mimic of a birch twig (Fisher 1930). Probably a great many more develop the simple and conspicuous patterns of red, black, yellow and white stripes and dots which, as Philip Sheppard was fond of pointing out, being the same colour as road signs, stand out well against vegetation, and which we recognize as warning colours. Why do they do this? Surely it is better not to be seen at all, than to be somewhat mangled before being dropped from the beak as too hot to handle? That there is indeed a disadvantage in becoming conspicuous is shown by what appears to be a correlation between diurnal flight and warning colour: most fully diurnal moths are warningly coloured (one thinks in Britain of the only truly diurnal geometrid, the Chimney Sweeper moth '*Odezia atrata*) which is jet black, compared with the ast, cryptically coloured nocturnal family to which it belongs); and Batesian mimicry, which involves a similar adoption of conspicuous colouring, is found frequently among butterflies but very rarely among caterpillars. It is quite uncertain in the case of moths whether warning colour allows them to come into the open and operate on solar power, flying by day being much more economical of energy than flying by night (e.g. Wasserthal 1975, Vielmetter 1958, Douwes 1976*a*, Douglas 1979) or whether being diurnal imposes enormous evolutionary pressure to become aposematic (Rothschild 1972*a*). Butterflies

as a group are diurnal, but many are not aposematic: many give the impression of avoiding predators by being thin and gristly which their dependence on solar rather than metabolic heat allows, and rather agile; on the whole, they are just not worth the energy required to catch them. However this may be, it looks as though warning colour evolves more readily when the organism is already conspicuous because it is flying around in full view: *Zygaena* larvae are toxic, slow moving and cryptic; the diurnal adults also toxic, are warningly coloured.

But even putting to one side the awkward thought that there are many aposematic caterpillars, this still does not explain the adoption of the 'road-sign' coloration. Two explanations, both of which are probably correct, have been put forward. The first is that there is an inbuilt tendency within the vertebrate nervous system to learn rapidly to associate these colours with a nasty experience. Certainly vertebrates do show asymmetries of this kind in response to colour. Goldfish being trained by electric shocks to stay in one half of a tank, or go to the other, learned more rapidly when a red light meant 'go' and a green light 'stay', than they did when the lights had the conventional meaning of traffic lights (stay on red, go on green) (Bisping *et al.* 1974). More recently Gibson (1980) showed that finches learned to avoid the negative experience of having prey disappear through a trap door when the prey was red than when it was cryptically coloured (blue) to match the background. It is not certain here whether it is the red colour, or the contrast with the background which induces the faster learning, but subjective experience certainly suggests that there is something unrestful about red and yellow colours: diazo slides are much pleasanter to look at if they are white on blue (the preferred colour with most lecturers) than if they are white on red. Red for us has strong connotations of warning and of decoration, as in 'The masque of the red death' or the use of the same root in Russian to mean both 'red' and 'beautiful'.

In addition, or alternatively, it may be that aposematic colouring has evolved to take warning patterns away, beyond any possibility of confusion, from the green and brown colours of the palatable, cryptic prey for which predators are constantly forming search images (Turner 1975*a*). The idea was succinctly put by Fisher (1930), who pointed out that it was essentially similar to the tendency of models to evolve away from their Batesian mimics: 'to be recognised as unpalatable is equivalent to avoiding confusion with palatable species.'

I believe that there is a further, overlooked factor, in the learning rather than searching behaviour of predators (see also Ch.12). Many aposematic species are poisonous, perhaps without being particularly distasteful (Rothschild 1971, 1972*b*, 1976, Rothschild *et al.* 1972*b*). In such cases there is a time-delay between eating the insect and receiving a nasty experience. Vertebrates are in fact capable of associating the symptoms of poisoning with a food item taken the appropriate time *before* the symptoms develop (e.g. Garcia *et al.* 1966, 1972), but if at that time they were consuming a series of insects, as for instance flycatchers and jacamars do, they may have difficulty knowing *which* insect caused the poisoning. Any insect conspicuously different from others will be much more readily associated with the unpleasantness.

A fourth possibility is that the conspicuous coloration causes a faster rate of learning simply because it induces a faster rate of attack. A predator attacks say ten conspicuous prey in a much shorter time interval than ten cryptic prey, and as learning and forgetting are time dependent, builds up its avoidance of the more frequently encountered prey much more quickly (see also Ch.12). (This is analogous to the greater protection given to a common warningly coloured form than to a rare one (below), the difference in abundance in this case being replaced with a difference in apparency.) Gittleman & Harvey (1980, also Gittleman *et al.* 1980) found that chicks more rapidly attacked, and then sooner avoided, unpalatable crumbs which contrasted with the background than those which matched it, although in this case a fifth possibility, which is in fact the preferred interpretation of the authors is not ruled out: that contrast by itself induces faster learning. That it was not simply colour was shown by reciprocal experiments in which either blue or green crumbs contrasted with, or matched the background. This of course is not quite the same as the theory that 'road-sign' colours are more easily learned, in themselves.

It remains to be seen which of the mechanisms, innately fast learning of bright colours, innately fast learning of contrasting colours, fast learning of prey that is different, greater initial rate of attack on conspicuous colours, and avoidance of mimicry of cryptic colours, is the significant mechanism in the origin of aposematic colouring. Although some experiments will be easy to devise (to choose between faster initial attack rate and rapid learning of contrast it would only be necessary to make the matching prey so common that they were attacked at the same rate as the contrasting prey), the coevolution of the vertebrate nervous system with the insects may have made it impossible for us finally to disentangle the problem.

Although there are thus at least five plausible explanations for the use of 'road-sign' colours to advertise distastefulness, there is a well known and much discussed difficulty in evolving the warning colour in the first place. Being distasteful can be individually advantageous if the predator drops you

relatively unharmed, but there is no advantage to the individual in attracting a predator by making itself conspicuous. This is most obviously the case if the 'advantage' of the bright colour is simply to generate faster learning through a faster rate of attack. The same applies to being poisonous: it profiteth one nothing to make the bird that ate one sick, as one cannot be delivered up whole, like Jonah out of the belly of the fish. The question was originally tackled by Fisher (1930), apparently the first to enunciate the principle of kin selection (see also Ch.12), put firmly on the map by Hamilton (1964*a*,*b*): if the death of an aposematic individual protects a relative, rather than an unrelated conspecific, then the gene causing the bright coloration, or toxicity, may be at an advantage. Harvey *et al.* (1981, 1982) have recently examined this situation mathematically, and have delimited the conditions under which aposematic coloration will evolve. The distasteful species must live, as do gregarious caterpillars, in family groups, which are rather widely scattered so that not very many families occur within the home range of any one predator. When a predator has eaten a few of the prey, it must learn to avoid them, and it must learn to avoid warningly coloured ones more quickly than similarly distasteful but cryptic prey. Finally, the warning pattern, while making the prey more memorable, must not make it excessively conspicuous. It seems that these conditions have been met in many aposematic species: aposematic larvae and bugs are frequently gregarious; and at least some warningly coloured butterflies have restricted home ranges and roost gregariously; the evidence for faster learning of bright colours has already been quoted; and a number of observers have noted that warningly coloured insects, although blatant in close up, are relatively camouflaged at a distance (review by Endler 1978).

I am uncertain about the statements that warningly coloured *Heliconius* are partly camouflaged in flight by a flicker effect (Papageorgis 1975), as at least one field observer believes the contrary—their patterns are adapted to make them *more* conspicuous against their normal background (Benson 1982). Hinton (1977) speculated that certain gregarious membracid bugs were black when young so as to form a black mass that would stand out against the background, whereas the adult colours, yellow spots on black, would tend to blend into grey on the young bugs. The contrast/camouflage story about bright colours can clearly be told both ways! But at least there are good grounds for believing that aposematic butterflies and dragonflies are not very much more conspicuous than cryptic butterflies when flying around in mid-air. It is surely significant that many aposematic butterflies have much duller colours on the underside, the surface exposed while at rest.

Järvi *et al.* (1981*a*,*b*) have recently challenged the view that kin selection is necessary for the evolution of aposematism, basing their view on experiments which show that Swallowtail (*Papilio machaon*) larvae are released, unharmed, by Great Tits. Here there is clearly an individual advantage in distastefulness, as there is in *Heliconius* and *Danaus*, where concentrations of the active compounds in the wings tend to facilitate their safe release (Boyden 1976, Brower & Glazier 1975). We need here to distinguish three aspects of aposematism: distastefulness, toxicity, and bright coloration (Harvey & Paxton 1981). Distastefulness, if produced by excreting the unpleasant substance on the outside of the prey, clearly has a strong individual advantage. Toxicity, without accompanying distastefulness and depending on delayed action within the predator's stomach, is of no individual advantage and can be selected only in kin groups (see also Ch.12). Bright colouring is the intermediate case: so long as the bright colouring is more easily remembered than cryptic colouring, a bright distasteful caterpillar is better protected than his cryptic brother in subsequent encounters *with the same individual predator*, but under most circumstances it seems unlikely that this form of selection will be strong enough to outweigh the disadvantage of attracting yet more predators by the bright colouring. In general we would expect family selection to have been influential, if not critical, in the evolution of bright colouring (but see Ch.12 for a somewhat different view).

Attempts to confirm many of the above hypotheses are likely to be hampered by further coevolution between predator and prey. The fact that warningly coloured butterfly larvae (Harvey *et al.* 1982) and even butterflies (Brown 1981, Turner 1981) are often gregarious, may not show that the gregarious habit was a precondition for the warning colour. First, in many circumstances it is advantageous for a predator that has encountered a distasteful prey, or an unrewarding 'patch' in its environment, to move on a bit before feeding again (Arnold 1978). Both this kind of predator behaviour and the effect of clumping the prey within the home ranges of a smaller number of predators (Turner 1975*b*), make it advantageous for distasteful prey to be gregarious. Although gregariousness can be individually advantageous for palatable prey, because of the 'selfish herd' effect, whereby a solitary individual is more likely to be eaten than a member of a clump (Hamilton 1971), this must often not be the case with camouflaged prey that are much smaller than their predator, who on discovering one will tend to wipe out the whole lot. Second, once bright coloration becomes the badge of toxic prey, there will be selection on predators to have an innate tendency to avoid such colours or to learn rapid avoidance of them. Schuler (1982) has recently presented evidence that there is just such an innate avoidance of 'wasp' patterns by starlings.

Any innate avoidance by the predators will produce individual selection for bright coloration. One could think of this as a very generalized kind of Müllerian mimicry. Third, there is an advantage to predators in finding toxic prey distasteful. Hence toxic prey will tend over time to become distasteful (again see also Ch.12).

The evolution of aposematism therefore seems likely to be a nested set of coevolutionary cycles between the prey and the predator, depending to some extent on kin selection but generating individual selective advantages at later stages. The whole scenario is roughly toxicity-clumping-distastefulness-blatant colour-gregariousness, with a slow trend from kin to individual selection as predators evolve in response to the evolution of their prey.

Are Batesian and Müllerian Mimicry Different?

Mimicry of the kind I am discussing (there are of course many other kinds—Wickler 1968, Vane-Wright 1976) is conventionally placed into two classes: Batesian mimicry, the resemblance between a palatable species and an unpalatable one and Müllerian mimicry, a mutual resemblance between two or more unpalatable species. Conventional wisdom states that in Batesian mimicry the model loses what the mimic gains in protection from predators, whereas Müllerian mimics all gain from the resemblance. Given that palatability is a complete spectrum, from the very nice to the very nasty, does this division make any sense? Are Batesian and Müllerian mimicry the extreme, limiting cases of a continuous range of phenomena (Huheey 1976, 1980*b*, Rothschild 1981*b*), or is it useful to divide the spectrum into two halves, say at the actual point of neutrality (Benson 1977)?

