in the second class, 40%, but the degree of heterozygosity in the individual's genome is considerably different: 0.3 % in the first case and 11.3% in the second.

For the enzymatic polymorphism the amount of heterozygosity per individual observed in D.m. falls in the range (.08 - .25) already observed on Drosophila species.

However, genetic variants studied in our two classes are probably different. In fact, for the rosy locus the electrophoresis technique allows one to see functional allozymic variants whereas the visible recessive phenotype technique shows the mutation corresponding to a "non functional" allozymic variant.

Thus, systematic comparisons in natural populations of the amount of polymorphism for the two mutation classes will provide interesting information on polymorphism, its nature and its maintenance.

Ce travail a été effectué dans le cadre de l'Equipe de recherche associée n°406 du CNRS: "Analyse et mécanisme du maintien du polymorphisme".

Jayasuriya, V.U. de S. and W.E. Ratnayake
Vidyodaya Campus, University of Sri Lanka, Nugegoda, Sri Lanka. Screening of some pesticides on Drosophila melanogaster for toxic and genetic effects.

The mutagenic effects of chemicals were discovered by Auerbach (1943) using Drosophila melanogaster. Sobels (1973) has very convincingly shown that Drosophila melanogaster is still the organism par-excellence for mutagenic studies. Although D. melanogaster has been used extensively for mutagenic studies, this organism has not been widely used for screening mutagenicity of pesticides. Instead, pesticides have been tested widely for genetic and cytogenetic effects on prokaryotes like E. coli, phage and on eukaryotes like Vicia faba and Allium cepa which are plants (Epstein and Legator 1970).

Benes and Sram (1969) studied the mutagenic effects of 34 chemicals (of which 16 were pesticides), by injecting relatively high concentrations into adult D. melanogaster flies and scoring for sex linked lethal mutations. The pesticides tested were mostly insecticides and they did not show any significant mutagenic activity.

In the present study a larval feeding method was employed for testing the mutagenic effects of pesticides. Initially, various concentrations of different pesticides (six - see Table 1) obtained by the serial dilution method were made up in Drosophila food medium and 50 1st instar larvae were introduced into 3" x 1" vials containing about 8 - 10 ml of treated food medium.

Table I. Pesticides used in the study.

1. Fenbar (fenitrithion)	
2. Endrin	1,2,3,4,10,10Hexachloro 6,7-epoxy-1,4,4a,5,6,7,8,8a-octahydro-1,4-endo-endo5,8-dimethanonaphthalene
3. Gamalin	Benzene hexa chloride
4. Deenol (D.D.T.)	1,1,1, Trichloro 2,2 bis(p-chlorophenyl) ethane
5. Nicotox	3-(1 Methyl-2-pyrrolidyl) pyridine
6. Stam 34	DDVP

The developmental period and percentage of emergence of adult flies were recorded. The results are summarized in Table II. Data obtained from these dosage mortality studies show that within a certain dose range, which varied from one pesticide to another, percentage mortality increased at different rates. It was also observed that development of D. melanogaster larvae was affected. In general, low concentrations did not affect the developmental period, but at increasing non-lethal concentrations of pesticides development was delayed. This is comparable to observations of Ramel & Magnusson (1969) for organic mercury compounds.

The approximate LD 50 concentrations of pesticides obtained from the above studies were then used to treat 1st instar larvae which were subsequently used in genetic tests. The genetic tests carried out were the Muller-5 technique for sex linked lethal mutations and the induced crossing over test in males. Oregon-K (wild type) larvae were treated for the former Test, while + + + +/dp b cn bw larvae were treated for the latter test. Table III summarizes the data obtained from these tests.

The sex linked lethal frequencies for the pesticides tested are almost at the spontaneous

level (0.52%). Nicotox, however, showed a sex linked lethal frequency between 1-2%. But due to the fact that only three lethals were detected out of 249 chromosomes, it is not possible to infer that this pesticide is definitely mutagenic. With regard to induced crossing over, however, almost every single pesticide has produced crossing over in the 1st three day brood. Approximately 1500 chromosomes were scored in these experiments. This would indicate that the pesticides do have some effect on the genetic material.

The fact that no sex linked lethal mutations were detected for any of the pesticides that were tested, it is almost certain that gross genetic damage is not produced by these pesticides. The induction of crossing over in males does indicate, however, that the pesticides do have some effects on the chromosomes of Drosophila. If the induced cross overs which are produced are due to single strand breaks on the DNA (Ratnayake 1970), it is possible that in the event that mutations are produced by these pesticides, they may occur as mis-sense mutations. Hence the sex linked lethal test is perhaps unable to detect such mutations. In order to detect such mis-sense mutations it would be advisable to use the specific locus method. We propose to carry out such tests in the future.

As cancers may arise due to somatic crossing over, a possible carcinogenic role of pesticides is indicated by the fact that they produce crossing over in the larval testes.

