A PRELIMINARY STUDY OF THE GENETICS OF DDT RESISTANCE IN HOUSEFLIES

By D. A. MAELZER* and R. L. KIRK*

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Summary

A detailed study is reported of the knockdown responses of a highly resistant "Illinois" strain of houseflies and of a non-resistant "Canberra" strain to topical applications of DDT and to exposure to a residual film of DDT. The dosage response curve for the Illinois strain is highly asymmetric, no increase in mortality being observed above a dose of 4 μg. DDT per fly. It is suggested that the resistant population is genetically heterogeneous, being composed of "weak" and "strong" individuals with respect to resistance to DDT.

Eight crosses between Canberra and "weak" Illinois flies were analysed using knockdown response to a dose of 8 μg. DDT per fly as a criterion of resistance. Although the pattern of response was highly variable there was little difference between F_1 and F_2 generations, the F_2 being only slightly more variable than the F_1. No flies in the F_2 fell into the "strong" category.

Five crosses between Canberra and "strong" Illinois flies were analysed similarly. There was a clear-cut segregation in the F_1 and F_2 generations into "strong" and "weak" individuals. The segregation suggests that high resistance in the Illinois strain is controlled by a dominant gene superimposed on the partially resistant constitution of the "weak" type.

The development of DDT resistance in a number of laboratory and field resistant strains of house flies is reviewed briefly and an attempt made to interpret these in the light of the present experiments.

I. INTRODUCTION

Since the observation by Sacca (1947) that field populations of the housefly, *Musca domestica* L., had become resistant to DDT and the subsequent demonstration by Lindquist and Wilson (1948) that a DDT-resistant strain could be selected in the laboratory, a large number of papers have appeared on the resistance of field and laboratory populations of houseflies to DDT and other insecticides. Recently two groups of workers have discussed the possible genetic mechanisms which control the resistance of houseflies to DDT. Bruce and Decker (1950*a*, 1950*b*) studied the resistance of offspring resulting from crosses between a field resistant (DDT 111) and a susceptible laboratory strain. The F_1, F_2, and F_{15} generations are reported to have LD50 values intermediate between those of the two parental strains and reciprocal crosses gave identical results. Bruce and Decker suggested that resistance might be described as a multiple gene character which causes indifferent physiological and, perhaps,

* Department of Zoology, University of Western Australia, Nedlands, W.A.

morphological changes. Using a different criterion for the assessment of resistance, Harrison (1951) concluded that DDT resistance is controlled by a single pair of allelomorphs. Individual flies from a resistant strain of Italian origin (Torre in Pietra) were crossed with susceptible flies, the progeny being tested by exposure to a DDT residue in glass cylinders. Knock-down time of each individual fly was noted and the population divided into resistant and susceptible flies on this basis. Results indicated that the F_1 had a resistance similar to, though not identical with, that of the non-resistant strain. In the F_2 generation 75 per cent. of the flies resembled the non-resistant strain.

The discrepant results in these two investigations were obtained on different strains of flies, and using different methods of assessing resistance; further, the published figures do not enable one to assess the importance of all the factors involved in such crosses. In an attempt to clarify the effect of some of these factors we have carried out a more intensive study of crosses between a highly resistant and a non-resistant strain of houseflies.

II. Materials and Methods

The resistant strain used in these experiments was obtained from the Multi-X stock (Bruce and Decker 1951) and was kindly supplied to us by Dr. Bruce of the Illinois Natural History Survey. Stock cultures of these flies have been maintained in our laboratories since October 1951 without further exposure to DDT. Non-resistant flies were derived from a laboratory strain maintained since 1940 by the Division of Entomology, C.S.I.R.O., Canberra.

Flies were reared at 28°C. with constant illumination. Larvae were bred in a slightly modified Peet-Grady medium (West 1951) and the adults allowed to emerge into wire frame cages covered with cloth and plastic. The adults were fed on a mixture of 50 per cent. milk and 5 per cent. sucrose soaked into cotton wool. Handling of the flies was facilitated by immobilizing them with CO_2.

Single-pair crosses were carried out in 4-oz. glass jars and virgin females were ensured by separating pupae into individual specimen tubes and sorting the emerging flies into appropriate pairs.

