The Genetic Basis of a Species-Specific Character in the *Drosophila virilis* Species Group

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ABSTRACT

The genetic basis of the species-specific dorsal abdominal stripe of *Drosophila novamexicana* was examined. The dorsal stripe is present in *D. novamexicana* and absent in all other members of the *Drosophila virilis* species group. Interspecific crosses between *D. novamexicana* and genetically marked *D. virilis* revealed that all four of the autosomes (except the tiny dot chromosome, which was not marked) and the sex chromosomes (the *X* and *Y* chromosome effects could not be disentangled) showed a significant effect on the width of the dorsal stripe. All the autosomes act approximately additively; only minor interactions were detected among them. No significant maternal effects were found. This means that a minimum of five loci are involved in the character difference between the two species, and this is the maximum number that this technique could discern. These results suggest that, based on the number of factors involved in the character difference, the inheritance of this character should be considered polygenic, but because chromosome *2* (the largest chromosome in the species) contributed over half of the variance toward the character difference, it is best to consider the inheritance oligogenic based on effect. The implications of these findings are discussed in light of the importance of macromutation in speciation and the sex chromosome theory of speciation.

" . . . we know virtually nothing about the genetic changes that occur in species formation."

LEWONTIN (1974, p. 159)

THIS lack of information is unfortunate, because to understand fully macroevolutionary change it is vitally important to know what genetic changes have occurred. This absence of knowledge has created a controversy about the actual genetic mechanisms of speciation and evolutionary change. Consequently, there has been a recent resurgence of interest in the genetics of speciation and species differences.

One approach to help in answering these questions regarding the genetics of evolutionary change is to study the genetic differences among closely related species (MAYNARD SMITH 1983). By analyzing these differences it may be possible to infer what genetic changes have occurred and begin to understand the genetics of macroevolutionary change. This information could also contribute to our understanding of the modes of speciation. For example, it may be possible that we could determine what the population structure was like during speciation by knowing the genetic differences among closely related species. TEMPLETON (1981, 1982a,b) has distinguished two different genetic architectures that might be expected to occur given the population structure of a species in the process of speciation. He indicates that one might expect only one or a small number of genes to contribute to a character difference if speciation occurred by a founding event, but that speciation in a large geographic population would be expected to proceed with many segregating factors. However, BARTON and CHARLESWORTH (1984) suggest that it may be easier to get a polygenic architecture by genetic drift. Along similar lines, WRIGHT (1982a,b) has suggested that a species occupying a newly available niche might be expected to be differentiated by only a few genes. In this instance, even if the mutations have unfavorable pleiotropic effects, they may be superior enough due to a lack of competition from other species. The theory of rapid evolutionary change resulting from only a few genetic changes has also been discussed by LANDE (1983) and MAYNARD SMITH (1983).

The *Drosophila virilis* species group presents an opportunity to examine the genetic differences among some closely related species. The 11 species recognized in the group are separated into two monophyletic groups, or phylads (THROCKMORTON 1982). The *D. virilis* phylad is of particular importance because all four species can hybridize to a certain extent (THROCKMORTON 1982). Although a variety of morphological characters have been mentioned as potential characters for separating these species (PATTERSON 1943), the primary ways of distinguishing the members of this group are by their karyotypes and crossabilities. However, *Drosophila novamexicana* is a

notable exception to the other members of the species group, because it is much lighter in color (PATTERSON and STONE 1949), and has a distinctive dorsal stripe on the abdomen that easily separates this species from all other members of the *D. virilis* group. The stripe can be described as a paler mid-dorsal anteroposterior stripe running down the abdominal tergites (see Figure 1). In addition, the darker lateral areas of the tergites are more lightly pigmented than in the other species. It should be mentioned that *Drosophila flavomontana* is also relatively light in overall body color, but it does not possess a stripe and it is not nearly as pale in color as *D. novamexicana*.

