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The genetics of warning colour in Peruvian hybrid zones of *Heliconius erato* and *H. melpomene*

BY J. MALLET†

Galton Laboratory, Department of Genetics and Biometry, University College London, Wolfson House, 4 Stephenson Way, London NW1 2HE, U.K.

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[Plates 1–3]

Heliconius warning colour is a good example of a genetic system shaped by strong selection. The genetics of colour patterns in interracial hybrid zones within both *H. erato* and *H. melpomene* was investigated. Within each species, the loci controlling these pattern differences are mostly homologous to those known from other races, but have somewhat different phenotypic effects. The precise genetic control varies geographically, even for nearly identical colour patterns. Independent evolution of the same pattern is unlikely; instead evolution of the genetic system is hypothesized to have occurred while stabilizing selection preserved the pattern itself.

Single genes often control more than one pattern element. This apparent pleiotropy is in part due to tightly linked loci within 'supergenes': rare recombinants (possibly mutants) in genes controlling 'dennis' and 'ray' patterns were found in both species. However, supergenes, which are likely in polymorphic Batesian mimicry, are not expected to accumulate in Müllerian mimics because polymorphisms, which would favour their evolution, are too transient. The existence of supergenes in *Heliconius* suggests that major switch genes are gradually built up within a locus rather than evolving wholly by macromutation or by selection for tighter linkage of mimetic genes. This gradual evolution at a single locus might be necessitated by a lack of other sites that can control warning patterns.

These genes are strongly epistatic, and heterozygotes and hybrid homozygotes have 'fuzzier' (less sharply defined) and more variable patterns than the pure races. The genetic system controlling colour pattern in *Heliconius* is clearly canalized and coadapted to produce efficient warning signals.

> '*As analysis is carried down toward the immediate effects of gene replacements, the relation to selective value tends to become more remote and contingent.*'
>
> (Wright (1968, p. 55) *Evolution and the genetics of populations*, vol. 1. University of Chicago Press.)

† Present address: Department of Entomology, Mississippi State University, Drawer EM, Mississippi State, Mississippi 39762, U.S.A.

Introduction

In nearly 90 years since the rediscovery of Mendel's work, little progress has been made in understanding the genetics of speciation. Speciation probably requires geographic separation (allopatric or parapatric), and it clearly involves the evolution of traits that have strong effects on fitness. Some have claimed that speciation may often be induced by founder events, which cause 'genetic revolutions' at strongly selected, coadapted genes (Mayr 1970; Templeton 1980). Because species differences are likely to be selected and coadapted, knowledge of allozymes, restriction enzyme maps, and other neutral or nearly neutral genetic elements can only trace divergence: it cannot help much with interpretation.

Geographic variation in *Heliconius* butterflies provides a useful model system for studying geographic divergence and speciation. Within geographic races, *Heliconius* warning patterns are under strong stabilizing selection. Where such races meet, they are separated by narrow hybrid zones. These hybrid zones can be stabilized by frequency-dependent predator attacks on rare morphs that immigrate from populations of a different race (Benson 1972; Brown *et al.* 1974; Turner 1984; Sheppard *et al.* 1985; Mallet 1986; Mallet & Singer 1987; Mallet & Barton 1989*a*). Such frequency-dependent predation is not normally considered to be a reproductive isolating 'mechanism', but the postmating isolation produced by warning colour and mimicry is nevertheless strong (selection coefficients, s, may be greater than 10% (Mallet & Barton 1989*a*)). This level of selection is similar to that affecting a translocation heterozygote, but without the reproductive compensation that so often mitigates the selection against chromosomal rearrangements. *Heliconius* colour patterns are thus a clear example of a genetic system that leads to multiple fitness peaks, including reproductive isolation.

In the lowlands of the Río Mayo and upper Río Huallaga, Peru, Müllerian mimetic 'postman'-patterned races of *Heliconius erato* and *H. melpomene* fly together. Each hybridizes with its respective Amazonian 'rayed' race near a ridge of mountains to the northeast of the town of Tarapoto (Lamas 1976; Mallet & Barton 1989*a*). The pure races and a description of their patterns are shown in figure 1, plate 1. Their colour patterns are similar to some of those analysed by Sheppard *et al.* (1985); however, no previous crosses have involved Peruvian stocks. In this paper, I investigate the genetics of these coincident hybrid zones by means of crosses within each species, and make comparisons with similar crosses between allopatric races (Sheppard *et al.* 1985). The work was done to investigate coadaptation and the existence of supergenes, as well as to use the genetics to estimate linkage disequilibria, and indirectly, selection pressures (Mallet & Barton 1989*b*; Mallet *et al.* 1989). For this reason phenotypes are conservatively lumped in the statistical analysis if there is any likelihood of confusion, though possible polygenic effects were also noted.

Methods

Crosses

All crosses were done in Tarapoto over a period of three months in eight 2 m high × 1.8 m × 1.8 m insectaries. Potted adult and larval host plants (respectively

Lantana spp. (Verbenaceae) and *Passiflora* spp. (Passifloraceae)) were placed in each cage. Adult food was supplemented with cut *Lantana* flowers in water for pollen and nectar, and 10–20% sugar water presented with yellow and red 'Tuffy' washing-up scourers in red plastic cups as visual attractants. Females were kept singly in each of seven of the cages; the eighth cage housed males. Further details of insectary maintenance are given by Turner (1974).

Wild-mated females were collected from hybrid and pure populations near Tarapoto, and were allowed to lay eggs. Multiple mating is rare in *Heliconius* (Boggs 1979), and sperm precedence in such cases (L. E. Gilbert, unpublished results), backed up by electrophoretic evidence (Mallet & King 1989), allows the assumption of a single male parent for the offspring. In *erato*, female progeny were mated with wild-caught or reared males to test hypotheses of inheritance. In *melpomene*, time permitted only progeny from wild-caught females to be reared.

Females will lay eggs only on new growth of preferred host plants (Smiley 1978; Gilbert 1982). Near Tarapoto, *erato* feeds on *Passiflora trifasciata*, *P. tricuspis*, and an unnamed species (the third egg-mimicking species figured by Gilbert (1982)); *melpomene* is on *P. menispermifolia*. Other host plants offered were only rarely used for oviposition; larvae died when given other *Passiflora*. Eggs were collected daily, and kept separately to avoid cannibalism and to record hatch rates. Larvae were reared individually with excess food. After pupation, rotting food was removed and pupae were suspended in their containers, if necessary, to allow emergence without deformation.

Analysis of the broods

Hypotheses for the inheritance of colour patterns were developed and then tested. Goodness-of-fit and homogeneity G-tests are used throughout. The additivity of the G-statistic (twice the difference in ln likelihood) allows tests to be combined and partitioned at will. The difference in ln likelihood may itself be used as a measure of inference (Edwards 1972), but a more standard significance-testing approach is used here; G approximates to a χ^2 distribution (*, $p < 0.05$; **, $p < 0.01$; ***, $p < 0.001$). Yates's correction was not used, because this destroys the additivity of G (the small sample-size effects are anyway removed by combining tests). G-tests are little affected by small samples, so long as n is somewhat above the number of cells in a table (Sokal & Rohlf 1981). In my own simulations of 2×2 homogeneity tables with cell proportions $\{0.01, 0.09, 0.09, 0.81\}$, a nominal significance ($G > 3.84$, $p < 0.05$) gave $0.02 \leqslant$ actual $p \leqslant 0.09$ when sample sizes were above 11; more symmetrical distributions do better. In making these tests, the most likely (in the strict statistical sense) parental genotypes have been assumed. This is justified because in large broods, which provide most of the information for the tests, the most likely parental genotypes are significantly more likely than other genotypes that produce different ratios; in small broods, little information is provided, so very little error is made in assuming the most likely configuration. Likelihood is also used to estimate 'support limits' for parameters i.e the interval in which $G_1 \leqslant 4$ (see Edwards 1972).

Phenotypic and genotypic nomenclature

Pattern elements are described according to the nomenclature given in figure 1. Forewing and hindwing are abbreviated 'FW' and 'HW', respectively. Genotypic nomenclature (in italics) follows previous work (Sheppard *et al.* 1985), whenever possible. To avoid unnecessary confusion, the separate phenotypic nomenclature used during the formulation of hypotheses is not presented here. Phenotypes are instead characterized by using allelic names in standard typeface. These genes interact strongly, so that only the entire phenotypic formula will give the correct appearance of the butterfly. (See the legends of the tables for details, and the figures for examples.) Loci (italicized) are referred to by their pairs of alleles separated by a slash, with the Amazonian allele cited first.

Results

The results of the crosses are shown in table 1 (*erato*), and table 2 (*melpomene*). These tables are the primary data on which the tests have been made, and show phenotypes and sexes of parents and offspring in each brood. The pure races and names of phenotypic components are shown in figure 1.

Genetics of erato

The hypothesis and tests

On the basis of the field phenotypic ratios it was initially hypothesized that each of three genes controlled separate pattern elements. However, the broods provided evidence for strong genetic interactions between the elements. It became obvious that yellow HW bar is strongly affected by FW band shape, as full yellow HW bars are only produced in individuals with broad, long FW bands, both in the broods (figure 2, plate 1), and in the field collections. The gene for absence/presence of yellow HW bars is named Cr/Cr^h; that for short, narrow as opposed to broad, long FW band shape and yellow HW bar modification is named *Sd/sd*. Thus full yellow HW bars are formed only in Cr^hCr^h *sdsd* genotypes.

