contrast density and, possibly, S-wave velocity or the intermittent presence of the layer would explain the lack of long-period S-wave near-vertical reflections[24]. □

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Haldane's rule has multiple genetic causes

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HALDANE'S rule states that "When in the F_1 offspring of two different animal races one sex is absent, rare, or sterile, that sex is the heterozygous [heterogametic or XY] sex"[1]. This rule represents one of the few patterns characterizing animal speciation[2,3]. Traditional explanations of Haldane's rule[1,4–6] claim that heterogametic hybrids are unfit because they lack an X chromosome that is 'compatible' with the autosomes of one species. Recent work[2,7] shows that this explanation is incorrect for hybrid sterility: contrary to prediction, homogametic hybrids carrying both X chromosomes from the same species remain fertile. Until now, similar tests have not been performed for hybrid inviability. Here I show that homogametic hybrids who carry both X chromosomes from the same species are inviable. These results show that the genetic causes of Haldane's rule differ for hybrid sterility versus inviability. Haldane's rule does not, therefore, have a single genetic basis.

The cross of *Drosophila simulans* females to *D. teissieri* males obeys Haldane's rule: only hybrid females appear[8] (Table 1). Hybrid males (the heterogametic sex in *Drosophila*) die during the first or second larval instar. The traditional Haldane–Dobzhansky–Muller[1,4–6] explanation of Haldane's rule claims that these males are inviable because they are genetically 'unbalanced': they lack some X-linked gene product from *D. teissieri* required by the *D. teissieri* autosomes to produce fit adults. Perhaps the strongest test of this theory involves producing hybrid *females* who carry both X chromosomes from the same species and a haploid set of autosomes from each species (Fig. 1). Because these 'unbalanced' female hybrids also lack an X chromosome from one species they should also be unfit[7]. Although repeated tests have disproved this explanation of Haldane's rule for hybrid sterility—unbalanced females remain fertile[2]—this simple test has not been performed for cases of Haldane's rule for inviability owing to the unavailability of the required genetic stocks.

As an attached-X chromosome (C(1)RM) is available in *D. simulans*, I was able to perform this critical test in the *D. simulans*–*D. teissieri* hybridization: the *D. simulans* C(1)RM female X *D. teissieri* male hybridization produces hybrid females who are just as genetically unbalanced as the inviable F_1 hybrid males. These females carry two *D. simulans* X chromosomes, a haploid set of autosomes from each species, a Y from *D. teisseri*, and cytoplasm from *D. simulans*, just as do the inviable F_1 males. As Table 1 shows, the results of this test are simple: no C(1)RM adult hybrid females ever appeared. Unbalanced females are, therefore, lethal. Sexing of hybrid larvae and pupae revealed that these females die in the same developmental stage as F_1 $X_{simulans}/Y_{teissieri}$ males (first or second larval instar).

The inviability of these females strongly suggests that the genetic basis of Haldane's rule differs for hybrid sterility and hybrid inviability. To test this possibility further, I did a similar analysis in a second hybridization. The *D. melanogaster* female X *D. simulans* male hybridization yields only females, whereas the reciprocal cross reportedly yields many males with a few rare females[9–11], that is, this direction of the cross is reportedly an exception to Haldane's rule. As such, this hybridization has been considered an inappropriate place to test the basis of Haldane's rule (if F_1 females are already inviable, the interpretation of any inviability of 'unbalanced' females is obviously muddied). However, we have recently discovered that many, if not all, extant strains of these species yield abundant hybrid females in both directions of the species cross, at least at lower temperatures (18°–22 °C) (see Table 1 for details). The *D. melanogaster*–*D. simulans* hybridization clearly obeys Haldane's rule.

This species pair thus affords another opportunity to test the basis of Haldane's rule for inviability. Although unbalanced hybrid females from this cross (carrying an attached X from *D. melanogaster*) have been repeatedly described as inviable[11,12],

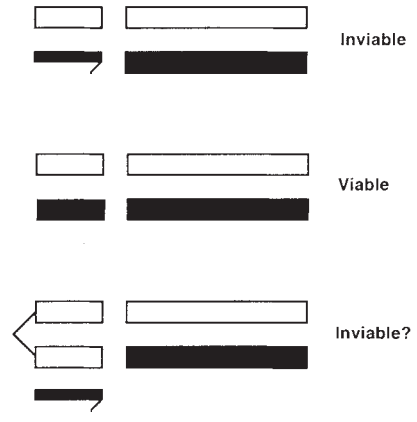

FIG. 1 Test of the genetic basis of Haldane's rule. The chromosomes of one species (for example, *D. simulans*) are shown in white and those of the other species (for example, *D. teissieri*) in black. Sex chromosomes are shown at the left (X on top; Y, with 'hook', on the bottom). Haploid sets of autosomes shown at the right. All genotypes carry cytoplasm from the 'white' species. The top genotype represents F_1 male hybrids and the middle genotype represents F_1 female hybrids. The bottom genotype depicts hybrid females who carry an attached X chromosome from the 'white' species. The traditional explanation of Haldane's rule predicts that this 'unbalanced' genotype will be inviable. (Population genetic models (H.A.O., unpublished) show that this theory requires an additional assumption: the alleles causing reproductive isolation act as loss-of-function mutations; that is more recessive alleles must have greater homozygous effects on hybrid fitness.)

