Decreased Metabolic Rate as an Acrolein Resistance Mechanism in *Drosophila melanogaster*

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Using several mutant strains of *Drosophila melanogaster*, with different degrees of spontaneous activity, we have studied whether a decreased metabolic rate, measured by oxygen consumption, might be one of the mechanisms involved in acrolein resistance, such as predicted by Hoffmann and Parsons (1989a, b). The results agree with this prediction.

**KEY WORDS:** *Drosophila melanogaster*; acrolein; spontaneous activity; resistance; oxygen consumption.

**INTRODUCTION**

Recently the role of environmental stress as a factor in evolution, especially in periods of quick change, has received increasing support (for review see Parsons, 1987, 1989). In fact, the genetic control of stress resistance tends to be mainly additive (Parsons, 1989) and this genetic architecture implies the possibility of rapid genetic change under directional selection. There is strong evidence that a possible underlying mechanism for increased stress resistance is a reduction in the metabolic rate of resistant individuals (Parsons, 1989; Hoffman and Parsons, 1989a, b; Sierra *et al.*, 1989). The hypothesis that increased resistance to a range of environmental stresses is associated with a reduction in metabolic rate led Hoffmann and Parsons (1989a) to make three predictions

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about genetic variation for stress resistance: "(1) increased stress tolerance will tend to be associated with decreased metabolic rate; (2) genetic correlations between tolerance of different environmental stresses will tend to be positive; (3) stress tolerance and life-history traits will tend to be genetically correlated, and in *Drosophila* these correlations will tend to be negative, except with longevity." [In this context we prefer to use the term resistance instead of tolerance according to Levitt (1980).]

These predictions have been checked by Hoffman and Parsons (1989a, b), who analyzed increased desiccation resistance, produced by artificial selection in *D. melanogaster*. In a series of recent papers (Sierra and Comendador, 1989; Comendador *et al.*, 1989; Sierra *et al.*, 1989), we have demonstrated that in *D. melanogaster* most of these predictions are in agreement with our results on selecting artificially for increased acrolein resistance: Acrolein resistant strains, selected at 17 and 24°C, show an increased developmental time, a reduction in productivity, and increased anoxia resistance. Furthermore, in agreement with Hoffmann and Parsons (1989b), the genetic control of resistance is additive, although at 24°C we also detected dominance in the direction of acrolein sensitivity (Comendador *et al.*, 1989).

Acrolein is an unsaturated aldehyde of three carbons, and therefore, there is a possibility that resistance to this compound is the result of specific biochemical changes, such as are known for some insecticides (Wood, 1981) and heavy metals (MacNair, 1981). However, this is not the case, since acrolein-resistant strains are also resistant to a variety of aldehydes (Barros *et al.*, 1990); moreover, neither of two described enzymes that may use short-chain aldehydes as a substrate, aldehyde oxidase and aldehyde dehydrogenase, is able to explain the resistance increase (Sierra *et al.*, 1989). All the evidence obtained to date points out that, according to the hypothesis of Hoffmann and Parsons (1989a), the most important mechanism for acrolein resistance is a depression in metabolic rate, which implies lower respiratory and energetic requirements. In this way, the uptake of acrolein by flies is reduced. This mechanism is unspecific and could be involved with resistance against other stressful environmental changes. (Sierra *et al.*, 1989).

In this work, we tried specifically to test the prediction that a reduction in metabolic rate is associated with increased stress resistance, using an experimental design which does not use artificially selected strains.

We have used a variety of mutant strains to examine the correlation between metabolic rate and acrolein resistance. These mutants are either hyperkinetic, such as the *Sh* and *Hk* mutants (see review by Ganetzky and Wu, 1986; Tanouye *et al.*, 1986), or hypokinetic, such as *shakA₂*