Two examples of this interaction are given in figure 2. The female parent of PE10 had no dennis, only a shadow HW bar on the underside, and a broad, fuzzy and short FW band: presumed genotype $CrCr^h$ *Sdsd* (i.e. like her offspring in figure 2.1). Because she was captured in a predominantly postman population, her mate was almost certainly Cr^hCr^h *sdsd*. The interpretation of the phenotypes of her offspring is then as follows (abbreviations for probably equivalent yellow HW bar phenotypes used by Sheppard *et al.* (1985) in their figure 7 and appendix 5 are included in quotation marks): figure 2.1, $CrCr^h$ *Sdsd*, fuzzy broad, short FW band (one narrow), with shadow HW bars on the underside, 'nb+fs+fd'; figure 2.2, $CrCr^h$ *sdsd*, broad, long FW band, with strong yellow marks in the region of the tip of the HW bar position, the remainder of the bar strongly shadowed, 'fr?'; figure 2.3, $CrCr^h$ *sdsd*, broad, long FW band, with near complete yellow HW bar but with strong black scaling passing along the veins, 'bb'; figure 2.4, Cr^hCr^h *Sdsd*, fuzzy, broad, short FW band, with partial yellow HW bar, not strongly expressed at the tip, and with fuzzy edges, 'ea'; figure 2.5, Cr^hCr^h *sdsd*,

full postman phenotype, with broad, long FW band and fully-formed yellow HW bar, 'bs'. All other relevant broods also conform to this hypothesis of Cr/Cr^h and *Sd/sd* interaction (PE9, 22, 21, 27, 29, 30), though broods on rayed backgrounds tend to have partial yellow HW bar phenotypes less strongly expressed (PE21, figures 2.6–2.8).

Sd/sd and Cr/Cr^h are phenotypically dominant in that the heterozygote looks considerably more like the phenotype of the rayed race than that of the postman race, though there are intermediates: this is also true for the *Sd* and *Cr* loci studied by Sheppard *et al.* (1985). As a first hypothesis, it was supposed that heterozygotes at *Sd/sd* always had an intermediate band shape (phenotype Sdsd) and that heterozygotes at Cr/Cr^h always produced a shadow (phenotype $CrCr^h$). However, the broods showed that the alleles *sd* and Cr^h were only sometimes penetrant in heterozygotes. For example, in PE3, the female parent was an apparent homozygote *CrCr SdSd*, having a narrow, short FW band, and a complete lack of HW bar or shadow. However, some of her offspring were Cr^hCr^h or sdsd, so the female must have been heterozygous at both genes. The female parent of PE18 was $CrCr^h$ sdsd, and yet there was a scarcity of $CrCr^h$ offspring: most were Cr–. The offspring in PE18 should all have carried *sd* alleles: yet six are phenotypically Sd–. It is clear the heterozygotes $CrCr^h$ and *Sdsd* cannot reliably be distinguished from their respective homozygotes *CrCr* and *SdSd*.

Dennis, ray and forewing band colour appear all to be inherited at a single locus, which is assumed to be the same as the D^R/d of Sheppard *et al.* (1985). In contrast to the previous loci, the genotypes D^RD^R, D^Rd and *dd* – see figure 3, plate 2, and Sheppard *et al.* (1985) – can all be distinguished because the dominance of the red pattern components (*d* produces a red, as opposed to yellow, FW band; D^R produces red-orange dennis and ray compared with its absence) goes in opposite directions in the two races. D^Rd heterozygotes do vary in the faintness of the ray and in the amount of yellow in the red FW band, but misidentifications are not possible because of the opposing dominance: they always have dennis, ray, and red FW bands.

The final hypothesis is then that the FW band colour and dennis-ray pattern are controlled by D^R/d (yellow FW band, dennis and ray as opposed to red FW band, no dennis or ray); that *Sd/sd* controls shape of FW band (short, narrow rather than long, broad), as well as interacting with Cr/Cr^h in forming the yellow HW bar (absence or presence). The hypothesis can now be tested for fit to Mendelian ratios in the broods. Broods PE12, 14, 21, 28, 31 are backcross-type broods for D^R/d. None of these broods deviated significantly from the Mendelian 1:1 expectation, giving a sum of $G_5 = 5.4$. Summing the numbers between broods, the ratio overall was not significantly different from 1:1 ($G_1 = 0.3$); by subtraction $G_4 = 5.1$ measures the heterogeneity between broods. Similarly for 1:2:1 broods at D^R/d (PE13, 15, 25), no individual brood had a high G, the sum being $G_6 = 6.6$. The ratio overall was not significantly different from expectation ($G_2 = 2.5$), and heterogeneity was slight ($G_4 = 4.1$). Broods segregating for Cr/Cr^h and *Sd/sd* were tested similarly, assuming complete dominance (i.e. that *CrCr* cannot be distinguished from $CrCr^h$ and that *SdSd* is indistinguishable from *Sdsd*); none show any evidence of deviation from the hypothesis. Over all genes and broods, summing the individual

TABLE 1. BROODS OF *ERATO*

(Parent and offspring phenotypes (males/females) for each brood. Wild-caught parents have four-digit numbers. Site of capture is recorded. Kilometre posts are along the Tarapoto–Yurimaguas road; most are near km 60 (Pongo de Cainarache), the approximate centre of the hybrid zones. Reared parents have both a brood number and an individual number; square brackets indicate most likely genotypes; italics indicate allelic genotypes absolutely required by the genetic hypothesis, and by the phenotypes of progeny and parents; male genotypes without identity numbers indicate wild matings. Explanation of phenotypes (genotypes in square brackets) is given below, with Amazonian genotypes first.)

phenotype [genotype]	description
D^RD^R [D^RD^R]	dennis, ray + yellow FW band.
D^Rd [D^Rd]	dennis, ray + red (or red overlaid with yellow) FW band.
dd [*dd*]	no dennis or ray, red FW band.
Sd– [*SdSd*, *Sdsd*]	narrow FW band, not extending into the discal cell, nor near the anal wing margin
Sdsd [*Sdsd*]	on the upperside, broader FW band, extending into the discal cell, and with a fuzzy proximal edge, not extending near the anal wing margin. On the underside, narrower, often similar to Sd–
sdsd [*sdsd*]	broad, compact FW band with crisp edges, extending well into the cell, and anally almost to the wing margin. Also adds yellow markings at the tip of the HW bar position. This genotype is necessary for full expression of the yellow HW bar
Cr– [*CrCr*, *CrCr*^{h}]	no trace of HW bar, either as a shadow or as flecks of yellow in the centre of the bar position
$CrCr^h$ [*CrCr*^{h}]	shadow HW bar on the underside (Both CrCr and $CrCr^h$ may have yellow spots at the margins of the bar positions, anally or distally.)
Cr^hCr^h [*Cr*^{h}*Cr*^{h}]	full yellow bar with sharp tip if also genotype *sdsd*; fuzzy yellow bar with blunt tip if also genotype *SdSd* or *Sdsd*

brood		phenotype	[genotype]	where captured
PE1				
female parent 1678		D^RD^R Cr– Sd	[*D*^{R}*D*^{R} *Cr*Cr *Sd*Sd]	km 72, Davidcillo
male parent		—	[*D*^{R}D^R CrCr sdsd]	—
	2/0	D^RD^R Cr–Sdsd		
PE3/PE6				
female parent 1725		D^RD^R Cr– Sd–	[*D*^{R}*D*^{R} *CrCr*^{h} *Sdsd*]	km 58
male parent		—	[*d*dCr*Cr*^{h} Sd*sd*]	—
	0/1	D^Rd Cr– Sdsd		
	1/2	D^Rd $CrCr^h$ Sdsd		
	0/1	D^Rd $CrCr^h$ sdsd		
	0/1	D^Rd Cr^hCr^h Sd–		
PE4				
female parent 1677		D^RD^R Cr– Sd–	[*D*^{R}*D*^{R} *Cr*Cr *Sd*Sd]	km 72, Davidcillo
male parent		—	[*D*^{R}D^R CrCr SdSd]	—
	2/0	D^RD^R Cr– Sd–		
PE7[a]				
female parent 1735		dd Cr^hCr^h sdsd	[*dd Cr*^{h}*Cr*^{h} *sdsd*]	km 48
male parent		—	[*dd Cr*^{h}*Cr*^{h} *sd*sd]	—
	11/8	dd Cr^hCr^h sdsd		
PE9				
female parent 1676		D^RD^R $CrCr^h$ Sd–	[*D*^{R}*D*^{R} *CrCr*^{h} *Sd*Sd]	km 72, Davidcillo
male parent		—	[*D*^{R}D^R *Cr*^{h}Cr^h Sdsd]	—
	0/1	D^RD^R Cr– Sdsd		
	1/1	D^RD^R $CrCr^h$ Sdsd		
	0/1	D^RD^R Cr^hCr^h Sd–		
	1/1	D^RD^R Cr^hCr^h Sdsd		