The dorsal stripe may just be a convenient way of measuring the lighter color of *D. novamexicana*, which is probably the important character. The importance of the lighter color may have to do with adaptation to a desert environment. Members of the *D. virilis* species group are mainly cold climate species, but *D. novamexicana* lives in the much warmer southwest desert region of the United States (THROCKMORTON 1982). This suggests that the lighter color of *D. novamexicana* may be due to selection for ultraviolet radiation protection (JACOBS 1974), heat-desiccation tolerance (DAVID *et al.* 1985), thermal regulation (NEEDHAM 1974), or a combination of these factors. In the genus *Drosophila*, it is known that different species form similar clines of color pattern (DAVID *et al.* 1985; CAPY, DAVID and ROBERTSON 1988), which is a strong indication that natural selection is acting on coloration. These observations imply that the lighter color may be a novel adaptation for *D. novamexicana*, which in any case represents a radical departure from the other members of the *D. virilis* species group. These are the kind of characters that are the most interesting to study genetically. Therefore, I have undertaken an analysis of this character to determine its genetic basis.

MATERIALS AND METHODS

The *D. virilis* strain (National Drosophila Species Resource Center No. 15010-1051.83) used in the crosses had all its autosomes marked with recessive markers, except for chromosome *6*, which is the tiny dot chromosome (only about one map unit long). The markers, their chromosomal location and map units, as given by ALEXANDER (1976), are as follows: *broken* (*b*, 2-188.0); *tiny bristles* (*tb*, 3-104.0), *gap-L2* (*gp*, 3-118.5); *cardinal* (*cd*, 4-32.2); *peach* (*pe*, 5-203.0). The *D. novamexicana* strain (National Drosophila Species Resource Center No. 15010–1031.5; Moab, Utah, 1975B) was selected because it has the widest dorsal stripe (see Table 1) and lightest body color of the four strains examined. This greatly facilitated the measurement of the dorsal stripe in the hybrids. The other three strains that were examined, and their corresponding National Drosophila Species Resource Center stock numbers are as follows: (15010-1031.4), Moab, Utah, 1975A; (15010-1031.7), Patagonia, Arizona; (15010-1031.8), San Antonio, New Mexico, June 1947.

The flies were raised in half pint milk bottles on a 4:1 mixture of Carolina Biologicals Drosophila instant media to corn starch. About 0.5 g of live baker's yeast was also added. The incubator was on a 12:12 light-dark cycle and kept at approximately 24°. All crosses were performed as mass matings with about 30 flies of each sex placed together in a half pint bottle and the adults were changed to new bottles when larvae were observed (about once a week).

The autosomal effects were examined by measuring the female flies from a backcross analysis. Males of *D. virilis* were crossed to females of *D. novamexicana*, and then the male F_1 hybrids were crossed to the females of *D. virilis*. Because only the F_1 male hybrids were used in the cross, no recombination could occur, so that entire chromosomal effects were examined. I was unable to use F_1 progeny from the opposite cross of females of *D. virilis* to males of *D. novamexicana*, because the male offspring from this cross are often sterile (PATTERSON and STONE 1949) or have low fertility (ORR and COYNE 1989). I attempted this cross, but did not obtain enough backcross offspring to perform a genetic analysis. I did not perform a backcross through the female F_1 hybrids, because the map distances are large and several of the *D. virilis* genetic markers are at the end of the chromosomes. Although there are some fixed chromosomal differences between *D. virilis* and *D. novamexicana* on all the major chromosomes, they constitute only a small part of the genome (PATTERSON and STONE 1952). This would mean that most of the genome would be unmarked due to recombination in the females.

The sex chromosome effect was measured by analyzing the reciprocal male F_1 hybrids. By comparing the width of the dorsal stripe between these two crosses, I could assess the effects of the sex chromosomes. This is because the male offspring from these crosses are genetically identical, with the exception of their *X* and *Y* chromosomes and the origin of their maternal cytotype. Unfortunately, the *X* and *Y* chromosome effects are confounded using this approach, and no test was possible for disentangling these effects. I was unable to separate the *X* and *Y* chromosome effect for two reasons: (1) I could not make the appropriate backcross due to the infertility of the male F_1 hybrids, as mentioned above, and (2) I was unable to make the backcross in the opposite direction, because there are no autosomal marker stocks for *D. novamexicana*. However, the sex chromosome effect measured here probably represents the *X* chromosome effect, because it is well known that the *Y* chromosome is to a large extent genetically inert when it comes to morphological characters (DRONOMRAJU 1965; WILLIAMSON 1976), so it is unlikely that it would contribute to the dorsal stripe. In order to avoid confounding maternal effects with the sex chromosome effect, I compared the reciprocal female F_1 hybrids from the crosses mentioned above. In this case, both female F_1 hybrids have the same genetic constitution, and differ only in their maternal cytotype.

