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Development and complementation of lethal mutations at the dumpy locus of *Drosophila melanogaster**

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SUMMARY

The genetical and developmental aspects of six dumpy mutants of *Drosophila melanogaster* have been investigated. The mutants o^{bm}, l^m and *olv* (possibly alleles), *lv*, lv^1 and lv^I (possibly alleles) were known to be lethal when homozygous. Previously the lethal effect has been treated as a uniform effect. However, the lethal stage is not the same for all homozygotes, being egg/larval (E/L) for the three *lv* alleles, egg (E) for *olv*, larval/larval ecdysis (L/L) for l^m and E/L and larval (L) for o^{bm}. Not all heterozygous combinations are lethal, i.e. o^{bm}/l^m and o^{bm}/lv^I are not lethal. Phenotypically the lethal heterozygotes fall into two patterns: (i) combinations not involving the allele o^{bm} and (ii) combinations involving o^{bm}. In the former, the mutant with the developmentally later expression is 'dominant' to the mutant with the developmentally earlier expression. In the latter, the genotypes manifest different proportions of individuals at the lethal stages E, E/L and L. Previous observations suggested that the lethality of the homozygote o^{bm}/o^{bm} was associated with the presence of an independent lethal in the stock. Observations presented here suggest that the lethality is a function of the o^{bm} allele itself. Complementation between some of the lethal mutants is not in accordance with the general rule for dumpy that compounds manifest the traits they have in common.

1. INTRODUCTION

An interpretation of complementation patterns of pseudoallelic series is still likely to be an essential prerequisite for a complete understanding of gene expression during the development of higher organisms. The dumpy series of pseudoalleles provides a good deal of interest for developmental studies because of their wide range of phenotypes and multiplicity of sites.

The dumpy mutants produce three major phenotypic expressions, all of which are recessive to wild type: a lethal effect (*l*), obliquity of the wings (*o*), and hypodermal irregularities of the thorax called vortices (*v*). Carlson (1959) named each mutant allele according to the major effects which it manifests, using the nomenclature *o*, *v* and *l*. Mutants have been found which manifest either only one effect, i.e. *o*, *v* or *l*, or any possible combination *ov*, *ol*, *lv* and *olv*. There is some intragenic

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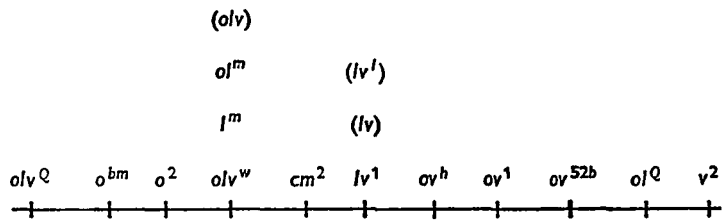
complementation – for example, *l/ov* is phenotypically wild type; and heterozygous combinations of pseudoalleles express the particular traits they have in common – for example, *ol/v* manifests oblique wings, and *lv/v* manifests thoracic abnormalities.

The dumpy map has many sites associated with the various combinations of *o*, *l* and *v* (Carlson, 1959; Southin & Carlson, 1962; Grace, reported by Jenkins, 1967). The most recent map (Grace, reported by Jenkins, 1967) lists eleven sites (Text-fig. 1). It is difficult to interpret the map because: (i) there is discontinuity in the *o*, *l* and *v* effects along the linear genetic map; (ii) different phenotypes sometimes have the same map position; (iii) similar phenotypes sometimes have different map positions. In addition there are many mutants which have not yet been mapped precisely.



Text-fig. 1. The arrangement of the mutant sites within the dumpy region (Grace, reported by Jenkins, 1967). The mutants o^{bm}, *olv*, l^m, *lv*, lv^l and lv^I were used in this investigation. The possible positions of three of these, (*olv*), (*lv*) and (lv^I), which were not given by Grace, are also shown here (from Southin & Carlson, 1962).

This paper is concerned with the lethal effect only. The expression and development of ten mutants, known to be lethal when homozygous, has been explored in order to gain some insight into the dumpy syndrome and gain a better understanding of some aspects of normal development. Six of these mutants are described here, namely o^{bm}, *olv*, l^m, *lv*, lv^1 and lv^I; the following four – ol^s, olv^{bm}, o^2lv^1 and ol^sv^2 – will be described in a later paper. The expression and development of five alleles which produce thoracic abnormalities has previously been reported (Metcalfe, 1970).

