It has previously been demonstrated that a fraction of catalase is compartmentalized in the glyoxosomes of maize (Scandalios 1974) and the liver peroxisomes of mammals (Holmes 1971). In our studies, the use of triton X-100 increased the amount of soluble extracted enzyme by 1/3 in adult extracts and by two-fold in larval extracts. These results may indicate that a fraction of the catalase activity is compartmentalized or membrane-bound to subcellular organelles in Drosophila.

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Biemont, C. Université Lyon 1, Villeurbanne, France. Parental effect and in-breeding depression in D. melanogaster.

Natural populations of Drosophila carry genetic loads consisting of deleterious variants which reduce the viability of their carriers when homozygous as a result of inbreeding (see Lewontin 1974). Dying of inbred offspring ranges from early embryogenesis to larval and pupal stages. Recently, in D. melanogaster, I interpreted such effects in terms of a single gene hypothesis (Biemont 1978, 1979). An Is (in-breeding sensitivity) gene, located on chromosome III, with alleles Is- and Is+ is involved in morphogenetic events. Its expression in homozygous (Is-/Is-) embryos depends on the presence in one parent of an Is+ allele which promotes embryogenesis. Crosses between Is-/Is- sibs produce embryonic deaths, of a level that varies according to the regulation of the expression of the Is- allele. We now report further evidence supporting the parental control of expressivity of the gene involved.

Brother-sister couples which laid eggs showing blocking in development were selected and the male and female separated. Once females from these couples of presumed Is-/Is- constitution were no longer producing fertilized eggs, they were crossed with Cy/Pm H/Sb males. Is-/Is+ Cy Sb flies from different sibships were then intercrossed leading to four classes of offspring with phenotypes

\[ +: \left( \frac{++}{++} + Is- \right), Cy: \left( \frac{Cy + Is-}{++ + Is-} \right), \]

\[ Sb: \left( \frac{++}{++} + Is- \right) \text{ and Cy Sb:} \left( \frac{Cy+ + Is-}{++ + Sb +} \right). \]

In each class, brothers and sisters were mated and viability of their offspring was evaluated as the proportion of wild type individuals (all +Is-/+Is-) obtained from the eggs laid by the sibs. Therefore, to each Is- Is+ Cy Sb x Is- Is+ Cy Sb parental couple, is associated the inbreeding response of the four classes of their offspring. Since our study is based on the egg-to-adult survival of wild type flies, I have eliminated the Cy Sb class in the progeny of which only 1/16 of wild type flies is expected; the number of such flies obtained was too small for valuable statistical analysis. The rank correlation coefficient of Spearman reveals a significant link between the values of the + and Cy classes (Fig. 1) \( r = 0.57; t = 2.5; P < 0.05 \). This correlation was not significant either between

Fig. 1. Egg-to-adult survival of wild type offspring from the + class, versus egg-to-adult survival of wild type offspring from the Cy class. For comparison between the two classes, values of the Cy class were multiplied by 4 since only 1/4 of the flies were theoretically expected to be wild type.
classes + and Sb (r = -0.019; t = 0.63; P > 0.05) nor between classes Cy and Sb (r = 0.14; t = 0.53; P > 0.05). Consequently, inbreeding depression is similar in the + and Cy classes but independent of that in the Sb class. As a result of our experimental scheme, all wild type individuals descending from the brother-sister matings were homozygous Is-/Is-. So, the difference between the +, Cy and Sb classes seems associated with the homozygous Is-/Is- constitution of the flies of the + and Cy class as compared with the Is-/Is+ heterozygous state of the flies of the Sb class. This observation suggests that the proportion of wild type flies, therefore the mortality rate during development, depends on the genomic constitution of the parents. The extent of inbreeding depression appears to characterize the parental couple, thus suggesting regulation by cytoplasmic factors, as previously inferred (Bimont 1978).

Such a parental effect has to be taken into account when inbreeding effects with different mating systems, or various natural populations, are compared. Indeed, whatever the nature of the implied gene, variation of its frequency in populations may influence the extent of viability depression after inbreeding and thus estimate of genetic load.

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Bishop, C.P. and A.F. Sherald*. University of Virginia, Charlottesville, Virginia; *George Mason University, Fairfax, Virginia. Isolation of two third chromosome mutants conferring resistance to α-methyl dopa.

α-methyl dopa (α-MD) is an in vitro inhibitor of dopa decarboxylase (DDC) and it was originally thought that α-MD might be used to screen for mutants with altered levels of the DDC enzyme. Although the original screen for resistance to α-MD produced two strains with elevated levels of DDC (Sherald and Wright 1974), screens for sensitivity to the inhibitor produced mutants with no effect on the enzyme (Sparrow and Wright 1974). Furthermore, it has been subsequently shown that the greater the number of DDC gene copies, the greater the sensitivity to α-MD (Wright, unpublished). Sensitivity to α-MD, it was discovered, was due to a locus, 1(2)amd, other than the structural locus for DDC (Wright et al. 1976a, 1976b). Since the 1(2)amd locus maps very close to the structural gene for DDC, the mutants with both elevated resistance and enzyme activity may be control mutants (Marsh and Wright 1979).

The two α-MD resistant mutants we report here were isolated from a total screen of 1,715 EMS mutagenized (Lewis and Bacher 1968) progeny from a lethal free third chromosome bw; st stock. They were isolated by survival on 0.8 mM DL α-MD, well above the concentration that is lethal to wild type flies (less than 0.4 mM). A total of 80 putative resistant mutants were recovered, 18 of which showed resistance upon retesting and two (PR40 and PR45) of these were selected for further study.

Table 1 shows that the locus responsible for resistance clearly segregates with the mutagenized third chromosome. Preliminary mapping of one of the mutants, PR45, places the locus between hairy (3-26.5) and thread (3-43.2) (Lindsley and Grell 1968). Using the L form of α-MD, which is roughly twice as lethal as the DL form, the LD50 for the two mutants has been established at 0.325 mM L α-MD for PR40 (bw; Tm3 Ser Sb/st*40) and 0.35 mM L α-MD for PR45 (bw; Tm3 Ser Sb/st*445). The LD50 for control stocks was below 0.1 mM L α-MD.

In addition to showing dominant resistance to α-MD, these mutagenized third chromosomes are recessive lethal. During preliminary mapping of PR45, replacement of large portions of the third chromosome did not permit construction of a homozygous resistant stock. Crosses between the two resistant mutants produced very few flies (roughly 5% of expected) carrying both resistant chromosomes, indicating that the two chromosomes fail to complement. The fact that the two independently isolated mutants are lethal in trans configuration and that a homozygous resistant stock could not be established even after replacement of significant portions of the third chromosome suggests that dominant resistance and recessive lethality may be due to hits in a single locus.

It is not surprising that more than one locus can affect resistance to a lethal substance. The function of the 1(2)amd locus and the locus reported here are unknown. The sites of possible action could include uptake or detoxification of the compound or alterations in the target protein. The genetic relationship between 1(2)amd locus and the third chromosome...