In order to account for the effect of body size, the length of the thorax was measured along with the dorsal stripe width. The stripe width was then normalized with the thorax length for the statistical analyses. The width of the dorsal stripe and the length of the thorax were measured by using an ocular micrometer at 42× magnification. The dorsal stripe measurement was taken on the third tergal segment of the abdomen. The thorax length was determined by measuring from the anterior most part of the thorax to the posterior part of the scutellum.

RESULTS

The means for the dorsal stripe width of the four strains of *D. novamexicana* examined are presented in

TABLE 1

Mean dorsal stripe width for *D. novamexicana*

Strain	*N*	Mean stripe width	Standard deviation	Standard error
Moab, Utah (0.5)	20	397.4	88.76	19.84
Moab, Utah (0.4)	20	321.7	85.65	19.15
San Antonio, New Mexico (0.8)	20	265.6	45.26	10.12
Patagonia, Arizona (0.7)	20	235.6	63.01	14.09

All measurements are in micrometers. See MATERIALS AND METHODS for a complete description of the strains.

TABLE 2

Means and standard errors for the stripe width and thorax length

Genotype	*N*	Stripe width	Thorax length	Dorsal stripe/(thorax length)^2 ($\times 10^5$)
+ + + +	15	181.8 ± 6.4	1454 ± 6.9	85.85 ± 2.87
b + + +	15	98.3 ± 7.9	1437 ± 7.8	47.55 ± 3.96
+ *gp* + +	15	175.7 ± 4.3	1438 ± 8.2	85.00 ± 2.38
+ + *cd* +	15	171.3 ± 5.2	1438 ± 6.2	82.80 ± 2.49
+ + + *pe*	15	159.2 ± 9.2	1413 ± 6.5	79.56 ± 4.42
b gp + +	15	66.9 ± 12.4	1419 ± 9.8	32.71 ± 5.95
b + + *pe*	15	66.1 ± 7.0	1410 ± 10.6	32.90 ± 3.28
b + *cd* +	15	79.1 ± 8.8	1437 ± 9.6	38.42 ± 4.23
+ *gp cd* +	15	147.9 ± 5.5	1412 ± 6.4	74.12 ± 2.81
+ *gp* + *pe*	15	153.9 ± 4.2	1386 ± 7.8	80.04 ± 2.08
+ + *cd pe*	15	148.7 ± 3.9	1405 ± 7.0	75.25 ± 1.92
b gp + *pe*	15	73.9 ± 8.5	1410 ± 13.6	37.14 ± 4.24
b gp cd +	15	4.3 ± 3.0	1364 ± 6.0	2.29 ± 1.60
b + *cd pe*	15	50.4 ± 8.4	1397 ± 7.8	25.88 ± 4.41
+ *gp cd pe*	15	130.5 ± 5.2	1364 ± 4.5	70.04 ± 2.63
b gp cd pe	15	54.8 ± 11.1	1427 ± 10.0	26.85 ± 5.45

All measurements are in micrometers.

Table 1. The variances among these strains were not homogeneous (SOKAL and ROHLF 1981) as determined by an F test ($F_{4,19} = 3.84$, $P = 0.0188$), so a ln transformation was performed to correct this distributional problem ($F_{4,19} = 2.82$, $P = 0.0542$). An analysis of variance (ANOVA) on the ln transformed data set revealed that there were significant differences among the strains ($F_{3,76} = 18.17$, $P = 0.0001$). As mentioned in the MATERIALS AND METHODS, the Moab (0.5) strain was selected for analysis because it had the widest stripe. Since the most extreme phenotype was selected, the genetic analysis should be viewed with some caution, because it is possible that a different result would be found if another strain was examined. However, it is unlikely that the character difference in this analysis is due in a large part to intraspecific polymorphisms. In fact, it is quite likely that this is only a minor part of the genetic basis of the stripe in this analysis, due to the extreme nature of the character difference between these two species.

