Genet. Res., Camb. (1962), **3**, *pp.* 405–416
With 1 *text-figure and* 1 *plate*
Printed in Great Britain

Manifestation of eight lethals in *Coelopa frigida* (F.)

By B. Burnet*
Department of Zoology, King's College, Newcastle upon Tyne, 1

(*Received* 10 *January* 1962)

Lethal factors figure prominently in studies of animal genetics. Recently an entire volume has been devoted to the comparative study of lethal factors in all their aspects (Hadorn, 1961). In the genus *Drosophila*, in which many such factors are known, attention is focused on two main questions. Firstly, what is the nature of the developmental disorder which leads to preadult mortality. Secondly, to what extent is the effect of natural selection acting on the heterozygotes an important factor controlling the frequency of recessive lethals. Although *Drosophila melanogaster* is an obvious choice for such studies with insect material it is by no means a necessary one, and very little comparable information is available for other acalyptera Diptera. This paper reports a study of a small number of recessive autosomal lethals in the seaweed fly *Coelopa frigida* (F.) some of which bear a striking resemblance to certain well-known mutants in *Drosophila melanogaster*. Isolation of embryonic lethals *E/L1* to *E/L5* from natural populations of the species has already been described (Burnet, 1961), whereas *E1*, and lethal *glassy* occurred as spontaneous mutations in the writer's inbred laboratory lines. Lethal *Delta* originated as a spontaneous mutation in the material of Dr U. Thompson, and the development of the homozygotes was subsequently studied by Dr S. A. Bawady. The description of lethal *Delta* given below is summarized from the unpublished accounts of these workers.

MATERIALS AND METHODS

Lethals are propagated in single pair cultures. Only the progeny of pairs giving a lethal segregation are kept in each generation. Cultures are made up in half-pint milk bottles with a pad of cellulose wadding moistened with sea water (3 g. wadding, 30 c.c. sea water), and a piece of split *Laminaria* stipe about 5 cm. long. The females lay their eggs as a clutch of 76 eggs on average, in overlapping rows, which hatch within a few minutes of each other.

The parents are removed as soon as the first clutch is laid, and after the first instar larvae have hatched, the clutch of empty egg cases and such unhatched eggs as may be present, is scored. The quantity of *Laminaria* is then made up to 60 g. and cultures incubated at 25° C. After the initial count of hatched and unhatched eggs the chorions are dissolved away with a 5% solution of sodium hypochlorite to examine the morphological type of dead embryos.

* Now at the Agricultural Research Council's Poultry Research Centre, West Mains Road, Edinburgh, 9.

Fixation of lethal embryos and larvae for histological examination is carried out in formol–alcohol–acetic acid mixture, puncturing to aid penetration. The material is double embedded using celloidin in methyl benzoate before paraffin wax. Sectioned embryos are stained in Gentian violet. Feulgen stained and unstained whole mounts of lethals *Delta*, *E/L2* and *E/L4* were also made. Sections of lethal *glassy* larvae were prepared with Hansen's trioxyhaematin as nuclear stain and Xylidine Ponceau and light green as counter stains.

THE PATHOLOGY OF LETHAL HOMOZYGOTES

E1. In embryos homozygous for lethal *E1* development is arrested at an early stage before blastoderm formation. Dechorionated embryos are indistinguishable from unfertilized eggs in whole mount preparations. Necrotic changes in the egg cytoplasm become evident at about the time of eclosion of normal sibs. Cessation of development, therefore, takes place at a very early ontogenetic stage, but the pathology has not been worked out in detail.

E/L1. Embryos homozygous for lethal *E/L1* resemble their normal sibs at the time of eclosion from the egg but fail to hatch. The main tracheal trunks are air filled but contain occasional liquid spaces and the lateral tracheal trunks are frequently filled with liquid. In a few cases hatching movements can be observed but many embryos are immobile. Some of the embryos succeed in opening the operculum and emerge wholly or partially, but those which do become free of the egg case collapse and become flaccid. The lethal larvae are unable to feed and die soon after emergence. Kalliss (1939) has described a lethal X-chromosome deficiency Df(1)26–2 in *Drosophila melanogaster* involving the loci *yellow* and *achaete* in which the lethal embryos are unable to hatch. Kaliss found that the main tracheal trunks were not properly evacuated due to permeability to water of the larval cuticle. Lethal *E/L1* is similar in the loss of turgor after hatching and in failure of evacuation of the tracheal system. However, Kaliss found that the cephalopharyngeal apparatus was improperly sclerotized so that the mouth hooks bent and prevented eclosion from the egg case. In this respect the two lethals differ as the cephalopharyngeal apparatus of lethal *E/L1* embryos was apparently normal, neither is there any indication of sex linkage for lethal *E/L1*.