TABLE 1 (*cont.*)

brood		phenotype	[genotype]	where captured
PE10				
female parent 1733		dd $Cr Cr^h$ Sdsd	[*dd CrCr^h Sdsd*]	km 48
male parent		—	[dd *Cr^h*Cr^h *sd*sd]	—
	3/1	dd $Cr Cr^h$ Sdsd		
	3/2	dd $Cr Cr^h$ sdsd		
	0/1	dd $Cr^h Cr^h$ Sd–		
	1/5	dd $Cr^h Cr^h$ Sdsd		
	3/4	dd $Cr^h Cr^h$ sdsd		
PE11/PE19				
female parent 1770		dd $Cr^h Cr^h$ sdsd	[*dd Cr^{h}Cr^h sdsd*]	Chazuta
female parent 1757		dd Cr^h Cr^{h}sdsd	[*dd Cr^{h}Cr^h sdsd*]	Chazuta
male parents		—	[dd *Cr^h*Cr^h *sd*sd	—
	11/11	dd $Cr^h Cr^h$ sdsd		
PE12				
female parent 1778		D^Rd $Cr Cr^h$ Sdsd	[*D^{R}d CrCr^h Sdsd*]	km 62
male parent		—	[*D^R*D^R CrCr Sd*sd*]	—
	1/0	$D^R D^R$ Cr– Sdsd		
	1/0	D^Rd Cr– Sdsd		
	0/1	D^Rd $Cr Cr^h$ Sd–		
	1/0	D^Rd $Cr Cr^h$ Sdsd		
	1/1	D^Rd $Cr Cr^h$ sdsd		
PE13				
female parent 1780		D^Rd Cr– Sdsd	[*D^{R}d Cr*Cr *Sdsd*]	km 62
male parent		—	[*D^{R}d* CrCr^h Sd*sd*]	—
	2/0	$D^R D^R$ $Cr Cr^h$ Sdsd		
	0/1	D^Rd Cr– Sd–		
	1/0	D^Rd Cr– Sdsd		
	1/0	D^Rd $Cr Cr^h$ Sd–		
	1/0	D^Rd $Cr Cr^h$ sdsd		
	0/1	dd Cr– Sd–		
	0/1	dd $Cr Cr^h$ sdsd		
PE14				
female parent 1781		D^Rd Cr– Sd–	[*D^{R}d Cr*Cr *Sd*Sd]	km 62
male parent		—	[*D^R*D^R CrCr^h Sdsd]	—
	1/6	$D^R D^R$ Cr– Sd–		
	3/3	$D^R D^R$ Cr– Sdsd		
	1/0	$D^R D^R$ $Cr Cr^h$ Sdsd		
	1/4	D^Rd Cr– Sd–		
	3/2	D^Rd Cr– Sdsd		
	1/1	D^Rd $Cr Cr^h$ Sdsd		
PE15				
female parent 1777		D^Rd $Cr Cr^h$ Sd–	[*D^{R}d CrCr^h Sd*Sd]	km 62
male parent		—	[*D^{R}d Cr*Cr Sdsd]	—
	0/4	$D^R D^R$ Cr– Sd–		
	1/1	$D^R D^R$ Cr– Sdsd		
	0/3	$D^R D^R$ $Cr Cr^h$ Sd–		
	1/0	$D^R D^R$ $Cr Cr^h$ Sdsd		
	2/4	D^Rd Cr– Sd–		
	3/1	D^Rd Cr– Sdsd		
	3/1	D^Rd $Cr Cr^h$ Sd–		
	1/1	D^Rd $Cr Cr^h$ Sdsd		
	0/3	dd Cr– Sd–		
	4/2	dd Cr– Sdsd		
	1/1	dd $Cr Cr^h$ Sd–		
	2/3	dd $Cr Cr^h$ Sdsd		

TABLE 1 (*cont.*)

brood		phenotype	[genotype]	where captured
PE18/PE20				
female parent 1799		D^RD^R $CrCr^h$ sdsd	[D^RD^R $CrCr^h$ *sdsd*]	km 62
male parent		—	[D^RD^R CrCr *Sd*Sd]	—
	1/5	D^RD^R Cr– Sd–		
	10/2	D^RD^R Cr– Sdsd		
	1/0	D^RD^R $CrCr^h$ Sdsd		
PE21[b]				
female parent PE3.2		D^Rd Cr– Sdsd	[D^Rd $CrCr^h$ *Sdsd*]	—
male parent 1776		D^RD^R Cr^hCr^h sdsd	[D^RD^R Cr^hCr^h *sdsd*]	km 62
	2/0	D^RD^R Cr– Sdsd		
	0/2	D^RD^R $CrCr^h$ Sd–		
	4/1	D^RD^R $CrCr^h$ Sdsd		
	6/4	D^RD^R $CrCr^h$ sdsd		
	0/3	D^RD^R Cr^hCr^h Sd–		
	3/2	D^RD^R Cr^hCr^h Sdsd		
	5/1	D^RD^R Cr^hCr^h sdsd		
	0/1	D^Rd Cr– Sdsd		
	4/6	D^Rd $CrCr^h$ Sdsd		
	3/2	D^Rd $CrCr^h$ sdsd		
	2/2	D^Rd Cr^hCr^h Sdsd		
	6/4	D^Rd Cr^hCr^h sdsd		
PE22				
female parent PE10.9		dd Cr^hCr^h Sdsd	[*dd* Cr^hCr^h *Sdsd*]	—
male parent 1776 (male parent of PE21)		D^RD^R Cr^hCr^h sdsd	[D^RD^R *CrCr sdsd*]	km 62
	1/0	D^Rd Cr^hCr^h Sd–		
	0/1	D^Rd Cr^hCr^h sdsd		
PE25				
female parent 1835		D^Rd $CrCr^h$ Sdsd	[D^Rd $CrCr^h$ *Sdsd*]	Pongo de Aguirre
male parent		—	[D^Rd $CrCr^h$ *sd*sd]	—
	0/2	D^Rd $CrCr^h$ sdsd		
PE26				
female parent PE13.1		dd Cr– Sd–	[*dd* $CrCr^h$ *Sd*Sd]	—
male parent 1746		dd $CrCr^h$ Sdsd	[*dd* $CrCr^h$ *Sdsd*]	km 62
	1/1	dd Cr– Sd–		
	5/4	dd Cr– Sdsd		
	4/0	dd $CrCr^h$ Sd–		
	3/6	dd $CrCr^h$ Sdsd		
	5/2	dd Cr^hCr^h Sd–		
	1/3	ddCr^hCr^h Sdsd		
PE27				
female parent PE18.1		D^RD^R Cr– Sdsd	[D^RD^R *Cr*Cr *Sdsd*]	—
male parent 1759		dd Cr^hCr^h sdsd	[*dd* Cr^hCr^h *sdsd*]	Chazuta
	1/0	D^Rd $CrCr^h$ Sd–		
	2/2	D^Rd $CrCr^h$ Sdsd		
	3/2	D^Rd $CrCr^h$ sdsd		

TABLE 1 (*cont.*)

brood		phenotype	[genotype]	where captured
PE28				
female parent PE15.9		dd Cr– Sdsd	[*dd Cr*Cr *Sdsd*]	—
male parent 1790		D^Rd $CrCr^h$ Sdsd	[D^Rd $CrCr^h$ *Sdsd*]	km 62
	1/1	D^Rd Cr– Sd–		
	5/2	D^Rd Cr– Sdsd		
	0/1	D^Rd Cr– sdsd		
	1/0	D^Rd $CrCr^h$ Sd–		
	3/3	D^Rd $CrCr^h$ Sdsd		
	0/7	D^Rd $CrCr^h$ sdsd		
	2/5	dd Cr– Sd–		
	3/3	dd Cr– Sdsd		
	1/1	dd Cr– sdsd		
	0/2	dd $CrCr^h$ Sd–		
	2/1	dd $CrCr^h$ Sdsd		
	4/2	dd $CrCr^h$ sdsd		
PE29				
female parent PE7.16		dd Cr^hCr^h sdsd	[*dd* Cr^hCr^h *sdsd*]	—
male parent		dd Cr–Sdsd	[*dd* $CrCr^h$ *Sdsd*]	km 61
	0/2	dd $CrCr^h$ Sdsd		
	3/3	dd $CrCr^h$ sdsd		
	2/3	dd Cr^hCr^h Sdsd		
	5/5	dd Cr^hCr^h sdsd		
PE30				
female parent PE15.20		dd Cr– Sdsd	[*dd Cr*Cr *Sdsd*]	—
male parent PE9.6		D^RD^R Cr^hCr^h Sdsd	[D^RD^R Cr^hCr^h *Sdsd*]	—
	1/2	D^Rd $CrCr^h$ Sd–		
	1/3	D^Rd $CrCr^h$ Sdsd		
	3/0	D^Rd $CrCr^h$ sdsd		
PE31				
female parent PE21.7		D^Rd $CrCr^h$ sdsd	[D^Rd $CrCr^h$ *sdsd*]	—
male parent 1844		D^RD^R Cr– Sd–	[D^RD^R *Cr*Cr *Sd*Sd]	Pongo de Aguirre
	0/1	D^RD^R Cr– Sd–		
	2/2	D^RD^R Cr– Sdsd		
	0/1	D^RD^R $CrCr^h$ Sdsd		
	1/1	D^Rd Cr– Sdsd		

[a] Some variation in shape of FW band in this brood. Five males and five females had FW bands that did not reach as near to the anal margin as normal for sdsd phenotypes. In other respects the phenotype was normal sdsd. This suggests variation in the allele *sd*, or at other genes affecting the shape of FW band but without effect on the yellow HW bar.

[b] One male dd Cr– Sdsd, almost certainly a contaminant, has been excluded from this brood; the phenotype dd is otherwise absent, and Cr– is a rare variant in the rest of the brood.

brood Gs gives $G_{29} = 26.8$, summing the overall ratio tests $G_9 = 5.2$, and heterogeneity $G_{22} = 21.6$. None of these tests is significant, so the hypothesis cannot be rejected.

There is still a possibility that small deviations from Mendelian ratios are masked by the small sizes of these broods. Many of the broods seem to have a slight excess of homozygotes: a positive correlation between uniting gametes (F)

within broods could explain this. The correlation would occur within a single pair brood, even though the pair may have mated at random. This heterozygote deficit F is not really the same as an inbreeding coefficient, although it is defined similarly so that in idealized crosses $Aa \times aa$, $F = 2\,fr(aa) - 1$; in $Aa \times Aa$ broods, $F = 1 - 2\,fr(Aa)$ if heterozygotes are detectable, or $4\,fr(aa) - 1$ if A is dominant, where fr represents the actual frequency of the genotype in the offspring. The likelihoods of different values of F for the 1:2:1, 3:1, and 1:1 cases have been obtained separately for all genes and brood types. The ln likelihoods were then added to obtain the overall support limits of F. This gives a powerful test with an effective sample size of 566 offspring. The test shows that F is not significantly different from zero ($G_1 = 3.0$), the support limits being $-0.01 \leqslant F \leqslant 0.16$.