The results of the crosses are presented in Tables 2–5 and Figures 1 and 2. The F_1 hybrids were largely intermediate when compared to the parental species,

TABLE 3

Four factor ANOVA table for the body size corrected data set

Source	d.f.	Sum of squares	Mean square	*F* value	*P*
2	1	141,799.6	141,799.6	712.073	0.0001
3	1	3,377.3	3,377.3	16.960	0.0001
4	1	6,788.6	6,788.6	34.090	0.0001
5	1	4,619.5	4,619.5	23.198	0.0001
23	1	930.2	930.2	4.672	0.0317
24	1	767.7	767.7	3.855	0.0508
25	1	559.9	559.9	2.812	0.0950
34	1	1,359.7	1,359.7	6.828	0.0096
35	1	131.9	131.9	0.662	0.4166
45	1	695.3	695.3	3.492	0.0630
234	1	114.3	114.3	0.574	0.4494
235	1	4.8	4.8	0.024	0.8768
245	1	657.3	657.3	3.301	0.0706
345	1	785.9	785.9	3.947	0.0482
2345	1	1,034.9	1,034.9	5.197	0.0236
Error	224	44,606.5	199.1		

This analysis was performed on the backcross data set corrected for body size by the transformation stripe width/(thorax length)^2.

but they do resemble the ancestral condition of no stripe slightly more. Using a scale where the female parental phenotypes of *D. novamexicana* = 1 and *D. virilis* = −1, the female F_1 hybrids have a degree of average dominance = −0.11 (MATHER and JINKS 1971).

All the marked autosomes showed a significant effect on the width of the dorsal stripe (Tables 3 and 5; Figure 2). There was a significant correlation between the dorsal stripe and the body size variable of thorax length ($t = 4.599$, d.f. = 239, $P = 0.0001$, $r^2 = 0.082$). In order to remove the effect of body size, two correction factors were evaluated (COYNE 1983). The first was the stripe width/thorax length and the second was the stripe width/(thorax length)^2. Both of these corrections still produced a significant association between the thorax length and the new variable: stripe width/thorax length ($t = 3.817$, d.f. = 239, $P = 0.0002$, $r^2 = 0.058$) and stripe width/(thorax length)^2 ($t = 3.043$, d.f. = 239, $P = 0.0026$, $r^2 = 0.037$). But the correlation was best removed by the stripe width/(thorax length)^2 correction factor, so this transformation was used for the body size corrected analysis. A four factor ANOVA was used to test for the effects of the autosomes. Both the uncorrected (SPICER 1990) and body size corrected (Table 3) data sets showed significant effects for all four autosomes.

In addition, the four factor ANOVA was used to test for interactions among the autosomes in the backcross (STEEL and TORRIE 1980). The ANOVA on the uncorrected data set revealed only 2 significant interactions at the $P < 0.05$ significance level (SPICER 1990), but the ANOVA on the body size corrected data set indicated 4 out of the 11 possible interactions were significant (Table 3). This suggests that the chromosomes are acting in a predominantly additive

TABLE 4

Means and standard errors for the F_1 hybrids

Genotype	N	Dorsal stripe	Thorax length	Dorsal stripe/(thorax length)^2 ($\times 10^5$)
F_1♂ (♀nov × ♂vir)	15	128.7 ± 3.33	1428 ± 5.07	63.06 ± 1.51
F_1♂ (♀vir × ♂nov)	15	90.4 ± 4.12	1350 ± 2.81	49.62 ± 2.26
F_1♀ (♀nov × ♂vir)	18	179.1 ± 6.04		
F_1♀ (♀vir × ♂nov)	18	174.0 ± 6.15		

All measurements are in micrometers. The sex chromosome effect is statistically significant, but the maternal effect is not. See text for the statistical analysis.

TABLE 5

Average effects and maternal effect

Chromosome	Dorsal stripe	Dorsal stripe/(thorax length)^2 ($\times 10^5$)
XY	38.28	13.44
2	96.89	48.61
3	18.38	7.50
4	23.60	10.63
5	23.60	8.77
Maternal effect	5.07	

The average effects are all statistically significant, but the maternal effect is not. See text for the statistical analysis.

way, although it appears that nonadditive effects are also present.