2. MATERIALS AND METHODS

The mutants used in this investigation, their source and Carlson's notation are given in Table 1. In this report the base symbol *dp* is omitted from the formulae of the mutants. The mutant o^{bm}, l^m and lv^1 have been precisely located on the genetic map (Text-fig. 1). The possible positions of *olv*, *lv* and lv^I are also indicated in Text-fig. 1. The mutants *olv* and l^m are possibly alleles at the olv^w site: *lv* and lv^I probably occupy the same site as lv^1 (Southin & Carlson, 1962).

Lethality of heterozygotes. All heterozygous combinations of the lethal mutants were tested for lethality by crossing the six stocks reciprocally and examining the progeny for *dp* flies. Since all the mutants except lv^I are balanced against *Cy*,

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lethality of the heterozygous combinations of *dp* would give rise to all *Cy*-winged progeny in the F_1. Crosses involving the allele lv^I, which is in coupling with *Cy* and balanced against *S*, were examined for the presence of *dp* (*Cy*, non-*S*) individuals; *S* and *S*, *Cy* being +.

Lethal phenotypes. To identify the lethal phenotypes the stocks were first outcrossed to a wild-type stock (Oregon R) to free them from the balancers *Cy* or *S*, and then crossed together to give, in the F_2 generation, homozygous lethals and all possible heterozygous combinations. Approximately 20 pairs of 4- to 8-day-old F_1 flies were left to lay eggs on food slants over a period of 2 h. The progeny was scored for lethals by inspecting at each stage of development. (i) *Egg stage*: at this

Table 1. *The dumpy alleles used in this investigation*

Original symbol	Carlson's notation	Occurrence	Stock	Source	Reference
o^{bm}	o^{bm}	X-ray	$dp^{o^{bm}}/CyB1\ L$	Phildelphia	Carlson & Southin (1959)
l^m	l^m	UV	$dp^{l^m}/CyB1\ L$	Ohio	Lindsley & Grell (1967)
T	*olv*	Spontaneous	$dp^T/S^2CyB1\ 1b^3\ cn^2\ L^4\ sp^2$	H. Meyer, Wisconsin	Altenburg Muller (1920)
tx	*lv*	Spontaneous	$dp^{tx}b/CyIns^{cn2}$	Philadelphia	Lindsley & Grell (1967)
lv^1	lv^1	Spontaneous	$dp^{lv^1}/CyB1\ L$	Ohio	Carlson & Southin (1962)
tx^I	lv^I	Spontaneous	*S sp cn M(2) S7* bw^D/ dp^{tx^I} *Cy*Ins*05 pr cn2 sp*	H. Meyer, Wisconsin	Lindsley & Grell (1967)

stage the unfertilized eggs were separated from those in which arrested development was due to a lethal, by dechorionating the eggs, mounting in Ringer solution and examining microscopically. (ii) *Larval stage*: immediately after hatching from the egg, and at the beginning of each instar, the larvae were counted and transferred to a new food slant.

Histological techniques. Flies were fixed in hot alcholic Bouin's fluid and double-embedded by the method of Symmons (1962) in ester wax (Steedman, 1960). Sections, 8 μ thick, were stained with Ehrlich's acid haematoxylin and counterstained with eosin.

3. RESULTS

Lethality of heterozygotes. The six mutants are lethal when homozygous. All heterozygous combinations of the lethal mutants were tested for lethality. The results (Table 2) show that almost all heterozygous combinations are lethal. The exceptions involve o^{bm}; o^{bm}/lv^I and o^{bm}/l^m are not lethal.

Viable o^{bm} heterozygotes have previously been reported – o^{bm}/olv, o^{bm}/lv and o^{bm}/ol – but the homozygote o^{bm}/o^{bm} was found to be lethal (Lindsley & Grell, 1967). In order to explain these results it was suggested that the lethality of the

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homozygotes might result from the presence of an independent lethal in the o^{bm} stock. This explanation also succeeded in accounting for the apparent departure of these observations from the hypothesis that heterozygotes manifest the traits they have in common. However, it does not accommodate all the observations on the o^{bm} heterozygotes to be presented here. Furthermore, the heteroallelic combinations o^{bm}/olv and o^{bm}/lv which have previously been reported as viable (Lindsley & Grell, 1967) are not found to be so here. (See Discussion for comment on both these

Table 2. *The complementation pattern of the alleles used in this investigation*

		Alleles					
		o^{bm}	l^m	olv	lv	lv^1	lv^l
						2 %+	
Alleles	o^{bm}	L	+	L	L	L	+
	l^m	.	L	L	L	L	L
	olv	.	.	L	L	L	L
	lv	.	.	.	L	L	L
	lv^l	.	.	.	.	L	L
	lv^I	.	.	.	.	.	L

L = lethal, no *dp* adults observed. + = hatch into adults.