Evidence for recombination within the D^R/d supergene

The pleiotropic effects of D^R/d on dennis, ray, and FW band colour have led Sheppard *et al.* (1985) to believe that a 'supergene', which they called D^{Ry}/d^{rY}, consists of three separate linked elements. No obvious crossovers between components were found in the present broods; however, one rayed individual with no dennis was found in the hybrid zone (figure 4.1, plate 2). This aberration was unique among 1571 *erato* collected from the hybrid zone region. Dennis-only (*DD rr*) *erato* races occur in the wild in the Guianas, but only a few ray-only (*dd RR*) *erato* specimens are known; see form *anaitis* Riffarth, from a Bolivian hybrid zone (Turner & Crane (1962), p. 150 as var. *vesta*; Ackery & Smiles (1976), figure 351).

Linkage and other types of interaction

In *Heliconius* (Suomalainen *et al.* 1973; Turner & Sheppard 1975), as in many other Lepidoptera (Robinson 1971) chiasmata and crossings-over occur only in males, the homogametic sex. Thus recombination between two genes in a doubly heterozygous female shows that those genes are unlinked. By this means, D^R/d is shown to be linked neither to Cr/Cr^h in PE21 nor to Sd/sd in PE21 and 28, and Cr/Cr^h is shown to be unlinked to Sd/sd in PE10, 21 and 29.

Interactions between unlinked genes could occur in the broods if coadaptation between the chromosomes affects survival (see, for example, Burton 1987). In *erato* there is no evidence for such genotypic interaction for D^R/d and Cr/Cr^h (PE21, 25) $G_3 = 0.1$; for D^R/d and Sd/sd (PE12, 13, 21, 25, 28) $G_7 = 4.1$; and for Cr/Cr^h and Sd/sd (PE3, 10, 21, 25, 29) $G_5 = 1.9$. These tests also show that there is little, if any, differential mortality of genotypes. But the genetic hypothesis does incorporate substantial interaction (epistasis) at the phenotypic level. One example is in the formation of the yellow HW bar (figure 2, and see above). Red FW bands are also usually wider than yellow bands on the upperside. This is especially true in *Sdsd* heterozygotes: red FW bands may reach to vein Cu1*b* on the upperside whereas yellow bands rarely stretch beyond Cu1*a* except as a small spot. On the undersides both types of band are reduced (compare figures 2.1, 2.4 with 2.6, 2.8). This interaction between FW band shape and colour is obvious when yellow and red are overprinted in D^Rd heterozygotes (figure 3.8). Less extreme, but none the less obvious, differences can also be seen in *sdsd* and *SdSd* homozygotes (compare

figure 3.1, 3.4 with 3.7, 3.10 and figure 3.3, 3.6 with 3.9, 3.12). Thus D^R/d and *Sd/sd* are epistatic in their effect on FW band shape. *Sd/sd* can also affect the expression of D^R/d; D^R-*sdsd* genotypes have reduced ray within the area occupied by yellow HW bar, even though the bar itself may be absent (figures 2.7, 3.3, 3.6 and 3.9).

Sex also affects the expression of *Sd/sd*; *Sdsd* heterozygotes more often have Sd– phenotypes (i.e. indistinguishable from *SdSd* genotypes) in females than in males (figure 3). Of seven broods tested (PE13, 14, 15, 18, 21, 26, 28) five had a significant interaction between Sd– or Sdsd phenotype and sex. Summing gives overall $G_7 = 35.3^{***}$. However, in PE13 (a small brood) and PE28 (a fairly large brood) there were no significant interactions, suggesting that other genes are also involved in the interaction between sex and *Sd/sd*. The effect of sex on FW band shape can also be seen in the field, for instance at Santa Rosa de Davidcillo (72 km from Tarapoto on the Tarapoto–Yurimaguas road), where *H. erato* is nearly fixed for D^R, *Cr* and *Sd* (see Mallet & Barton 1989*a*). Of the 103 *Sd*– and *Sdsd* males at this locality, 35 were scored as Sdsd, whereas only two of 52 such females were scored Sdsd ($G_1 = 21.4^{***}$). Phenotypes of known genotype did not interact with sex; for D^R/d, PE13, 14, 15, 21, 28 and 31, $G_8 = 10.7$. Similar tests can be made for genotypic interaction of *Sd/sd* and Cr/Cr^h with sex by lumping Sdsd with SdSd and $CrCr^h$ with CrCr: for *Sd/sd*, PE10, 13, 21, 28, 29 and 30, $G_7 = 10.6$; for Cr/Cr^h, PE10, 21, 26 and 29, $G_4 = 3.5$. Sex does not have a noticeable effect on the scoring of Cr– compared with $CrCr^h$ (PE13, 14, 15, 21, 26 and 2, $G_6 = 3.6$), in contrast to its effect on the scoring of *Sd/sd*.

Minor genes affecting colour pattern

These broods show phenotypic variability in hybrid genotypes. This occurs both in one-locus heterozygotes (in the breadth of the FW band in *Sdsd* genotypes, for example) and in multilocus homozygotes not found in pure populations (in *CrCr sdsd* genotypes, for example). The 'modifiers' that cause this variability cannot easily be identified without other phenotypic marker effects (see Sheppard *et al.* 1985); however, between-brood differences in expression indicate that some of this variation is genetic. For example, D^Rd heterozygotes may have pure red FW bands, or red bands 'overprinted' with yellow (figure 3.7–3.9): in PE21, only one of 30 D^Rd genotypes has yellow overprinted; in PE27, nine out of ten are overprinted, whereas in PE28 there are 12 of 24. Broods are strongly heterogeneous for overprinting (PE14, 15, 21, 27, 28 and 30, $G_5 = 37.9^{***}$; to simplify table 1, overprinting data are not shown). Similarly, dominance of *Sd/sd* and interactive variability between sex and *Sd/sd* are probably under genetic control (see above).

A further variable feature of hybrids (not recorded in table 1) is the presence of yellow dots near the HW anal margin and fuzzy yellow dots near the centre of the HW in unbarred phenotypes (Cr– and $CrCr^h$; figure 3.4, 3.8). These dots are not significantly correlated with other phenotypes in the crosses, and the HW central dot can even be found in some pure rayed populations (J. Mallet, personal observations in Cuzco Department, Peru).

Another minor phenotypic trait that does not seem to have a simple explanation

is the *ca.* 0.5 mm diameter anal yellow spot of the FW, homologous to the base of the 'yellow line' in East Brazilian *erato*. In Peru this spot is normally present in rayed individuals (figure 3.4, 3.8), and absent in the postman race. However, it does occasionally appear in postman individuals, where it may even form a short medial line up to 3 mm long. This pattern element (not recorded in table 1) does not appear to be affected by genes on any of the chromosomes carrying D^R/d, Cr/Cr^h, or Sd/sd, nor does it interact with sex (G-tests on original data). The rayed race also has larger yellow spots on the body.

Comparison with the results of Sheppard et al.

The *Cr* and *Sd* loci have been named because of the similarities of their action and interaction to loci of the same name described by Sheppard *et al.* (1985). The allele Cr^h is assumed to be a new allele with effects somewhat intermediate between Cr^P and *cr*; see Discussion. Sheppard *et al.* (1985, page 571) recognize a further locus, *Yl*, which they point out could be allelic with *Sd* because recombinants are unknown. Another putative locus, *St*, is also closely linked and has effects on band shape that are similar to those of *Sd*. *St* is perhaps also allelic with *Sd* if the single known recombinant was mistaken (their page 560). Sheppard *et al.* (1985) suggested two additional unlinked loci (Ly^B/ly^b and *Ybs/ybs*) that may modify the yellow HW bar, but these loci could not be distinguished here.

The FW band shapes produced in these broods are reminiscent of phenotypes produced in the broods involving the Ecuadorean twin-banded *H. erato notabilis*. In that race, the band sometimes does not split on the FW upperside when it is split on the underside. In the present crosses, FW band shape is determined by *Sd/sd* and can differ between upper and under surfaces: there is also a difference in shape depending on the colour of the band. Sheppard *et al.* (1985) interpreted similar variants in band splitting as due to gene *Ur* for 'upperside response', on the basis of two rather small broods (their page 565). Their brood 10A could be explained simply on the basis of *Sd* (and tightly linked loci), as was done here: heterozygotes at the locus could allow splitting of the red band on the underside but not on the upperside. Their brood 3D could be similarly explained if the expression of heterozygotes at *Sd* is variable, as here. Their brood 5A shows a very significant association between upperside split bands and yellow band colour, similar to the interaction of shape with colour in the current broods. All of these effects could be due to the *Sd–St* complex of loci; the *Ur* locus may be unnecessary.

Description of plate 1

Figure 1. Geographic races of *H. erato* (left) and *melpomene* (right). Top, 'postman' races with broad, long red FW band and yellow HW bar; *H. e. favorinus* and *H. m. amaryllis*. Bottom, 'rayed' races with narrow, short yellow FW band, orange-red FW 'dennis' (proximal patch) and similarly coloured HW 'ray'; *H. e. emma* and *H. m. aglaope*.

Figure 2. Examples of interaction between Cr/Cr^h and Sd/sd in the yellow HW bar of *erato*. Left, uppersides: right, undersides. 1–5, from brood PE10; 6–9, PE21. 1–4 females; 5–9, males. Genotypes: 1, *dd* $CrCr^h$ *Sdsd*; 2, *dd* $CrCr^h$ *sdsd*; 3, *dd* $CrCr^h$ *sdsd* with unusually well-developed HW bar; 4, *dd* Cr^hCr^h *Sdsd*; 5, *dd* Cr^hCr^h *sdsd*; 6, D^RD^R $CrCr^h$ *Sdsd*; 7, D^RD^R $CrCr^h$ *sdsd*; 8, D^RD^R Cr^hCr^h *Sdsd*; 9, D^RD^R Cr^hCr^h *sdsd*.