To answer this question we need to devise a system for predator behaviour (I shall write 'system' not 'model' for obvious reasons), and the answer we get will depend on how we think predators behave. Chiefly from the experiments of J. V. Z. and L. P. Brower, we know that after a number of encounters with unpalatable prey, a predator will make a 'decision' not to attack anything it 'recognizes' as this type of prey. At intervals it may make a 'mistake' and attack this kind of prey again (reviews Brower 1963, Rettenmeyer 1970, Turner 1977*a*). As it is clear that 'mistakes' of this kind allow predators to sample and adapt to a changing world, we might think of them as deliberate reversals of policy rather than as failures of memory. The opposite of course will happen to palatable prey: the predator learns to attack it after a number of pleasurable encounters. As unfamiliar prey is not automatically attacked, but is approached with some caution (e.g. Coppinger 1969, 1970, Shettleworth 1972*a*,*b*, Morrell & Turner 1970) we can think of neutral palatability *either* as the point in the middle of the spectrum where tasting the prey results in neither an increase nor a decrease in the rate of attack, *or* as the watershed between prey which provoke an 'attack' decision and those which provoke avoidance.

The simplest way of imitating (modelling) this system is by Monte Carlo simulation: imagine a jacamar sitting on a branch, catching insects. The general supply of flies and beetles keeps it moderately satisfied. The much lower density of passing butterflies (say one within reach every five minutes) is too small a part of the diet to affect its level of hunger. An unfamiliar butterfly has a 50% chance of being attacked. If it turns out to be nasty, the

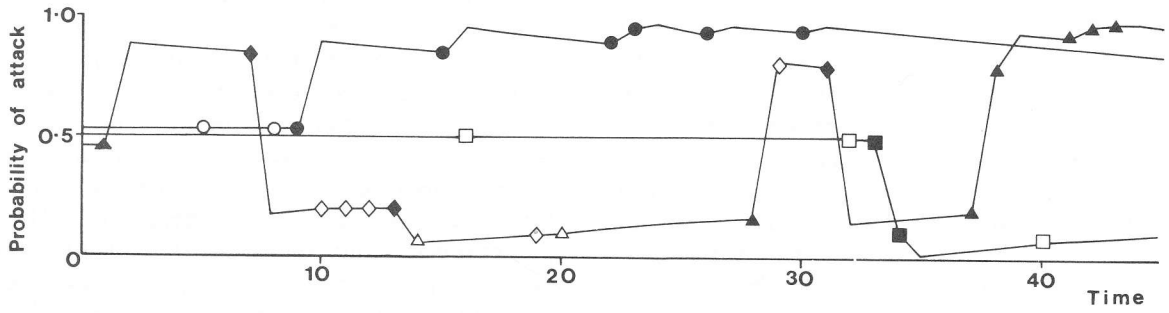

Fig. 14.1. Part of a computer simulation of a predator preying on a random sequence of four butterflies: nice (circles), nasty (squares), model (diamonds) and mimic (triangles). Graph shows current probability of the bird attacking the four types (model = mimic, as they cannot be distinguished on sight). Open symbols—prey not attacked; closed symbols—prey attacked. For example, the predator fails to attack the first two 'nasties' that it sees, then attacks the third and fourth, after which it has 'learned' to avoid them. Conversely, after ignoring the first two 'nice', it discovers that they are palatable and attacks them regularly. The model and mimic cause the predator repeatedly to 'change its mind': it attacks the first mimic, and the following model, avoids three models, makes a 'mistake' and attacks a model, avoids a model *and* two mimics, attacks a mimic, ignores one model but attacks the next, after which a 'mistake' leads it into attacking a series of mimics which appear in a cluster with no models to protect them. Original data, from simulation on a programmable calculator.

probability of subsequent attack is reduced, say to 0.5 × 0.2 = 0.1. Conversely, a pleasant butterfly increases the probability of attack to, say, 1 − (0.5 × 0.2) = 0.9. The bird also 'forgets': every five minutes the attack probabilities are reduced (or increased) to 98% of their current difference from the base line of 0.5. Figure 14.1 shows part of a computer run in which the bird encounters at equal frequency four kinds of butterfly: a nice one, a nasty one, and an indistinguishable pair of mimics. The mimic is as nice as the nice species, and the model is as nasty as the nasty species. The system, as can be seen, imitates the behaviour of a predator whose experiences build up to 'decisions' to attack or not attack a particular kind of prey, these decisions being reversed after a variable length of time.

The results of a series of runs with this system are shown in Fig. 14.2. The unpleasantness of the nasty and model butterflies is constant throughout, but the palatability of the 'nice' one and the mimic is varied right across the palatability spectrum, from inducing

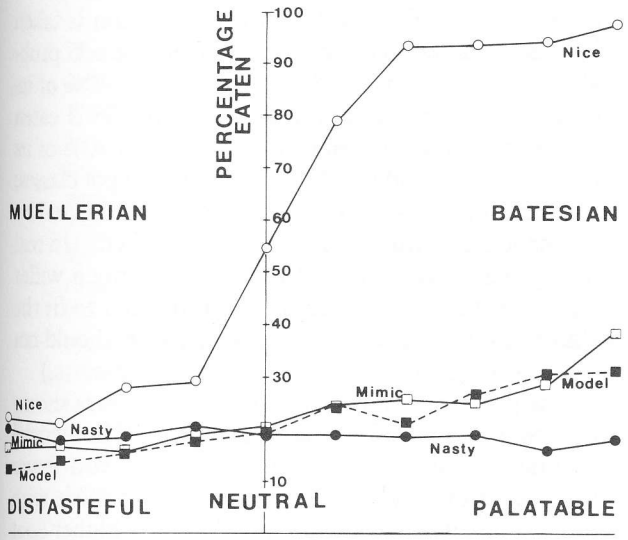

Fig. 14.2. Mimicry and the palatability spectrum. Attack rate on four types of prey, by a predator operating on the stochastic learning system shown in Fig. 14.1. The 'nasty' prey (closed circles) and the model (closed squares) are unpalatable throughout the experiment, reducing subsequent attack probability to 20% of its current value if they are eaten. The palatability of the 'nice' prey (open circles) and the 'mimic' (open squares), which is indistinguishable in pattern from the model, varies according to the values shown on the horizontal axis. To the left of the neutral point they are unpalatable, palatable to the right; at the extreme left all four prey are equally unpalatable. Note that to the left of neutrality both model and mimic are protected; to the right the mimic is better protected than the nice prey, which acts as a control, but the model is predated more than the nasty prey. Original runs on a programmable calculator, in part carried out by Ms E. Kearney as an honours project in the Department of Genetics, University of Leeds.

a 100% decision to attack, to being as unpleasant as the nasty and the model. The result is the one anticipated by conventional wisdom: when the mimic is palatable, the model-mimic pair is eaten much less than the nice butterfly, and a little more than the nasty; in the unpalatable half of the spectrum, all four butterflies are somewhat protected, but it is the model-mimic pair that receive the greatest protection (as predicted by Sheppard & Turner 1977).

The point of neutrality thus divides the palatability spectrum into two qualitatively different zones, as Benson (1977) postulated. On one side both species benefit; on the other the mimic benefits at the expense of the model. Müllerian mimicry and Batesian mimicry seem to me to be good names for the zones as the evolutionary consequences of being on one side or the other are, as I will show, rather different; although in the real, fluctuating world, a species whose palatability is in the region of neutrality will be neither quite one nor the other.

How much is this division of the spectrum into Müllerian and Batesian zones a result of the particular system which I have used? A system in which the predator decides firmly, rather than probabalistically, not to eat or to eat a particular pattern, and then reverses that decision after a particular time (the nastier or nicer the experience the longer the time) produces a result which is fundamentally the same except that the Batesian mimic and its model tend to be eaten at the same rate no matter how nice the mimic is. (The Monte Carlo system, although it works on a different computer algorithm, is basically the same system except that it allows the decision period to vary about its own mean.) If the predator makes a decision to avoid unpleasant prey not for a particular time, but until it has seen a particular *number* of these prey, then as was pointed out by Huheey (1976), who solved this system explicitly, there is no Müllerian mimicry as conventionally understood, as in a pair of mimics the nastier always suffers from the resemblance, even if its mimic is itself also distasteful. But on the whole I think it very unlikely that a predator who has decided to give Monarch butterflies a miss, starts counting the number it sees and then attacks, say, the eleventh one! Time-dependent reversal or forgetting seems much more likely to be the general rule, and therefore I believe that Fig. 14.2 represents the usual situation in the real world.

The other empirical fact about predator learning which must be understood is that the conditioned stimulus is 'generalized': that is to say the predator will treat as 'the same' not only an identical insect, but one that is somewhat like it. The probability that something is treated as 'the same' declines as its appearance departs more and more from the conditioned signal, is greater for really nasty experiences than for moderately nasty ones (Duncan

& Sheppard 1965, Goodale & Sneddon 1977), and is also heavily context-dependent: a pattern treated as a mimic of the model when a particularly attractive alternative food is available may be attacked a large per cent of the time if the alternative food is less attractive (Schuler 1974). It is important to note that generalization seems to be qualitatively the same when the conditioned stimulus is an unpleasant taste (Goodale & Sneddon 1977) as when it is an electric shock (Duncan & Sheppard 1965), so allowing some valid conclusions about mimicry to be drawn from shock experiments (Fig. 14.3).

For the purposes of this discussion, I am going to imagine the generalization of warning patterns to be constant for any one population of predators, and that predator behaviour with respect to model-mimic systems is like that shown in Fig. 14.2. This amounts to saying that I am ignoring some of the variance put into the system by such complexities of the real, fluctuating world as varying population sizes, changing availability of alternative prey, and the clumping of model populations.

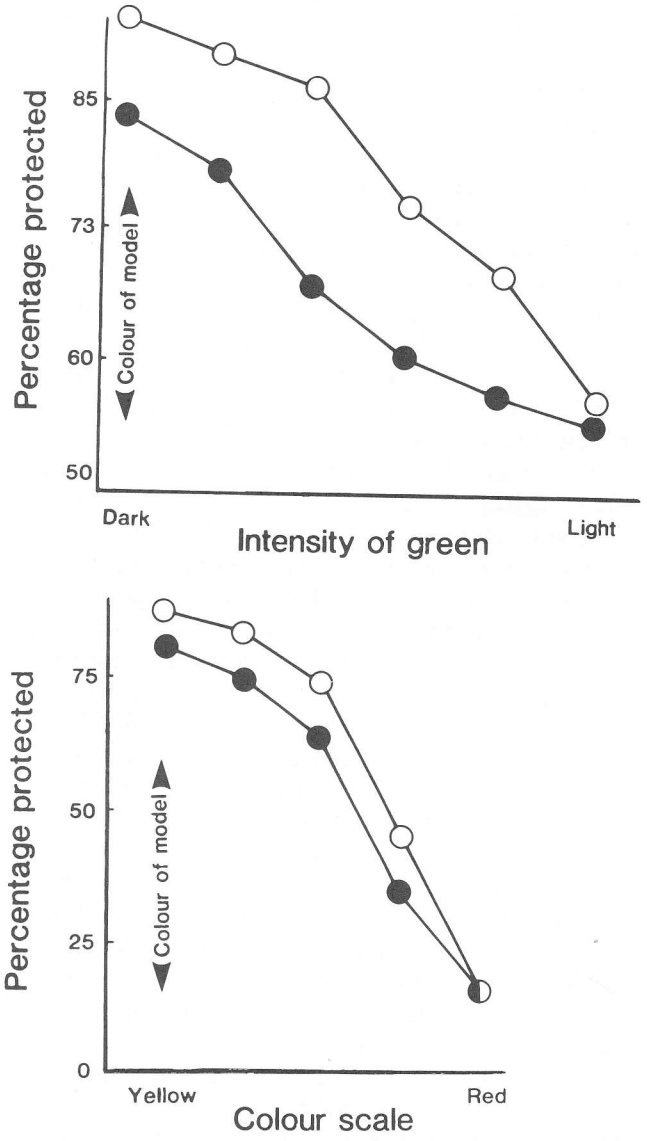

Fig. 14.3. Generalization curves produced in experiments by Duncan & Sheppard (1965) (upper) and Goodale & Sneddon (1977) (lower). The graphs show the predation rate on mimics as they depart in colour from the colour of the model. Open symbols: very unpleasant model; closed symbols: less unpleasant model.

