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## *PSEUDO-ALLELISM AT THE VERMILION LOCUS IN DROSOPHILA MELANOGASTER*

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A number of cases have been reported in Drosophila[1–6] and Aspergillus,[7, 8] and apparent cases in Neurospora[9, 10] and cotton,[11] in which recombination occurs between mutants which otherwise classify as allelic by the usual phenotypic and genetic criteria. Such mutants have been designated as pseudo-alleles. In addition to recombination, pseudo-alleles manifest a differential phenotype depending upon whether the mutants are compounded in coupling or in repulsion. Thus in a case of pseudo-alleles which can be designated as $m_1$ and $m_2$, both of which are recessive to wild-type, the phenotype of individuals of the genotype $m_1 + / + m_2$ is mutant, while that of individuals $m_1 m_2 / + +$ is wild-type. Alternative explanations have been proposed to account for this situation. On the one hand, it has been reasoned that pseudo-alleles represent separate genes whose loci and functions are distinct. The phenotypic differences alluded to are then interpreted as being the consequence of position effect.[1, 2, 4] Alternatively, it has been argued that pseudo-alleles are components of a larger "physiological" gene, whose functions are indistinguishable but whose component parts are separable by crossing-over.[8, 12] The phenotypic differences associated with pseudo-alleles are therefore those expected according to the usual rules of dominance.

These alternative interpretations imply conflicting definitions of the gene. On the one hand the gene as defined in terms of linkage relations, mechanism of action and mutability is one and the same. On the other the limits determined for the gene in terms of linkage do not necessarily coincide with the limits of the gene in terms of function.

It is the purpose of this report to discuss a case of pseudo-allelism at the vermilion eye color ($v$) locus in *D. melanogaster* and to determine what bearing it has on the interpretation of pseudo-allelism and the gene.

*Phenotypic Differentiation of v Mutants.*—In an earlier discussion of several sex-linked, recessive $v$ mutants of *D. melanogaster*, it was noted that while they are essentially phenotypically indistinguishable, it is possible to separate them into two classes on the basis of their phenotypes in the presence of a non-allelic suppressor mutant.[13] Certain of the $v$ mutants (collectively designated as $v^s$) are suppressed, while others (collectively designated as $v^u$) are unsuppressed. It was

also noted that $v^s$ and $v^u$ mutants react in identical fashion in gynandromorphs and when developing larvae are supplied either formyl-kynurenine or kynurenine.[13] Under the latter conditions both types of $v$ mutants synthesize brown eye pigment.

Additional data are now available which serve for further comparison of the $v$ mutants. It has been known that $v^s$ mutants when subjected to partial larval "starvation" synthesize brown eye pigment.[14] Tests of the effects of larval "starvation" on the eye color of $v^u$ mutants have been performed and demonstrate that they fail to synthesize brown eye pigment under these conditions,[15] thereby presenting a second means of distinguishing between $v$ mutants. It has also been reported that $v^s$ mutants raised in an optimal nutritional regime accumulate non-protein tryptophane.[16] A comparison of the non-protein tryptophane contents of

TABLE 1

COMPARATIVE PHENOTYPIC RESPONSES OF THE TWO CLASSES OF $v$ MUTANTS

| $v$ MUTANT | SUPPRESSOR | LARVAL "STARVATION" | FORMYL-KYNURENINE | KYNURENINE | TRYPTOPHANE ACCUMULATION |
|---|---|---|---|---|---|
| $v^s$ | + | + | + | + | yes |
| $v^u$ | − | − | + | + | yes |

+ = brown pigment formed; − = no brown pigment formed.

TABLE 2

TESTS OF $v$ MUTANTS TO SUPPRESSOR, $su^2$-$s$

| ORIGIN OF MUTATION | SUPPRESSED | UNSUPPRESSED | $N$ |
|---|---|---|---|
| Spontaneous | 3 | 3 | 6 |
| X-ray and gamma | 0 | 15 | 15 |
| Ultra-violet | 0 | 1 | 1 |

