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A comparative genetical study on DDT resistance in adults and larvae of the mosquito *Aedes aegypti* L.

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1. INTRODUCTION

Studies by Brown & Abedi (1962) and Klassen & Brown (1964) have shown that DDT resistance in larvae of *Aedes aegypti* is inherited as an incompletely dominant gene (designated *DDT* or R^{DDT}) on linkage group II although inheritance is complicated by modifiers (Wood, 1965). Coker (1966) reports that a gene on linkage group II confers resistance in adults although the linkage relationships of this *R* gene appear to be different from the larval gene.

Larval resistance involves an increase in dehydrochlorination of DDT to harmless DDE by the enzyme DDT-dehydrochlorinase (Chattoraj & Brown, 1960; Abedi *et al.*, 1963; Kimura & Brown, 1964). The biochemical basis of adult resistance is not known.

In most studies the scoring of susceptible (+/+), hybrid (R^{DDT}/+) and resistant (R^{DDT}/R^{DDT}) phenotypes in segregation has been subject to experimental error because of the difficulty in finding 'discriminating concentrations' of DDT to separate phenotypes with complete certainty. Only after intensive selection with a DDT substitute insecticide, WARF (N,N-dibutyl-*p*-chlorobenzenesulphonamide) which raised DDT resistance to a high level, was a discriminating concentration of DDT found which killed all susceptible individuals but no F_1 hybrids. But in this study resistance in adults no longer behaved as a single gene (Pillai & Brown, 1965).

By breeding from the most DDT-resistant F_2 progenies of single-pair matings in the TRINIDAD (DDT-resistant) strain, Wood (unpublished results) has produced a strain 'T' with a higher level of DDT resistance than has been attained by straightforward selection (larval L.C.50 > 100 p.p.m, 24 hours exposure). Crosses with a susceptible strain (Wood, 1967) showed less than 3% overlap between *R*/+ and +/+ phenotypes and less than 4% between *R*/+ and *R*/*R*. Resistance behaved as a single gene in reciprocal F_2's and backcrosses to susceptible (+/+) but there was evidence of modifiers influencing the expression of the heterozygote in backcrosses to the resistant (*R*/*R*) parent, resistance in the heteroyzgote being significantly higher than expected.

2. MATERIALS AND METHODS

Two strains have been employed: the DDT-resistant T strain (see above) and a susceptible strain (64) marked on all three linkage groups. Strain 64 is homozygous for one recessive larval marker *y* (yellow) on linkage group II, one pupal and early

adult marker *re* (red eye) on linkage group I and two adult markers, *s* (silver spots on lateral abdomen reduced or absent), on linkage group II and *blt* (black tarsus) on linkage group III.

Stocks were reared at 27 ± 0·5°C. and 70 ± 5 % relative humidity. Eggs, laid on filter paper, were hatched in hay infusion. The larval diet was supplemented with powdered dog-biscuit from the second day after hatching and from the fifth day pupae were separated into clean water for emergence in population cages. Adult males were fed on sugar, females on mice anaesthetized with nembutal.

DDT tests were performed at 70 ± 5 % relative humidity, 25 ± 1°C. air temperature or 23 ± 2°C. water temperature, by the methods recommended for adult and larval mosquitoes by World Health Organization (1963).

Single-pair crosses were made between 64 and T, nine pair matings for each reciprocal cross. F_1 (T × 64) and F_1 (64 × T) progenies were tested with DDT at larval and adult stages. Backcrosses to strain 64 were made from a single T × 64 progeny and a single 64 × T progeny, chosen at random. Twenty such backcrosses were made, divided equally between the four possible combinations: F_1 (T × 64) ♀ × 64 ♂, 64 ♀ × F_1 (T × 64) ♂, F_1 (64 × T) ♀ × 64 ♂, 64 ♀ × F_1 (64 × T) ♂. Backcross progenies were tested with DDT, at larval and adult stages.

Evidence provided below indicates that larval and adult resistance derive from different loci, which will be designated R^{DDT1} and R^{DDT2} respectively.

3. DDT TESTS ON F_1 LARVAE AND ADULTS IN COMPARISON WITH STRAIN 64

The results of DDT tests on strain 64 and F_1 hybrids (T × 64 and 64 × T) are given in Table 1. Five hours' exposure to 0·5 p.p.m. DDT separated hybrid and susceptible larvae with less than 3 % overlap. Two and a half hours' exposure to

Table 1. *Mortality in strain 64 and in F_1 progenies from crosses between 64 and T, tested with 0·5 p.p.m. DDT for 5 hours at the larval stage, or with 2 % DDT for 2½ hours as adults (females)*

	Larvae			Adults (♀♀)		
Strain or F_1	Numbers tested	Numbers dead	% mortality ± S.E.	Numbers tested	Numbers dead	% mortality ± S.E.
64	250	250	100	102	102	100
F_1 (T♀ × 64♂)	250	1	0·4 ± 0·4	—	—	—
F_1 (64♀ × T♂)	150	10	7·5 ± 2·1	117	1	0·8 ± 0·8

2 % DDT separated hybrid and susceptible adults (females) with less than 1·0 % overlap. In terms of 95 % confidence limits the potential overlap is no more than 4·4 % in larvae and 2·4 % in adults. A suitable concentration of DDT to separate hybrid and susceptible male adults could not be found.

