

The Phenogenetics of a Super-suppressor in *Drosophila melanogaster* IV.* Sub-phenotype Interactions

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Abstract

The existence of a mutant in *D. melanogaster* that has interactive phenotypes with several $su(Hw)^2$ -suppressible mutants that also have other suppressors has permitted the division of suppressor genes into two classes. The first class, of which $su(Hw)^2$ is the only established member, are characterized by exerting their effect during the information transfer process.

Introduction

The case that the mutant suppressor of *Hairywing* in *Drosophila melanogaster* is involved in information transfer, specifically at the translation level, has been presented in the three previous papers in this series (Lee 1970, 1973a, 1973b). In the second of these it was established that there is a prerequisite for suppression which is independent of the perceptible phenotype, and this led to the question of whether this specificity resided in the gene product or the gene itself. To investigate this question it was necessary to delve below the level of the phenotype; this was made possible by the utilization of specific suppressor genes in combination with the curious mutant *divers*.

Materials and Methods

When homozygous the mutant *divers*² produces interactive phenotypes with a number of $su(Hw)^2$ -suppressible mutants that also have other suppressors (Table 1). The purpose of the investigation was to observe the fate of such interactions when the allele responsible for the interaction was suppressed.

The mutant *dvr*², while having no obvious phenotypic effect alone,

1. gives spirally curled wings in the presence of a *yellow* allele,
2. gives crumpled wings in the presence of a *forked* allele, and
3. acts as an enhancer of *cut*⁶ (Lindsley and Grell 1968).

A $y^2\ ct^6\ dvr^2\ f^1$ stock was made homozygous for the suppressors $su(Hw)^2$ and $su^w f$ and heterozygous for the dominant suppressors $Su(y^{51G})$ and $Su^P(1)f$ in four separate stocks. It should be noted that $Su(y^{51G})$ had previously been found to also suppress y^2 (Lee, unpublished data). These stocks were then examined for the primary (suppressible) phenotype and for the *dvr*²-interactive phenotype.

Unlike the other three, $su(Hw)^2$ is a multiple suppressor and so it was necessary to eliminate the possibility that it also suppressed *dvr*². To do this a $y^1\ ct^6\ dvr^2\ f^1$ stock was tested with $su(Hw)^2$, with the result that neither y^1 nor its interaction with *dvr*² were suppressed while *ct*⁶ and *f*¹ exhibited normal suppression. Therefore *dvr*² is not $su(Hw)^2$ -suppressible. The original *dvr*¹ allele (since lost) showed a further lethal interaction with *sc*. This was tested for *sc*¹ and *dvr*² using a multiply marked first chromosome, and no such interaction was detected.

* Part III, Aust. J. Biol. Sci., 1973, 26, 903-9.

Results and Discussion

The results as presented in Table 2 were definitive. While $su(Hw)^2$ simultaneously suppressed both the primary mutant phenotype and the *divers*-interactive phenotype of all three mutants [note again the similarity with point reversions of dvr^2 -interactive mutants which, as Green (1961) showed, fail to exhibit the *divers* interaction], none of the other three suppressors had any effect on the interactive phenotype, despite effectively suppressing their respective primary mutant phenotypes. In other words,

Table 1. Names, symbols and location of mutants^A referred to in the text

Mutant	Symbol	Map position on chromosome I
Cut	<i>ct</i>	20·0
Divers	<i>dvr</i>	28·1
Forked	<i>f</i>	56·7
Whittinghill's suppressor of forked	<i>su^wf</i>	65·9
Dominant first chromosome suppressor of forked	<i>Su^D(I)f</i>	not located
Suppressor of yellow-51G	<i>Su(y^{51G})</i>	not located
Scute	<i>sc</i>	0+
Yellow	<i>y</i>	0+

^A Lindsley and Grell (1968).

Table 2. Effect of suppression of the primary mutant phenotype on the *divers*-interactive phenotype

Suppressor	Mutant	Primary phenotype	Interactive phenotype	Type of suppressor
<i>Su(y^{51G})</i>	y^2	Suppressed	Not suppressed	Class II
<i>Su^D(I)f</i>	f^1	Suppressed	Not suppressed	Class II
<i>su^wf</i>	f^1	Suppressed	Not suppressed	Class II
<i>su(Hw)^2</i>	y^2, f^1, ct^6	Suppressed	Suppressed	Class I

Table 3. Two models of suppression in *D. melanogaster*

Mutant	Level at which effect occurs:	
	Model 1	Model 2
Class II suppressor	Phenotype (metabolism)	Primary gene product
<i>divers</i>	Primary gene product	Translation
Class I suppressor	Translation	Transcription

despite not being $su(Hw)^2$ -suppressible, there is no phenotypic indication of the presence of dvr^2 in the $y^2 ct^6 dvr^2 f^1$ stock when it is homozygous for $su(Hw)^2$. This statement requires some qualification, since 9·87% of males of this genotype had slightly snipped wings, indicative of marginal expression of dvr^2 -enhanced ct^6 . Nevertheless it can be concluded that we are dealing with two distinct classes of suppressor, the difference between them being the level at which each is functioning.

Denoting $su(Hw)^2$ as a class I suppressor and the others as class II suppressors we can say that the presence of *divers* reveals that an individual or population carrying a mutant suppressed by a class II suppressor is actually 'more mutant' or less similar to wild type than if the same mutant was suppressed by a class I suppressor. Therefore the class I suppressor must be more basic in its action. These results only establish the ranking of effects and not the levels at which they are occurring, i.e. *divers* has its effect prior to that of class II suppressors but subsequent to class I suppressors. Examples of two feasible models are shown in Table 3. Evidence presented in the other papers of this series (Lee 1970, 1973*a*, 1973*b*) strongly supports the first model. If we accept that the mutants with which we are dealing produce their effect via an altered gene product the only possible conclusion is that class I suppressors have their effect prior to the formation of this product and consequently that the prerequisite specificity for suppression must be a function of the information transfer process leading to the formation of this gene product.

References

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