TABLE 3

CROSSING-OVER BETWEEN $v^1$ AND $v^{36f}$ (EXPERIMENTS 1–3, ♀ ♀ WITH FREE X CHROMOSOMES; EXPERIMENTS 4 AND 5 ♀ ♀ WITH ATTACHED-X CHROMOSOMES)

| | GENOTYPE OF ♀ ♀ | | | |
|---|---|---|---|---|
| | X chromosomes; | Autosomes* | $v^+$ ♂ ♀ | $N$ |
| 1. | $v^{36f}/ras^2\ v^1\ m\ f$; | $Cy/+$; $In(3)CXF,\ D/+$ | 1 ♂ = $v^+\ m\ f$ | 34,556 |
| 2. | $v^{36f}/ras^2\ v^1\ m\ f$; | $Cy/+$; $Sb,\ In(3R)C/+$ | 1 ♂ = $v^+\ m\ f$ | 33,600 |
| 3. | $v^{36f}/ras^2\ v^1\ m\ f$; | $Cy/+$; $In(3)CXF,\ D/Sb,\ In(3R)C$ | 0 | 37,448 |
| 4. | $ras^2\ v^1\ f/v^{36f}\ m\ f$; | $Cy/+$; $Ubx/+$ | 1 ♀ = $v^+\ f$ | 45,651 |
| 5. | $ras^2\ v^1\ f/v^{36f}\ m\ f$; | $Cy/+$; $In(3)T,\ Me/+$ | 3 ♀ ♀ = $v^+\ f$ | 23,951 |

* See Bridges and Brehme[20] and Lewis[5] for description of autosomal inversions.

$v^s$ and $v^u$ mutants cultured under such conditions shows that they accumulate like amounts of non-protein tryptophane, and it is not possible to distinguish between them on this basis.[15] Table 1 comprises a summary of the several phenotypic and biochemical comparisons which have been made with the $v$ mutants. It may be noted that only the suppressor and larval "starvation" serve to distinguish among the $v$ mutants.

*Crossing-over Between v Mutants.*—It was suggested earlier in view of the separability of $v$ mutants on the basis of the suppressor that the $v$ locus is pseudo-allelic.[13] This possibility has been tested, using the mutants $v^1$ (suppressed) and $v^{36f}$ (unsuppressed). In these experiments the mutants raspberry-2 eye color ($ras^2$), 0.2 unit to the left, and miniature wing ($m$) 3.1 units to the right of the $v$ locus, were used as crossing-over markers. Females of the genotype $ras^2\ v^1\ m/v^{36f}$ and heterozygous for several autosomal inversions were constituted and crossed to $ras^2\ v^1\ m$

♂♂. The results of these crosses are listed in table 3. It may be noted that two $v^+$ $m$ ♂♂ were recovered. The occurrence of $v^+$ individuals associated with crossing-over is indicative of pseudo-allelism. This may be best accounted for here by assuming that $v^1$ lies just to the left of $v^{36f}$ such that the genotype of the parental ♀♀ was $ras^2\ v^1\ +m/+\ +\ v^{36f}\ +$. A single crossover occurring between $v^1$ and $v^{36f}$ would yield $+\ +\ +\ m$ or $ras^2\ v^1\ v^{36f}$ individuals.

It may be noted that on the basis of pseudo-allelism, in addition to the $v^+$ $m$ individuals recovered from ♀♀ $ras^2\ v^1\ m/v^{36f}$ the complementary crossover type, viz., $ras^2\ v^1\ v^{36f}$ should occur. There was no evidence for the occurrence of this type