E/L3 and *E/L5*. Embryos homozygous for lethals *E/L3* and *E/L5* appear to be fully developed but fail to eclode from the egg. No hatching movements were ever observed.

E/L2 and *E/L4*. Homozygotes for lethals *E/L2* and *E/L4* show gross morphological defects of the embryonic head. The cephalopharyngeal apparatus is abnormal and there are various stages of arrest in the involution of the head. The segmentation posterior to the head, and the tracheal system, is normal in both cases. The pseudocephalon of the normal embryo is shown in Plate I, Figs 1 and 2. In Fig. 3 the mouth hooks and fields of chitin teeth have been prevented by failure of head involution from entering the pseudocephalon of an *E/L2* mutant. The lateral pharyngeal sclerites are distorted and thickened and the epi- and hypostomal

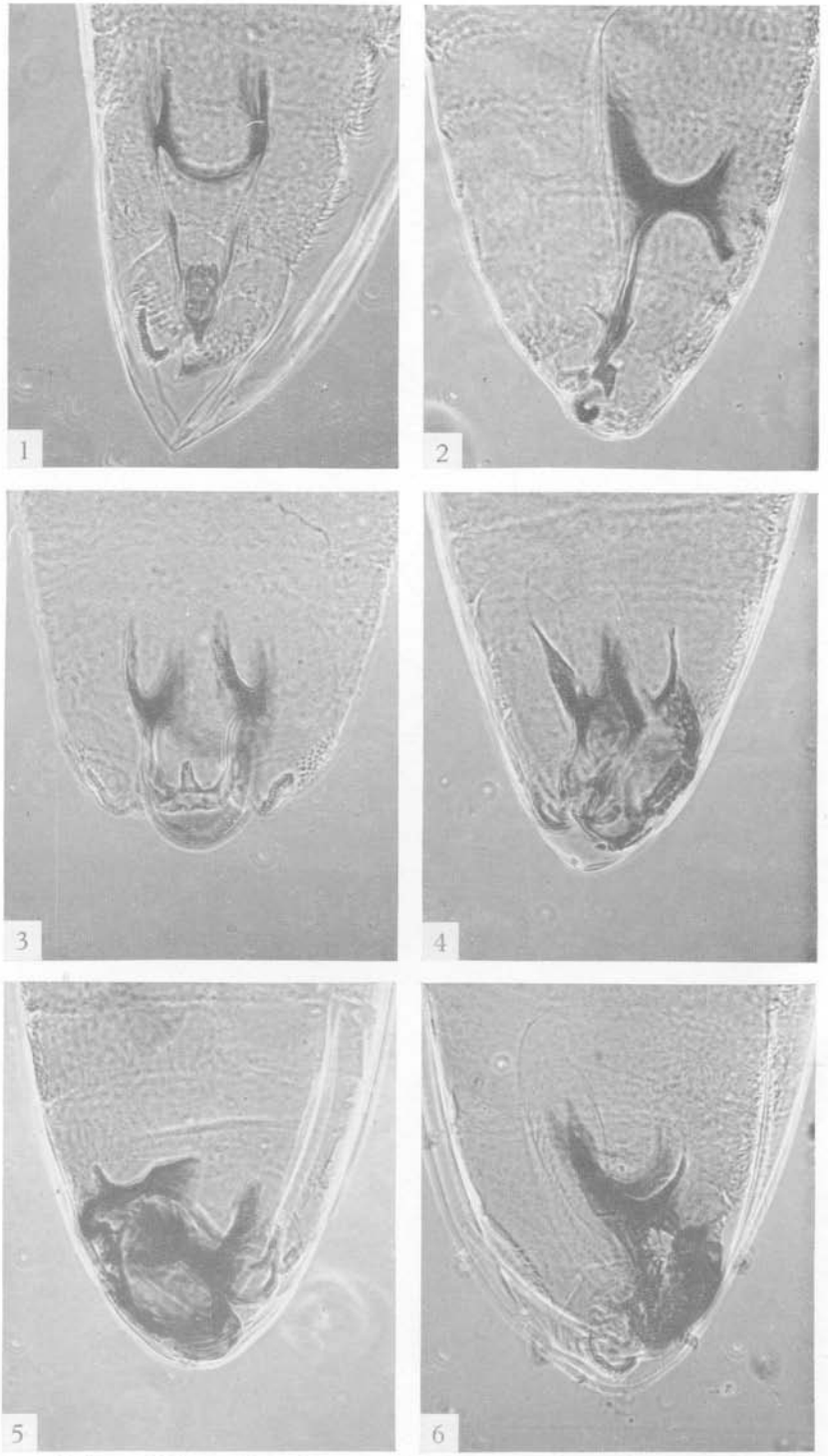


Fig. 1. Dorsal view of the cephalopharyngeal apparatus of a wild type embryo just before eclosion from the egg.

Fig. 2. Wild type. Lateral view.

Fig. 3. Homozygote for lethal *E/L2*, ventral view, showing incomplete head involution. The epi- and hypostomal sclerites are malformed and protrude beyond the level of the mouth hooks.

Fig. 4. Incomplete head involution and severe distortion of the cephalopharyngeal apparatus in a homozygote for lethal *E/L2*.

Fig. 5. Homozygote for lethal *E/L4* showing early failure of head involution, chitinized ectodermal rudiments of the cephalopharyngeal apparatus lie on the outside of the embryo.

Fig. 6. Failure of involution at an advanced stage in a homozygote for letahl *E/L4*. A dense mass of chitin lies at the back of the median tooth.