Müllerian Mimicry—Gradual Convergence

The two systems of predator behaviour are now combined to show the way in which close Müllerian mimicry might evolve between two rather similar warningly coloured species. Both butterflies are imagined to have patterns which vary in the number of white spots on the wings, but they are not perfect mimics. Species A ranges from having no spots up to four spots, whereas the range in B is from one to five (Fig. 14.4). Densities and unpalatability, both equal in the two species, are the same as at the extreme left of Fig. 14.2, and generalization is taken into account by reducing the current attack probability for the 1 spot and 3 spot classes to 40% of its current value every time a 2 spot butterfly is eaten (the 2 spot attack probability is reduced to 20% of its current value), and similarly for all other spot classes; the flanking classes are thus somewhat protected by an encounter with the class in between them. (In real life, generalization would probably be much wider than this: the simplification, used in order to fit the algorithm into a programmable calculator, should not make any qualitative difference to the results.)

The protection afforded to the two species is shown by the heavy line in Fig. 14.4 (left). The crucial finding is that in species A the classes with fewer spots are on the whole less protected than those with more spots, whereas in species B it is the higher spot numbers that are less protected. The net result is to select for increase of spots in A, decrease in B, and, in short, for convergence of the two species onto the same spot distribution. Given that there is at least some genetic influence on spot number, the butterflies will, in the fullness of time, become very accurate Müllerian mimics.

For reasons which will become clear later, I shall not now give an actual example of this process in a wild species. However, the evolution of any mimicry which, as with Müllerian mimicry, does not involve an increase in conspicuousness, probably proceeds in small steps in this way. It is not difficult to imagine the gradual perfection of a leaf mimic from a simply-camouflaged green insect. Mimicry of snakes, almost the only kind of Batesian mimicry known in caterpillars (significantly always of cryptically coloured snakes) also probably evolves in this way. It is certainly possible to build up an

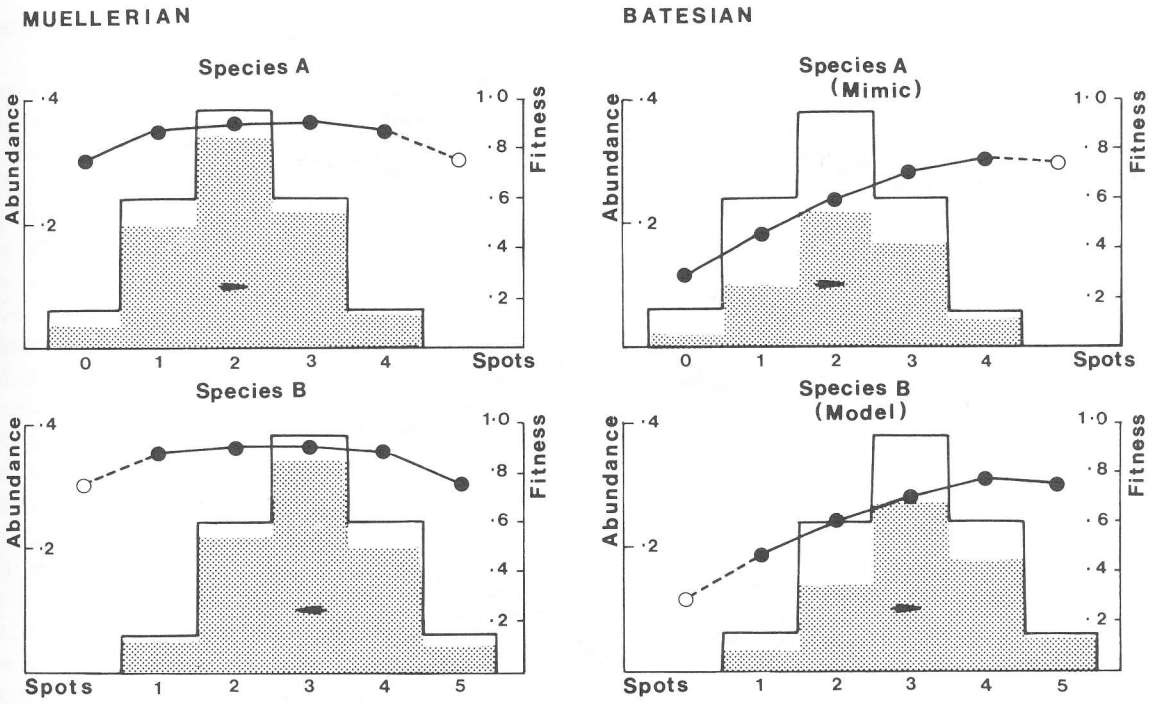

Fig. 14.4 left. Gradual evolution of Müllerian mimicry. Two species, both alike in density and unpalatability, have overlapping but not identical variation in the number of spots on the wing (outlined histograms). A predator, acting on the same stochastic learning system as in Figs 14.1 and 14.2, cannot distinguish them, but does generalize to the extent of avoiding adjacent spot-classes at 75% of the avoidance generated by the spot-class it has just encountered. The heavy line shows the 'fitness' (1 *minus* predation rate) of the various spot numbers, which is naturally the same for both species. The shaded histograms, showing the spot distribution after one round of such predation, show that the spot-distributions of the two species are converging toward each other (arrows, not to scale, show direction). One prey encountered in every time interval; other parameters the same as at the extreme left of Fig. 14.2.

Fig. 14.4 right. Gradual 'advergence' of a Batesian mimic to its model. The situation is the same as at the left, except that species A is now highly palatable (reducing probability of subsequent *avoidance* by 80% whereas the model, species B, reduces the probability of subsequent *attack* by 80%). In contrast to the Müllerian situation, both species are now selected for *increased* spot number (arrows, not to scale), but as shown by the shaded histograms (survivors after selection) the mimic is selected for much more rapid increase than the model, so that eventually both the species will have the same pattern distribution. All figures are original simulations of 5000 bird-butterfly encounters.

acceptable cryptic snake-like pattern from a number of small mutations. Take the caterpillar in Fig. 14.5a, a snake mimic not unlike the Elephant Hawk moth caterpillar, with frontally placed eye spots, and an intricate cryptic pink and brown pattern with short diagonal lines along the back such as are seen in many noctuids. The surprise is that this animal is nothing more than a domestic silkworm, but carrying four rather unusual mutations (Fig. 14.5): *Moricaud*, which comes from the wild ancestor (*Bombyx mandarina*) gives it the general, brown ground colour and the basis for the eye spots; the details of the pattern have been added by three mutations which have occurred within the domestic strain, *Zebra*, *Multilunar* and *quail*. Individually these add a little to the pattern, and it is instructive to see how few genetic changes are needed to turn a fairly plain brown caterpillar into a quite intricately patterned cryptic mimic. Aposematic colouring can also be achieved quite easily: a worm carrying *Multilunar* and *Striped* (an allele of *Moricaud*) is black with bright orange dots on each segment (Fig. 14.5g).

It is important to note, as there has been some genuine confusion on this point (Rothschild 1981*b*) that the individual genes may be identifiable (i.e. be 'major' genes) even when evolution has been gradual: whether a gene is a 'major' gene or a 'polygene' simply depends on our skill in genetic analysis; whether it produces 'minor' or 'major' changes in the colour pattern depends on its mode of action and the amount it changes the pattern relative to the amount of generalization by the predators.

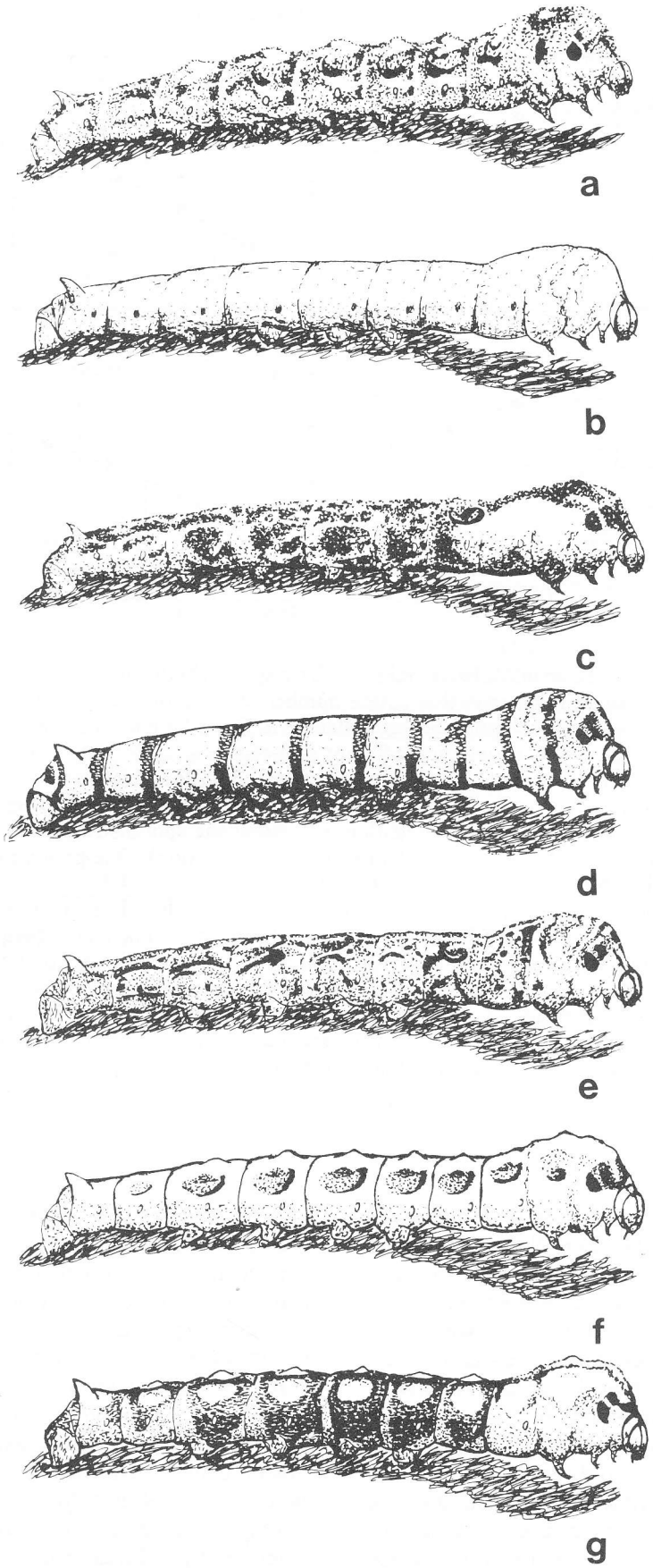

Fig. 14.5. Genetics of a caterpillar pattern which mimics a snake (a). The pattern can be built up on a white ground (b) by the four mutants *Moricaud* (c), *Zebra* (d), *quail* (e), and *Multilunar* (f). This last mutant, when combined with the mutant *Striped* (but not with the others), produces an aposematic pattern (g).

