

Kerver, J.W.M. and R. Bijlsma. University of Groningen, The Netherlands. The effect of DDT on the activity of the pentose phosphate cycle in *D. melanogaster*.

The problem that insects develop resistance to insecticides has gained much attention in the years 1950-1970. Especially the resistance to chlorohydrocarbon insecticides—of which DDT (1,1,1-trichloro-2,2-bis(p-phenyl)-ethane) is the best known representative—is well studied.

(For a review see Perry and Agosin 1974). The resistance can be obtained by means of different mechanisms such as: avoidance of the insecticide by the insect, changes in penetration through the cuticula and increased detoxification by degradation of the insecticide. The degradation of chlorohydrocarbon insecticides is found to occur in many different ways in insects. In *D. melanogaster* two major ways of DDT degradation are regarded as important: degradation via hydroxylation to dicofol (2,2-bis(p-chlorophenyl)-1,1,1-trichloroethanol) and via dehydrochlorination to DDE (2,2-bis(p-chlorophenyl)-1,1-dichloroethylene). These reactions both require energy in the form of NADPH which is thought to be supplied by the pentose phosphate cycle. This may imply that treatment of *Drosophila* with DDT accelerates the activity of the pentose cycle. This was investigated by studying the effect of DDT treatment on flies by measuring the activity of two enzymes of the pentose cycle: glucose-6-phosphate-dehydrogenase (G6PD) and 6-phosphogluconate-dehydrogenase (6PGD).

Adult flies (5-10 days old) were transferred to food supplemented with 0.005% DDT and the in vitro enzyme activities of G6PD and 6PGD were measured (for method see Bijlsma 1978) at subsequent days and compared with the activities of flies on food without DDT (control). Fig. 1 shows the ratio of the activity of flies on DDT food and the activity of flies on control food (DDT/control) in the course of the experiment. This figure also shows the percentage dead flies on DDT-supplemented food in the course of time (on control food hardly any flies died). It is evident that the activity of both enzymes increases in flies on DDT, especially in females, and after 96 hours this increase is significant ( $P < .01$ ) in all cases. During the experiment quite a number of flies died and it is therefore possible that the change in activity of both G6PD and 6PGD is due to selection in favor of flies with high activity, rather than to an increase of enzyme activity in the individual flies. Therefore the experiment was repeated for lower concentrations of DDT: 0.0025% and 0.001%. On these concentrations a lower number

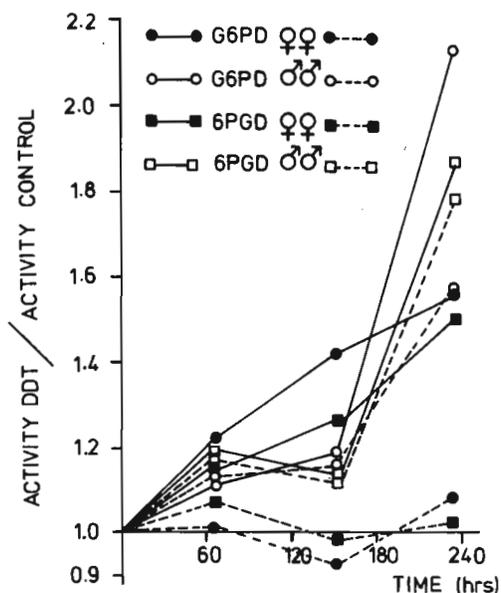


Fig. 1. The ratio of activity of flies on DDT-supplemented food (0.005% DDT) and the activity of flies on normal food at subsequent times of treatment (solid lines), together with the percentage flies that died at the same times (broken lines).

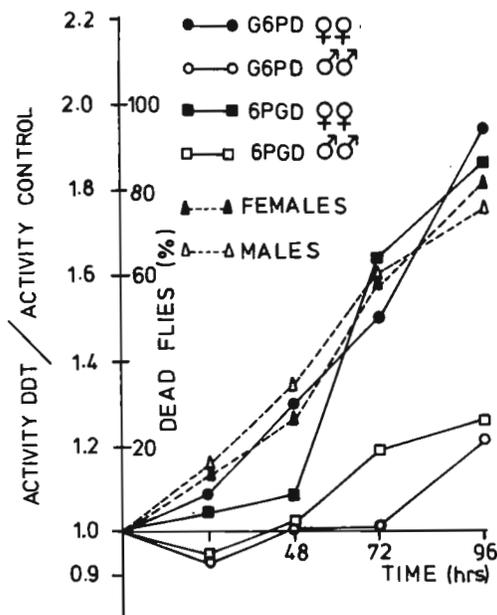


Fig. 2. The ratio of the activity of flies on DDT-supplemented food and the activity of flies on normal food at subsequent times of treatment. The data are given for two concentrations of DDT: 0.0025% (solid lines) and 0.001% (broken lines).