In all cases the testing procedure followed was to sort the pupae into cages and test at a standard age of 5-8 days after emergence. The DDT used was a commercial sample of 80 per cent. purity. Testing was carried out either by topical applications to the dorsal surface of the thorax of 0.002 ml. of DDT in acetone delivered from an Agla micro-injection syringe or by exposure to a DDT residue of approximately 10 mg./sq. ft. in 1000-ml. beakers. After testing by topical application flies were transferred for observation to clean bottles containing food. The same conditions of temperature and light used for breeding were maintained throughout. When knockdown time was being recorded the following criterion was used:

Flies were considered to be knocked down only when they showed a permanent loss of equilibrium. Frequently, this occurs a considerable time after the flies are first knocked down (Yeager and Munson 1949).

TABLE 1

NO. OF CANBERRA FLIES DEAD 24 HR. AFTER RECEIVING A MEASURED DOSE OF DDT

DDT (μg. per fly)	0·032		0·048		0·064		0·08		0·096		0·112		0·128		0·16		0·192	
	No. Used	No. Dead	No. Used	No. Dead	No. Used	No. Dead	No. Used	No. Dead	No. Used	No. Dead	No. Used	No. Dead	No. Used	No. Dead	No. Used	No. Dead	No. Used	No. Dead
Male	40	5	80	23	60	48	60	49	61	59	80	79	81	80	60	60	40	40
Female	40	5	80	2	60	3	63	9	60	11	78	38	80	42	60	50	40	37
Total	80	10	160	25	120	51	123	58	121	70	158	117	161	122	120	110	80	77

TABLE 2

NO. OF ILLINOIS FLIES DEAD 24 HR. AFTER RECEIVING A MEASURED DOSE OF DDT

DDT (μg. per fly)	0·16		0·4		0·8		1·2		1·6		4		8		16		32		64		96	
	No. Used	No. Dead	No. Used	No. Dead	No. Used	No. Dead	No. Used	No. Dead	No. Used	No. Dead	No. Used	No. Dead	No. Used	No. Dead	No. Used	No. Dead	No. Used	No. Dead	No. Used	No. Dead	No. Used	No. Dead
Male ..	42	5	41	16	43	20	41	21	40	21	39	17	37	20	40	16	44	24	39	24	29	11
Female ..	41	3	40	0	41	11	41	16	40	13	41	28	40	16	41	17	41	22	40	19	36	20
Total	83	8	81	16	84	31	82	37	80	34	80	45	77	36	81	33	85	46	79	43	65	31

III. Results

(*a*) *Dosage-mortality Curves*

In order to study the variability of response to applications of DDT in the two strains of flies at our disposal, initial dosage-mortality tests were carried out. The results of these tests are given in Tables 1 and 2. The log dosage-probit mortality curves for the two strains are plotted in Figure 1.

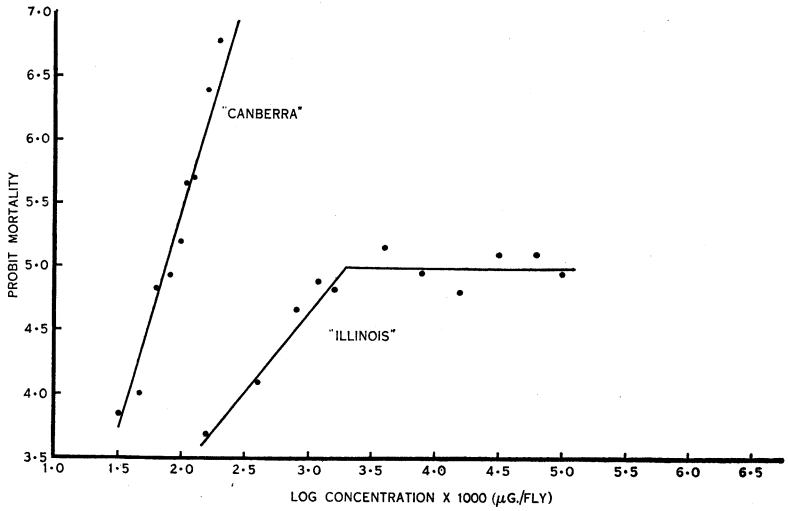

Fig. 1.—Dosage-mortality curves for two strains of houseflies.

While the Canberra flies gave a dosage-mortality response similar to other strains of DDT-susceptible flies, the resistant Illinois flies gave a linear log dosage-probit response relationship up to a concentration of DDT of 4 μg. per fly. At this level approximately 50 per cent. of the flies were alive after 24 hr. and continued to live normally after that. Further increases in the concentration of DDT up to a maximum of 96 μg. per fly resulted in no further increase in mortality.