Batesian Mimicry—Two Phase Evolution

Whatever its causes, the phenotypic 'distance' between warningly coloured species and more or less cryptic, palatable prey presents a serious problem for the evolution of Batesian mimics. Or rather, it presents a serious problem for evolutionary theory: the problems of Batesian mimics appear to be less than insuperable, as there are plenty of them in the world! That there is a real 'problem' for the insects is shown by the fact, already mentioned, that Batesian mimicry of aposematic models is very rare in caterpillars, indicating that the increase in conspicuousness exerts a heavier price than the gain from resembling the model. An analogous resistance seems to occur in butterflies, in which the original non-warning colour may function as a sexual or other social signal. Silberglied (Ch.20) presents evidence that in *Colias* the male colour is used in signalling to other males (specifically the UV flash repels rivals, Silberglied & Taylor 1978) and hence may be an important component of male mating success. Female colour is known from many studies (Chs 20, 21, 23—but see also Ch.22) to affect male choice, but the single mating of the sperm storing females compared with the multiple matings of the males will make sexual selection considerably stronger, perhaps by one or two orders of magnitude, in males than in females. Sexual selection on the behaviour of the two sexes, as in most animals, will render the males much less discriminating than the females (Ch.20, Turner 1978—but see also Ch.23). Female colour is therefore expected to be subject only to weak stabilizing selection, whereas male colour, whether by selection for communication with other males, as both Silberglied (Ch.20) and Vane-Wright (1980) suggest, or alternatively because of discrimination by the females (Ch.21), is likely to be highly resistant to change. Hence the limitation of mimicry to females

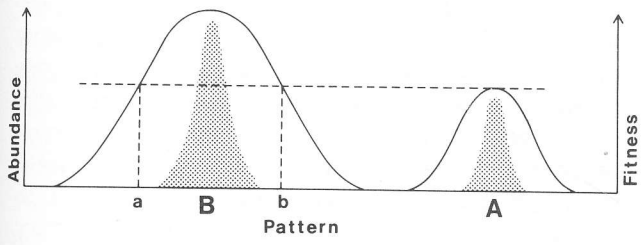

Fig. 14.6. Mimicry evolving by a single large mutation. Two species, whose phenotypes are shown by the shaded distributions, are rather dissimilar, but mimicry will evolve if species A, whose pattern has its own fitness (heavy curve) generated by whatever function (e.g. social or cryptic) it serves, can produce a mutation which need not perfectly resemble the model (B) (curve of protection generated by distastefulness and warning colour), but which has only to lie in the phenotype range *ab*. From Sheppard (1962) and Sheppard *et al.* (1984).

in a great many butterfly species provides good, although indirect, evidence that there is evolutionary resistance to the development of Batesian mimicry.

The 'problem' can be illustrated as in Fig. 14.6 (Sheppard 1962). The existing pattern has some function, which produces a peak of fitness in that part of the adaptive space. The generalization of the predators produces a peak of fitness round the pattern of the potential model. But between these is a region of the phenotype space where neither advantage can be experienced: a move into this by the potential mimic loses it the cryptic, or sexual, or perhaps thermoregulatory function of its original pattern, but fools not a single predator! The 'solution', as we all now know, is the occurrence of a mutation of comparatively large effect, great enough to place the pattern of the mimic somewhere in the region (*ab* in Fig. 14.6) where the protection given by the model confers greater fitness than that conferred by the original pattern.

Punnett (1915), working long before generalization by predators was understood, supposed that the mutation had to land the mimetic pattern bang on target, with very high quality mimicry. Goldschmidt (1945) also supposed that this happened, largely because it was consonant with other evolutionary theories which he held. Fisher (1927, 1930) on the other hand, again because the view fitted better with his other evolutionary theories, supposed that mimicry evolved gradually by the accumulation of many genes each of very small effect. It was Nicholson (1927) who proposed the compromise theory, later adopted by Sheppard (1962) and by Ford (1964), that mimicry evolves in two stages, and that once the first mutation, which may be of quite major effect, is established, the rather inaccurate resemblance it achieves can be considerably enhanced by the action of further 'modifier' genes. The only requirement is that the necessarily inaccurate mimicry produced by the first mutation confers greater fitness than whatever adaptation was served by the original pattern. When we remember that the original pattern must have been refined by thousands of generations of natural selection, it is not surprising that mimicry does not arise very often and that most species are non-mimetic.

Although the best studied mimics from a genetic point of view are the three polymorphic *Papilios* (*dardanus*, *memnon* and *polytes*), the considerable genetic complexity which arises from the polymorphism itself (see below) makes them rather poor exemplars of the major-gene/modifier system. The clearest demonstrations of the initial major mutation in Batesian mimics come from experiments with monomorphic species, of necessity conducted by rather difficult inter-species crosses. Thus Clarke & Sheppard (1955*a*) showed that mimicry of *Battus philenor* by the black swallowtail *Papilio polyxenes*

is largely produced by a single gene converting the butterfly to black from the yellow colour of its relatives. The mimicry is refined by several further loci which control the details of the yellow spotting. Significantly, the black but non-mimetic species *Papilio brevicauda* carries the major black mutation but not the modifiers (Clarke & Sheppard 1955*a*, Sheppard 1961*b*). Similarly, Platt (1975) has shown that mimetic *Limenitis* differ from their non-mimetic relatives by a major mutation which wipes out the 'white admiral' bars which are the most prominent feature of the non-mimics.

Although in the polymorphic *Papilios* the original major mutation cannot be unambiguously identified, they do provide excellent evidence for the existence of modifying genes which specifically improve the mimetic pattern, and that pattern only. Thus the Ethiopian population of *P. dardanus* is polymorphic for mimetic forms and for a yellow non-mimic. The models are tail-less, but all the local forms of *dardanus* have tails, which presumably serve an aerodynamic function and must be favoured by selection in the males and the yellow females. By an elegant study, Clarke & Sheppard (1962*b*) showed that the population contained genes which specifically shortened the tails of the mimetic forms, but not of the non-mimetic yellow females. They were later able to demonstrate an analogous set of modifying genes in *Papilio polytes*, this time specifically *increasing* the length of the tails in the mimics, which copy a distasteful *tailed* swallowtail (Clarke & Sheppard 1972). Similar *specific modifiers* (Charlesworth & Charlesworth 1976) were found altering the colour pattern of a form of *P. dardanus* in parallel with the geographical cline of its model, *Amauris niavius*, for the amount of black on its wings, and the colour pattern of a form of *P. polytes* in response to geographical variation in the model, *Pachliopta aristolochiae* (Clarke & Sheppard 1960*b*, 1972).

It may now seem *passé* to say, yet again, that these results do not accord with Goldschmidt's theory that mimicry would arise by systemic mutations which immediately produced high grade mimicry. But the revival of interest in Goldschmidt's ideas (e.g. Gould 1980) makes it important not to ridicule him (that particular sport being long out of fashion), but to show where his ideas were wrong, in order to sort out those parts of his work which can be profitably pursued. On mimicry, Nicholson (1927) was the one who was closest to the mark, and Goldschmidt even cites Nicholson in support of his own theory. Batesian mimicry usually evolves in two phases: the establishment of a major mutation which produces only rather poor mimicry, followed by the selection of further genes which refine the resemblance of the mimic to the model. I have emphasized this point again, because in thinking about problems connected with the evolution of mimicry it is most important to keep these two processes separate in one's mind. The two phases involve very different phenomena.

Müllerian Mimicry— Two Phase Evolution Too?

We have now arrived at what, after the seminal work of Clarke and Sheppard, has been seen as the conventional picture of the evolution of mimicry: Batesian mimicry evolves by the two phase process; Müllerian mimicry can evolve by gradual convergence (Fig. 14.4). It was Fisher (1930) who produced an argument to show that Müllerian mimicry can arise in this way, as part of his attempt to defeat the saltational theories of Punnett. Fisher's presentation gives the impression even after quite careful reading that he has proved that Müllerian mimicry does not evolve by major mutations, although in fact he never says as much. Thus Sbordoni *et al.* (1979) noting that the moth *Zygaena ephialtes*, which they had shown to be distasteful, had used major gene mutations to produce its mimetic pattern, described it as a mimic with a mixture of Batesian and Müllerian properties.

Consider a rainforest, with a great diversity of warningly coloured butterflies, presenting many different patterns. Those resembling each other closely enough will converge, by the process described in Fig. 14.4, until they are good Müllerian mimics. In this way, like planets forming from a cloud of gas, clusters of mimetic species will arise, and form what we call mimicry 'rings'. Species occupying the spaces in between the rings will be pulled into them, but sooner or later these focal patterns, having absorbed all the available species, will stabilize. If they differ too much from each other they will not be able to converge, for birds will never mistake one for the other (Sheppard *et al.* 1984). Hence, in the South American rainforests there are no fewer than five different mimicry rings, all very distinct to the human eye, and probably to the avian eye as well (Papageorgis 1975) (Fig. 14.7). Likewise in the West African rainforest there are two butterfly rings (Owen 1974*b*) and among European bumblebees two rather distinct patterns, each comprising several species (Plowright & Owen 1980). Whether the little scenario I gave for the origin of these rings is correct is not very important—what matters is that the presence of all five of the South American butterfly rings throughout the American tropics (with some geographical variation), argues that they are persistent and relatively stable in evolutionary time.