of flies died during the experiment, less than 40% on 0.0025% DDT and less than 10% on 0.001% DDT. The results are shown in Fig. 2. Also in this experiment a significant increase in the activity of G6PD and 6PGD is found in flies on DDT, though at the lowest concentration only the males respond. In spite of some differences in reaction between males and females it is evident that DDT treatment can strongly increase the activity of G6PD and 6PGD in adults. Preliminary experiments suggest that this also holds for larvae.

References: Bijlsma, R. 1978, *Genet. Res. Camb.* 31:227-237; Perry, A.S. and M. Agosin 1974, *The Physiology of Insecta*, vol. VI, Academic Press, New York.

King, R.C. and B.D. Buckles, Jr. Northwestern University, Evanston, Illinois. Three mutations blocking early steps in *Drosophila* oogenesis: *fs(4)34*, *fs(2)A16*, and *fs(1)231M*.

Two major deficiencies, *Df(4)M* and *Df(4)G*, allow the right arm to be partitioned into three unequal parts (Hochman, 1974). Hochman showed that *fs34* is not included in either deficiency, and therefore it probably resides somewhere between the middle of subdivision 102B and the beginning of subdivision 102E (see King, 1975, his Fig. 1). The heterozygotes used for the maintenance of the stock population are of genotype *fs(4)34/ci<sup>D</sup>*. The *fs(2)A16* mutation was induced by Bakken (1973) using EMS. She studied ovarian whole mounts from homozygotes and concluded that the ovarioles lacked clear cut germaria. The mutation has not yet localized on chromosome 2. Heterozygotes have the genotype *fs(2)A16/SML*.

The results of our electron microscopic studies are summarized in the accompanying illustration of sections from 2 day ovaries (Fig. 1). The *fs34* ovariole begins with a normal terminal filament which is anchored to the tubular epithelial sheath. The anterior end of the germarium contains abnormally large numbers of oogonial cells and lacks clusters of cysto-

The *fs(4)34* mutation was recovered by B. Hochman from a male collected in December of 1963 from a natural population of *Drosophila melanogaster* living in Lake County, Florida. Hochman (1972) reported that the mutant resided on the 4th chromosome, and it is still the only female sterile gene known for this microchromosome.

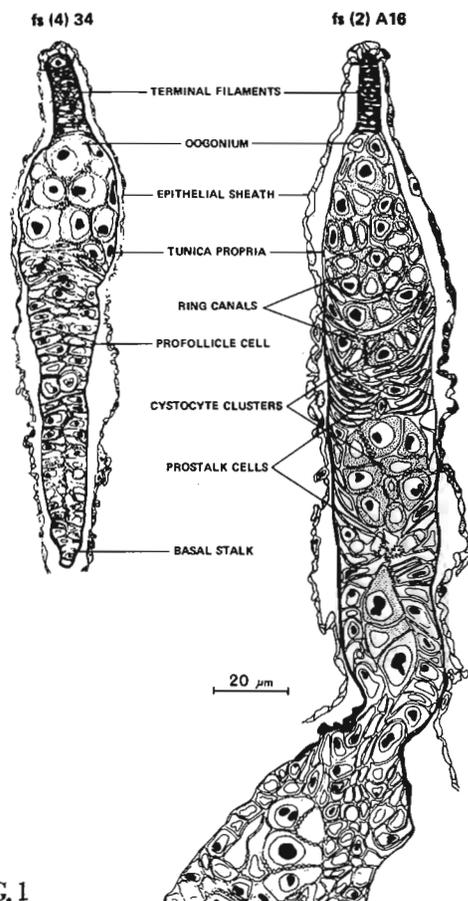


FIG. 1



FIG. 2