Phase contrast (× 180)

(*Facing p.* 406)

sclerites are fused and protrude from the head. The median tooth is missing. Figure 4 is typical of a great many *E/L2* homozygotes in showing an anomalous mass of diffuse chitin in the dorsal portion of the pseudocephalon. The posterior forks of the lateral plates, longitudinal shafts and median tooth can be recognized within the mass.

E/L4 homozygotes show a similar spectrum of stages of head involution as shown in Figs. 5 and 6. In many lethal embryos head involution fails entirely (Fig. 5), and at the anterior end of the embryo there is a sepia-coloured cap. Figure 6 shows a case of late failure of involution in which the mouth hooks and chitin teeth have almost reached their final position. A common feature of these cases of late failure is the fusion of the epi- and hypostomal sclerites in a dense mass of heavily pigmented chitin at the back of the median tooth. The morphological effects of lethals *E/L2* and *E/L4* are very similar to those of lethals Df(2) vg^C and vg^D, in *Drosophila melanogaster* described by Bull (1954) in which similar abnormalities of the mouth hook apparatus are caused by failure of involution and contraction.

Delta

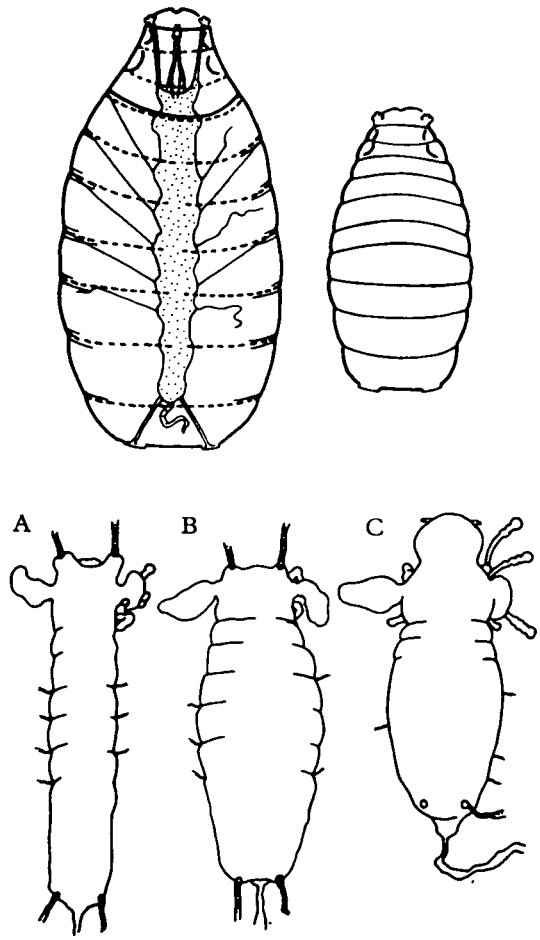
Delta is a dominant mutant acting as an embryonic lethal in homozygotes. The heterozygotes are distinguished by delta-shaped thickening of the longitudinal wing veins at the wing margin and at the junctions with the cross veins. There is occasional nicking of the wings and a slight reduction of eye size.