The sex chromosomes also have a significant effect on the width of the dorsal stripe (Tables 4 and 5), which, as I mention in the MATERIALS AND METHODS, probably represents the *X* chromosome effect. There was a significant association between the width of the stripe and length of the thorax in the male F_1 hybrids ($t = 7.165$, d.f. = 29, $P = 0.0001$, $r^2 = 0.635$). As in the autosomal analysis, two corrections were evaluated in an attempt to remove the effect of body size on the stripe measurements. However, both correction factors still showed a significant correlation with thorax length: stripe width/thorax length ($t = 6.118$, d.f. = 29, $P = 0.0001$, $r^2 = 0.572$) and stripe width/(thorax length)^2 ($t = 4.937$, d.f. = 29, $P = 0.0001$, $r^2 = 0.446$). As in the autosomal analysis, the correlation was best removed by the stripe width/(thorax length)^2 correction factor, so this transformation was used for the body size corrected analysis. An *F* test revealed that the variances were homogeneous, so no additional tranformation was needed. A *t* test on the uncorrected ($t = 7.220$, d.f. = 28, two-tailed $P = 0.0001$) and body size corrected ($t = 4.937$, d.f. = 28, two-tailed $P = 0.0001$) data sets both showed a significant effect that can be attributed to the sex chromosomes. There was no significant ($t = 0.589$, d.f. = 34, two-tailed $P = 0.5601$) maternal effect present (Tables 4 and 5).

These results show that all the marked autosomes and the sex chromosomes have a measurable effect

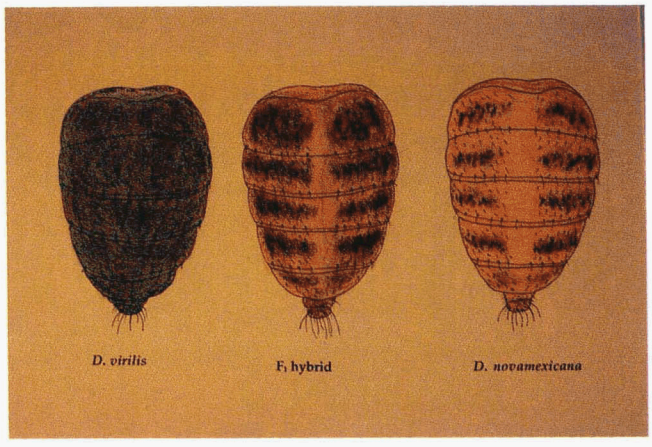

FIGURE 1.—The dorsal abdominal stripe: *D. virilis*♀, F_1 (*D. virilis*♀ × *D. novamexicana*♂) hybrid♀, *D. novamexicana*♀. Notice the overall lighter body color of *D. novamexicana* as compared to *D. virilis*.

on the width of the dorsal stripe. This means that a minimum of five loci are responsible for the character difference between *D. novamexicana* and *D. virilis*. This finding, that at least five loci are responsible for this character difference, is the maximum this technique could distinguish, because only five chromosomes were marked and no recombination was permitted to occur.

MAYNARD SMITH (1983) suggests that if five or more loci of about equal effect are present then the genetic architecture should be considered as polygenic. If only the number of factors affecting the dorsal stripe is taken into account, then it would appear that this character should be considered polygenic. But in this instance it is clear that the chromosomal effects are not about equal, because chromosome *2* contributes over half of the variance toward this character difference (Table 5). By MAYNARD SMITH's (1983) definition, it is probably best to consider the genetic basis of this character as oligogenic in terms of effect. However, due to this preliminary analysis of the dorsal stripe (only total chromosomal effects were measured), the oligogenic interpretation may be a premature assessment of the true nature of the genetic architecture. It is well known that many polygenic characters show a predominantly large effect of one particular chromosome [see CHARLESWORTH, COYNE and BARTON (1987), Table A3], but further genetic analysis reveals that many loci are involved. An example of this is the sternopleural bristles of *D. melanogaster* in which about 75% of the variance for this character is contributed by one chromosome, but a recombination analysis shows that over 17 genetic factors on this chromosome control the character difference (SHRIMPTON and ROBERTSON 1988a,b). In the present analysis on the dorsal stripe, chromosome *2* is genetically the largest chromosome (257.6 map units) of *D. virilis*, representing over 25% of the genome of this species (GUBENKO and EVGEN'EV