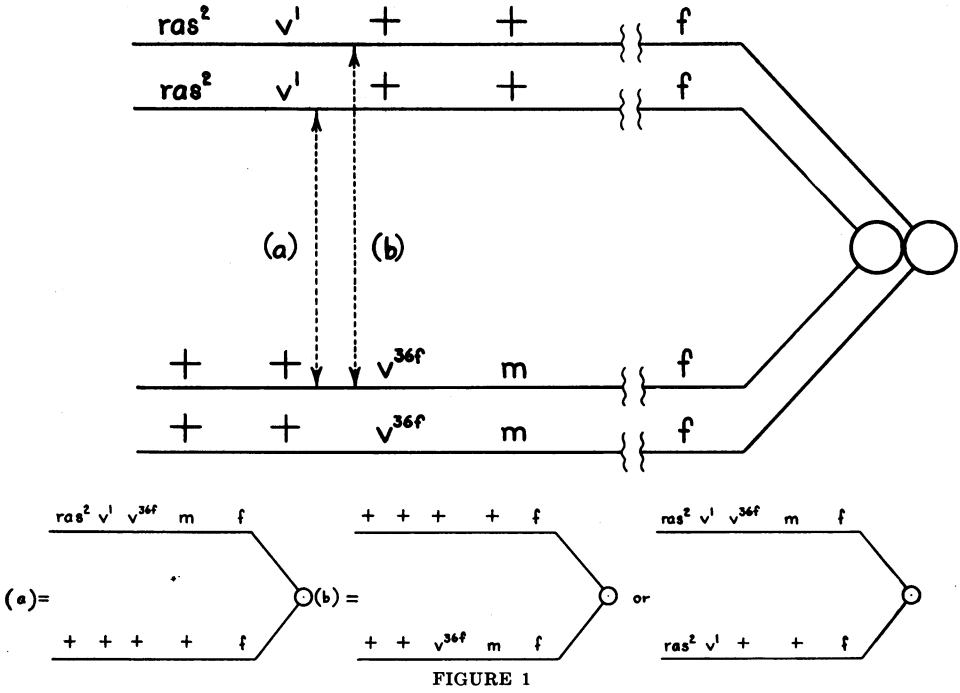


FIGURE 1

Scheme for detecting crossing-over between pseudo-alleles $v^1$ and $v^{36f}$ in attached-X ♀♀. The upper diagram represents the genotype of the attached-X ♀♀. The reciprocal crossover is designated (*a*), the non-reciprocal (*b*), and the corresponding crossover products shown below are designated (*a*) and (*b*).

in the initial crosses. In fact, it appears highly unlikely that individuals of the genotype $ras^2\ v^1\ v^{36f}$ could be differentiated phenotypically from ♀♀ $ras^2\ v^1$ or $ras^2\ v^{36f}$. Since both the $v^1$ and $v^{36f}$ mutants are incapable of utilizing tryptophane, it appears reasonable to conclude that the mutants coupled together on the same X chromosome would also fail to utilize tryptophane. Accordingly, it would be expected that the phenotype of individuals homozygous for $v^1\ v^{36f}$ would be inseparable from that of individuals homozygous $v^1$ or $v^{36f}$.

There does exist at least one method which permits the recovery from ♀♀ $v^1/v^{36f}$ of both products of crossing-over between the pseudo-alleles. This involves crossing-over in attached-X ♀♀. This technique was first utilized for recovering

simultaneously both products of unequal crossing-over in Bar eye in *D. melanogaster*,[17] and has more recently been used to establish pseudo-allelism at the white locus in *D. melanogaster*.[5] Accordingly attached-X ♀ ♀ of the genotype $ras^2\ v^1\ +\ f/+\ +\ v^{36f}\ m\ f$ were constituted, autosomal inversions introduced, and these ♀ ♀ were then crossed to $v\ f$ ♂ ♂. The ♀ progeny of these crosses were examined and $v^+\ f$ ♀ ♀ were sought. In figure 1, the genotype of the attached-X ♀ ♀ is illustrated; in addition, the products of crossing-over between the *v* pseudo-alleles are listed. As indicated in figure 1, three products of crossing-over between the pseudo-alleles can occur, but only the two producing $v^+$ phenotypes are detectable. The $v^+$ phenotypes will occur as a result of either a reciprocal or a non-reciprocal crossover. It may be noted that $v^+$ ♀ ♀ occurring as a result of a reciprocal crossover between the pseudo-alleles will possess both chromatids involved in the crossover, i.e., will have one X chromosome carrying the wild-type alleles of the *v* pseudo-alleles while the homologous X will carry the two mutants pseudo-alleles. These ♀ ♀ will be of the genotype indicated for type (*a*) in figure 1 and will be phenotypically $v^+\ f$. That such is the case can be determined by a progeny test. If the $v^+\ f$ ♀ is the result of a reciprocal crossover, there should occur among her attached-X ♀ progeny individuals of the phenotype $ras^2\ v\ m\ f$. These, the result of a subsequent non-reciprocal crossover occurring to the right of *m* in the $v^+\ f$ ♀, represent the individuals homozygous for both pseudo-alleles and each X chromosome is genotypically $ras^2\ v^1\ v^{36f}\ m\ f$.

The $v^+\ f$ ♀ ♀ occurring as a result of non-reciprocal crossover between the pseudo-allelic loci will possess one crossover chromatid and one non-crossover chromatid. Their genotype corresponds to (*b*) of figure 1 and also will be phenotypically $v^+\ f$. On progeny testing, these ♀ ♀ should produce among the attached-X ♀ progeny the phenotype $v^{36f}\ m\ f$. This occurs as a result of a non-reciprocal crossover occurring to the right of *m* in the $v^+\ f$ ♀.