Results of crosses giving *dp* adults

	Progeny						
	2 dumpy alleles		1 dumpy allele		No dumpy alleles		
Genotype tested	No.	Phenotype	No.	Phenotype	No.	Phenotype	χ^2 for agreement with 1 *dp*:2+
o^{bm}/l^m	90	+	208	*Cy*	.	.	1·3
o^{bm}/lv^l	5	Blistered wings and thoracic abnormalities	248	*Cy*	.	.	111·9*
o^{bm}/lv^I	82	*Cy* and low penetrance of thoracic abnormalities	99	*S*	91	*S*, *Cy*	1·3

* $P < 0{\cdot}001$.

points.) Since the heterozygote o^{bm}/l^m is viable, it follows that if the l^m is an allele of *olv*, it is behaving in accordance with the previous report (Lindsley & Grell, 1967). The three *lv* alleles interact differently with o^{bm} although they are phenotypically inseparable in combination with all other alleles with which they have been tested (Lindsley & Grell, 1967).

The lethal phenotypes. Previously the lethal effect of the dumpy locus has been treated as one individual effect. But, in fact, there is a variety of lethal expressions, which will now be described. Six lethal stages were observed, namely egg (E),

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egg/larval boundary (E/L), larval ecdysis (L_1/L_2 and L_2/L_3) and larval lethals or 'lethal sinuous' (L_1 and L_2). These names in all cases refer to the phenotypes of the lethals and not to particular genotypes.

Table 3. *Lethal stages of homozygotes*

Genotype	Lethal stages*						Total lethals	Total non-lethals	χ^2 for agreement with 3:1
	E	E/L	L_1	L_1/L_2	L_2	L_2/L_3			
olv/olv	92·4	7·6	.	.	.	.	92	260	0·2
lv/lv	4·9	95·1	.	.	.	.	123	404	0·8
lv^1/lv^1	2·5	97·5	.	.	.	.	280	779	1·2
lv^I/lv^I	3·8	96·2	.	.	.	.	52	120	2·5
l^m/l^m	.	2·3	.	96·2	.	1·5	131	421	0·5
o^{bm}/o^{bm}	6·8	89·0	2·1	.	2·1	.	146	456	0·98

E, Egg lethals; E/L, egg/larval lethals; L, larval lethals; L/L, larval ecdysis lethals.
* The percentage of lethals occurring at each lethal stage is given.

Table 4. *Lethal stages of heterozygotes: I. Combinations not involving the allele* o^{bm}

Genotype	Lethal stages*						Total lethals	Total non-lethals	χ^2 for agreement with 3:1
	E	E/L	L_1	L_1/L_2	L_2	L_2/L_3			
olv/lv	5·4	94·6	.	.	.	.	56	201	1·4
olv/lv^1	9·6	90·4	.	.	.	.	178	541	0·02
olv/lv^I	5·5	94·5	.	.	.	.	127	432	1·4
lv/lv^1	3·5	96·5	.	.	.	.	260	808	0·2
lv/lv^I	1·8	98·2	.	.	.	.	56	174	0·05
lv^1/lv^I	1·3	88·7	.	.	.	.	62	199	0·2
olv/l^m	.	5·2	.	94·8	.	.	58	220	2·5
lv/l^m	.	8·8	.	91·2	.	.	68	249	2·1
lv^1/l^m	.	7·5	.	92·5	.	.	241	784	1·2
lv^I/l^m	.	7·7	.	92·3	.	.	62	192	0·04

KEY. See Table 3.

The lethal stages for the six homozygotes are given in Table 3. Phenotypically, the mutants fall into four groups, namely *olv*, the three *lv* mutants, l^m and o^{bm}. It can be seen that (i) the E lethal stage is characteristic of the genotype *olv/olv*; (ii) the three *lv* mutants are all affected in a similar manner, the majority dying at the E/L stage; (iii) the lethal stage of l^m/l^m occurs at the time of larval ecdysis, much later than the lethal stage of *olv/olv*; the fact that these two homoallelic mutants do not interact in the same way with o^{bm} may be related to the difference in lethal expression; (iv) the mutant o^{bm} differs from the other alleles in that a few deaths occur in the middle of the first or second larval instar, whilst the majority occur at the E/L stage.