Any explanation of the results of crosses between Illinois and Canberra flies must take into account the apparent lack of homogeneity in the Illinois population. A method of testing individual flies was developed, therefore, which would enable some discrimination to be made between flies resembling the Canberra stock in their resistance, and those resembling either "weak" Illinois flies, i.e. those unable to withstand a single dose of 4 μg. per fly, or "strong" Illinois flies, i.e. those able to survive a dose between 4 and 96 μg. per fly. It was found that a dose of 8 μg. DDT per fly gave the most clear-cut difference between these three possible types if knockdown time of the flies was recorded. Table 3 and Figure 2 present the knockdown times for Canberra and Illinois stocks.

The Canberra flies show a very rapid knockdown response; 50 per cent. of flies being knocked down after 1 hr., 100 per cent. after 2 hr. Knockdown response of the Illinois flies overlaps slightly with that for the Canberra stock,

for although no flies are knocked down in 1 hr., 10 per cent. come down in the second hour. The number of flies knocked down then rises steadily up to 6

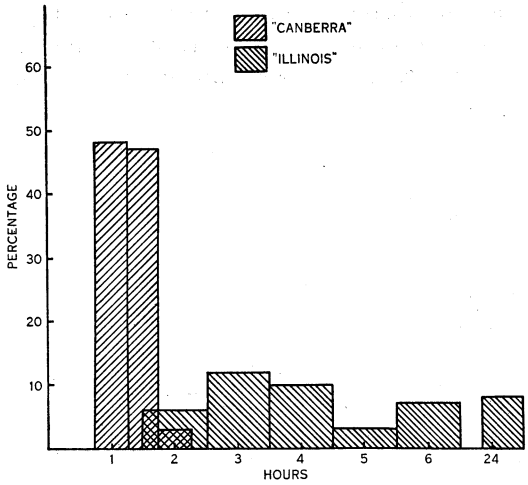

Fig. 2.—Frequency histograms for knockdown by DDT (8 μg. per fly).

TABLE 3

KNOCKDOWN TIMES FOR FLIES RECEIVING A TOPICAL APPLICATION OF 8 μG. DDT IN 0.002 ML. ACETONE

		No. Knocked Down After									
Canberra Strain	No. Used	30 Min.	45 Min.	60 Min.	90 Min.	120 Min.	3 Hr.	4 Hr.	5 Hr.	6 Hr.	24 Hr.
Male	17	10	10	10	17	17					
Female ..	14	—	—	5	13	14					
Total	31	10	10	15	30	31	A further 80 flies were tested and observed only after 120 min. They gave 100 per cent. knockdown				
Illinois Strain											
Male	37	—	—	—	—	3	9	17	18	18	20
Female ..	40	—	—	—	—	2	5	5	6	13	16
Total	77	—	—	—	—	5	14	22	24	31	36*

* A further 82 flies were tested and observed only after 120 min. They gave 14 per cent. knockdown.

hr., when 40 per cent. of the flies are involved. Subsequently little further increase occurs, 53 per cent. of flies remaining alive at the end of 24 hr.

A somewhat similar knockdown response is obtained if the flies are exposed to a DDT residue of 10 mg./sq. ft. in 1000-ml. beakers (not more than 10 flies in each beaker) (Table 4). Canberra flies are all knocked down at 180 min., only 14 per cent. of Illinois flies being knocked down in the same period of time. The knockdown of Illinois flies increases to 49 per cent. at the end of 7½ hr., and remains at this level up to 10 hr. At this stage there is still no mortality among the control flies, but thereafter mortality rises in both control and experimental series. This is due, almost certainly, to starvation, since the flies in these tests, unlike those in the individual dosage response tests, were not fed during the 24-hr. test period.

The knockdown pattern, both in response to individual doses and to exposure to a DDT residue, is in agreement with the information from the dosage-mortality curves. Canberra flies are not only killed by a lower dose of DDT than are the Illinois flies, but at a single dose level corresponding to a point on the "plateau" of the Illinois dose response curve, are knocked down very much more quickly. The knockdown pattern for Illinois flies suggests, as does the dosage-mortality curve, that this stock is heterogeneous for factors influencing resistance, approximately 50 per cent. of the stock having resistance characteristics at a "high" level.