In the initial tests made as listed in 4 of table 3, one $v^+\ f$ attached-X ♀ was recovered. On progeny testing, this ♀ produced a total of 113 attached-X ♀ progeny, of which 105 were phenotypically $v^+\ f$ and 8 $ras^2\ v\ m\ f$. Since the eye color of $ras^2\ v^1\ v^{36f}\ m\ f$ ♀ ♀ is inseparable from that of ♀ ♀ $ras^2\ v^1$ and since the ♀ ♀ carry the markers expected for a reciprocal crossover, it may be concluded that these attached-X ♀ ♀ are genotypically homozygous $ras^2\ v^1\ v^{36f}\ m\ f$. The attached-X's of ♀ ♀ $ras^2\ v^1\ v^{36f}\ m\ f$ were detached and tested to the suppressor. As expected, the suppressor failed to alter the eye color. Thus it can be concluded that the crossover carrying the two pseudo-alleles has been recovered.

Three additional cases of crossing-over between the *v* pseudo-alleles in attached-X ♀ ♀ have been recovered in an experiment where a different combination of autosomal inversions was used. These are listed in 5 of table 3.

*Cytology of the v Locus.*—In a number of cases of pseudo-allelism in *D. melanogaster*, evidence has been submitted which points strongly to the association of the pseudo-alleles with a doublet as observed in the salivary gland chromosomes.[4] To determine whether such an association exists in the case of the *v* pseudo-alleles four independent *v* deficiencies were examined cytologically. While the limits of the deficiencies varied, all had one feature in common, viz., the absence of the doublet 10A 1–2 as listed in Bridges' revised salivary gland X chromosome map.[18] The association of the *v* locus with a cytologically visible doublet supports the interpreta-

tion of pseudo-allelism, but it cannot be concluded with absolute certainty that $v^1$ is localized in the left half (10 A1) and $v^{36f}$ in the right half (10 A2) of the doublet.

*Mutation to $v^s$ and $v^u$.*—In the course of this study a total of 22 independently occurring $v$ mutations have been assembled and tested to the suppressor. These mutants, a number of which were contributed by other investigators, all arose from $v^+$ and include both spontaneous and induced mutants. In table 2 the results of the tests of the mutants with the suppressor together with their mode of origin are summarized. It is significant to note that whereas among the spontaneous mutants both $v^s$ and $v^u$ types occur, only $v^u$ mutants have been found in the induced group. Or phrased slightly differently, while $v^s$ mutants occur spontaneously, they have thus far failed to occur among the induced $v$ mutants. Does this observation mean that there is a difference in induced mutability between the pseudo-allelic $v$ loci? This question cannot be conclusively answered at this time for a number of reasons. The data on crossing-over between the pseudo-alleles have been derived from spontaneously occurring mutants. Thus far crossing-over between a $v^s$ mutant and an x-ray induced $v^u$ mutant has not been observed although admittedly extensive trials have not been made. Two x-ray $v^u$ mutants were tested with $v^1$ in attached-X ♀ ♀, paralleling experiment 4 of table 3. In each case no $v^+$ ♀ ♀ were found among 40,000 progeny. These negative results are not conclusive. If the observed crossing-over between $v^1$ and $v^{36f}$ is a reasonable approximation of the frequency of crossing-over between the pseudo-alleles (see table 3), the negative results noted can readily be accounted for on the basis of chance. The relatively low frequency of crossing-over between $v^1$ and $v^{36f}$ has for the time being discouraged large scale testing for crossing-over between other $v$ mutants.

If it is assumed that the suppressor does distinguish between the pseudo-allelic loci, it is possible to interpret the origin of induced $v^u$ mutants in one of three ways: (1) All induced mutants are "point" mutations allelic to $v^{36f}$. (2) All induced mutants involve both loci either as small rearrangements or "point" mutations at both loci. (3) Induced mutants include both types (1) and (2).