1

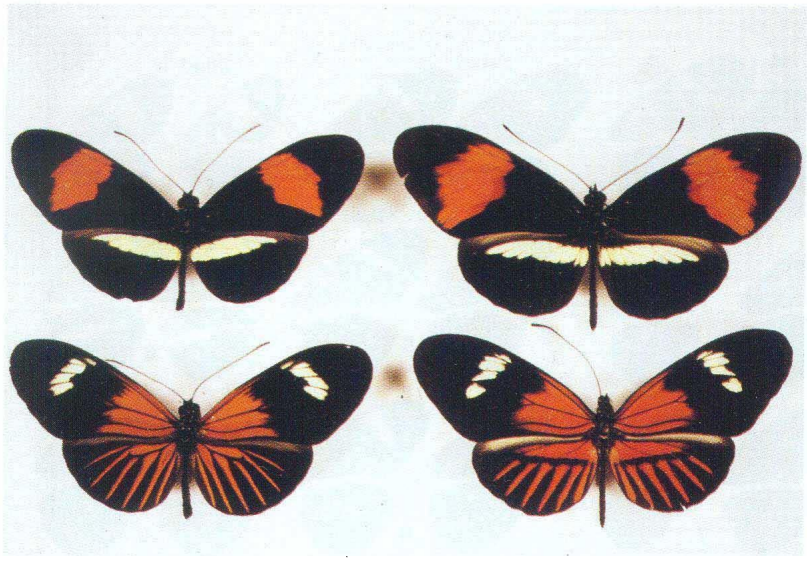


FIGURE 1. Geographic races of *H. erato* (left) and *melpomene* (right). Top, 'postman' races with broad, long red FW band and yellow HW bar; *H. e. favorinus* and *H. m. amaryllis*. Bottom, 'rayed' races with narrow, short yellow FW band, orange-red FW 'dennis' (proximal patch) and similarly coloured HW 'ray'; *H. e. emma* and *H. m. aglaope*.

2

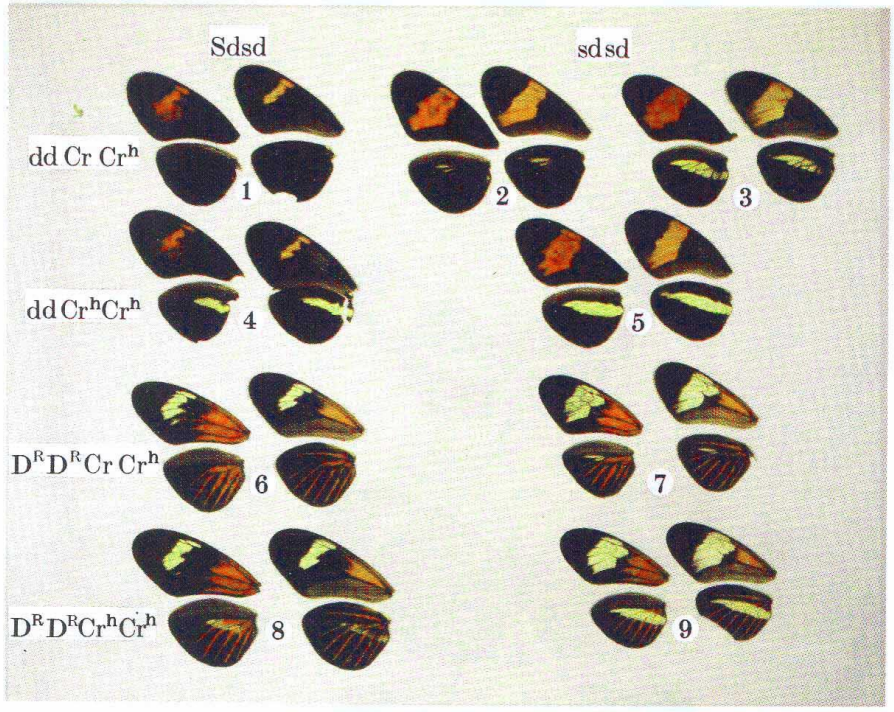
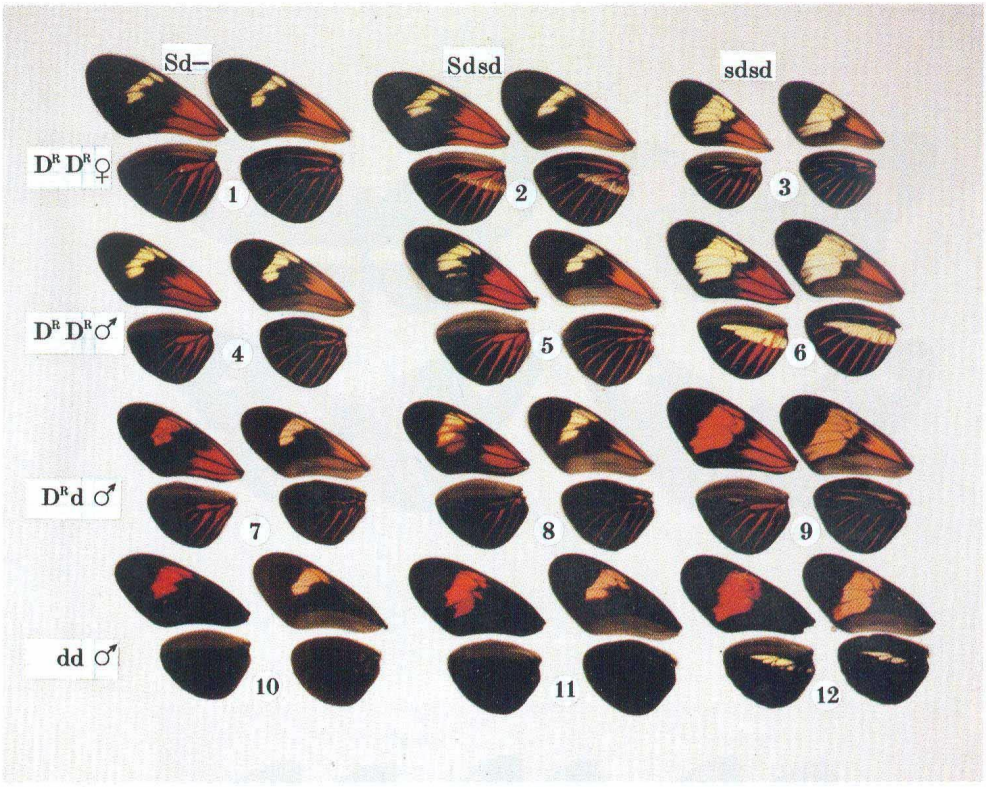


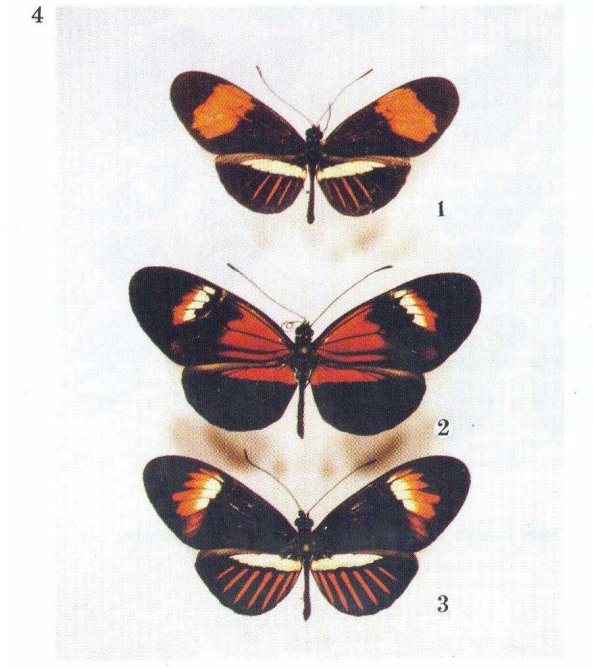
FIGURE 2. Examples of interaction between Cr/Cr^h and Sd/sd in the yellow HW bar of *erato*. Left, uppersides: right, undersides. 1–5, from brood PE10; 6–9, PE21. 1–4 females; 5–9, males. Genotypes: 1, *dd* $CrCr^h$ *Sdsd*; 2, *dd* $CrCr^h$ *sdsd*; 3, *dd* $CrCr^h$ *sdsd* with unusually well-developed HW bar; 4, *dd* Cr^hCr^h *Sdsd*; 5, *dd* Cr^hCr^h *sdsd*; 6, D^RD^R $CrCr^h$ *Sdsd*; 7, D^RD^R $CrCr^h$ *sdsd*; 8, D^RD^R Cr^hCr^h *Sdsd*; 9, D^RD^R Cr^hCr^h *sdsd*.

3



DESCRIPTION OF PLATE 2

FIGURE 3. Interaction of sex and D^R/d with *Sd/sd* in the shape of the FW band of *erato*. Also shows HW bar phenotypes. 1,4,8, from PE31; 2–3,5–6,9, PE21; 7,10–11, PE15; 12, PE28. 1–3, females; 4–12, males. Genotypes: 1, D^RD^R $CrCr^h$ *Sd–*; 2, D^RD^R Cr^hCr^h *Sdsd*; 3, D^RD^R $CrCr^h$ *sdsd*; 4, D^RD^R *Cr– Sd–*; 5, D^RD^R $CrCr^h$ *Sdsd*; 6, D^RD^R Cr^hCr^h *sdsd*; 7, D^Rd $CrCr^h$ *Sd–*, no FW yellow overprinted; 8, D^Rd *Cr– Sdsd*, FW yellow strongly overprinted; 9, D^Rd $CrCr^h$ *sdsd*, no FW yellow overprinted; 10, *dd* $CrCr^h$ *Sd–*; 11, *dd* $CrCr^h$ *Sdsd*; 12, *dd Cr– sdsd*.