Three questions are considered here with regard to the heterozygous combinations. (i) Can the phenotype be predicted? (ii) What is the interaction of alleles

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within a map site? (iii) Do any individual mutants discriminate between what appear to be phenotypically identical alleles?

One possible prediction is that an early acting mutant is 'recessive' to a later acting one. In this case the phenotype of the heterozygote would be similar to that of the mutant which is blocked at the later stage in development. The phenotypes of almost all heterozygous combinations, except those which involve the allele o^{bm}, follow this prediction (Table 4). The developmentally earlier mutant, *olv* (E phenotype), is 'recessive' to the developmentally later mutants, i.e. the *lv* alleles (E/L phenotype) and l^m (L/L ecdysis). Similarly, the three *lv* alleles are all 'recessive' to the developmentally later l^m mutant. These observations also show that the interaction between alleles which share the same map site, i.e. l^m and *olv* and also the three *lv* alleles, follow this prediction.

Table 5. *Lethal stages of heterozygotes: II. Combinations involving the allele* o^{bm}

	Lethal stages*								Total	χ^2 for
Genotype	E	E/L	L_1	L_1/L_2	L_2	L_2/L_3	*dp** adults	Total *dp*	non-*dp*	agreement with 3:1
o^{bm}/olv	7·9	79·5	8·7	.	3·9	.	0	127	392	0·8
o^{bm}/lv	2·4	29·3	9·8	.	58·5	.	0	82	268	0·5
o^{bm}/lv^1	1·4	27·0	6·4	.	62·8	.	2·4	296	878	0·01
								Total Adults†		
o^{bm}/lv^I	.	.	.	.	5	.		237		
o^{bm}/l^m	.	.	.	.	.	.		223		

KEY. See Table 3.

† Since the stocks were first outcrossed to wild type to free them of markers and then crossed together to give the required genotypes, it is not possible to separate *dp* adults from *non-dp* unless they manifest *v* or *o* effects with complete penetrance (Table 2).

The phenotypic interactions of the heterozygotes involving the allele o^{bm} (Table 5) are different from the others. The heterozygote o^{bm}/olv is similar to the homozygote o^{bm}/o^{bm}, as predicted. However, in the heterozygous combinations o^{bm}/lv, o^{bm}/lv^1 and o^{bm}/lv^I there are shifts towards developmentally later expression than is shown by either mutant of each phenotype. This interaction was not predicted.

In addition the allele o^{bm} differentiates between the three *lv* alleles. There are on the one hand *lv* and lv^1, similar alleles where the majority of lethals occur at the L_2 stage, and on the other hand lv^I, where only a few L_2 lethals occur, the majority of larvae developing through to the adult.

The phenotype of o^{bm}/l^m is of particular interest. It is difficult to predict because these two mutants manifest different lethal phenotypes at the larval stages, i.e. L_1/L_2 ecdysis is developmentally later than L_1 but developmentally earlier than L_2. Complementation occurs between these mutants since the genotype is viable and the flies are phenotypically wild type.