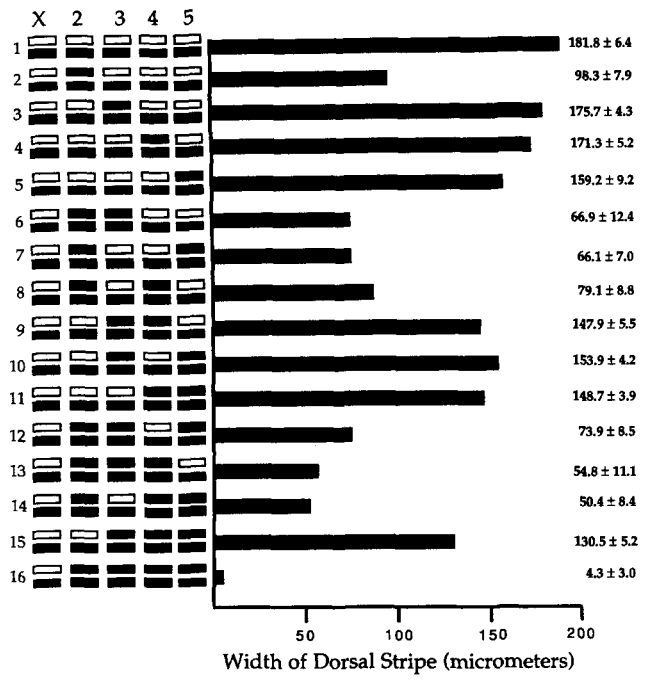

FIGURE 2.—Histogram of the mean dorsal stripe width for each genotype from females of the backcross F_1♂ (♀*D. novamexicana* × ♂*D. virilis*) × ♀*D. virilis*. The dark bars represent the *D. virilis* chromosomes and the open bars the *D. novamexicana* chromosomes. Each genotype was represented by $n = 15$ flies. Standard errors are given for each mean. See MATERIALS AND METHODS for the recessive markers that were used.

1984). Consequently, a more refined analysis may well reveal a polygenic system as opposed to the oligogenically inhertied system suggested in the present study. However, if the effect on chromosome *2* is determined to be produced by a single gene, then a purely polygenic interpretation for this character may not be tenable. Only further genetic analysis can determine the nature of this effect for certain.

DISCUSSION

The neo-Darwinian view is that macroevolution can best be explained as the result of microevolution, and that no special processes are needed to account for it (WRIGHT 1978; CHARLESWORTH, LANDE and SLATKIN 1982). But this does not mean that genes of large effect have been totally discounted (TEMPLETON 1982a; WRIGHT 1982a,b; LANDE 1983; MAYNARD SMITH 1983). This is what makes the genetics of coloration particularly interesting, because there is already literature suggesting that genes of large effect may be important in this instance. In fact, the early proponents of saltatory evolution cited these cases as their prime evidence (LEVINTON 1988). With this background it seems that the present result, showing that the dorsal stripe is controlled by several genetic factors, is somewhat at odds with the current literature on color differences among species (MAYNARD SMITH 1983), although it should be mentioned that only a few such studies exist. In Drosophila, the only interspecific cross dealing with coloration concerns the difference in pupal case color between *Drosophila americana* and *D. virilis* (STALKER 1942). In *D. virilis* the pupal case is either a gray or black, while in *D. americana* it is red. This genetic difference is due primarily to a dominant factor(s) on chromosome *5* together with modifiers on the 2–3 fusion. Some intraspecific studies on the thoracic pigmentation of *D. melanogaster* and *D. simulans* show that several loci are producing the observed differences (DAVID *et al.* 1985; CAPY, DAVID and ROBERTSON 1988). But genetic analyses on the intraspecific abdominal tergite coloration of *Drosophila polymorpha* (DA CUNHA 1949) and several members of the *Drosophila montium* species group (OHNISHI and WATANABE 1985), indicate that only one autosomal locus is responsible. More studies, like this one, are needed before a consensus can be reached on the genetics of coloration.