DESCRIPTION OF PLATE 2

FIGURE 4. Probable recombinants within supergenes controlling dennis and ray in *erato* and *melpomene*. 1, *erato* male no. 1981; genotype *dd R– Cr^hCr^h sdsd*, captured near km 63 Tarapoto–Yurimaguas. 2, *melpomene* male no. 1995 from near Yumbatos, km 61 Tarapoto–Yurimaguas; genotype *D– rr N^N– B– Yb–*. 3, *melpomene* male offspring from brood PM16; genotype *dd R– N^N– B– ybyb*.

5

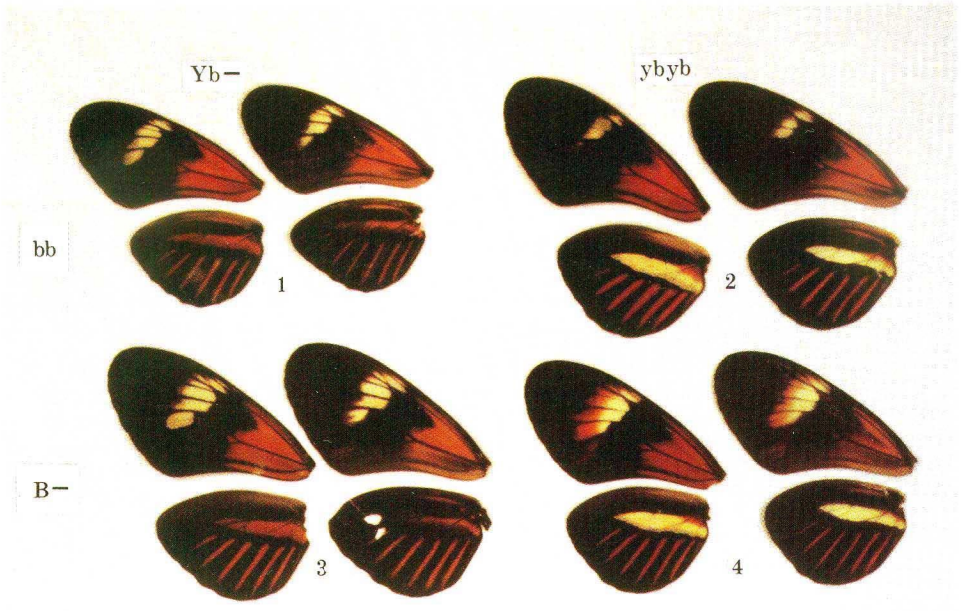


FIGURE 5. *H. melpomene* from brood PM16. Female progeny. Genotypes: 1, D^R– N^N– *bb Yb*–; 2, D^R– N^N– *bb ybyb*; 3, D^R– N^N– *B*– *Yb*–; 4, D^R– N^N– *B*– *ybyb*.

6

FIGURE 6. *H. melpomene* from brood PM24. 1, female parent, captured at Pongo de Aguirre, Río Huallaga; 2–5, male progeny. 1, *dd* N^BN^B *B*– *ybyb*; 2, D^R– N^N– *B*– *Yb*–; 3, D^R– N^N– *B*– *Yb*–; 4, apparent D^R– N^N– *B*– *ybyb*; 5, D^R– N^BN^B *B*– *ybyb* (the yellow HW bar on the upperside has been discoloured by emergence in a humid environment). An excess of N^N– phenotypes (yellow FW band with narrow red outer) is probably caused by gene *m*/*M*; apparent N^N– phenotypes are produced in *mm* N^BN^B genotypes.

In *erato* dennis–ray patterns were always orange-red, and the red FW bars were always crimson; in these crosses there was no evidence for a separate locus determining a colour switch (see Sheppard *et al.* (1985) for the locus *or*/*Or*).

The purpose of this section, which was added at the request of Professor J. R. G. Turner, one of the authors of Sheppard *et al.* (1985), is to point out that the basic genetics of *H. erato* races may be considerably simpler than has been realized. The pure phenotypes of *H. erato* are remarkably constant and appear to be affected by rather few loci. In contrast, single- and multi-locus hybrid genotypes generate variable phenotypes with strong epistasis. This may have led to an overestimate of the number of loci involved. Another possibility is that the genetics of other races of *H. erato* is more complex than that of the races studied here. Only further work on the genetics of the races will resolve the matter.

Genetics of melpomene

The hypothesis and tests

Because a total of only 92 adults in five broods were reared for *H. melpomene*, all of which have wild male parents of unknown phenotype, little more can be done than test whether the genetics here conform to previous hypotheses for similar races (Sheppard *et al.* 1985). The genetic hypothesis, together with phenotypic data from the broods, is given in table 2. Essentially, a supergene (block of tightly linked loci) D^R/d controls presence as opposed to absence of both rays and dennis (the same name is used in *melpomene* as in *erato*, even though homology has not been proved and the locus does not control FW band colour in *melpomene*); N^N/N^B controls absence compared with presence of yellow FW band, as well as a narrow or broad red band (if present); b/B controls absence and presence of red FW band; and Yb/yb controls absence or presence of the yellow HW bar. Evidence exists that N^N/N^B and Yb/yb, as well as b/B and D^R/d, are both linked pairs (Sheppard *et al.* 1985).

Dennis and ray are monomorphically present in all broods. (In PM16 all offspring are rayed, though a single individual lacks dennis; see figure 4.3 and figure 5, plate 3.) Thus nothing can be said about Mendelian ratios of D^R/d. As dennis and ray is present in all the offspring of PM24, but lacking in the female parent (figure 6, plate 3), the gene(s) determining this pattern must be dominant, agreeing with the hypothesis.

N^N/N^B segregates in only one brood, PM24 (figure 6). Because the female parent

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Description of plate 3

Figure 5. *H. melpomene* from brood PM16. Female progeny. Genotypes: 1, D^R– N^N– *bb Yb*–; 2, D^R– N^N– *bb ybyb*; 3, D^R– N^N– *B*– *Yb*–; 4, D^R– N^N– *B*– *ybyb*.

Figure 6. *H. melpomene* from brood PM24. 1, female parent, captured at Pongo de Aguirre, Río Huallaga; 2–5, male progeny. 1, *dd* N^BN^B *B*– *ybyb*; 2, D^R– N^N– *B*– *Yb*–; 3, D^R– N^N– *B*– *Yb*–; 4, apparent D^R– N^N– *B*– *ybyb*; 5, D^R– N^BN^B *B*– *ybyb* (the yellow HW bar on the upperside has been discoloured by emergence in a humid environment). An excess of N^N– phenotypes (yellow FW band with narrow red outer) is probably caused by gene *m*/*M*; apparent N^N– phenotypes are produced in *mm* N^BN^B genotypes.

was N^BN^B (figure 6.1), and there are N^N– and N^BN^B phenotypes in the progeny, the male must have been N^NN^B, giving an expected 1 : 1 ratio in the progeny. This hypothesis is firmly rejected ($G_1 = 12.5^{***}$): there are too many N^N–phenotypes. PM24 alone gives little idea of the cause, but an unlinked gene *m/M* can also produce N^N-like phenotypes when homozygous *mm* in N^BN^B individuals (*m/M* was first found in crosses between Costa Rican postman and rayed Brazilian races (Mallet & Gilbert 1989). Another possible explanation, sperm mixing from separate field matings, is seemingly ruled out by evidence for sperm precedence (L. E. Gilbert, unpublished data). Sheppard *et al.* (1985) suggest that N^NN^B

TABLE 2. BROODS OF *MELPOMENE*

(Parents and offspring shown as in table 1, though all parents of *melpomene* broods were wild-caught. Explanation of phenotypes (genotypes in square brackets) is given below, with Amazonian phenotypes first.)

phenotype [genotype]	description
D^R– [D^RD^R, D^Rd]	dennis and ray
dd [*dd*]	no dennis or ray
R– [*RR*, *Rr*]	rays present (in PM16: in other broods, rays are always present, and shown as superscript; in D^R– phenotypes, absent in dd phenotypes)
rr [*rr*]	rays absent (not found in any of these broods, except in dd phenotypes).
N^N– [N^NN^N, N^NN^B]	yellow FW band present. Red FW band, if present, narrow and pushed distad
N^BN^B [N^BN^B]	no yellow FW band. Red band, if present, broad
bb [*bb*]	red FW band absent
B– [*BB*, *Bb*]	red FW band present
Yb– [*YbYb*, *Ybyb*]	yellow HW bar absent
Ybyb [*Ybyb*]	yellow HW bar present as a shadow, or enlarged dennis bar, which has similar shape and position to yellow bar
ybyb [*ybyb*]	yellow HW bar present

brood		phenotype	[genotype]	where captured
PM2				
female parent 1751		D^R– N^N– B– Yb–	[D^RD^R N^NN^N *Bb Yb*Yb]	km 62
male parent		—	D^RD^R N^NN^N *bb* YbYb]	—
	1/1	D^R– N^N– bb Yb–		
	2/1	D^R– N^N– B– Yb–		
PM5				
female parent 1752		D^R– N^N– bb Yb–	[D^RD^R N^NN^N *bb Ybyb*]	km 62
male parent		—	[D^RD^R N^NN^N *bb ybyb*]	—
	9/3	D^R– N^N–bb Yb–		
	4/6	D^R– N^N– bb ybyb		
PM16				
female parent 1775		D– R– N^N– B– ybyb	[*Dd R*R N^NN^N *Bb ybyb*]	km 62
male parent		—	[D*d* Rr N^NN^N *bb Ybyb*]	—
	1/0	D– R– N^N– bb Yb–		
	2/2	D– R– N^N– bb Ybyb		
	0/2	D– R– N^N– bb ybyb		
	3/1	D– R– N^N– B– Ybyb		
	0/1	D– R– N^N– B– ybyb		
	1/0	dd R– N^N– B– ybyb		