A significant difference in larval tolerance to 0·5 p.p.m. DDT occurred between the reciprocal crosses ($P < 0·01$) although no consistent differences were found between individual F_1 progenies, within each reciprocal cross.

4. DDT TESTS ON BACKCROSS LARVAE

Backcross progenies, having been reared to the fourth larval stage, were divided into two fractions one of which was tested with DDT at once, as early fourth-stage larvae, the other kept until the adult stage when females were tested 2–8 days after emergence.

Resistance segregated in the backcross in coupling with y^+ and s^+. Details of larval resistance are given in Table 2, part A. Linkage between R^{DDT1}, y and s on linkage group II is confirmed. Crossover frequencies in male and female heterozygotes did not differ.

Mortality in these larval tests was unexpectedly low ($37{\cdot}9 \pm 1{\cdot}4\,\%$) compared with the pilot tests (Wood, 1967), indicating a significant depletion of +/+ (homozygous susceptible) segregants. In recombination of R^{DDT1} with y, there was seen to be a depletion in the parental fraction $+y/+y$ and in the recombinant fraction ++/++; and the segregation of $+:y$ was also distorted ($\chi^2 = 9{\cdot}45$, $P < 0{\cdot}01$). Because of the associated influence upon the $+:y$ ratio, the distortion R^{DDT1}:+ is most probably due to differential mortality of the + (DDT-susceptible) phenotype prior to testing (rather than to incomplete penetrance of 'susceptibility'). On this assumption the recombination fraction $R^{DDT1} - y$ is $12{\cdot}3 \pm 0{\cdot}9\,\%$; no correction is required (Bailey, 1961). On the alternative but less likely assumption of differential mortality of both R^{DDT1} and y alleles, the recalculated recombination fraction $R^{DDT1} - y = 11{\cdot}6 \pm 1{\cdot}7\,\%$.

Backcross survivors of larval tests ($R^{DDT1}/+$) were reared to pupae and scored for *re* (linkage group I) and then to adults to be scored for *s* (linkage group II) and *blt* (linkage group III). A small count indicated that linkage group I has no influence on larval DDT resistance (Table 2, part A). The segregation of *re* with sex (Mm = ♂, mm = ♀) showed a recombination value of $re - M = 2{\cdot}3 \pm 0{\cdot}9\,\%$ (Table 2, part B). The inequality of male and female numbers results from the presence in the T strain of 'distorter' (D) the dominant allele closely linked with sex (M) which produces an excess of males (Hickey & Craig, 1966).

By the adult stage mortality following (larval) DDT exposure had risen to $49{\cdot}8 \pm 2{\cdot}2\,\%$. This indicates a post-treatment mortality of $11{\cdot}9/62{\cdot}1 \times 100 = 19{\cdot}2\,\%$ of the larval survivors. The larval survivors are all R^{DDT1+} because the larval discriminating dose is known to kill 100 % +/+ (64) larvae. The recombination fraction $R^{DDT1} - s = 3{\cdot}0 \pm 0{\cdot}7$. The recombination fraction $y - s = 7{\cdot}9 \pm 0{\cdot}6$ (Table 2, part B). We have seen that recombination $R^{DDT1} - y = 12{\cdot}3 \pm 0{\cdot}9\,\%$. The order of these three loci on linkage group II is therefore $R^{DDT1} - s - y$.

Recombination between R^{DDT1} and *blt* on linkage group III was $39{\cdot}5 \pm 2{\cdot}1\,\%$ (Table 2, part A) suggesting an influence of linkage group III on larval resistance (see discussion). The reduced recombination $R^{DDT1} - blt$ was mirrored by an apparent linkage between *s* and *blt* ($41 \pm 2{\cdot}1\,\%$ recombination) (Table 2, part A) although in adults derived from larvae unexposed to DDT, *s* and *blt* assorted independently ($47{\cdot}9 \pm 1{\cdot}1\,\%$ recombination) as did *y* and *blt* (Table 2, part B).