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References: Auerbach, C. 1943, DIS 17:48-50; Benes, V. and R. Sram 1969, Industrial Med. Vol. 38 12:50-52; Epstein, S. and M.S. Legator 1971, The Mutagenicity of Pesticides, MIT press; Ramel, C. and J. Magnusson 1969, Hereditas 61:231-254; Ratnayake, W.E. 1970, Mut. Res. 9:71-83; Sobels, F.H. 1973, Asilomar Conference on Environmental Mutagens.

Table II. Dosage/mortality of pesticides tested.

Pesticide	% Concentration	% Mortality	Remarks
Fenbar	0.00004	32.0	-
	0.00006	53.4	-
	0.00008	80.0	-
	0.00009	90.0	development delayed 1-3 days
	0.00010	92.3	" " " "
Gamalin	0.001	20.5	-
	0.002	83.5	development delayed 1-2 days
	0.003	100.0	" " " "
Nicotox	0.002	10.0	-
	0.004	14.7	-
	0.008	18.0	-
	0.010	75.0	development delayed 3 days
	0.012	90.0	" " " "
Endrin	0.0001	10.7	-
	0.0002	45.7	-
	0.0003	65.7	-
	0.0004	75.0	development delayed 3-4 days
	0.0005	89.4	" " " "
Deenol	0.0010	18.0	-
	0.0015	23.0	-
	0.0020	29.0	-
	0.0025	53.0	50% of emergence delayed 1 day
	0.0050	100.0	" " " " " "
Stam 34	0.035	33.7	emergence delayed 2-4 days
	0.0435	86.7	" " " "
	0.0875	100.0	" " " "

Table III. Results of Sex-linked lethal and induced crossing over experiments.

	Sex linked lethal test					Induced crossing over test		
Pesticide	% concentration	% emergence	No X chromosomes tested	No of lethals	% lethals	Cross over types	No of cross overs	Total scored
Fenbar 1	0.00008	69.6	474	1	0.21	1 bw 1 cn bw	2	2047
2	0.00008	58.3	-	-	-	2 dp b cn	2	1409
3	0.00008	63.	-	-	-	1 bw 1 cn bw 1 dp b 1 b cn bw	4	2306
Endrin 1	0.002	59.3	230	1	0.43			
2	0.003	64.0	-	-	-	-	-	-
3	0.004	61.0	-	-	-	1 dp b	1	1285
Gamalin 1	0.001	60.0	405	2	0.50	4 bw 1 dp b cn	5	2246
2	0.001	70.0	-	-	-	2 bw	2	1668
Deenol 1	0.0025	49.0	314	-	-	1 bw 1 dp b bw	2	2061
Nicotox 1	0.008	72.6	249	3	1.20	-	-	2081
Stam 34 1	0.035	52.0	240	2	0.83	-	-	1650
Control		98.0	381	2	0.52	-	-	4963

Postlethwait, J.H. University of Oregon, Eugene. Molting of Drosophila first instar larval cuticle induced by a metamorphosing host.

At metamorphosis third instar larval tissues degenerate. To find whether larval tissues of the first instar larva molt or degenerate during metamorphosis, the anterior thirds of first instar larvae were transplanted into ready to pupate hosts. As figure 1A shows, the implanted first instar larval cells did not degenerate; rather they molted to produce second or third instar mouth hooks (see Table 1). In contrast, anterior thirds of first instar larvae when implanted into adult females only occasionally molted to form second instar mouth hooks and never molted twice to form third instar mouth hooks (Figure 1B and Table 1). Thus mouth hook molting in a metamorphosing host is due at least in part to the host's endocrine organs since more molts occur in a metamorphosing host than in a non-metamorphosing host.

To ascertain whether the implant's ring gland was responsible for sparing the epidermal cells from degeneration or whether this property was inherent in the cells themselves, first instar mouth hooks without brains or ring glands were implanted into metamorphosing or non-metamorphosing (adult) hosts. The results (Table 1) show that mouth hooks without first instar endocrine organs can molt repeatedly in a metamorphosing host. In one case (Figure 1C) we even observed a total of four pairs of mouth hooks which represents one more molt than occurs in situ. This may have been accomplished in a manner similar to the supernumerary molts found by Žďarek and Slama (1), who injected ecdysone into last stage fly larvae shortly after their molt. In the case reported here, the rapid succession of high ecdysone titers during metamorphosis may have elicited three molts from the first instar epidermis. Only fifteen percent of control mouth hooks implanted into adults molted once. These studies show that the ability to molt rather than degenerate is a property of the first instar epidermis rather than the immediate effect of the first instar brain-ring gland complex upon these cells.

The competence of the imaginal discs of the first instar larva to respond to the metamorphosing host by differentiation can also be studied in these experiments. In only seven percent of the transplants of anterior thirds of first instar larvae into mature third instar larval hosts was there any indication of differentiation of adult parts. In one case there