In homozygous lethals abnormalities become apparent with a delay in blastoderm formation which in normal embryos is completed during the 2nd hour of development, but not in the lethals until the 3rd hour. The blastoderm cells of the lethal are smaller than normal and the lateral cell boundaries poorly defined. Development becomes progressively more abnormal due to failure of gastrulation. The ventral furrow of the lethal is shallower than in the normal and the cells of the germ band are loosely and irregularly arranged. The posterior midgut rudiment forms as an invagination which in normal and lethal embryos contain the pole cells. In the normal embryo a region of rapid mitotic activity lies in the postero-ventral lip of this invagination which causes it to move antero-dorsally. In the lethal the midgut rudiment is associated with two centres of mitotic activity, and after the antero-dorsal movement of the invagination to about one-quarter of the length of the embryo from the caudal end, their activity causes the closure of the invaginated posterior midgut rudiment which undergoes no further development. Both endodermal and mesodermal derivatives fail to differentiate. The anterior and posterior midgut and malphigian tubules are suppressed and there is no hindgut. The body and gut musculature is completely absent. Some ectodermal derivatives are formed, the foregut differentiates into oesophagus and pharyngeal rudiments. Salivary gland anlagen and tracheal pits appear at the 8th hour of embryogenesis but fail to develop further.

glassy

The homozygous mutants for lethal *glassy* become recognizable as early third instar larvae but can be classified with certainty only from the middle of third

instar. The mutant larvae are, apart from the mouth hook apparatus and gut contents, completely glassy in appearance and become bloated with haemolymph so that by late third instar they are twice the volume of their normal sibs. Many of the larvae become so bloated that feeding and movement are prevented. The fat body is rapidly reduced in such larvae, a proportion of which form a puparium



Text-fig. 1. Upper left, puparium of a *glassy* homozygote with the pupa visible through the translucent cuticle. Upper right, puparium of a wild type sib. Lower, three typical stages at which pupal development ceases. A, abdomen uncontracted, cryptocephalous, wing and leg buds everted. B, partial abdominal contraction. C, abdomen contracted, cephalic complex everted.

which is much larger than normal (Text-fig. 1). The larvae frequently fail to contract normally before pupation and the pupal case often does not develop the dark pigmentation of normal puparia but remains a translucent corn colour through which the pupa can be examined (Table 1). All stages from brown patches to normal pigmentation can be found, and some mutants which have not accumulated a large volume of haemolymph succeed in forming a normal puparium.

The stage at which pupal development is arrested is shown in Table 2. In a series of 184 mutant larvae, 89 successfully formed a puparium, 13 of these failed to pass the prepupal stage and remained as pseudopupae. Of the 76 which successfully completed the prepupal ecdysis in 54 abdominal contraction failed. The pupa resembled that shown in Text-fig. 1*a*, the wings and legs were everted but the head remained within the thorax. In 12 cases there was partial or complete abdominal

Table 1. *Contraction and pigmentation of the puparium in homozygotes for lethal* glassy

	Puparium uncontracted		Puparium contracted		
Died as third instar larvae	Glassy or corn colour	Brown	Glassy or corn colour	Brown	Total
190	53	32	14	10	299

contraction but the head was not everted (Text-fig. 1*b*). In 10 cases with abdominal contraction the head was everted (Text-fig. 1*c*), but in only one of these were the external genitalia formed. The effect of removing excess haemolymph from *glassy* larvae was examined by puncturing with a sterile needle and allowing the larvae to deflate to normal size, they are not harmed by this treatment and remain glassy in appearance. The larvae re-accumulate the excess haemolymph and become

Table 2. *The stage at which development ceased in homozygotes for lethal* glassy

		Pupae				
		Wings and legs everted				
		Acephalous		Head everted		
Larvae	Pseudo-pupae	Abdomen uncontracted	Abdomen contracted	No external genitalia	External genitalia	Total
95	13	54	12	9	1	184

bloated as before but the onset of pupation is delayed by about 2 days over the controls.