(*b*) *Crosses between Canberra and "Weak" Illinois Flies*

A large number of reciprocal crosses between single Canberra and Illinois flies were set up and the flies allowed to lay their eggs. The eggs were removed and the parents tested for resistance with a single topical application of 8 μg. DDT per fly. Illinois parents that survived less than 6 hr. were classed as "strong." None of the parents was knocked down between 6 and 24 hr.

Eight crosses between "weak" Illinois and Canberra flies were successfully carried through to an F_2 generation, and tests completed on samples of the F_1 and F_2 populations. The results are recorded in Table 5.

One point emerges clearly as a result of these crosses, in contrast to those in which a strong Illinois parent is involved. In the F_1 generations no flies survived more than 6 hr., but in the F_2 generations a small proportion (1.8 per cent.) survived more than 6 hr., but none of these six flies lived for 24 hr.

A more detailed examination of the data in Table 5 reveals that the knockdown pattern for F_1 and F_2 flies is in no case identical with either that for Canberra flies alone or for "weak" Illinois flies alone. Approximately 80 per cent. of the F_1 and F_2 flies were knocked down at the end of 2 hr. and a further 15 per cent. in the succeeding 2 hr. But the figures reveal considerable variation from cross to cross for the knockdown pattern in the first and second hour, both among F_1 and F_2 flies.

This variability in response over a time period critical for discriminating typically "Canberra" knockdown responses from "Illinois" responses suggests that no simple genetic factor is involved whose effects are clear-cut. It seems likely that both genetic and environmental factors contribute to the observed differences in knockdown pattern of F_1 and F_2 flies during the first and second hours. Until further work enables an assessment to be made of the relative

TABLE 4

KNOCKDOWN TIMES FOR FLIES EXPOSED IN 1000-ML. BEAKERS TO A DDT RESIDUE OF 10 MG./SQ. FT.

Canberra Strain	No. Used	No. Knocked Down After																
		60 Min.	75 Min.	90 Min.	120 Min.	150 Min.	180 Min.	210 Min.	240 Min.	270 Min.	300 Min.	330 Min.	360 Min.	390 Min.	420 Min.	450 Min.	600 Min.	24 Hr.
Male	31	13	25	27	—	30	31											
Female	22	5	8	15	—	17	22											
Total	53	18	33	42	—	47	53	* A further 120 flies were tested and observed only at 150 min. They gave 100 per cent. knockdown.										
Knockdown (%)		34	62	79	—	88	100											
Illinois Strain																		
Male	22	—	—	2	3	5	8	10	12	12	12	12	12	12	12	12	12	12
Female	35	—	—	—	—	—	—	—	—	1	5	6	10	12	15	16	17	24
Total	57	—	—	2	3	5	8	10	12	13	17	18	22	24	27	28	29	36 *
Knockdown (%)		—	—	4	5	9	14	18	21	23	30	32	39	42	47	49	51	63

* Control flies in 1000-ml. beakers gave nil mortality after 600 min.

importance of these genetic and environmental factors little significance should be attached to the calculation of variance for the F_1 and F_2 populations.

(c) *Crosses between Canberra and "Strong" Illinois Flies*

The picture emerging from crosses between Canberra flies and Illinois flies which survive an application of 8μg. per fly is entirely different from that for the "weak" crosses discussed above.

Five such crosses were carried through to completion, and the results of mortality tests carried out on samples of the F_1 and F_2 generations are given in Table 6. Each fly was given a dose of DDT of 8μg. and the mortality after 24 hr. recorded.

The results fall into two distinct groups on the basis of the reaction of the F_1 generation. Crosses 9 and 10 show no mortality among the F_1 offspring, whereas crosses 11, 12, and 13 show more non-resistant than resistant flies in the F_1, the mortality at the end of 24 hr. being 64 per cent. A χ^2 test applied to the data in Table 6 reveals that crosses 9 and 10 are significantly different from 11, 12, and 13. Furthermore, crosses 11, 12, and 13 also differ significantly from one another in the F_2 generation. The difference between these two groups at first suggests that a single dominant gene exercises a major influence in determining survival to the test dose applied. If this is the case the Illinois parents entering crosses 9 and 10 would be homozygous, while those entering crosses 11, 12, and 13 would be heterozygous for this gene. On this basis we should expect 75 per cent. of the F_2 population in crosses 9 and 10 to be resistant individuals. On the basis of random mating among the observed population in the F_1 of crosses 11, 12, and 13, we should expect 33 per cent. resistant individuals in the F_2 generations. It will be seen that the fit to this hypothesis is reasonably good for crosses 9 and 10. But it must be noted that, in the three crosses 11, 12, and 13 where resistant and non-resistant flies are found together in the F_1 and F_2 generation, the number of non-resistant flies is above expectation in every case. This deviation is significant.