TABLE 2. (*cont.*)

brood		phenotype	[genotype]	where captured
PM17				
female parent 1771		D^R– N^N– B– Ybyb	[D^RD^R N^NN^N *Bb Ybyb*]	km 62
male parent		—	[D^RD^R N^NN^N *bb* YbYb]	—
	3/3	D^R– N^N– bb Yb–		
	3/3	D^R– N^N– bb Ybyb		
	1/3	D^R– N^N– B– Yb–		
	5/3	D^R– N^N– B– Ybyb		
PM24[a, b]				
female parent 1841		dd N^BN^B B– ybyb	[*dd* N^BN^B *B*B *ybyb*]	Pongo de Aguirre
male parent		—	D^RD^R N^NN^B BB *Ybyb*]	—
	6/8	D^R– N^N– B– Ybyb		
	4/5	D^R– N^N– B– ybyb		
	4/1	D^R– N^BN^B B– ybyb		

[a] This brood can be explained by assuming a locus, *m/M*, that interacts with the other genes controlling FW band. N^N-like bands in the presence of *b* alleles can be produced in two ways: first in genotypes N^N– *B*– *M*–, and secondly in genotypes N^BN^B *B*– *mm*. The most likely parents in PM24 are then female *Mm* N^BN^B *BB*, and male *mm* N^NN^B *B*–. One half of the N^BN^B offspring would appear N^N–like, giving three quarters with N^N–like phenotypes overall. A gene similar to *m/M* (presumably the same) was first found in crosses between Costa Rican postman *melpomene* and north Brazilian rayed stock (Mallet & Gilbert 1989), and was shown to be unlinked to N^N/N^B. The *m* allele has an unknown function in the rayed races, presumably enhancing the yellow of the FW band in N^NN^N *mm bb* genotypes.

[b] A presumed contaminant with phenotype D^R– N^N– bb Yb– has been excluded.

heterozygotes may often be distinguished by the intermediate width of the red band. PM16 has individuals with narrow red bands that could be N^NN^N (figure 5.3), but so does PM24 (figure 6.2), where they must be N^NN^B. This unexplained variation may be due to the action of *m/M*, or other interacting genes. In view of these problems, I have here lumped N^NN^N with N^NN^B as the phenotype N^N–.

The gene *b/B* segregates in PM2, 16, 17, and conforms to a 1:1 hypothesis in all ($G_3 = 0.3$); *Yb/yb* also conforms to expectation (PM5, 16, 17, 24, $G_4 = 2.9$). *Ybyb* heterozygotes, which according to the hypothesis show as shadow bars on the underside HW, are not always visible, especially on rayed backgrounds. For instance, the female parent of PM5 had no obvious shadow (phenotypically Yb–), and yet produced some ybyb offspring, and so must have been *Ybyb*. Similarly, one offspring in PM16 was scored Yb–, but had a ybyb mother, and so must have been *Ybyb*.

Interaction and linkage

The genes N^N/N^B and *Yb/yb* interact strongly: in PM24 no N^BN^B Yb– phenotypes were found ($G_1 = 8.0$**), probably owing to linkage, which has also been observed in other crosses (Sheppard *et al.* 1985). Some of the previous crosses have relatively high levels of recombination between the two markers in males (as in PM24 which has *ca.* 32% 'recombinants', all of phenotype N^N ybyb), whereas others have recombination fractions of about 1% (Sheppard *et al.* 1985; Mallet & Gilbert 1989). Once again, the unlinked recessive allele *m* can explain these anomalies: *mm* N^BN^B *ybyb* genotypes appear similar to N^N– *ybyb* recombinants. See footnote to table 2 for details.

The other genes were also tested for interaction. According to previous work (Sheppard *et al.* 1985), b/B and D^R/d are linked, but none of the present broods segregated for D^R/d. However, the pairs b/B and D^R/d, as well as N^N/N^B and Yb/yb, are in stronger linkage disequilibrium than other unlinked pairs in the hybrid zone, which supports the hypothesis of linkage (Mallet *et al.* 1989). Interactions between the other gene combinations, and those involving sex were also investigated: none were significant. There is of course very strong phenotypic epistasis of N^N/N^B, b/B, and m/M in producing the narrow yellow or broad red FW band.

Much work remains to be done on the colour-pattern genetics of *melpomene*. There is considerable variation within phenotype classes, perhaps due to modifiers. Orange compared with red colour of dennis, ray, and FW band may have a simple mode of inheritance as reported by Sheppard *et al.* (1985), but these broods give little evidence one way or the other.

Evidence for crossing-over within the D^R/d supergene

In PM16, all individuals carried dennis and ray except one, which had rays without dennis (figure 4.3). This is potentially a crossover within the D^R/d supergene; as far as I know no *melpomene* has ever been recorded of this phenotype. A related species, *H. timareta* is, however, polymorphic for ray-only phenotypes (see Turner & Crane (1982); as *H. melpomene contiguus*). Because $\frac{1}{13}$ is not significantly different from $\frac{1}{4}$, the expected fraction of rayed-only phenotypes in a $D^R d^R \times D^R d^r$ cross, it cannot be proved whether the mutation or crossover occurred in PM16, or was inherited from farther up their lineages.

A single individual collected in the field among 903 *melpomene* over the whole study had the reciprocal phenotype: dennis and no ray (figure 4.2). There is one race of *melpomene* fixed for this dennis-only phenotype in the Guianas (Turner 1971), but the phenotype has not to my knowledge been reported elsewhere.

Are the races incompatible?

There was no evidence for incompatibility between the races in either species. 1. The majority of females were highly fertile when given enough nectar and pollen. Small brood sizes resulted only from death or removal of the female parent. 2. The hatch rate of eggs was 89% in *erato* and 92% in *melpomene*, with most failures due to parasitoids and handling. 3. Larval survival was low (40% in *erato*, and 17% in *melpomene*), but was probably mainly caused by disease and shortages of larval food, the latter especially in *melpomene*. 4. The sex ratio of hybrid broods has support limits of 44–54% males (PE11 from a pure population was excluded) in *erato*, and 43–63% in *melpomene*; and there is no evidence of heterogeneity between broods (in *erato* hybrid broods, $G_{19} = 20.5$; in *melpomene* $G_4 = 0.6$). Thus there is no evidence for reduced viability of the heterogametic sex: a 'Haldane's rule' (Haldane 1922; Jones & Barton 1985). 5. It has already been noted that there is no evidence for coadaptation between autosomes that affects viability. 6. There was no evidence for mating preferences in *erato*; it was easy to obtain matings of virgin females with healthy males of all phenotypes. 7. In *erato*, there was no significant excess of homozygotes (F; see above), and field D^R/d phenotypic ratios conformed to Hardy–Weinberg (Mallet *et al.* 1989).

These tests are consistent with reproductive compatibility and random mating, though the small numbers involved mean that weak incompatibility or slight deviations from random mating would be difficult to detect. The results agree with the general conclusions of a variety of studies of *Heliconius*, in which no premating or postmating reproductive isolation between races has ever been shown (Sheppard *et al.* 1985), apart from that generated by the effect of differing warning colour.

Discussion

Comparisons with the genetics of other races of erato and melpomene

Most of the genes noted here are similar to those reported by Sheppard *et al.* (1985). However, Sheppard's *erato* alleles have different effects to those in Peru. For example, as in Peru, Sheppard's *Cr/cr* interacts with *Sd/sd* (the latter closely linked with, if not identical to, Yl^t/yl^T) to produce a yellow HW bar in East Brazilian postman *erato*; but in Brazil the allele *cr* also produces cream rectangles (hence the name '*Cr*'; the superscript '*h*' introduced here refers to the Huallaga region) on the HW margin, as well as affecting the expression of a median FW yellow line; neither is present in Peru. In Peru *Sd* produces a narrow FW band, whereas in Brazilian rayed populations the band produced by *Sd* (or *Yl*) is often broad or broken into separate spots. Without crosses between the allopatric races, it is not certain that the genes are the same, but because the details of gene action and interaction are similar across races (and notably different between species), homology within each species seems most likely. Linkage, like that between N^N/N^B and *Yb/yb* in *melpomene*, provides further evidence of homology. In the future it will be possible to use linkage in *erato* to identify Cr/Cr^h in other races; starch-gel electrophoresis has shown that Cr/Cr^h is linked to the Aconitase locus (Mallet & King 1989).

It is interesting that the Peruvian yellow HW bar is more similar to that of the postman race of Central America than to the bar of the East Brazil race, even though genetically the Peruvian bar is more similar to that of East Brazil. In Central America, the bar produced by the Cr^P allele does not interact strongly with other genes (Sheppard *et al.* 1985). These geographic differences in epistasis are similar to those in *Papilio dardanus*, in which background modifiers are important for the expression of geographically restricted mimicry (Clarke & Sheppard 1960). In *H. erato*, some phenotypes are constant between geographical areas, even though their genetic control may differ. Possibly these similar phenotypes evolved in parallel; this would seem likely as *erato* is mimetic. Another possibility is that, in spite of the stabilization of the phenotype by selection, the genetic determination of that pattern has diverged in isolated or distant populations without altering the pattern.

The first hypothesis, of parallel mimetic evolution, would require that races of *erato* and *melpomene* converged on a species with the yellow HW bar phenotype independently in five disjunct areas: Central America, central Colombia, southern Colombia, Peru and Brazil. Because no potential model species now exists in sympatry with any postman race except in East Brazil, where postman *H. erato* partly overlaps with *H. besckei*, the hypothesis seems unlikely. Instead the distribution of the postman races of *erato* and *melpomene* suggests that they may

have originally been connected, but were displaced to the perhiphery by rayed races evolving in the Amazon basin. Drift (or selection other than that stabilizing the pattern) may have led to the differences in genetic control, presumably before the East Brazil race separated from the Peruvian postman race. This evolution of the control of colour pattern gives a clue as to how reproductive isolation may arise geographically: geographic differences in reproductive compatibility can evolve through drift or selection, in spite of the strong selection that maintains reproductive compatibility within each population.