Table 2. *Test of linkage relationships of* R^{DDT1} *(larval resistance)*

Mating $64 \times (T \times 64)$: $\frac{ys}{ys}\ \frac{blt}{blt}\ \frac{re}{re}\ \frac{+}{+} \times \frac{ys}{++}\ \frac{blt}{+}\ \frac{re}{+}\ \frac{+}{R^1}$

A: Larvae exposed to DDT to score R^1 and + phenotypes.
B: No DDT treatment, to score other markers.

A or B	Genotype of heterozygous parent	Phenotypes of progeny				Total tested	% kill ± S.E.	Genes tested	% Recombination ± S.E.
A	$\frac{+}{R^1}\ \frac{y}{+}$	$\frac{+\,y}{415}$	$\frac{++}{44}$	$\frac{R_1\,y}{105}$	$\frac{R_1\,+}{547}$	1211	37·9 ± 1·4	R^1-y	12·3 ± 0·9
A	$\frac{+}{R^1}\ \frac{s}{+}\ \frac{blt}{+}$	$\frac{R^1\,s\,blt}{4}$	$\frac{R^1\,s\,+}{12}$	$\frac{R^1\,+\,blt}{207}$	$\frac{R^1\,+\,+}{311}$	1064	49·8 ± 2·2	R^1-s R^1-blt $s-blt$	3·0 ± 0·7 39·5 ± 2·1 41·0 ± 2·1
		$\frac{+\,s\,blt}{}$	$\frac{+\,s\,+}{}$	$\frac{+\,+\,blt}{}$	$\frac{+\,+\,+}{}$				
		Could not be separated							
A	$\frac{+}{R^1}\ \frac{re}{+}$	$\frac{+\,re}{}$	$\frac{++}{}$	$\frac{R^1\,re}{51}$	$\frac{R^1\,+}{52}$	200	48·5 ± 4·9	R^1-re	49·5 ± 5·0
		Could not be separated							
B	$\frac{y}{+}\ \frac{s}{+}\ \frac{blt}{+}$	$\frac{y\,s\,blt}{373}$	$\frac{y\,s\,+}{375}$	$\frac{y\,+\,blt}{50}$	$\frac{y\,+\,+}{73}$	2044	—	$y-s$ $y-blt$ $s-blt$	7·9 ± 0·6 49·3 ± 1·1 47·9 ± 1·1
		$\frac{+\,s\,blt}{22}$	$\frac{+\,s\,+}{17}$	$\frac{+\,+\,blt}{537}$	$\frac{+\,+\,+}{597}$				
		Parental	Parental	Recombinant	Recombinant				
B	$\frac{M\ re}{m\ +}$ or $\frac{m\ +}{M\ re}$	♀♀ 61	♂♂ 241	♀♀ 2	♀♀ 5	309	—	$re-M$	2·3 ± 0·9

R^{DDT1} is abbreviated to R^1.

Table 3. *Test of linkage relationships of* R^{DDT2} *(adult resistance)*

Mating $64 \times (T \times 64)$: $\frac{ys}{ys}\ \frac{blt}{blt}\ \frac{+}{+} \times \frac{ys}{++}\ \frac{blt}{+}\ \frac{+}{R^2}$

A: With no larval exposure to DDT.
B: After larvae exposed to DDT to eliminate +/+ segregants at R^1 locus.

A or B	Genotype of heterozygous parent	Phenotypes of progeny				Total tested	% kill ± S.E.	Genes tested	% Recombination ± S.E.
A	$\frac{+}{R^2}\ \frac{y}{+}\ \frac{s}{+}$	$\frac{R^2\ y\ s}{70}$	$\frac{R^2\ +\ s}{7}$	$\frac{R^2\ y\ +}{9}$	$\frac{R^2\ +\ +}{195}$	583	55·2 ± 2·1	$y-s$	4·6 ± 0·0
		$\frac{+\ y\ s}{120}$	$\frac{+\ +\ s}{4}$	$\frac{+\ y\ +}{7}$	$\frac{+\ +\ +}{191}$			R^2-s	47·2 ± 2·1
								R^2-y	46·8 ± 2·1
A	$\frac{+}{R^2}\ \frac{blt}{+}$	$\frac{+\ blt}{250}$	$\frac{+\ +}{72}$	$\frac{R^2\ blt}{34}$	$\frac{R^2\ +}{227}$	583	55·2 ± 2·1	R^2-blt	18·2 ± 1·6
B	$\frac{+}{R^2}\ \frac{blt}{+}\ \frac{s}{+}$	$\frac{R^2\ s\ blt}{0}$	$\frac{R^2\ s\ +}{0}$	$\frac{R^2\ +\ blt}{7}$	$\frac{R^2\ +\ +}{53}$	129	54·3 ± 4·4	$blt-s$	37·2 ± 4·3
		$\frac{+\ s\ blt}{0}$	$\frac{+\ s\ +}{0}$	$\frac{+\ +\ blt}{41}$	$\frac{+\ +\ +}{28}$			R^2-s	53·5 ± 4·4
								R^2-blt	27·1 ± 3·9

R^{DDT2} is abbreviated to R^2.