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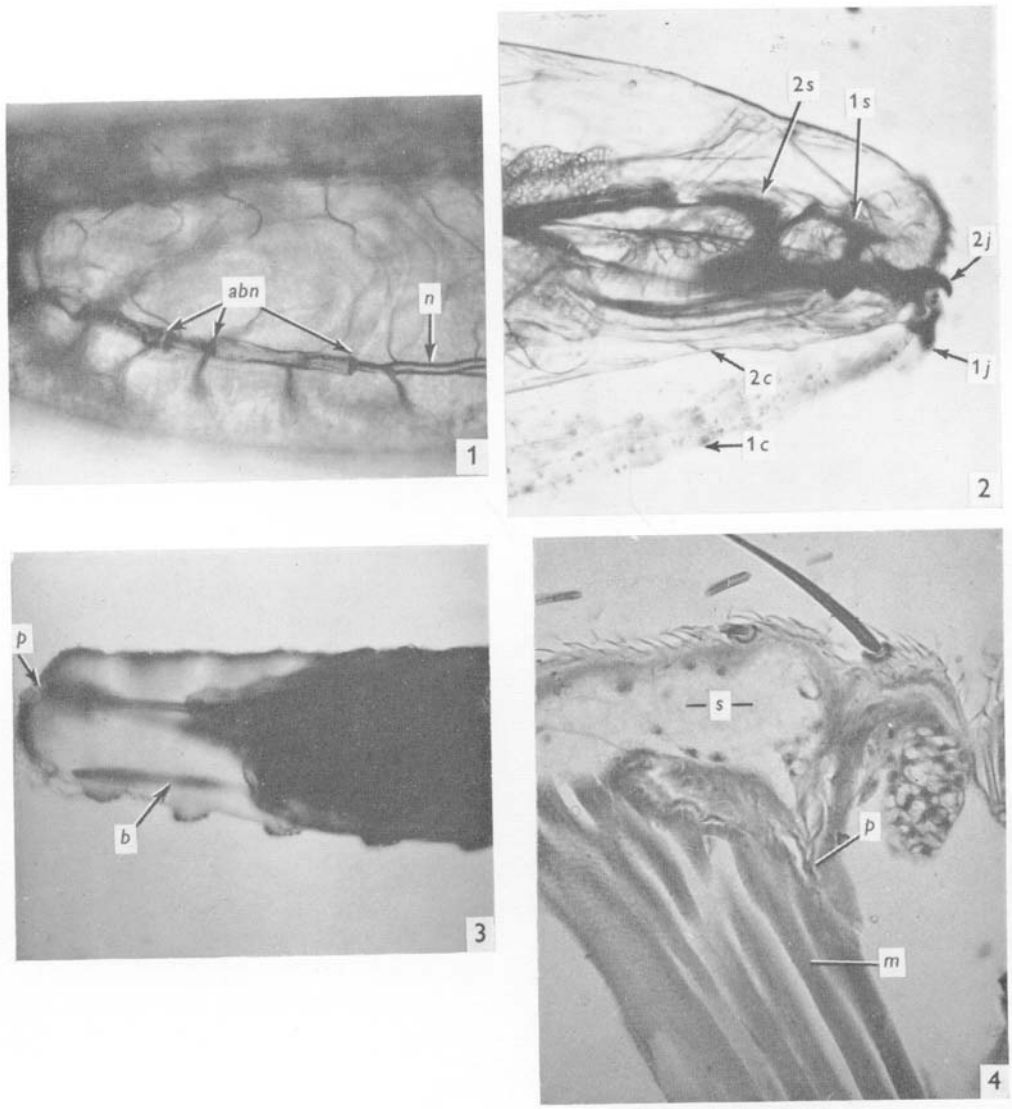

Fig. 1. Posterior region of an E/L lethal (*lv*/*lv*), showing trachael trunks with both normal (*n*) and abnormal (*abn*) regions filled with air.

Fig. 2. Anterior region of a L_1/L_2 ecdysis lethal (l^m/l^m): first instar jaws thrown off (1*j*); first instar suspensoria still within body (1*s*); withdrawn from first instar coat (1*c*); and, second instar jaws (2*j*), suspensoria (2*s*) and coat (2*c*) completely duplicated.

Fig. 3. Posterior region of a L_1/L_2 ecdysis lethal (l^m/l^m) withdrawn from posterior spiracles (*p*) but still attached to the ventro-posterior part of the body wall (*b*) which has been stretched.

Fig. 4. Longitudinal section of lv^l/o^{bm} adult, showing an extracoxal depressor muscle (*m*) attached to a chitinous plate (*p*). The space (*s*), above the plate would normally be occupied by the muscle.

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Developmental aspects

Egg lethal (*E*). These lethals appear morphologically like fully developed wild-type eggs, except for regions of the tracheal trunks and branches where the supporting rings of chitin are insufficient in number and are incomplete in shape. The tracheal system fails to fill with air. These abnormalities by themselves are probably not sufficient to prevent eclosion from the egg membrane since a first instar lethal with no tracheal trunks has been described by Oster (1952). The E lethals show little or no muscular movement when artificially hatched from the egg membrane. Six *olv*/*olv* eggs aged 22 h from the time of deposition were compared with wild type, and no histomorphological differences were found under the light microscope.

Egg/larval boundary lethal (*E*/*L*). The majority of these lethals fail to hatch from the egg membrane. They differ from E lethals in that sections of the tracheal system fill with air. Those sections which do not fill with air are not necessarily abnormal in structure (Plate 1, fig. 1), thus suggesting that the two factors are independent. The E/L lethals, unlike the E lethals, exhibit co-ordinated locomotion when artificially hatched from the egg membrane.