Once distinct rings of this kind form, then further Müllerian mimicry will arise in the same way as Batesian mimicry. Fig. 14.6 could equally represent two protected, warningly coloured species. If the less protected of them (A) can produce an approximate

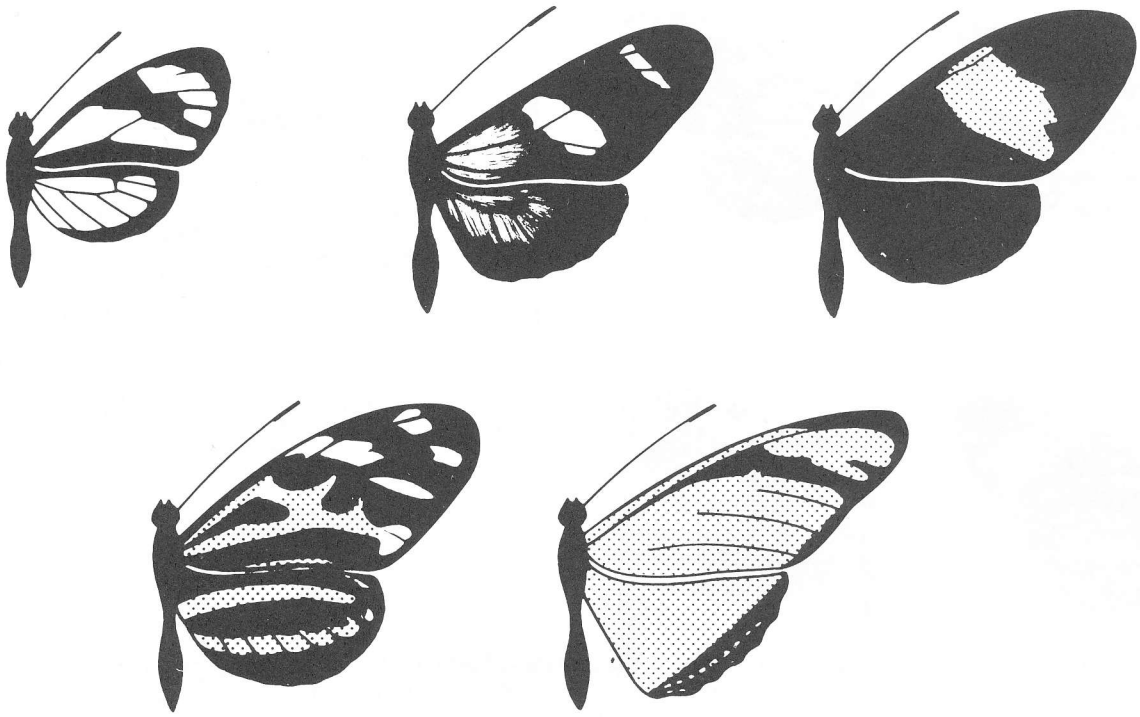

Fig. 14.7. The five mimicry rings to which most of the long winged aposematic butterflies of the South American rain forest belong, represented here by one species from each ring, as they appear in Trinidad. From Sheppard *et al.* (1984).

resemblance to the better protected one (B), then the two will become Müllerian mimics by a two stage process which, as with Batesian mimicry, will involve the substitution first of a major mutation, and second of an indeterminate number of modifiers, some of which may of course be individually detectable mutations themselves, which refine the resemblance.

A rather clear example of the first step was discovered by Keith Brown and Woodruff Benson, in company with Philip Sheppard, in the population of *Heliconius hermathena* near Faro on the Amazonas (Fig. 14.8). In most of its scattered colonies in the Amazon basin this butterfly is not a Müllerian mimic. It has its own stable pattern of yellow bars and red splotches, and is apparently so different from the other sympatric *Heliconius* that no single mutation can initiate mimicry. But at Faro it flies with a local population of the polytypic *Heliconius melpomene* (see Fig. 14.9) which differs from normal *hermathena* only in lacking the yellow bars. In this one population a single gene which largely removes the bars has risen to a high frequency, making *hermathena* and *melpomene* into quite good Müllerian mimics (Brown & Benson 1977).

However, research in *Heliconius* has to yield precedence to studies on the European moth *Zygaena ephialtes*, most recently reviewed by Sbordoni *et al.* (1979). We are fortunate in having excellent studies not only of the genetics of this species (Bovey 1941 and later, Dryja 1959), but also of its ecology and behaviour, and of the reactions of predators (Bullini *et al.* 1969, Sbordoni & Bullini 1971).

In northern Europe the moth is a comimic of other distasteful members of its own genus; in much of southern Europe it has departed from this certainly ancestral pattern and become a Müllerian mimic of the sympatric moth *Amata phegea* (Ctenuchidae). Crosses between the northern ancestral populations and the southern *Amata*-mimics show that most of the difference resides in two genes, one converting the red colour to yellow, and the other (unlinked) increasing the black (or dark green) markings and converting most of the spots on the wings to white. (Dryja (1959) believed, as all subsequent commentators have noted, that this 'gene' was a complex of two loci, and that some aberrant moths appearing in his broods were the result of crossing over between them. Dryja did not publish the data for this conclusion in full and as his material was destroyed by military action, it is unfortunately impossible to verify it.) There are then two possibilities (if we exclude the rather improbable eventuality that near-perfect mimicry was produced by both mutations occurring at the same time): either the pattern or the colour must have changed first. Thanks to experimental and field work by Sbordoni, Bullini and their colleagues, we know as certainly as one can know anything in evolutionary biology that the first change was in the pattern. Changing the colour from red to yellow produces a bright

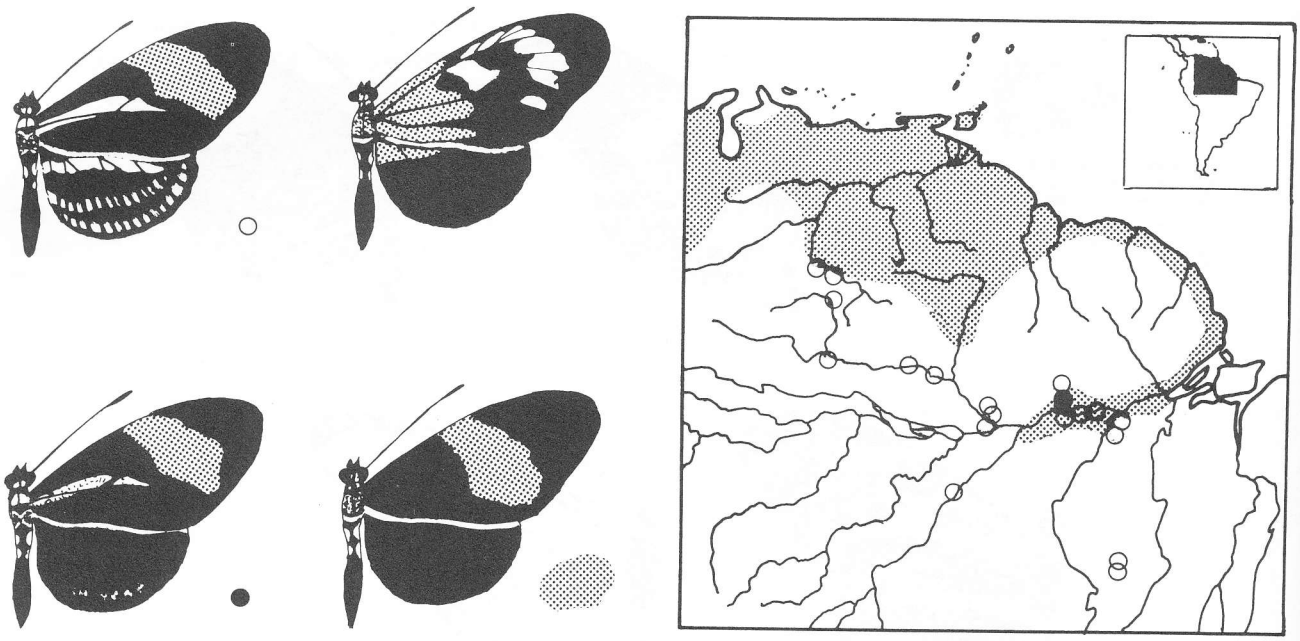

Fig. 14.8. Evolution of approximate mimicry by a single mutational step in *Heliconius hermathena*. The most widespread form of this localized Amazonian species (top left) bears no resemblance to the related species, such as *H. melpomene* and *H. erato* (top right) with which it flies. But at one locality (Faro) where it flies with *melpomene* and *erato* races (bottom right) from which it differs only in having yellow bars, a single mutation has largely removed the yellow bars from a majority of the population of *hermathena* (bottom left).

yellow *Zygaena* moth that does not look remotely like *Amata phegea*. On the other hand, changing the pattern without changing the colour produces a moth that bears a passable resemblance to *phegea*, except for some small marks near the thorax and the stripe on the abdomen being the wrong colour. Now this is known to be an adequate mimic of *phegea*, not only from experiments with caged birds, but from the fact that in an unusual population consisting entirely of this form, the moths adjust their posture to correspond with whichever of the other comimics (*Zygaena* spp. or *A. phegea*) is in flight at the time (Sbordoni & Bullini 1971). This is very good evidence that wild birds treat this form as a mimic both of *A. phegea* and of the other *Zygaena*.

Thus in terms of the system for the evolution of Müllerian mimicry in Fig. 14.6, one must imagine that mimicry was initiated by the pattern mutation, which carried *ephialtes* into the region of the phenotype space which was protected by *phegea*, without in this case totally losing the protection of the *Zygaena* mimicry ring: presumably the *Zygaena* and *Amata* protection curves overlap slightly. In most southern populations, this mimicry has been further refined by changing the colour from red to yellow, a change a geneticist would think of as a major gene, but which, as only a very few marks on the moth are now coloured, is in terms of the perceptual generalization of the predators, a rather minor change.

It could be that both *Z. ephialtes* and *H. hermathena* are unusual in using a rather large mutation to initiate mimicry, and that most other Müllerian mimics evolved by convergence of the gradual kind from patterns that were already rather similar. But in two other *Heliconius* species at least, changes in the mimetic pattern have involved genes which appear to be major to us, and probably also to their predators. *Heliconius melpomene* and *Heliconius erato*, almost always strict parallel mimics of one another, have diverged to an astounding degree *within* each species, so that their score of geographical races have only relatively recently been correctly assigned to just the two species (Fig. 14.9) (Emsley 1965, Turner 1965). Our genetic studies of six races of *melpomene* and eight of *erato* show that a relatively small number of major changes are involved in differentiating each race (Table 14.1), but, as predicted by the two stage theory, there are further minor genes which alter the expression of the major mutations. For example, the red rays of the Amazonian races of *melpomene* (produced by a single dominant allele) become much wider when placed on the genetic background of the East Brasilian race, which lacks these rays; in such a backcross hybrid, the rays are rather like the markings of two close relatives *Heliconius cydno* and *H. ethilla*. Thus the Amazonian races carry genes whose specific function is to make the rays narrow, in mimicry of *H. erato* and of several other *Heliconius* species.

As Dixey (1909) pointed out, there are two factors that determine which of a pair of distasteful species will be the better protected, and hence act as the model: distastefulness and abundance, a common