It is possible to explain the discrepancy if the dominant gene involved in these crosses also has the effect of reducing the fertility of the flies which carry it. This may indeed be the case. We have made a number of attempts to establish cultures of Illinois flies from crosses between pairs of "strong" flies. Such crosses have always yielded few adults in the first generation, and none of these stocks has been successfully maintained for more than two or three generations.

Other factors, however, are almost certainly involved. One can be illustrated from cross No. 13. The knockdown pattern for the Illinois parent was one displayed by a small percentage of flies in this stock. After receiving the standard dose of 8μg. DDT the fly was recorded in the knockdown stage after 3 hr. At 6 hr. the fly had recovered, and at 24 hr. was still alive. This "strong" fly gave the most discrepant results on the single dominant gene hypothesis for high resistance. But an examination of the knockdown pattern for flies in the F_1 and F_2 generation from this cross (Table 7) shows that a small number of

TABLE 5

KNOCKDOWN FREQUENCIES. FLIES FROM CANBERRA × "WEAK" ILLINOIS CROSSES

Each fly received a topical application of 8 μg. DDT

Cross No.	Illinois Parent	Time of Knockdown of Illinois Parent (hr.)	No. Tested		1 Hr.		2 Hr.		3 Hr.		4 Hr.		5 Hr.		6 Hr.		6-24 Hr.	
			♂	♀	♂	♀	♂	♀	♂	♀	♂	♀	♂	♀	♂	♀	♂	♀
F_1																		
1	♀	2-3	20	23	13	6	7	10	—	3	—	4	—	—	—	—	—	—
2	♀	3-4	21	20	6	6	14	12	1	2	—	—	—	—	—	—	—	—
3	♂	1-2	20	20	—	—	14	6	6	2	—	7	—	2	—	3	—	—
4	♂	2-3	15	15	—	—	15	15	—	—	—	—	—	—	—	—	—	—
5	♀	5-6	19	21	14	10	5	4	—	3	—	1	—	3	—	—	—	—
6	♀	5-6	23	20	9	5	14	4	—	11	—	—	—	—	—	—	—	—
7	♀	5-6	21	18	9	5	12	8	—	—	—	5	—	—	—	—	—	—
8	♀	5-6	41	43	28	17	13	9	—	11	—	6	—	—	—	—	—	—
Total F_1			180	180	79	49	94	68	7	32	—	23	—	5	—	3	—	—
F_2																		
1	♀	2-3	21	21	5	2	16	2	—	6	—	1	—	4	—	1	—	5
2	♀	3-4	20	21	7	2	4	5	9	6	—	6	—	1	—	1	—	—
3	♂	1-2	20	20	6	1	9	5	5	7	—	—	—	3	—	3	—	1
4	♂	2-3	20	20	15	18	3	2	1	—	—	—	1	—	—	—	—	—
5	♀	5-6	20	20	—	—	20	16	—	2	—	2	—	—	—	—	—	—
6	♀	5-6	20	21	—	—	20	20	—	1	—	—	—	—	—	—	—	—
7	♀	5-6	41	40	—	—	41	33	—	7	—	—	—	—	—	—	—	—
8	♀	5-6	21	20	—	—	21	16	—	2	—	2	—	—	—	—	—	—
Total F_2			183	183	33	23	134	99	15	31	—	11	1	8	—	5	—	6

flies in both generations manifest the same phenomenon as the Illinois parent, i.e. recovery after an initial knockdown. None of the flies in any of the other 12 crosses reported here showed this behaviour.

It seems likely that the Illinois parent in cross 13 was one which, though perhaps carrying the "strong" resistant gene, carried other factors which led to early knockdown. The fly would fall into an intermediate category between "weak" and "strong," and in consequence might be expected to yield a larger number of non-resistant flies among its offspring than flies which fall clearly into the "strong" category.