The fat body in *glassy* larvae is reduced or absent presumably as a consequence of starvation due to inability of badly bloated larvae to carry out normal feeding activities, but serial sections of mutant larvae and their normal sibs failed to reveal any gross abnormalities of the larval organs except that in general the tissues of *glassy* larvae were smaller than in normal sibs of the same age. Lethal *glassy* is in many ways similar to *ltr*(3)–20·7 *translucida* described by Hadorn (1951) in *Drosophila melanogaster*. This lethal also causes accumulation of haemolymph and

reduction of fat body. As in lethal *glassy* surplus haemolymph lies between the pupa and the puparium. Unlike lethal *glassy* the head is usually everted, there is advanced metamorphosis of the head and thorax, and the abdomen undergoes no differentiation. Whatever the cause of accumulation of haemolymph in *glassy*, its presence interferes with the contraction of the larvae prior to pupation. The shortening of the larval cuticle which forms the pupal case is due to muscular contraction. This is confirmed by Benz (1957) who in his study of l(2)–20·3 lethal *polymorph* in *Drosophila melanogaster* found that failure of contraction before pupation was associated with muscular dystrophy in the larvae. In addition to failure of the larva to contract before pupation in lethal *glassy* there is also failure of contraction in the pupa itself. The abdomen remains uncontracted and the head often fails to evert, though it can often be induced to do so in such cryptocephalous papae by gentle pressure on the thorax after the pupal case has been removed. It seems that failure of head eversion is a mechanical consequence of failure of abdominal contraction which leaves the pupal thorax jammed against the back of the larval cephalopharyngeal armature.

SPECIFICITY OF THE LETHAL CRISIS

As described above, a small proportion of *E/L1* homozygotes are able to eclose from the egg. Although these larvae die soon after as first instar larvae they succeed in passing the stage at which development is arrested in other lethal sibs. In 27 clutches segregating for lethal *E/L1* (Table 3). the dead first instar larvae were

Table 3. *Frequency of lethals in the progeny of matings between lethal heterozygotes*

Lethal	Total eggs	No. of lethals: Expected	No. of lethals: Observed	χ^2
Delta	1170	292	315	2·31
E1	2304	576	584	0·15
E/L1	2755	686	617	9·96*
	2572†	643	665	1·00
E/L2	2358	590	641	5·99‡
E/L3	2946	737	720	0·49
E/L4	1647	411	422	0·19
E/L5	1880	470	466	0·05
glassy	1542§	386	341	6·85*

* Significant at the 1% level.
† Embryos plus dead first instar larvae.
‡ Significant at the 5% level.
§ Third instar larvae.

added to non-hatching *E/L* stage embryos; these clutches now give a good agreement to the expected ratio of 3:1, indicating that the apparent deficit of *E/L1* homozygotes is caused by the limit of classification cutting across the period of expression of this lethal. A similar situation is described in the data of Brehme (1937) on the time of action of the *C1B* lethal in *Drosophila melanogaster*.

Lethals *E1*, *E/L3*, *E/L4*, *E/L5* and *Delta* give good agreements to expectation in single factor segregations and appear to be strictly phase-specific in their mode of action. However, for lethal *E/L2* Table 3 shows a significant excess of lethal embryos. Unfertilized eggs are not included in the counts and all embryos classified as lethals showed the same pattern of abnormality involving the embryonic head. It seems that, in a small proportion of the heterozygotes, lethal *E/L2* has incompletely recessive effects which are expressed during embryonic development.

With lethal *glassy* the situation is rather different to that in the embryonic lethals. In Table 2 development is shown to cease at various stages but arrest seems to occur at certain important morphogenetic events from the third instar onwards. The majority of lethal homozygotes are arrested before the larval-prepupal boundary or else in the pupa, in the short period after eversion of the wings and legs but before eversion of the head. Classification of lethal *glassy* is possible from middle third instar onwards when the homozygotes are recognizable with certainty, but in Table 3 the proportion of homozygotes, observed in 26 families is significantly below expectation (heterogeneity $\chi^2_{25} = 29{\cdot}44$, $P = 0{\cdot}3$–$0{\cdot}2$). This deficiency is due to a proportion of *glassy* homozygotes which die before reaching the middle of third instar. The effect is more pronounced when the larvae are crowded and is not due to incomplete penetrance of the *glassy* phenotype.