Larval ecdysis lethal or 'lethal stuck' (L_1/L_2 *and* L_2/L_3). The normal change from one larval instar to the next involves both the duplication of chitinous parts and the actual physical process of moulting (Bodenstein, 1944; Novak, 1966). In larval ecdysis lethals, both these processes are affected; they vary independently of each other, indicating that they are separate processes. In the majority of these lethals the larvae attempt to free themselves from their old chitin coat which always remains intact; the suspensoria are always retained within the body although the jaws are almost always 'thrown off' (1*j* in Plate 1, fig. 2); many larvae withdraw from their posterior spiracles which remain attached to the tracheal trunks (*p* in Plate 1, fig. 3); certain regions of the body wall remain connected to the chitin coat and become stretched during larval contraction (*b* in Plate 1, fig. 3); and the duplication of chitinous structures is often incomplete.

Larval lethal or 'lethal sinuous' (L_1 *and* L_2). These larvae no longer increase in size and are unable to develop into the next larval instar. Their mouthparts appear abnormally large for the body and the tracheae have a sinuous course, possibly as a result of disproportionate growth, hence 'lethal sinuous'. Comparison between 'lethal sinuous' and *lethal-meander* (*l-me*) (Hadorn 1956) – which it appears to mimic – revealed that the two mutants differ only in the time of death, which is at the third larval stage for *l-me*, and at the first or second for 'lethal sinuous'.

Only a small percentage of lv^I/o^{bm} individuals are lethal; four adult lv^I/o^{bm} were sectioned and compared with wild type. The flies, in addition to manifesting thoracic abnormalities with low penetrance, possess an additional chitin abnormality which affects only the dorsal attachment of the extracoxal depressor muscle (Plate 1, fig. 4). This direct muscle is, in the mutants, attached to a curved chitinous plate instead of to the wall of the scutum. The plate is joined to the

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thoracic wall only at its edges, appears to be composed solely of chitin, and assumes a knobbly appearance caused by additional pockets and small branches. It is, however, not present in l^m/o^{bm} adults. The plate is very different from the thoracic abnormalities, which are hypodermal pits or protrusions in the regions of attachment of indirect flight muscles (Metcalfe, 1970).

As pointed out above, the L_1/L_2 ecdysis lethal stage was characteristic for the homozygote l^m/l^m; and that l^m appeared to be 'dominant' when in combination with *olv* and the three *lv* alleles. However, it is clear from the above description of L_1/L_2 ecdysis lethals that these larvae are of variable expression with regard to both the amount of duplication of chitinous structures and the degree of moulting. This variability in expression was tested within and between four genotypes – l^m/l^m, l^m/lv^1, l^m/lv, l^m/olv – by scoring the L_1/L_2 ecdysis lethals for the manifestation of second instar mouthparts, i.e. jaws and suspensoria. The results given in Table 6

Table 6. *Variation in amount of duplication of mouthparts of L_1/L_2 ecdysis lethals*

Genotype	L_1 mouth-parts only	L_2 jaws	L_2 mouth-parts complete	Total lethals scored
l^m/l^m	3	70	27	128
l^m/lv^1	31	49	20	35
l^m/lv	37	53	10	38
l^m/olv	87	13	0	55

The percentage of lethals of each phenotype is given.

reveal that the genotypes tested do vary in expression since the proportions of individuals with completely duplicated mouth-parts is largest in the genotype l^m/l^m and smallest in l^m/olv, whilst l^m/lv^1 and l^m/lv behave similarly. Thus although the lethal stage is the same for all these genotypes, i.e. L_1/L_2 ecdysis, more detailed observations reveal that the heterozygotes have slightly earlier developmental expression than the homozygote l^m/l^m. This indicates that the mutant l^m does not behave as a complete 'dominant' to *olv* and the two *lv* alleles.

4. DISCUSSION

Metcalfe (1970) described the phenotypic patterns of five thoracic abnormalities (invaginations of the hypodermis) produced by the mutants v^2, cm^2, *ov*, *lv* and *olv*, both when homozygous and in all heterozygous combinations. Each allelic combination manifests a characteristic pattern of abnormalities. Although some manifestations remain constant for each allele it is not possible to predict a pattern for heterozygotes. Each of the mutants has its own characteristic manifestations and is morphologically distinct, not only with respect to the pattern of abnormalities manifested, but also their size and the number of indirect muscles lost (with which the invaginations are associated). Similarly, detailed observations reported here show that the lethal mutants are not phenotypically uniform.

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Since the lethal phenotypes are different it would be useful to describe the general genotypic relationships. There are clearly four phenotypic patterns among the homozygotes tested here. The interactions of the heterozygotes show that these patterns are not unrelated, as is indicated by the interactions which have been described within two main groups. (i) Almost all the mutants, except o^{bm}, interact similarly, i.e. the mutant with the developmentally later expression is 'dominant' to the mutant with the developmentally earlier expression. (ii) The o^{bm} heterozygotes, except o^{bm}/l^m, manifest different proportions of lethals at E, E/L, L_1 and L_2 stages.