The available genetic and cytological information indicates that none of the induced $v^u$ mutants is associated with gross chromosomal rearrangement. While it cannot be stated conclusively at this time whether induced $v^u$ mutants involve both pseudo-allelic loci, there is evidence available from studies of lozenge (*lz*) pseudo-alleles[2, 19] that *lz* mutants rarely if ever involve two loci simultaneously. Analysis of eight independent x-ray induced *lz* mutants demonstrates that each can be assigned, on the basis of crossing-over, to one of the three *lz* loci. If the induced $v$ mutants are homologous to the induced *lz* mutants in that only one locus mutates at a time, and if the suppressor does distinguish between the pseudo-allelic loci, then the results noted demonstrate differential mutability of the $v$ pseudo-allales. It is pertinent to point out here that the observations on induced mutation are not peculiar to the $v$ pseudo-alleles. In a study now in progress[19] on the forked (*f*) locus in *D. melanogaster*, it has been shown that it is possible to distinguish between *f* mutants by using a non-allelic suppressor mutant; pseudo-allelism is indicated for the *f* locus since crossing-over occurs between suppressed and unsuppressed *f* mutants; and all induced *f* mutants studied are unsuppressed. The analysis of the *f* locus will be discussed shortly.

*Discussion.*—The foregoing account of the genetics and cytology of mutants at

the *v* locus in *D. melanogaster* has established the existence of two pseudo-allelic *v* loci. It is of importance to reconsider here the bearing this case of pseudo-allelism has upon the general interpretation of pseudo-allelism. As emphasized at the outset, the specific question which the phenomenon of pseudo-allelism poses may be stated as follows: do pseudo-alleles represent closely linked genes with distinctive functions or do they represent an integrated functional unit of the chromosome whose component parts can be separated by means of crossing-over? At first glance, it would appear that the latter interpretation more closely fits the *v* pseudo-alleles. The phenotypic effects of the several *v* mutants alluded to previously indicate that all *v* mutants are incapable of utilizing tryptophane, the initial step in the biosynthesis of brown eye pigment. This is borne out by the observations that all *v* mutants accumulate non-protein tryptophane and respond similarly by synthesizing brown eye pigment when supplied either formyl-kynurenine or kynurenine. Thus, it would appear that *v* mutants are functionally inseparable. There are, however, two observations which militate against an interpretation of functional identity among the several *v* mutants studied. These are the formation of brown eye pigment by some but not all mutants, either in the presence of a non-allelic suppressor or when subjected to partial larval "starvation." It should be added here that there is a slight but consistent phenotypic difference which the experienced observer can use to distinguish between *v* mutants. The phenotypes of $v^s$ and $v^u$ individuals can be distinguished when in addition they are homozygous for the autosomal eye color mutant brown (*bw*). Thus while the eye color of $v^u$; *bw* flies is essentially white that of $v^s$; *bw* is not and a discernible quantity of brown eye pigment is formed.

The role of the suppressor mutant has significance for the interpretation and meaning of pseudo-allelism. As noted previously, the suppressor mutant $su^2$-*s* not only suppresses some *v* mutants but also the sex-linked recessive body color mutant sable (*s*). A number of alleles of $su^2$-*s* have been reported;[20] one has been described as suppressing not only *v* but the recessive, second chromosome eye color mutant purple (*pr*), while another has been described as suppressing the recessive, second chromosome mutant speck (*sp*). Accordingly, appropriate tests were made to determine whether $su^2$-*s* suppresses *pr* and *sp*. The results obtained demonstrated that $su^2$-*s* suppresses *pr* and *sp* in addition to *v* and *s*. An allele of $su^2$-*s*, $su^g$-*v*, also was found to suppress *pr* and *sp*.

These observations on the action of the suppressor support the contention that the $v^s$ and $v^u$ mutants have separate, distinctive actions. It is pertinent to point out here that *pr* eye color mutant is autonomous,[21] in contrast to both the $v^s$ and $v^u$ mutants which are non-autonomous in development.[13, 21] In addition *pr* flies do not accumulate non-protein tryptophane.[15] Thus the suppressor acts to suppress other mutants, but fails to suppress $v^u$ mutants which superficially appear to be closely related in their mode of action to the $v^s$ mutants. It therefore appears highly unlikely that the $v^s$ and $v^u$ mutants can be identical in their modes of action and yet be separable in their reactions in the presence of the suppressor. Since the mutants $v^1$ and $v^{36f}$ have been separated by crossing-over, by the suppressor, and by larval "starvation," the conclusion can be drawn that these mutants constitute different genes with distinctive loci and functions. These findings for the *v* pseudo-alleles are in accord with those noted for the bithorax-bithoraxoid pseudo-alleles[4] which can be

separated not only by crossing-over, but in addition on the basis of their distinctive phenotypic effects. In addition, the case of the inositolless mutants in Neurospora, which can be distinguished by means of a suppressor and which apparently cross over, parallels the results reported here for the *v* pseudo-alleles.[9, 22]