Out of a total of 795 *glassy* homozygotes examined eight individuals managed to complete development to adult flies. Two pair matings were made from these flies, and both produced all lethal progeny, thus proving that the parents were homozygous for the lethal factor. These individuals may be regarded as true breakthroughs in that they by-passed the lethal crises which caused failure of development and death of other homozygous *glassy* sibs. They are of some theoretical interest since they show that 'modifying factors' may from time to time be sufficiently powerful to suppress the developmental effects of a recessive lethal in homozygous state. Lethal *glassy* contrasts with the embryonic lethals in showing no strict specificity in its time of action.

THE VIABILITY OF LETHAL HETEROZYGOTES

Heterozygotes for lethal *Delta* can be distinguished as adults by the characteristic abnormality of the wing venation from which the mutant takes its name. In a back-cross experiment ($+/D \times +/+$) the F_1 progeny consisted of 867 Delta and 914 wild-type flies ($\chi^2 = 1{\cdot}24$, $P = 0{\cdot}25$) giving a good agreement to the expected ratio of *Delta* and wild-type flies in equality. Recessive lethals with dominant visible effects in the heterozygotes are not common and this creates the necessity for less direct methods of analysis. Let s be the relative viability of individuals heterozygous for a given recessive lethal in comparison with wild-type flies. Then an estimate of s is obtained in the following way. Flies heterozygous for the lethal are mated, giving progeny whose expected frequencies are: $2s(+/l)$ to $1(+/+)$. These progeny are pair-mated at random, and the proportion which give a lethal

segregation noted (+/l × +/l matings). s is then estimated by maximum likelihood from this proportion. We have:

	Type of mating		
	(+/l) × (+/l)	Rest	Total
Expected frequency	$4s^2/(1+2s)^2$	$(1+4s)/(1+2s)^2$	1
Observed number	a	b	n

the maximum likelihood function is:

$$L = 2a \log s + b \log (1+4s) - 2(a+b) \log (1+2s)$$

differentiating and solving for s we have:

$$s = \frac{a + \sqrt{(an)}}{2b}$$

$$V_s = \frac{(1+4s)(1+2s)^2}{16n}$$

It has been shown that after a female has laid her first fertile clutch a second male may be used to fertilize the subsequent clutch without giving rise to mixed progeny, thus providing a reliable method for making cross-tests for allelism between embryonic lethals, Burnet (1961). In this way we are able to identify lethal heterozygous pairs in an inbred line and use them to make an outcross to an unrelated stock. Outcrosses were made to a stock homozygous for three unlinked recessive visible factors so that the phenotype of both the F_1 eggs and the F_1 adults could be checked. In the case of the outcross of a lethal heterozygote the F_1 yields, of course, no lethal segregation. Making random pair matings among these individuals, 1 in 4 of these pairs are expected to give a segregation for the lethal in the next generation, and by similar reasoning to that above we have:

$$s = \frac{a + \sqrt{(an)}}{b}$$

$$V_s = \frac{(1+2s)(1+s)^2}{4n}$$

For a lethal which is completely recessive to its wild-type allele, the viability of the heterozygote is equal to wild type, and the expected value of s is unity. Results for seven lethal lines are shown in Table 4. The values for lethal *E1*, *E/L1*, *E/L2* and *glassy* do not differ significantly from unity, suggesting that these lethals have no incompletely recessive viability effects. On the other hand, there is evidence that, under the standard culture conditions at present employed, lethal *E/L3* causes a reduction in the viability of heterozygous carriers. Table 4 also shows that the viability index for lethals *E/L4* and *E/L5* is substantially higher than normal, the difference between the observed and expected values being significant at the 1% level of probability. Among the families scored for lethal *E/L4* one was

found in which all pair matings gave a lethal segregation. This family was excluded from the total shown in Table 4 and used to establish a separate line in which lethal *E/L4* was balanced against a second lethal in the homologous chromosome.

Table 4. *Index of viability* s *for lethal heterozygotes in seven inbred lines of* C. frigida

Lethal	Pairs examined	Lethal segregations	*s*	S.E.
E1	140	58	0·903	0·128
E/L1	347	169	1·15	0·105
E/L2	226	109	1·14	0·128
E/L3	234	90	0·816*	0·089
E/L4	427	179	1·60†	0·129
E/L5	390	249	1·98†	0·188
glassy	138	59	0·945	0·134

* Significant at the 5% level.
† Significant at the 1% level.