The knockdown pattern for the susceptible flies in crosses 9-13 (Table 7) resembles that for flies in crosses 1-8 (Table 5). Again there is considerable variation in the percentage knockdown in the first and second hour, but in no case is the pattern typical of either Canberra or Illinois flies but lies between these two extremes. This suggests that, whatever the nature of the factors determining the high resistance manifest by the "strong" Illinois flies, these factors are superimposed on a genetic constitution similar to that of the flies placed in the "weak" category.

TABLE 6

NO. OF FLIES ALIVE AND DEAD 24 HR. AFTER RECEIVING A DOSE OF 8 G. PER FLY DDT. CANBERRA × "STRONG" ILLINOIS CROSSES

Cross No.	F_1			F_2		
	No. Used	No. Dead	No. Alive	No. Used	No. Dead	No. Alive
9	38	0	38	39	11	28
10	41	0	41	42	13	29
11	40	24	16	244	162	82
12	24	13	11	165	131	34
13	30	23	7	83	70	13

IV. DISCUSSION

A consideration of the effect on DDT resistance of the crosses recorded above suggests that at least two types of genetic constitution are found among individuals in the resistant population. One type, when crossed with a non-resistant individual, gives rise to offspring with an intermediate resistance and these in turn produce an F_2 generation little different from the F_1. The resistance of such flies seems to be multifactorial. Flies of this type succumb to dosages varying over a range from 0.1 to 4.0 μg. DDT per fly. Part of this variation is due to differences in body weight, for which no allowance has been made in these experiments. Part may be due to uncontrolled environmental factors, of which food may be one of some importance. But it would seem plausible that some at least of the variation is due to different combinations of a group of genes controlling physiological or morphological characteristics of value in resisting the action of DDT.

TABLE 7

KNOCKDOWN FREQUENCIES. FLIES FROM CANBERRA × "STRONG" ILLINOIS CROSSES

Each fly received a topical application of 8 μg. DDT

Cross No.	Illinois Parent	No. Tested		No. Knocked Down After													
				1 Hr.		2 Hr.		3 Hr.		4 Hr.		5 Hr.		6 Hr.		24 Hr.	
		♂	♀	♂	♀	♂	♀	♂	♀	♂	♀	♂	♀	♂	♀	♂	♀
F_1																	
9	♂	20	18	—	—	—	—	—	—	—	—	—	—	—	—	—	—
10	♀	21	20	—	—	—	—	—	—	—	—	—	—	—	—	—	—
11	♀	18	22	7	—	1	9	—	4	—	2	—	—	—	—	1	—
12	♀	13	11	—	—	7	4	—	2	—	—	—	—	—	—	—	—
13*	♀	15	15	—	—	13	11	2	2	—	—	(7)	—	—	—	—	2
F_2																	
9	♂	22	20	6	2	—	2	1	2	—	—	—	—	—	—	—	—
10	♀	19	20	2	—	3	1	1	3	—	1	—	—	—	—	—	—
11	♀	123	121	69	54	8	10	6	10	—	1	—	1	—	1	—	2
12	♀	86	79	—	—	66	52	3	4	—	—	1	3	—	—	2	—
13*	♀	40	43	31	38	2	—	(2)	1	—	(3)	2	—	—	(2)	1	2

* The Illinois parent in cross No. 13 was knocked down at 3 hr. but subsequently recovered and was alive at the end of 24 hr. Several flies in the F_1 and F_2 from this cross exhibited the same phenomenon. The figures for recovery are given in parentheses.

The second type of fly in the resistant population has a genetic constitution similar to that of flies of type 1 but possessing in addition a genetic factor which enables the individual to survive single applications of DDT from 4 μg. up to at least 96 μg. These flies, when crossed with non-resistant individuals, show a segregation in the F_2 generation which suggests that the genetic factor involved is a single dominant gene. But there is also some evidence that flies carrying this gene are less fertile than those possessing its allelomorph only.