As for previous studies examining the genetic basis of *Drosophila* species differences, the evidence implies that most characters are polygenically inherited. STERN, SCHAEFFER and SPENCER (1944) initially suggested that a wide variety of characters studied in *D. virilis* phylad interspecific hybrids were polygenically controlled, but no complete analysis with markers was performed. The more recent studies on *D. melanogaster* subgroup hybrids by COYNE (1983), and COYNE and KREITMAN (1986) on genital morphology, and COYNE (1985) on sex comb tooth number and testis color; on Hawaiian Drosophila hybrids by VAL (1977), and TEMPLETON (1977) on head width, and CARSON and LANDE (1984) on the tibial cillia of males; and on *Drosophila auria* complex hybrids by HARA and KUROKAWA (1983, 1984) on sternite bristle number; all show several chromosomes as having an effect on the character differences. The only other study is that by SPENCER (1940) on arista number. SPENCER's (1940) results on the *D. virilis* × *D. americana* hybrids indicate polygenic inheritance for arista number, but MAYNARD SMITH (1983) considers this study inadequate because the differences between the species are too small. The dorsal stripe result presented here seems consistent with the previous observations, namely that differences between species are primarily under the control of several genes and are not monofactorial in nature.

It could be argued that the divergence of these two species occurred so long ago that a gene of major effect was initially responsible for the color difference, but that subsequently, modifiers have evolved. This must be considered a possibility for two reasons. The first is that *D. novamexicana* and *D. virilis* are not phylogenetically each other's closest relatives, which means that several speciation events have taken place since the divergence of these species (SPICER 1990).

Second, these two species have probably been separated for several million years (SPICER 1990), which would permit them ample time to diverge. However, given the view that monofactorial inheritance is a primary mode of evolutionary change, one might expect that a major locus would still be dominating the character. This does not appear to be the case for the present example. Although chromosome *2* does contribute the largest part of the variance toward the character difference (Table 5), all the chromosomes contribute a significant amount. However, if the contribution of chromosome *2* is a single gene, then the idea that the initial cause of the difference was due to a gene of large effect would have to be considered a strong possibility. Only a more refined genetic analysis of chromosome *2* will resolve this question.

These data may be useful for addressing the sex chromosome theory of speciation. This theory proposes that postzygotic reproductive isolation is produced by epistatic interactions, due largely to genetic changes on the sex chromosomes, which have accumulated by natural selection (CHARLESWORTH, COYNE and BARTON 1987). The rationale for this suggestion is based on the hemizygous nature of the sex chromosomes. Partially recessive mutations are only weakly selected on the autosomes, but on the sex chromosome they are immediately exposed to selection in males. Hence, selectively advantageous mutations, which are at least partly recessive, would become fixed disproportionately on the sex chromosomes relative to the autosomes. The implication of the sex chromosomes in fitness characters, such as viability and fertility, has been well documented (COYNE and ORR 1989), but there is a discrepancy among characters that do not strongly influence postzygotic isolation. Many studies on interspecific morphological traits reveal an approximately equal effect for all chromosomes, but others show a large *X* chromosome effect (CHARLESWORTH, COYNE and BARTON 1987). In Drosophila, the evidence is equivocal, because so few studies exist. The studies of VAL (1977), TEMPLETON (1977), and CARSON and LANDE (1984) on some Hawaiian Drosophila do seem to show a disproportionate *X* effect. But the studies of COYNE (1983, 1985), and COYNE and KREITMAN (1986) on *D. melanogaster* species group hybrids, and HARA and KUROKAWA (1983, 1984) on *D. auraria* complex hybrids, show an approximately equal effect for the sex chromosomes and autosomes. The present analysis on the dorsal stripe reveals no disproportionate effect for the sex chromosomes, which is in agreement with the sex chromosome theory of speciation (CHARLESWORTH, COYNE and BARTON 1987).

In conclusion, genetic analysis of the dorsal stripe appears to lend no support to the notion that macroevolutionary changes are primarily caused by genes of large effect. This assessment is based on a character that is quite distinct and one that might have been predicted to have a monofactorial mode of inheritance. It also concerns a character that has traditionally been considered a likely candidate for single gene effects, because color pattern genes have previously been shown to have large effects. But this was not found to be the case in this instance, because the dorsal stripe seems to be under the genetic control of several loci, although chromosome *2* does account for the majority of the variance. As for the sex chromosome theory of speciation, this represents another example of an additive character with no disproportionate *X* chromosome effect. This is the result predicted by the model, so the genetics of the dorsal stripe seems to represent another example consistent with the sex chromosome theory of speciation.

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