Further, the mutants l^m and o^{bm} are of interest with respect to one other genotype–phenotype interrelationship. The patterns of lethal expression subsequent to the E/L stage fall into one of two sequences: (i) L_1–L_2 and (ii) L_1/L_2 ecdysis–L_2/L_3 ecdysis, but never follow the pattern L_1–L_1/L_2 ecdysis, L_2–L_2/L_3 ecdysis. Developmentally, L lethals and L/L ecdysis lethals do not occur together in the same genotype. Genetically, larval (L) lethals are associated only with the allele o^{bm} and L/L ecdysis lethals only with the mutant l^m. These observations show separate expression of the two genotypes during development. The complementation between l^m and o^{bm} may possibly be due in part to this fact.

However, it should be noted that many examples of multiple allelic loci have been reported in which mutants complement each other in heterozygotes to produce a wild-type phenotype. It has been shown that the levels of enzyme activity in several such complements are much lower than that of wild type. For example, Glassman & Pinkerton (1960) found that flies heterozygous for *ma-l* (maroon-like) alleles were wild type in eye colour even though the amount of xanthine dehydrogenase was only 5% of the value for wild type.

Other than the *dp* locus, only one locus with recessive lethals has been explored in detail in *Drosophila*, i.e. the Notch locus. The pseudoallelisms of the recessive lethals has been critically demonstrated, but the lethal stage of the mutants used was not scored (Wellshons & Von Halle 1962). Poulson (1940) examined the phenotype of seven lethals; they all appeared phenotypically the same, expressing their lethality at the egg stage. None of the lethal mutants complemented each other in trans position.

The interactions of o^{bm} with the other mutants produce both viable (o^{bm}/lv^I, o^{bm}/l^m) and lethal phenotypes (o^{bm}/olv, o^{bm}/lv and o^{bm}/lv^1). There seem to be two possible ways of explaining these interactions. Firstly, they could be a function of the allele o^{bm} itself. Secondly, as previously suggested (Lindsley & Grell 1967), the lethality could result from the presence of a closely linked independent lethal in the o^{bm} stock. The observations presented here support the first explanation.

(i) The genotypes o^{bm}/olv, o^{bm}/lv and o^{bm}/lv^1 as well as o^{bm}/o^{bm} are lethal. This means that the same independent lethal should be present simultaneously in all these stocks and this seems improbable.

(ii) Furthermore, these genotypes do not all have the same phenotypes. This is not a reasonable expectation in the presence of the same independent lethal.

(iii) The expression of E/L lethals of o^{bm}/o^{bm} and the o^{bm} heterozygotes is

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morphologically the same in all details with that produced by other *dp* genotypes, e.g. the *lv* alleles.

(iv) Larval lethals are produced only by o^{bm}/o^{bm} and o^{bm} heterozygotes. This suggests that o^{bm} is different from the other mutants. However, preliminary observations on the *dp* mutant ol^s/ol^s indicate that this expression is not confined to o^{bm}, because some lethals occur at the larval stages L_1 and L_2 (phenotypically 'lethal sinuous').

The genotypes o^{bm}/olv and o^{bm}/lv are found to be lethal here. The difference in viability of these genotypes compared with the earlier report (Lindsley & Grell, 1967) might result from (i) the use of different *olv* and *lv* alleles, or (ii) the o^{bm} allele itself having undergone some subsequent modification, or (iii) some change having occurred in the genetic background of either the *olv* and *lv* stocks or the o^{bm} stock.

Carlson (1961) proposed that the *dp* locus has three complementation units which are involved in seven phenotypic patterns o, l, v, ov, ol, lv and olv. Heterozygotes manifest those traits in common to both alleles. The heterozygotes, o^{bm}/l^m and o^{bm}/lv^I, are found to be viable here. Thus these interactions of o^{bm} with the lethal mutants l^m and lv^I are not in accordance with the general rule that the compounds of *dp* manifest the trait they have in common, i.e. the lethal effect.

Another *dp* mutant, l^{mi} (lethal-isoallele) which does not conform to this general rule has been described (Meyer, 1970). This mutant is a wide-type isoallele, i.e. phenotypically wild type when homozygous but recognizable in various heterozygous combinations (Stern & Schaeffer, 1943). Like o^{bm}, it behaves differently with the lethal mutants. It also interacts differently with the *lv* alleles. But l^{mi} shows some interesting differences from o^{bm} in its interactions. (i) Both homoallelic mutants *olv* and l^m are lethal in combination with l^{mi} but only *olv* is lethal in combination with o^{bm}. (ii) In contrast with o^{bm}, the mutant l^{mi} is lethal in combination with lv^I but viable with *lv*.