The question of the basic difference in the mode of action of the mutants $v^1$ and $v^{36f}$ cannot be satisfactorily answered at this time. It has been noted that both mutants are blocked biochemically at the initial step in the biosynthesis of brown eye pigment and thus both accumulate non-protein tryptophane. The investigation of the genetic control of tryptophane desmolase in Neurospora is a case in point.[23] Here, it has been ably demonstrated that two allelic mutants, separable by an independent suppressor, both influence the same enzyme activity. By analogy, the case of the *v* pseudo-alleles may also represent a case where the same enzyme system is altered by different mutant genes in different ways. The fact that $v^s$ flies can synthesize brown eye pigment in the presence of the suppressor or when subjected to larval "starvation" suggests that they may, in fact, possess the necessary enzyme system which catalyzes the oxidation of tryptophane but that the particular enzyme(s) is either inactive or is inactivated by the $v^s$ gene or its products. The suppressor or larval "starvation" thus serve to activate the enzyme(s) or to remove its inhibitors. In the case of the $v^u$ mutants it would appear that a thus far irreversible alteration in the enzyme system has occurred such that it is incapable of catalyzing the oxidation of tryptophane. It must be emphasized that the foregoing interpretations of the mechanism of action of the $v^s$ and $v^u$ mutants are tentative. While there is no *a priori* reason to believe that the same enzyme system cannot be altered by different genes and therefore in different ways, there is as yet no information available which permits a critical discrimination among the possible mechanisms which might be operative here.

What interpretation can be submitted to explain the differential phenotype observed when the pseudo-alleles are in coupling versus repulsion? Since all *v* mutants are recessive to wild-type it readily follows that individuals of the coupling genotype $v^1\ v^{36f}/+\ +$ should be wild-type in phenotype. However, the fact that individuals of the repulsion genotype $v^1\ +/+\ v^{36f}$ are vermilion in phenotype clearly fails to follow the expectation based on dominance of the wild-type alleles. Thus any explanation of phenotype based on the assumption of simple dominance and recessivity fails. Rather it appears possible to interpret the resultant phenotypes in terms of the primary action of the mutant genes themselves along the lines previously discussed by Lewis.[4] It is assumed here that the primary action of both $v^1$ and $v^{36f}$ mutants involves a block at tryptophane, each accomplishing the block in a different way. The interpretation of the phenotypes of the mutants in coupling and repulsion must include the assumption that the primary gene-controlled reactions are here confined to the immediate vicinity of the gene. Moreover, it must be assumed that the immediate reactions governed by the genes on one chromosome are independent of those on the homologous chromosome. Thus in the case of individuals of the genotype $v^1\ v^{36f}/+\ +$, the utilization of tryptophane is blocked by the *v* mutants on the one chromosome, but this block does not impede the action of the + alleles on the homologous chromosomes in utilizing tryptophane and thus brown eye pigment is synthesized. However in the case of the individuals of the genotype $v^1\ +/+\ v^{36f}$, each mutant pseudo-allele acting independently blocks the

utilization of tryptophane and thus prevents the biosynthesis of brown eye pigment. Thus while the wild-type alleles of each pseudo-allele are present, their effects are rendered ineffective by the action of the adjacent *v* mutants and the resultant phenotype is vermilion rather than wild-type.

*Summary.*—A phenotypic comparison between two types of *v* mutants in *D. melanogaster* is presented.

Pseudo-allelism at the *v* locus is indicated since crossing-over between the mutants $v^1$ and $v^{36f}$ was obtained.

Cytologically the *v* loci appear to be associated with the doublet 10 A1-2 in the salivary gland chromosome.

The bearing of these results on the interpretation of pseudo-allelism and the nature of the genes involved is discussed.

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## *NUMERICAL PREDICTION OF CYCLOGENESIS**

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*1. Introduction.*—In a recent article[1] the writer and N. A. Phillips devised a sequence $E_n$ of mathematical models of the atmosphere capable of predicting the geopotentials of $n$ suitably selected isobaric surfaces, and then presented the results of a series of consecutive 24-hour numerical integrations that had been carried out with the models $E_1$ and $E_2$ for an intensely cyclogenetic situation.