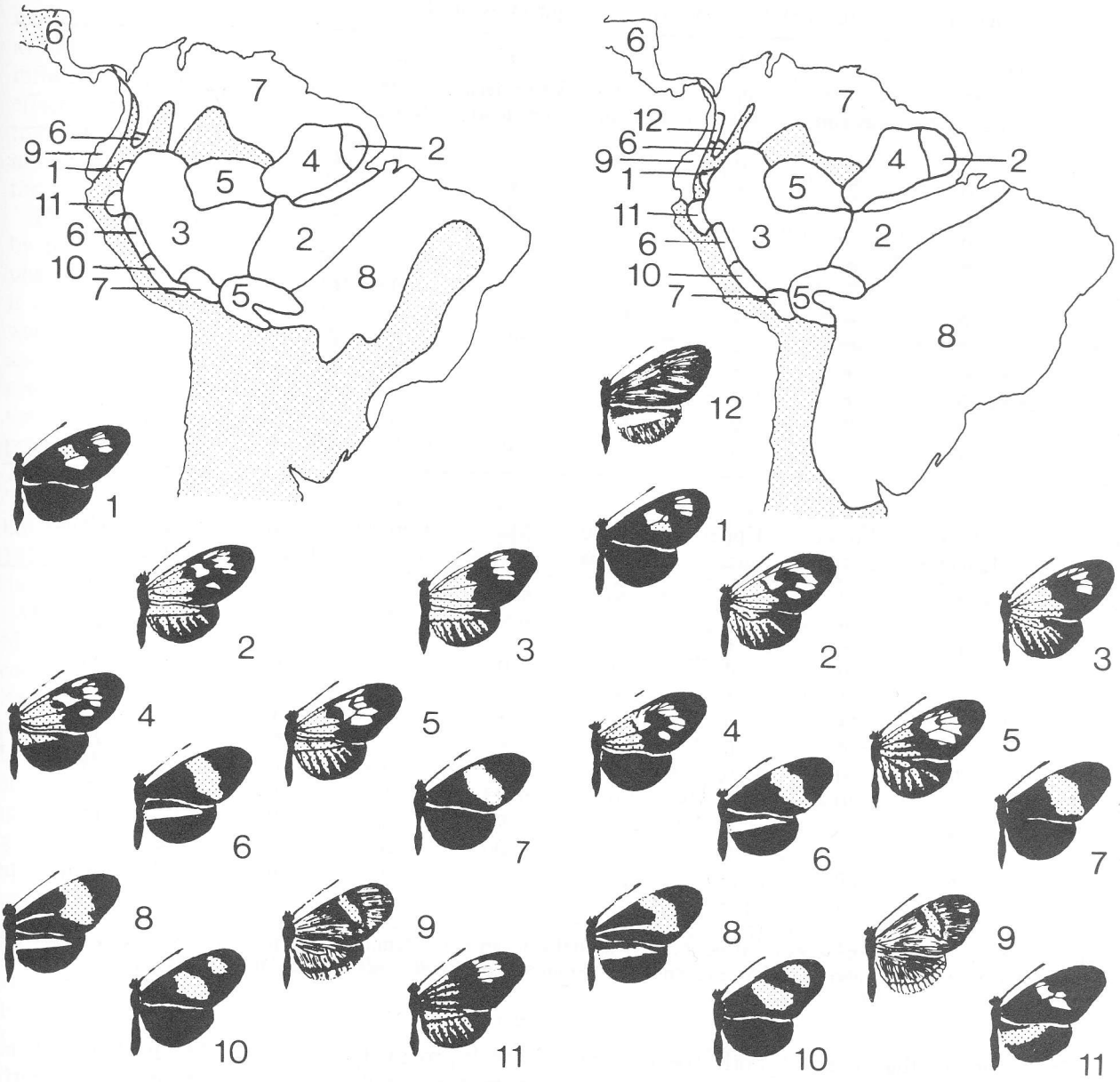

Fig. 14.9. Parallel Müllerian mimicry of the races of *Heliconius melpomene* and *H. erato*. From Turner (1981) courtesy of Annual Reviews Inc.

species being much better protected than a rare one. Sbordoni *et al.* (1979, also Bullini *et al.* 1969) have argued convincingly that although *Zygaena ephialtes* may in fact be nastier than *Amata phegea*, the much greater abundance of *Amata* (compared with *Zygaena*) which persists in undisturbed biotopes in Italy, and may have been general in southern Europe at the end of the glaciation, has caused *ephialtes* to converge to the pattern of *phegea*. Similarly we have postulated (Sheppard *et al.* 1984, also Turner 1977*b*, 1982) that the divergence of races within each of our two *Heliconius* species has been driven largely by marked spatial alterations in the abundance of the various warningly coloured butterflies of South America when the rain forest became fragmented into more or less isolated refuges during the latest cold dry period of the Pleistocene (Fig. 14.13), with *melpomene* and *erato* tending to mimic whichever was locally the most abundant and protected mimicry ring. (For reviews of Quaternary climate and vegetation in South America, see Prance 1982.)

Müllerian and Batesian Mimicry—Convergence or Advergence?

It is now important to bear in mind the distinction between the two phases of evolution. Although Batesian and Müllerian mimicry can and do evolve by the same process in the first phase, with the palatable or less protected species converging by a single mutation onto the better protected model (it

Table 14.1. Genetics of six races of *H. melpomene* and eight races of *H. erato*.

H. melpomene Race	1 East Ecuador	2 Lower Amazon	3 Upper Amazon	4 Guiana/ Manaus	7 Venezuela/ Trinidad	8 East Brasil	Gene
	+	0	0	0	+	+	*D*
	0	0	+	+	+	+	*R*
	0	+	+	+	0	0	*B*
	0	+	+	+	0	0	*N*
	0	0	0	0	0	+	*Yb*
	+	+	0	+	+	0	*C*
	0	+	+	0	0	0	*O*
	0	0	0	0	+	0	*F*
	0	+	+	+	+	+	*Rr*
	0	+	+	+	+	+	*S*
	+	0	0	0	0	0	*T*
	+	0	0	0	0	0	*Wh*

H. erato Race	1 East Ecuador	2 Lower Amazon	3 Upper Amazon	4 Guiana/ Manaus	5 Mato Grosso	6 Central America	7 Venezuela/ Trinidad	8 East Brasil	Gene
	+	0	0	0	0	+	+	+	*D*
	+	0	0	+	0	+	+	+	*R*
	0	+	+	+	+	0	0	0	*Y*
	0	0	0	0	0	0	0	+	*Yl*
	0	+	+	+	+	+	+	+	*St*
	+	+	0	+	+	+	+	+	*Sd*
	+	0	0	0	+	+	+	+	*Ly*
	0	0	0	0	0	0	0	+	*Cr*
	0	0	0	0	0	+	0	0	*P*
	0	+	+	0	0	0	0	0	*Or*
	+	0	0	0	0	0	0	0	*Ur*
	+	0	0	0	0	0	0	0	*Wh*
	0	+	+	+	+	+	+	+	*Ro*

+ indicates that this race has the recessive allele at this locus; 0 that it carries the dominant. The patterns produced by these alleles are shown in Fig. 14.9, which also indicates the race numbers. From Sheppard *et al.* (1984); also in Turner (1981, 1983*a*).

is fairly obvious that even if both species are distasteful, a mutation of the better protected species that resembles the less protected is at no advantage), the events in the second, 'modification' phase will be different. Müllerian mimics will converge mutually on some intermediate pattern; the model of a Batesian mimic, as has been frequently pointed out, is placed at a disadvantage by the mimicry, and should evolve away from the mimetic pattern. Brower & Brower (1972) coined the term 'advergence' for this process whereby mimic and model are involved in an 'arms race' (Dawkins & Krebs 1979) which the mimic somehow wins by evolving towards the model faster than the model can evolve away.

To the question 'how does the mimic win the race?' there are I believe two valid answers, both well expounded by Nur (1970), and dependent on considering the two distinct phases in the evolution of mimicry. Take first the gradual 'modifier' phase in which the mimic will evolve slowly toward the model. A hint as to why the model evolves at a slower rate is provided by Fig. 14.2: the advantage of being a mimic (compare predation on the nice butterfly with predation on the mimic) is considerably greater than the disadvantage of being a model (compare predation on the model with predation on the nasty butterfly). To show that this effect does indeed result in the mimic catching up with the model I have again simulated the situation of two species with varying numbers of white spots: all the conditions are the same, except that the species with the smaller number of spots is now a palatable Batesian mimic. Fig. 14.4 (right) shows that the rate of predation on each spot class is now skewed in such a way that both species are subject to selection for greater spot numbers, but that the mimic is much more strongly selected than the model. In fact for the particular numerical values used in this simulation, the mean spot number of the mimics will move during one generation of selection from 2.00 to 2.22, whereas the model mean moves only from 3.00 to 3.12, so that the 'gap' between the species has narrowed by 0.10 of a spot.

Given that spot number is inherited, both species will slowly increase their spot number, but stabilize as the mimic catches up with the model. It is not difficult to see that this result is a general one: the effect of the mimic is always to make phenotypes 'to the right' fitter, but as the model's phenotype is nearer to the right than is the mimic's, selection on the model will always be the weaker.

The model of course could escape from the mimic by producing a major mutation which carried its phenotype right away from the present pattern. But it cannot do this successfully, as the new mutant would be rare, would not be recognized by predators, and would be sampled by them: in warning coloration, nothing succeeds like being common (see the experiment by Benson 1972 on the increased predation rate on *Heliconius* with altered patterns). Thus the first phase of evolution, a large mutational 'jump', can be used by the mimic to initiate mimicry, but not by the model to escape (Nur 1970, Sheppard 1975). The only chance for the model is to make a 'jump' into a different, well-protected mimicry ring.

One further difference between Batesian and Müllerian mimicry is brought out by the two computer simulations: the final rate of convergence. Remember that all the parameters, except the palatability of the 'mimic', are the same in Fig. 14.4. In the Batesian case, as has been said, one generation of predation narrows the difference between the species by 0.10 of a spot. In the Müllerian case one species increases by 0.028 of a spot, and as both are equally unpalatable, the other declines by the same amount; thus the difference narrows by only 0.056 of a spot. The long-term response to these selection pressures will be determined by the genetic architecture of spot number, but again assuming that this is the same in the two cases, it is clear that the Batesian pair are converging nearly twice as quickly as the Müllerian pair. Marshall (1908) and many later students of mimicry have suggested that Batesian mimicry is expected to be the more accurate and it is said (although it would be very difficult to quantify) that Batesian mimics often show astoundingly close resemblance, whereas Müllerian mimics often show only a general similarity (e.g. Fisher 1930).

Even with equal heritabilities, the convergence of a Müllerian pair will not in general be equal: the less abundant or more palatable species will evolve faster and farther than the commoner or more distasteful one (Fisher 1930). In the limit, when one species is very rare (or of course, when it is of neutral palatability), this one will do all the evolving and the 'model' will remain unaltered. Whether this has happened in any *Heliconius* species we do not yet know, but on taxonomic grounds it is likely that few if any changes have occurred in *Amata phegea* as a result of its association with the normally much rarer *Zygaena ephialtes* (Sbordoni *et al.* 1979). It is sometimes suggested that the nastier of a pair of Müllerian mimics will evolve 'away' from the association, like the model in a Batesian system: Rothschild (1981*b*) for instance says that local and temporal variation in toxicity 'will mitigate against [*sic*] the tendency for the more poisonous species to, willy nilly, evolve away from the less poisonous and thus be forced out of the desirable companionship of its co-mimics.' It should now be clear from the two limiting cases of Müllerian mimicry (equal nastiness, or one mimic neutral) discussed that, given the system of predator behaviour postulated here, there is no such tendency for one of the Müllerian mimics to 'escape'. Unequal nastiness simply results in unequal but nonetheless mutual convergence.

Batesian Mimicry—Preadaptation

The frequency-dependent effect on aposematic species just mentioned is inevitably reversed for Batesian mimics: the commoner they become the less benefit accrues from the mimicry. The result is well known: mutations producing mimicry of another model are at an advantage when rare, so that the species tends to become polymorphic (Ford 1971). In a study of the first importance for our understanding of mimicry, Charlesworth & Charlesworth (1976) have investigated what happens to such mutations if they arise in a species which is already polymorphic for a mimetic and a non-mimetic form. If the new mutant is unlinked, or only loosely linked to the major gene which originally produced the mimicry, then only two outcomes are possible: either the new mimic is so advantageous that both the major allele and the new modifier spread to fixation, so converting the species to monomorphic mimicry of the new model, or the new mimic is not advantageous enough, and the new mutation does not spread. *Papilio dardanus* provides an example of this system: in subSaharan Africa both mimicry and loss of tails (unlinked genes) have reached fixation; i.e. all *dardanus* are tail-less and mimetic. In Ethiopia there is a polymorphism with non-mimics at 60–80% of the population, and here the *tail-less* allele has been unable to spread: both mimics and non-mimics have tails on the wings.