TABLE 1 Results of species crosses

Hybridization	Females	Males
D. simulans—*D. teissieri*		
D. simulans Fla. City ♀ × *D. teissieri* Congo ♂	51	0
D. simulans yw ♀ × *D. teissieri* Congo ♂	200	0
D. simulans C(1)RM, *yw*/Y ♀ × *D. teissieri* Congo ♂	0*	0
D. melanogaster—*D. simulans*		
D. melanogaster Oregon-R ♀ × *D. simulans* v^C ♂	87	0
D. melanogaster Bellows Falls ♀ × *D. simulans* v^C ♂	46	0
D. melanogaster Bellows Falls ♀ × *D. simulans* Fla. City ♂	190	0
D. melanogaster Oregon-R ♀ × *D. simulans vm* ♂	297	0
D. melanogaster Napa ♀ × *D. simulans* w^{2C} ♂	170	0
D. simulans v^C ♀ × *D. melanogaster* Oregon-R ♂	83	40
D. simulans v^C ♀ × *D. melanogaster* Bellows Falls ♂	176	169
D. simulans Fla. City ♀ × *D. melanogaster* Bellows Falls ♂	62	43
D. simulans vm ♀ × *D. melanogaster* Oregon-R ♂	186	471
D. simulans w^{2C} ♀ × *D. melanogaster* Napa ♂	105	111
D. melanogaster C(1)RM, *ywf*/Y♀ × *D. simulans* v^C ♂	0	350
D. melanogaster C(1)RM, *ywf*/Y♀ × *D. simulans vm* ♂	0	267

Number of adult hybrids appearing in species crosses. All hybrids are sterile. All markers are X-linked. Crosses performed at 22 °C and 18 °C (as results did not differ with temperature, data were pooled). In the third cross, numerous hybrid larvae and pupae appeared in ~30 vials (the $X_{teissieri}/Y_{simulans}$ hybrid males survive into the pupal stage). The timing of hybrid lethality was determined by scoring the sex of hybrid larvae/pupae using the X-linked *yellow* marker. The reciprocal cross (*D. teissieri* female × *D. simulans* male) fails, apparently because of complete sexual isolation[8]. The appearance of abundant females in the *D. simulans* ♀ × *D. melanogaster* ♂ crosses is not due to some rare hybrid inviability rescue mutation (H.A.O. and J. Coyne, unpublished results): numerous females appear when using all tested strains of both *D. simulans* and *D. melanogaster* (at least 7 strains of each species tested). Tested strains include freshly caught wild-type flies of both species as well as older laboratory mutant and wild-type stocks (including flies from the United States, Australia and Okinawa). Although hybrid females are abundant at 18–22 °C, few appear at 25 °C.

* One hybrid female appeared who was yellow$^+$, white$^+$, not the expected C(1)RM, yellow, white. This female clearly resulted from the breakdown of the C(1)RM chromosome (which occasionally occurs in the *D. simulans* stock) and thus carried one X chromosome from each species.

there is little reason for believing that this remains true given the unusual present crossing behaviour of these species. I thus crossed *D. melanogaster* attached-X females to *D. simulans* males. As Table 1 shows, the resulting attached-X hybrid females remain completely inviable. Moreover, these females die during the same developmental period as hybrid males (late larval/early pupal)[11].

Thus, in both hybridizations, hybrid females who are homozygous for the X from one species are as inviable as hybrid F_1 males bearing the same X. These results clearly support the traditional explanations of Haldane's rule for inviability (although population genetic theory shows that these explanations require subtle modification (see Fig. 1)). Most important, these findings qualitatively differ from those obtained in analogous tests of Haldane's rule for sterility[2] and thus show that the genetic basis of Haldane's rule in *Drosophila* differs for inviability versus sterility, as several workers have speculated[3,13]. Haldane's rule does not, therefore, have a single genetic cause, as it once appeared[2,4,5].

Nonetheless, the genetic bases of Haldane's rule for sterility versus inviability appear to differ in a logical way. In *Drosophila*, almost all mutations affecting fertility have sex-limited effects, whereas almost all mutations affecting viability do not[14]. Similarly, the genes causing hybrid sterility clearly have sex-limited effects: although unbalanced hybrid females are homozygous for the X-linked alleles causing hybrid male sterility, they remain fertile[2]. Moreover, when both hybrid sexes are partially sterile, mapping experiments show that different loci affect each sex[2]. On the other hand, the present results suggest that the genes causing hybrid inviability affect both sexes—females made homozygous for alleles causing hybrid male inviability are lethal. Although we cannot prove that the same loci cause the lethality of attached-X hybrid females and F_1 males, this conclusion is strengthened by the finding that, in both hybridizations, these females and males die at the same time during development. Moreover, in *D. melanogaster*–*D. simulans*, the fact that the hybrid 'rescue mutation' *Hmr* restores the viability of both F_1 hybrid males and attached-X hybrid females[11] strongly suggests that these hybrids suffer from the same developmental problem.

In conclusion, the behaviour of the factors causing reproductive isolation between species is strikingly similar to that of alleles affecting fitness within species: factors affecting fertility have sex-limited effects, but those affecting viability apparently do not. This parallel provides the clearest evidence to date that speciation involves the divergence of 'ordinary' genes with normal functions within species[15], as proposed by the founders of the modern synthesis. □

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Induction of immediate spatiotemporal changes in thalamic networks by peripheral block of ascending cutaneous information

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PERIPHERAL sensory deprivation induces reorganization within the somatosensory cortex of adult animals[1–6]. Although most studies have focused on the somatosensory cortex[1–6], changes at subcortical levels (for example the thalamus) could also play a fundamental role in sensory plasticity[7–11]. To investigate this, we made chronic simultaneous recordings of large numbers of single neurons across the ventral posterior medial thalamus (VPM) in adult rats. This allowed a continuous and quantitative evaluation of the receptive fields of the same sample of single VPM neurons per animal, before and after sensory deprivation. Local anaesthesia in the face induced an immediate and reversible reorganization of a large portion of the VPM map. This differentially affected the short latency (4–6 ms) responses (SLRs) and long latency