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Differences between Rates of Metabolism of Benzene Hexachloride in Resistant and Susceptible Houseflies

In a previous communication[1], the metabolism of γ-BHC was described after injection into three strains of flies: a susceptible strain (*S*), a strain resistant to γ-BHC (R_1), and a strain resistant to γ-BHC and DDT (R_2).

The rate of metabolism in the susceptible strain was not comparable with that of the resistant strains, since the former died rapidly, whereas the latter remained unaffected. Thus it was impossible to tell whether the difference in metabolism could be a cause of the difference in susceptibility.

Hitherto, the method of estimation did not permit the study of the metabolism at doses low enough to leave also strain *S* unaffected. However, the problem becomes accessible for experimentation if we assume that all isomers of BHC are metabolized by the same mechanism. Then the breakdown in the strains can be studied in comparable circumstances, as it is possible to estimate the α- and δ-isomer at concentrations that are not at all toxic to the flies.

The flies were injected into the thorax with BHC in an emulsion of peanut oil, and kept at 25° C. for varying periods of time, after which the amount of

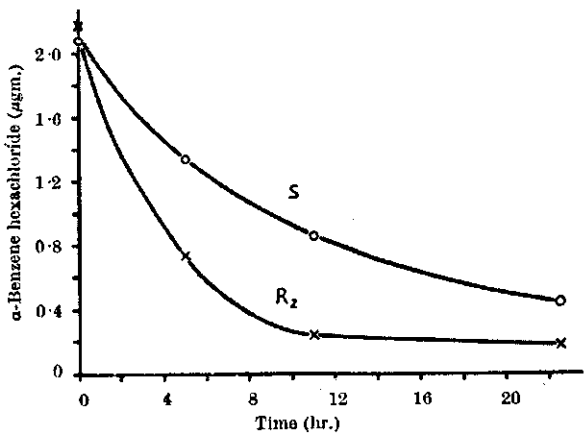

Fig. 1. Amount of α-BHC per fly plotted against time after injection. *S*, susceptible strain; R_2, γ-BHC and DDT-resistant strain. Fifteen flies used for each estimation

Table 1. BREAKDOWN OF α- AND δ-BHC
Thirty flies used for each estimation; horizontal lines represent injections on the same day

Isomer	Amount injected per fly (μgm.)	Time between injection and estimation (hr.)	Breakdown (per cent) S	R_2	R_1
α	0·83	4	36	60	80
	0·93	4	36	57	57
	0·67	4	45	55	65
	2·34	5	34	—	57
	2·34	5	43	—	71
	2·34	5	40	57	—
	2·34	5	23	50	69
δ	2·34	18½	28	43	—
	2·34	18½	29	32	—
	2·34	18½	25	32	—
	2·34	18½	27	46	47
	2·34	18½	27	39	37

BHC was estimated. The results are presented in Fig. 1 and Table 1. It is shown that there is a difference in breakdown of the α- and δ-isomer between the susceptible and resistant strains. This difference is somewhat larger in the case of the α-isomer. The breakdown of the δ-isomer is markedly slower than that of the α-isomer. As for technical reasons the experiments were not extended beyond 24 hr., it was impossible to obtain the complete breakdown curve for this isomer.

If our hypothesis is correct, the difference in rate of breakdown of γ-BHC between the susceptible and resistant strains has two causes: (1) a difference in breakdown capacity—a possible cause of difference in susceptibility; (2) a decreasing rate of breakdown in the susceptible strain as a result of the toxic action of γ-BHC—an effect of the susceptibility.

If we assume that the metabolism results in a detoxication, it is very probable that the difference in metabolism is a cause of the difference in susceptibility, although it is as yet impossible to estimate its importance. For example, it is not known how the rate of breakdown affects the concentration at the site of action. Work with more strains will perhaps be helpful in judging the significance of rate of breakdown for resistance, as it may reveal whether there exists a correlation between capacity of breakdown and level of resistance.

F. J. OPPENOORTH

Laboratory for Research on Biocides,
National Council for Agricultural Research T.N.O.,
Vondellaan 6, Utrecht.
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[1] Oppenoorth, F. J., *Nature*, 173, 1000 (1954).

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