The occurrence of two distinct genetic types of flies among the resistant strain analysed here emphasizes the danger inherent in basing discussions of resistance on LD50 values. Complete information on the dosage response pattern of the population studied should be supplied if a picture of the mechanism by which resistance is built up in field and laboratory strains is to be obtained. On the basis of what little information is available an attempt can be made to outline a general mechanism for the acquisition of resistance in strains of *Musca domestica.* Two intensively studied laboratory strains have been selected from non-resistant stocks by exposure over successive generations to DDT. The Orlando strain used in studies by Lindquist and Wilson (1948), Wilson and Gahan (1948), King and Gahan (1949), King (1950), Pratt and Babers (1950), and Lindquist *et al.* (1951) increased in resistance only slightly up to the 22nd generation. However, between the 22nd and 55th generation a 50-fold increase in resistance occurred, and the population was much more variable than before. The resistance does not seem to have increased much since the 55th generation, but King and Gahan (1949) reported that some flies in the population could not be killed by very high concentrations of DDT. King (1950) reported a decline in resistance of the Orlando strain when selection pressure was removed at the 53rd generation, and Lindquist *et al.* (1951) also demonstrated a decline in resistance at the 85-88th generation following removal of selection pressure at the 75th generation.

The DDT 1 strain selected by Bruce and Decker (1950*a*) from an NAIDM laboratory strain showed a somewhat similar development. There was little increase in resistance up to the twelfth generation but after that a very rapid increase was recorded for the next five generations. Bruce and Decker (1950*b*) did not observe any decline in resistance over 34 generations when the selection pressure was removed.

Two partially resistant field strains DDT 111 (Bruce and Decker 1950*a*) and Torre in Pietra (Harrison 1952) have also been subjected to selection for DDT resistance in the laboratory. Both these strains showed only the rapid phase of development of resistance. In the Torre in Pietra strain selection for eight generations yielded a population with characteristics similar to those reported here for the strain derived from Multi-X, i.e. a highly asymmetric dosage-mortality curve. Roma, another Italian strain partly resistant to DDT, has been subjected to selection by Harrison under conditions identical with those for Torre in Pietra. After eight generations no significant increase in resistance took place.

Further studies on selection in the laboratory of partially resistant field strains have been reported from California. Perry and Hoskins (1950) were

able to increase the resistance of the Laton strain five times at the LD50 value by only one generation of selection, and this Super Laton stock showed an increased variance. A Bellflower field strain from California had LD50 values higher than either Laton or Super Laton, and was more variable than either of these two strains.

This analysis indicates that most field populations of houseflies must be polymorphic with respect to factors controlling resistance to an insecticide like DDT and the evolution of a DDT-resistant strain must follow a pattern rather like the following:

The first stage is a relatively slow process involving the accumulation of a number of independent factors each of which, singly or in combination, confers resistance on the fly. Following this slow increase is a rapid rise in resistance of the population, owing to selection of a highly resistant phenotype controlled probably by a single pair of alleles. This highly resistant population can never be rendered homogeneous because of the low fertility associated with the high resistance factor. It seems likely that the high resistance factor cannot be selected initially because the selective advantage this factor tends to confer is less than its selective disadvantage owing to the lower fertility associated with it.

V. Acknowledgments

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VI. References

Bruce, W. N., and Decker, G. C. (1950*a*).—*Soap Sanit. Chem.* **26**: 122-5; 145-7.
Bruce, W. N., and Decker, G. C. (1950*b*).—*Pest Control* **18** (4): 9-16.
Bruce, W. N., and Decker, G. C. (1951).—*Pest Control* **19** (4): 9-11.
Harrison, M. (1951).—*Nature* **167**: 855-6.
Harrison, M. (1952).—*Bull. Ent. Res.* **42**: 761-8.
King, W. V. (1950).—*J. Econ. Ent.* **43**: 527-32.
King, W. V., and Gahan, J. B. (1949).—*J. Econ. Ent.* **42**: 405-9.
Lindquist, A. W., and Wilson, H. G. (1948).—*Science* **107**: 276.
Lindquist, A. W., *et al.* (1951).—*J. Econ. Ent.* **44**: 931.
Perry, A. S., and Hoskins, W. M. (1950).—*J. Econ. Ent.* **43**: 839-50.
Pratt, J. J., and Babers, F. H. (1950).—*Science* **112**: 141-2.
Sacca, G. (1947).—*Riv. Parassit.* **8**: 127-8.
West, L. S. (1951).—"The Housefly." pp. 1-584. (Constable: London.)
Wilson, H. G., and Gahan, J. B. (1948).—*Science* **107**: 276-7.
Yeager, J. F., and Munson, S. A. (1949).—*J. Econ. Ent.* **42**: 874-7.