Evidence for supergenes

Without further experiments, it is difficult to prove whether the dennis/ray variants in each of *erato* and *melpomene* (figure 4) are recombinants or mutations at their respective D^R/d genes, mutations at modifier genes, or non-genetic variations. These variants all come from the hybrid zones, and I know of only a few similar variants that have ever been collected elsewhere, all from other *erato* hybrid zones (Ackery & Smiles 1976). In a hybrid zone, recombination is likely because of the abundance of heterozygotes; in contrast, mutants and non-genetic variants should not be commoner in hybrid zones than in the pure races (unless there is hybrid dysgenesis, for which there is no evidence in *Heliconius*). Recombination therefore seems most likely to have produced these aberrations: the genes determining dennis and ray in both species are probably 'supergenes'.

D^R/d is known to be linked to b/B in *melpomene* (30% recombination in males: see Sheppard *et al.* (1985)), but nothing can be said about the order of genes on the chromosome from either of the two field-caught individuals. Consideration of PM16 gives support for the gene order *bDR*; *bRD* requires more crossovers. These crossovers could have occurred in any ancestor of either of the parents, and b/B and D^R/d are loosely linked, so the evidence for this gene order is weak.

Why are warning colours not polygenic?

A surprising fact about the genetics of mimicry, amply confirmed here, is that major changes can be wrought by single genes (including linked blocks of individual loci). It is often argued that many such examples of single genes with strong effects may be mistaken interpretations of multiple loci, perhaps with threshold effects. However, strong evidence for single genes (or at least linked blocks) is provided here by interaction between phenotypes, caused by pleiotropy, linkage, and epistasis, coupled with a lack of linkage interactions with other phenotypes. The linkage of Cr/Cr^h to *Aconitase* in *erato* is further evidence of the reality of one of these genes.

In colour pattern evolution, such major gene effects may be due to evolution in genetic 'background' (i.e. polygenic modifiers: Clarke & Sheppard (1960)), or in dominance at single genes (Stewart & Lees 1987), possibly caused by modifiers. Crosses between species of *Heliconius* demonstrate that the background is important for allelic expression in crosses at this taxonomic level (P. M. Brakefield, unpublished data; L. E. Gilbert, unpublished data). In interracial crosses, on the other hand, there is a greater importance of a few genes with major effects (this study; Sheppard *et al.* 1985): only a small residual effect of background is evident.

Some neo-Darwinists (see, for example, Charlesworth *et al.* 1982, p. 489) have pointed out that mutations affecting superficial colour patterns can have major effects because they are unlikely to interfere with the development of vital organs. But the pattern of a mimetic butterfly is itself a vital organ: the selection that predators exert on mimetic genes is often above 10% (Mallet & Barton 1989*a*), equivalent to selection against a rather severe developmental abnormality.

There have been various attempts to explain this neo-Darwinian anomaly. First, Sheppard (1959) showed how predation on polymorphic Batesian mimics should select against the production of intermediates. This could lead to the evolution of close linkage of controlling genes, to form supergenes. On the other hand, supergenes are not expected to coalesce in Müllerian mimics like *Heliconius*: in these species polymorphisms are transient and unstable, which should limit the selection for linkage. Instead, Nicholson (1927), Turner (1977) and Sheppard *et al.* (1985) have proposed a 'sieve' hypothesis for the evolution of Müllerian mimicry. Mutations of small effect would be little protected by their slight similarity to the model, and would lose the advantage of similarity to conspecifics. Only mutations that at a single step gain a close similarity to the model will be advantageous; selection will then perfect the mimicry in the usual polygenic way.

Such a sieve may explain single genes of major effect, but it cannot explain the evolution of supergenes. Yet Müllerian mimics do have supergenes. For example, in both *erato* and *melpomene* dennis and ray are obviously functionally related, as they co-occur in so many races and are usually the same orange-red colour. But, because they are separable by crossovers (or mutations), at least two tightly linked mutations seem to be needed to perfect the pattern in each species. Sheppard *et al.* (1985) further postulate that the yellow FW band is, in *erato*, a separate element in the same supergene, rather than a pleiotropic effect of one of the D/d or R/r genes. In addition, individual elements within supergenes may themselves be supergenes, because each element controls a sharp mimetic pattern that cleanly crosses compartmental boundaries in the wing (Sibatani 1980; Nijhout & Wray 1989). The genetically simple switch genes we now observe seem to have arisen somewhat gradually by evolution *within* each supergene, as well as by selection at unlinked background modifiers. These supergenes may owe their existence, not to selection for tight linkage, but to the constraints of the genetic system: there may simply be few genes that control qualitative shifts in colour pattern, and these may be found in linked blocks (see also Turner 1984). This is quite common for other functionally related traits that are not *a priori* expected to evolve closer linkage, and which may have arisen by gene duplication: for instance the bithorax complex that controls insect segmentation (Peifer *et al.* 1987), or genes involved in the ability to taste bitter compounds in mice (Lush & Holland 1989).

The dominance sieve and reconstruction of the ancestral pattern

An advantageous mutant will increase in frequency more readily if it is dominant; recessives will often be lost before being tested by selection. With local effective population sizes of about 1000, an advantageous dominant with $s \approx 0.1$ is 25 times more likely to become fixed that a similarly advantageous recessive

(Clarke *et al.* 1985). An additional possible reason for the evolution of dominant phenotypes is 'dominance drive', which favours dominant alleles simply because they produce selective asymmetries across warning-colour hybrid zones (Mallet 1986; Mallet & Barton 1989*b*). Such selection causes the dominant pattern to spread behind a moving cline.

This 'dominance sieve' has been used to reconstruct hypothetical all-recessive yellow-barred ancestors in both *erato* and *melpomene* (Turner 1976 *et seq.*; Sheppard *et al.* 1985). Some related species (*H. charitonia* and *H. nattereri* respectively) are yellow-barred as expected. However, the method makes a number of questionable assumptions. First, it is assumed that dominance is complete and unchanged throughout the history of the colour pattern radiation. In Peru partial dominance is frequent and, especially in *erato*, there is substantial dominance variability, at least some of which is genetic, and which could form the basis for evolution of dominance (see also the *Ybs* gene which affects dominance in *H. erato* (Sheppard *et al.* 1985)). Similar dominance variability is found in colour patterns of the mimetic butterfly *Hypolimnas misippus* (Smith & Gordon 1987). Second, although yellow and black outgroup species exist, other possible outgroups have red patterns, for example the non-mimetic species *Heliconius clysonimus* and *H. timareta*. Novel colour patterns have evolved so often in *Heliconius* that outgroup analysis may be useless. Third, at least some of the genes appear to be tightly linked blocks of loci that may have dominant and recessive components, for example D^R/d. The dominance sieve can give a possible ancestor, but one that is far from certain.

Evidence for coadaptation

The genes revealed by these crosses are pleiotropic and epistatic; for instance in the HW bar of *erato* (figure 2), or the FW band of *melpomene* (figure 6). Lines of determination leading from genotype to phenotype form a network (Wright 1980), diagrammed in figure 7 for *erato*. Genetic resolution in *Heliconius* is not yet detailed enough to distinguish pleiotropy from tight linkage, so many of the colour-pattern 'genes' we observe could in fact be linked blocks of genes, or supergenes. However, the epistasis is more certain. It would be interesting to know whether this epistasis is a form of coadaptation, i.e. was produced by selection, or is merely a fixed genetic feature. In the brine shrimp *Tigriopus*, coadaptation seems certain because multilocus hybrids do not mature (Burton 1987). In *Heliconius*, the low fitness of hybrids would instead be due to bird predation on less memorable colour-pattern morphs, and so will not be detectable as inviability during rearing. Pattern elements such as the FW band in *melpomene* and the yellow HW bar of *erato* are sharply contrasting bands of colour in the pure races. The fuzziness and variability of intermediate colour patterns suggests that canalization has broken down in hybrids. Because such fuzzy pattern elements are rare in pure races, and seem poor advertising to our eyes, an objective argument can be made that the genes controlling patterns such as the yellow HW bar of *erato* are coadapted (see also Clarke & Sheppard 1960).

Each pattern element is itself likely to be integrated with others to form a startling and memorable signal; a higher level of pattern coadaptation than that

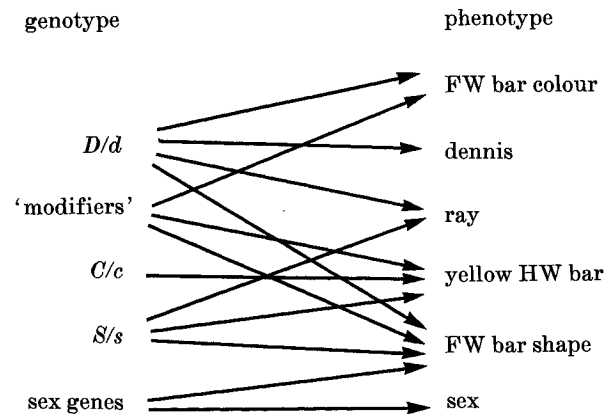

FIGURE 7. Pleiotropy and epistasis in *erato*. Arrows connect genes to the phenotypes they control (see Results for details). Divergent arrows indicate pleiotropy (including linkage within supergenes), convergent arrows epistasis. 'Modifiers' have not been identified, but are probably genetic because of significant between-brood differences. Each phenotypic element is itself integrated into an overall warning pattern upon which fitness depends; a higher level of coadaptation than shown in this diagram.

shown in figure 7. Colour and pattern combinations of *Heliconius* are visually striking today because less impressive combinations failed to deter predators in the past.

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