The high values for lethals *E/L4* and *E/L5* were examined further by making outcrosses of heterozygotes to an unrelated stock homozygous for three recessive factors: *white eye* (*we*) *sherry eye* (*sh*) and *cut wing* (*ct*). Table 5 shows that the value of *s* for lethal *E/L4* remains significantly higher than expectation after outcrossing whereas the value for lethal *E/L5* does not. The behaviour of lethal *E/L5*

Table 5. *Index of viability* s *for lethal heterozygotes in two outcross lines*

Lethal	Pairs examined	Lethal segregations	*s*	S.E.
E/L4	161	55	1·41*	0·185
E/L5	153	44	1·15	0·159

* Significant at the 5% level.

in the outcross suggests that the high value obtained for the inbred line is due to the presence of a deleterious factor in the homologous chromosome and not to any peculiar property of lethal *E/L5* itself. As mentioned above, the inbred lines are maintained under a system of mating in which only the progeny of matings between lethal heterozygotes are kept. The conditions are, therefore, ideal for the formation of a balanced system with any deleterious or lethal factor which appears in the homologous chromosome.

The value of *s* for *E/L4* is based on the total from five outcross cultures, all show a good agreement among themselves with respect to the excess of heterozygotes (heterogeneity $\chi^2 = 1{\cdot}31$, $P = 0{\cdot}85$). The persistence of the high value of *s* in the outcross suggests that the superior viability of heterozygotes for this lethal depends on properties of the lethal bearing chromosome itself. The mean clutch size in the inbred line for females heterozygous for lethal *E/L4* is 111 eggs. In segregating

cultures 25% of the eggs fail to hatch, giving an average 83 first instar larvae per culture. This is more than the standard culture bottle will support so that survival to adulthood at this density is less than 80% of the total initial number of larvae, (Burnet & Thompson, 1960). It seems that heterozygotes for lethal *E/L4* are at an advantage under these crowded conditions.

DISCUSSION

Comparative studies on the time of action and morphological pattern of damage of homozygous recessive lethals in *Drosophila melanogaster* show that, in general, such factors are specific to a particular phase of development (Hadorn, 1951). With the exception of lethal *E/L1* and *glassy*, the factors described above do not differ from this general rule. On the other hand, there is an increasing number of cases, Benz (1957), Seto (1954), in which the timing of lethality does not show this specificity. Hadorn (1951) has suggested that the absence of complete concordance in the timing of lethality between homozygous lethal sibs is due to residual genetic variation or to small environmental differences. In lethal *glassy*, timing of developmental failure is sensitive to environmental variations, significantly more mutants achieve contraction and pigmentation of the puparium at 18° C. than at 25° C., and under conditions of larval crowding fewer mutant larvae reach the pupal stage than in control cultures. The spread in distribution of lethality shown by this mutant, coupled with the observation that a very small proportion of mutants develop to fertile adults, underlines the arbitrary distinction between so called unconditional lethals and semi-lethals. If it can be shown that the effects of lethals may be ameliorated both by genetic modifiers and by environmental variables it may be expected that under certain conditions the penetrance of a recessive lethal may be reduced to zero. Suggestive evidence in this direction has already been given by Dobzhansky & Spassky (1944) who showed that homozygotes for a second chromosome AA.1015 in *Drosophila pseudoobscura* were lethal at 25½° C., semi-lethal at 21° C. and close to normal viability at 16½° C.

Of the eight lethals described in this study, detectable effects have been found in the heterozygotes of four. As attention has been concentrated on morphological effects, and effects on preadult viability, and bearing in mind that the tests on pre-adult viability are not on a scale large enough to detect small departures from expectation, the possibility of additional effects on these and other characters cannot be dismissed.

In a study of 75 sex-linked lethals in *D. melanogaster* Stern *et al.* (1952) found a range of values for the viability index of lethal heterozygotes ranging from 0·602 to 1·312, with a mean of 0·961 giving an overage a 5% reduction in viability. Both *E/L2* and *E/L3* have deleterious effects in the heterozygotes but in only the latter case were progeny tests large enough to detect the effect. That the reduction in the observed proportion of viable heterozygotes for *E/L3* was a spurious effect due to a slower rate of development can be discounted as each culture was exhausted of viable adult flies.