The phenotypic interactions of o^{bm} within the group of *lv* alleles do not follow the more usual pattern. However, instances are known of mutants at other loci whose behaviour is similar to that of o^{bm}. For example, three phenotypically identical *lozenge* (*lz*) alleles, known to occupy identical loci can be separated from one another because of the variation in expression when in combination with lz^k (Green, 1961).

Metcalfe (1970) from observations on the development of the thoracic abnormalities concluded that the primary action of *dp* is in the hypodermis and that the overlying chitinous structures are effected secondarily. The same conclusion is consistent with the observed expressions of the lethal phenotypes.

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REFERENCES

ALTENBURG, E. & MULLER, H. J. (1920). The genetic basis of truncate wing – an inconstant and modifiable character in *Drosophila*. *Genetics* **5**, 1–59.

BODENSTEIN, D. (1944). Induction of larval moults in *Drosophila virilis*. *Biological Bulletin marine biology Laboratory, Woods Hole* **86**, 113–124.

CARLSON, E. A. (1959). Allelism, complementation and pseudoallelism at the dumpy locus in *Drosophila melanogaster*. *Genetics* **44**, 347–373.

CARLSON, E. A. (1961). Limitations of geometrical models for complementation mapping of alleles. *Nature* **191**, 788–790.

CARLSON, E. A. & SOUTHIN, J. L. (1959). Preliminary genetic evidence supporting the complementary structure of the heredity material in *Drosophila* spermatozoa. *Genetics* **44**, 502–503.

CARLSON, E. A. & SOUTHIN, J. L. (1962). Comparative mutagenesis of the dumpy locus in *Drosophila melanogaster*. I. X-ray treatment of mature sperm – frequency and distribution. *Genetics* **47**, 321–336.

GLASSMAN, E. & PINKERTON, W. (1960). Complementation at the maroon-like eye-colour locus of *Drosophila melanogaster*. *Science* **131**, 1810–1811.

GREEN, M. M. (1961). Phenogenetics of the lozenge loci in *Drosophila melanogaster*. II. Genetics of lozenge-krivshenko (*lz*^{k}). *Genetics* **46**, 1169–1178.

HADORN, E. (1956). Patterns of biochemical and developmental pleiotropy. *Cold Spring Harbour Symposium of quantitative Biology* **21**, 363–373.

JENKINS, J. B. (1967). Mutagenesis at a complex locus in *Drosophila* with monofunctional alkylating agent, ethyl methanesulfonate. *Genetics* **57**, 783–793.

LINDSLEY, D. L. & GRELL, E. H. (1967). Genetic variations of *Drosophila melanogaster*. *Carnegie Institute of Washington*, publ. no. 627.

METCALFE, J. A. (1970). Developmental genetics of thoracic abnormalities of dumpy mutants of *Drosophila melanogaster*. *Genetics* **65**, 627–654.

MEYER, H. U. (1970). An iso-allele of the dumpy lethal. *Drosophila Information Service* **45**, 147.

NOVAK, V. J. A. (1966). *Insect Hormones*. London: Methuen.

OSTER, I. I. (1952). A study of U.V.-induced lethal mutations in *Drosophila melanogaster*. *Heredity* **6**, 403–407.

POULSON, D. F. (1940). The effects of certain *X* chromosome deficiencies on the embryonic development of *Drosophila melanogaster*. *Journal of experimental Zoology* **83**, 271–326.

SOUTHIN, J. L. & CARLSON, B. A. (1962). Comparison of micromaps obtained by direct and indirect methods of recombination in the dumpy region of *Drosophila melanogaster*. *Genetics* **47**, 1017–1026.

STEEDMAN, H. F. (1960). *Selection Cutting in Microscopy*. Oxford: Blackwell.

STERN, C. & SCHAEFFER, E. W. (1943). On wild type isoalleles in *Drosophila melanogaster*. *Proceedings of the National Academy of Sciences U.S.* **29**, 361–367.

SYMMONS, S. (1962). Comparative anatomy of the mallophagan head. *Transactions of the Zoological Society of London* **27**, 349–436.

WELSHONS, W. J. & VON HALLE, E. S. (1962). Pseudoallelism at the Notch locus in *Drosophila*. *Genetics* **47**, 743–759.

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