On the other hand, if the new mutation happens to be rather closely linked to the original major gene, then both loci remain polymorphic, and the butterfly becomes a polymorphic mimic of two different models. If further linked mutations occur in the vicinity of the others, producing mimicry of yet more models, an extensive poly-morphism builds up, in which the forms are controlled by clusters of tightly linked loci which at first sight appear to be multiple alleles at one locus. Thus in the highly polymorphic

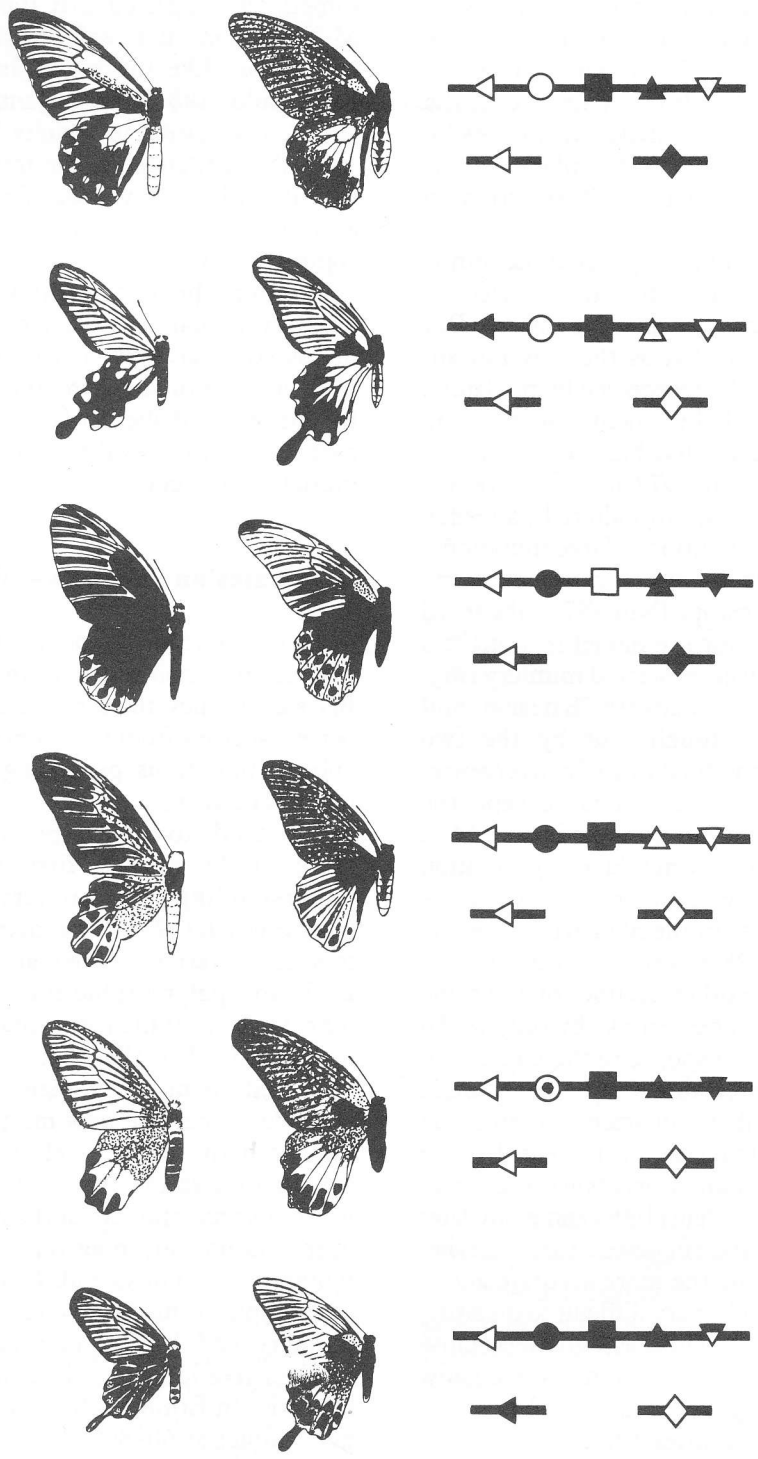

Fig. 14.10. Six of the forms of the batesian mimic *Papilio memnon* (centre) with their models (left). The diagrams (right) represent the way the forms are built up from combinations of a limited number of genes: ◁ tails (closed symbol, present; open, absent); ○ hindwing pattern (closed, dark; open, with extensive white marks; dot, with less extensive white); □ forewing (closed, dark; open, with white apex); △ 'shoulder' flash (mimics head or thorax colour of model—closed, red; open, white); ▽ abdomen (closed, black; open, yellow; particoloured, black with yellow tip); ◇ yellow suffusion of hindwing (closed, present; open, absent). Black bars represent chromosomes. Five of the genes (or six if one takes into account data showing that the 'gene' for hindwing pattern is at least two genes) are very tightly linked, and behave as if they were multiple alleles of a single locus. The remaining two are unlinked. Data from Clarke *et al.* (1968) and Clarke & Sheppard (1971).

Papilio memnon the occurrence of rare crossovers reveals that the cluster contains at least six loci (a 'supergene') affecting the colour and morphology of different parts of the wings and body (Fig. 14.10) (Clarke *et al.* 1968, Clarke & Sheppard 1971). In these circumstances, as Fisher first pointed out, selection will favour tightening of linkage between the loci. Evidence from the silkworm indicates that Lepidoptera have factors which exert very strong control over recombination in their own chromosome (Turner 1979, Ebinuma & Yoshitake 1982) and hence this tightening of linkage may be most effective (in males only of course; there is no recombination in females—Turner & Sheppard 1975). In *Papilio dardanus* and *Papilio polytes* the linkage has become so tight that the nature of the individual loci can no longer be discovered, given the limits on the number of offspring obtainable in butterfly breeding experiments (Clarke & Sheppard 1960*d*, 1972).

The curious and interesting thing about these findings is that the Fisherian theory of modification does not entirely account for the supergene: the loci involved must be fairly closely linked from the start, and the alternative view that selection will bring together loosely linked or unlinked loci to form the supergene is no longer tenable (see also Rothschild 1981*b*). Many people will find it improbable, if not smacking of special creation, that the loci should just happen to be appropriately linked. It is indeed improbable, and that is why there are so few spectacularly polymorphic mimics among butterflies: only a few species happen to have clusters of loci controlling wing pattern functions. These are the ones that attract our attention by becoming polymorphic: in a way we are performing a biased experiment. Even so, the 'successes' do not have things one hundred per cent in their favour; whereas *P. memnon* happens to have a tail locus linked to the colour pattern genes, *P. dardanus* and *P. polytes* do not, and consequently adjust less accurately to the presence of tailed and tail-less models.

The origin of supergenes in mimetic butterflies is in short an example of something I like to call a 'sieve': an evolutionary mechanism that picks out those species, or genes, which happen to have the required properties. The simplest example is provided by the dominance of most of the genes for industrial melanism in moths: although both dominant and recessive melanic mutations occur, it is the dominant ones that increase faster under natural selection and hence become the predominant industrial melanics (Haldane 1924, Sheppard 1975).

It is clear that a sieve operates also on the evolution of the colour patterns in mimetic butterflies. For

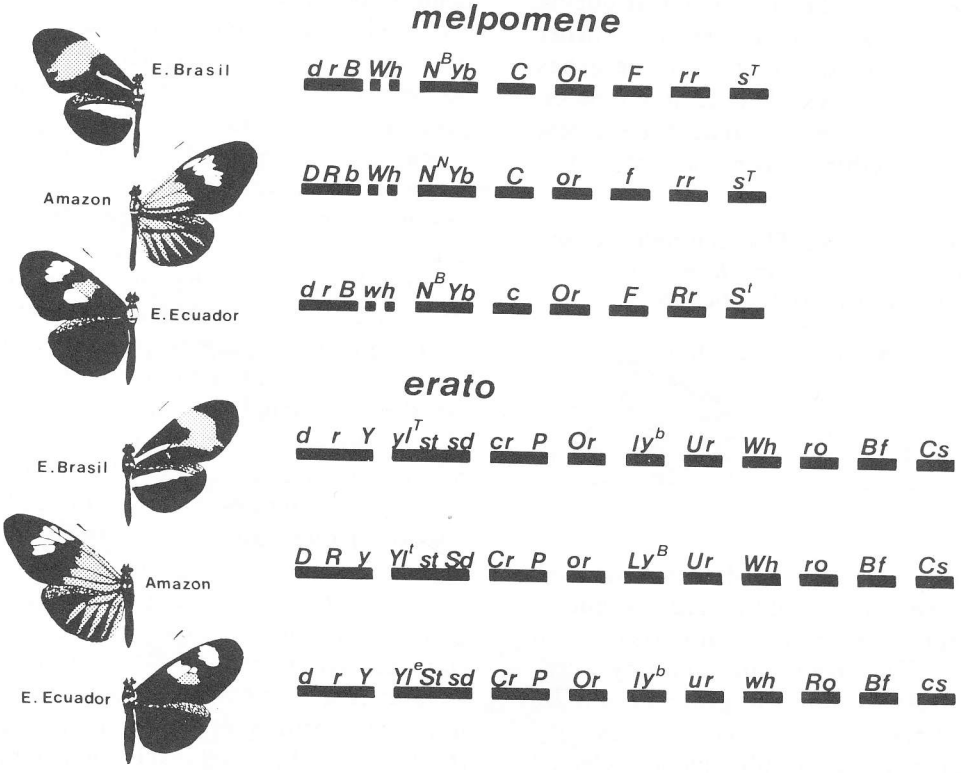

Fig. 14.11. The genetics of three parallel races of *Heliconius melpomene* and *Heliconius erato*. The way in which the alleles shown here combine to produce other races is shown in Table 14.1, where the dominant alleles are indicated by 0 and the recessive by +; in this figure dominance is indicated by a capital letter. Heavy bars are chromosomes. Data from Sheppard (1963), Turner (1972) and Sheppard *et al.* (1984).

example, when the three forms of *Papilio dardanus* which mimic *Amauris niavius*, *Amauris echeria* and *Amauris echeria septentrionis* are crossed into races where the forms do not occur, the genes producing the forms cease to be distinguishable: they produce, all three, the same pattern (Clarke & Sheppard 1960*c*, 1962*a*,*b*, 1963). Now, suppose that the *A. niavius* mimic was the first one. If the mutant converting this to *A. echeria* had occurred on *this* genetic background it would not have spread, for it would not have produced a new pattern, and likewise for the mutant changing this form to mimic *A. e. septentrionis*. In short, for these new forms to appear, some of their distinctive 'modifiers' must have existed in the population *before* the mutation occurred. The evolution of mimicry involves also a certain amount of preadaptation, or luck.

The occurrence of the fortunate and fortuitous linkage required for the evolution of supergenes is seen in *Heliconius*, in which a few of the genes controlling the mimetic patterns are linked, some of them rather closely (Fig. 14.11) (Sheppard *et al.* 1984). There is no balanced polymorphism in these species, and thus no prolonged period in which selection could produce, or substantially increase, the linkage between loci. A possible explanation for the linkage is that we expect a rather weak sieving effect which causes linked rather than unlinked modifiers to spread in the population if the mutations arise during the period when the major gene is spreading (Turner 1977*a*). However, as the distribution of the genes on the chromosomes is an excellent fit to a Poisson distribution (Table 14.2), there is no need to suppose that the linkage is anything other than random.

Table 14.2. Fit of distribution of loci controlling colour patterns between chromosomes in *H. melpomene* and *H. erato* to a random (Poisson) distribution.