Recently a remarkable case of single gene heterosis involving a second chromosome lethal l(2)55i in *D. melanogaster* has been analysed by Schnick, Mukai & Burdick (1960). Maintenance of this lethal at high frequency in a variety of outcross populations was found to be due to superior fecundity of the heterozygous lethal females. It is not possible with the data described above to determine whether the superior viability for *E/L4* heterozygotes is due to the effect of the *E/L4* locus *per se* or due to particular properties of the gene complex in the lethal bearing chromosome as a whole. The distinction between these two possibilities will depend on the performance of lethal *E/L4* in a variety of outcross populations. Sandler, Hiraizumi & Sandler (1959) have described the maintenance of an excess of heterozygotes for a second chromosome lethal in *D. melanogaster* due to distorted gametic segregation ratios. An excess of lethal-bearing sperm is produced by the males. If there are distorted gametic segregation ratios for either sex carrying lethal *E/L4* we should not expect the good agreement to expectation for the zygotic segregation ratios produced by matings between heterozygotes in Table 3, so that the excess of *E/L4* heterozygotes cannot be attributed to this cause. For the same reason the excess cannot be attributed to differential sperm competitive ability.

SUMMARY

The time of action and morphological pattern of damage of eight recessive autosomal lethals in *Coelopa frigida* (F.) is described. Seven embryonic lethals are phase-specific in their time of action but lethal *glassy* may cause death at any time from the beginning of the third instar onwards.

A method for estimating the preadult viability of heterozygotes for lethals is described. Heterozygotes for lethal *E/L3* show some impairment of viability, whereas, under conditions of larval competition heterozygotes for lethal *E/L4* are superior in viability to their wild type sibs.

This work was carried out during tenure of a post-graduate studentship from the Department of Scientific and Industrial Research. The author wishes to express thanks to Dr Barnet Woolf for his assistance in deriving the viability index, and to Dr Ursula Philip for her interest and encouragement.

REFERENCES

Benz, G. (1957). Untersuchungen uber die Wirkung der Letalfactoren von *Drosophila melanogaster*. *Z. indukt. Abstamm-. u. VerebLehre*, **88**, 78–114.

Brehme, K. S. (1937). The time of action of the C1B lethal in *Drosophila melanogaster*. *Amer. Nat.* **71**, 567–474.

Bull, A. L. (1954). Experimental studies of three cases of embryonic lethality in *Drosophila melanogaster* produced by over-lapping deficiencies for the vestigial locus. *Proc. 9th int. Congr. Genet.* (*Bellagio*), 1153–1156.

Burnet, B. (1961). On the distribution of recessive embryonic lethals in a natural population of *Coelopa frigida*. (Fab.) *Genet. Res.* **2**, 249–271.

Burnet, B. & Thompson, U. (1960). Laboratory culture of *Coelopa frigida* (Fab.) (Diptera: Coelopidae). *Proc. R. ent. Soc. Lond.* A, **35**, 85–89.

Dobzhansky, Th. & Spassky, B. (1944). Genetics of natural populations. XI. Manifestation of genetic variants in *D. pseudoobscura* in different environments. *Genetics*, **29**, 270–290.

HADORN, E. (1951). Developmental action of lethal factors in *Drosophila*. *Advanc. Genet.* **4**, 53–85.

HADORN, E. (1961). *Developmental Genetics and Lethal Factors*. London: Methuen & Co. Ltd.

KALISS, N. (1939). The effect on development of a lethal deficiency in *D. melanogaster*: with a description of the normal embryo at the time of hatching. *Genetics*, **24**, 244–270.

SANDLER, L., HIRAIZUMI, Y. & SANDLER, I. (1959). Meiotic drive in natural populations of *Drosophila melanogaster*. I. The cytogenetic basis of segregation distortion. *Genetics*, **44**, 233–250.

SCHNICK, S. M., MUKAI, T. & BURDICK, A. B. (1960). Heterozygote viability of a second chromosome recessive lethal in *Drosophila melanogaster*. *Genetics*, **45**, 315–329.

SETO, F. (1954). Time of action of a series of recessive lethal factors in *Drosophila melanogaster*. *J. exp. Zool.* **126**, 17–32.

STERN, C., CARSON, G., KINST, M., NOVITSKI, E. & UPHOFF, D. (1952). The viability of heterozygotes for lethals. *Genetics*, **37**, 413–449.