	No. of loci per chromosome				
	0	1	2	3	TOTAL
Observed cases	24	12	3	3	42
Expected cases	22.0	14.2	4.6	1.1	39.9

I have said that what we need to do with the theories of Punnett and Goldschmidt is not to ridicule them, but to determine what parts of them might be correct. The theory that mimicry arises perfect from the start is shown in general not to be true: both Müllerian and Batesian mimics give evidence for the occurrence of subsequently selected modifying genes. But the theory that homologous genes could produce the same pattern in model and mimic, suggested in that simple form by Punnett (1915) and in the form of homologous developmental pathways by Goldschmidt (1945), is to some extent confirmed by a comparison of the genetics of *Heliconius melpomene* and *H. erato*. It seems too much of a coincidence that these two butterflies, which although not sister-species are still quite closely related, should both have a single linkage group which controls the yellow colour of the forewing band, the 'Dennis' marks on the forewing, and the rays on the hindwing, and probably in each case comprising three loci (*B*, *D*, *R* in *melpomene*, *Y*, *D*, *R* in *erato*), and another linkage group which in both species controls the yellow hindwing bar and white hindwing margin (Fig. 14.11). This is good evidence that homologous genes are being used in both butterflies. But the whole of the resemblance between the two species is not produced in this way. The superficially similar forewing bands of the East Ecuadorian races (Fig. 14.11) are produced by genes whose mode of action is quite different: there is for instance no analogue in *melpomene* of the *Ro* gene which rounds out the tip of the band in *erato*.

The problem with the homology hypothesis is not that it is wrong, but that Goldschmidt tried to make it explain too much. The less closely related are a pair of mimics, the less likely it will be that they will use homologous developmental pathways (Nicholson 1927). To some extent I have cheated with the silkworm mimic of the snake (Fig. 14.5); the ease with which this intricate pattern is produced is no doubt in part due to the fact that, on the background produced by the *Moricaud* gene introduced from the wild ancestor *Bombyx mandarina*, the effect of the mutations in *B. mori* may be to evoke developmental pathways which existed in the wild ancestor. The delicate reticulate pattern added by the *quail* mutation is not produced simply by that mutation, but by the interaction of *quail* with many other genes in the genome. The cryptic snake pattern is therefore not produced merely by the four mutations shown in Fig. 14.5, but by a considerable array of genes controlling developmental pathways, some of which may have been modified during the history of wild *Bombyx* to produce refined cryptic patterns. In fact, as even domestic *mori* retain the display behaviour appropriate to a snake mimic (which of course looks utterly meaningless when performed by a white silkworm), there is a strong presumption that the wild ancestor *was* a snake mimic.

Thus, although homologous developmental pathways cannot entirely explain mimicry (who would suppose that snakes and *Bombyx* caterpillars share a significant number of pathways?), there are two important principles implicit in Goldschmidt's thinking which, in the excitement which followed on disproof of his saltational theory, have been overlooked. First, a single mutation may be able to produce a refined and well-adapted pattern if it switches on again a hidden developmental pathway that has been refined by the selection of modifiers

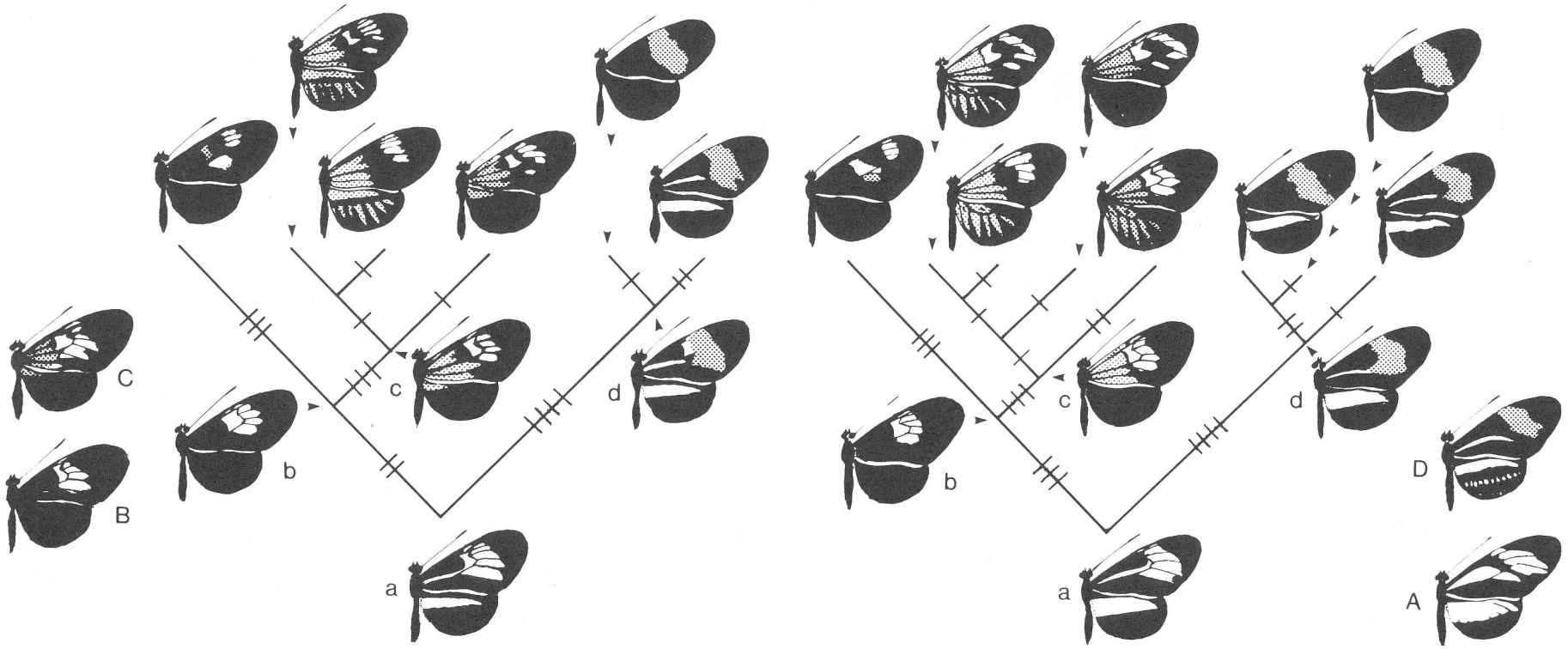

Fig. 14.12. Cladogeny of some races of *H. melpomene* (left) and *H. erato* (right). Minimum trees have been constructed using the genetic data in Table 14.1, by applying the dominance-sieve theorem and the weighted invariant step method (Farris *et al.* 1970). Of several equal length trees obtained, the most geographically probable have been selected for illustration. Reconstructed ancestors (a, b, c, d) may have been Müllerian mimics of patterns which are still extant in the relict species A, B, C, D (*H. nattereri*, *H. timareta*, *H. elevatus*, *H. hermathena*). Cross bars, placed conventionally in the centre of the branch, denote the substitution of a major gene mutation.

at some time in the past and then switched off. The selection in domestication of the *plain* alleles which remove all colour from the silkworm must have left largely intact, though subject to mutational damage, the developmental pathways, controlled by other gene loci, that produced the cryptic coloration of the wild ancestor. Second, more generally, the effect of a particular mutation, and therefore its success or failure in producing a new adaptation, depends critically upon the background genome in which it occurs. A mutation that produced tolerable mimicry on one genetic background might be hopelessly maladaptive on another. The resulting selection of only those mutations which 'suit' the genetic background, results in cases like the *Amauris*-mimics of *P. dardanus*, in which the genome appears to be preadapted to mimicry.

Müllerian Mimicry—Evolution by Jerks

As this last idea is somewhat akin, although by no means identical to some of the notions of Goldschmidt (1945—the chief difference being that whereas he maintained that the genome was set up in such a way as to produce immediate, high grade mimicry, I am making the more modest suggestion that it has to be set up in such a way that approximate mimicry is possible), it will be interesting to round off this discussion by looking at another theory in which Goldschmidt's idea of bang-on target systemic mutations have been invoked: the punctuational model of evolution (Gould 1980, Stanley 1979). According to this theory, evolution takes place by an alternation of long periods of stasis (no change) with short periods of very rapid evolution in which both speciation and the origin of new adaptive phenotypes take place.

Our genetic studies on *Heliconius melpomene* and *H. erato* have now reached the stage where we can use cladistic methods to reconstruct the evolution of the races. The technique is quite simple: the 'dominance sieve' mentioned above in relation to industrial melanism tells us that of any pair of alleles the recessive one is likely to be ancestral, and given that theorem the minimum tree can be estimated readily by the weighted invariant step method (Farris *et al.* 1970). The trees obtained (Fig. 14.12 shows those which correspond best with the current geographical distribution of the races) suggest that these two species have been mutual Müllerian mimics during the whole of their adaptive radiation, having both started as black butterflies with a yellow barred pattern not unlike the existing species *H. charitonia* and *H. nattereri*; perhaps they both inherited it from a common ancestor (Sheppard *et al.* 1984; Turner 1981). This is encouraging as it is far more likely that the beautiful parallel variation we see today would have been produced by parallel evolution than that all the races had converged, in pairs, from very disparate ancestors. Whether the trees and reconstructed ancestors are 'right' of course we cannot say: I nurse a suspicion that the common ancestors are 'wrong' in one feature, but am unable to prove this within the rules of cladistics. What is important is that, whatever the exact form of the tree, they must, like the ones figured, include the substitution of a few major genes in each branch (shown as cross bars).

Fig. 14.13. Approximate location of South American rain forests at the peak of the last glaciation, *ca* 18 000 years BP, deduced from a combination of biogeographical and palaeoecological data. After K. S. Brown, in Prance (1982).

Now if race formation took place in glacial refuges (Fig. 14.13), these trees span the 30 000 years of the most recent glacial cycle, at least. Complete substitution of a gene under selection of only 1% takes less than 4000 generations, or 400 years in *Heliconius* (Turner 1982). Thus if we could find fossil butterfly patterns, what we would see in these species would be the alternation of periods of stasis with rapid periods of change. In short, punctuated equilibria. However there is nothing in the process which is not perfectly describable in terms of population genetics and neoDarwinism. There are no systemic mutations, or catastrophic speciation events; merely the substitution of new dominant genes under natural selection.

I believe that we may here be seeing an example of an important evolutionary process: the appearance of new phenotypes, taking place quite rapidly in evolutionary time, when a species comes to occupy a new ecological niche. I have argued here that

Heliconius take up new patterns (an analogy for ecological niches) when some other pattern becomes well protected; niches of course are a sort of photo-negative of this: they tend to be occupied when they are 'empty', whereas mimicry rings become increasingly occupied when they are 'full'. I have elsewhere (Turner 1977*b*, 1981, 1982, also Sheppard *et al.* 1984) advanced the view that changes in pattern abundance, and hence protection, tended to be marked, persistent and therefore influential when the rainforest, fragmented by cool dry conditions during glacial maxima, underwent progressive extinction of parts of its fauna and flora (Fig. 14.13). Extinction empties ecological niches, and remaining species may evolve to occupy them. In ecologically stable periods, when all niches are occupied, there will be little evolution. Rapid changes occur in the comparatively rare circumstance that a new, imperfect adaptation, not yet improved by the long selection of 'modifier' genes, confers higher fitness than the old, refined adaptation. The removal of competition by the extinction of species will provide precisely this condition.

How often such changes involve major mutations remains to be seen. Although morphological changes seem more generally to be produced by a number of genes of individually small effect, single major genes are certainly involved not only in mimicry but in the evolution of host-resistance and parasite virulence (Sidhu 1975).

It is here, rather than in any new theory of systemic speciation, that we should seek for the causes of evolution by jerks.

Acknowledgements

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