5. DDT TESTS ON BACKCROSS ADULTS

Adults from larvae not exposed to DDT at the larval stage were subjected to a concentration of DDT designed to discriminate $R^{DDT2}/+$ and $+/+$ phenotypes. Adult resistance was closely associated with linkage group III (Table 3, part A). The recombination frequency $R^{DDT2} - blt = 18{\cdot}2 \pm 1{\cdot}6$. But, unlike larvae, adult resistance was not associated with linkage group II. The recombination frequencies are $R^{DDT2} - s = 47{\cdot}2 \pm 2{\cdot}1$ and $R^{DDT2} - y = 46{\cdot}8 \pm 2{\cdot}1$ (Table 3, part A).

A second series of DDT tests was made on adults derived from the *survivors* of larval DDT tests, i.e. stock which were entirely composed of $R^{DDT1}/+$ individuals. Mortality in these adults, $54{\cdot}26 \pm 4{\cdot}4\%$ (Table 3, B), was not significantly less than in adults unselected with DDT at the larval stage, $55{\cdot}23 \pm 2{\cdot}1\%$. Thus the larval R^{DDT1} factor on linkage group II confers no resistance in adults. Moreover, if linkage group III has some influence on larval resistance (see Discussion) this particular effect of linkage group III does not appear to influence adult resistance.

Adult mortality in the main series of backcrosses was $55{\cdot}23 \pm 2{\cdot}1\%$. This value deviates slightly but significantly from the expectation of 50% ($\chi^2 = 6{\cdot}38$, $P < 0{\cdot}02$). This is accounted for by the fact that 2·5 hours' exposure to 2% DDT was not always the most satisfactory discriminating concentration. This was established when DDT tests on strain 64 were carried out in parallel with the backcross tests. Then it was evident that whereas on some occasions a full exposure of 2·5 hours was required to kill 100% of strain 64, the same result could be achieved with exposures as short as 1·75 hours on other occasions. Therefore, in later tests, backcross mortality was scored as soon as mortality in strain 64 was complete. When results from such tests alone are considered (Table 4)* the backcross mortality is $50{\cdot}0 \pm 3{\cdot}0\%$ (unselected larvae), agreeing with expectation. In such tests, the crossover

Table 4. *Test for linkage between* blt *and* R^2 (*adult resistance*)

$$\text{Mating: } 64 \times (T \times 64): \frac{blt\ +}{blt\ +} \times \frac{+\ R^2}{blt\ +}$$

Pretreatment with DDT	Phenotypes of Progeny: + *blt*	++	R^2 *blt*	R^2 +	Total	% kill	% Recombination
None	112	23	22	113	270	$50{\cdot}0 \pm 3{\cdot}0$	$16{\cdot}7 \pm 2{\cdot}3$
Larvae	19	21	5	25	70	$57{\cdot}1 \pm 5{\cdot}9$	$37{\cdot}1 \pm 5{\cdot}8$

Adults were exposed to 2% DDT for 105–150 min. to score R^2 and + phenotypes. Larval pretreatment consisted of 5 hours exposure to 0·5 p.p.m. DDT to eliminate +/+ segregants at the R^1 locus. R^2 indicates the R^{DDT2} locus for adult resistance.

frequency $R^{DDT2} - blt = 16{\cdot}7 \pm 2{\cdot}3\%$ which does not differ significantly from the value $18{\cdot}2 \pm 1{\cdot}6\%$ calculated from the total data. Mortality in adults derived from selected larvae (Table 4B) also agreed with expectation. However, selection had significantly raised the recombination value $R^{DDT2} - blt$ to $37{\cdot}1 \pm 5{\cdot}8$, perhaps

* They are also included in the data presented in Table 3.

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because there was a relative excess of +/+ among the larval survivors (as also appeared to be the case in Table 3).

6. CROSSOVER FREQUENCIES IN MALE AND FEMALE HETEROZYGOTES

Klassen & Brown (1964) have compared the percentage recombination in linkage group II from male and female heterozygotes. Crossing-over in the male was no less than in the female for short distances but for longer distances (15–20 units) crossing-over in the male was slightly but significantly lower. Crossover values for male and female heterozygotes in the present study are compared in Table 5. They are not significantly different.

Table 5. *Crossing-over in male and female heterozygotes* $(64 \times T)$ *and* $(T \times 64)$

	♂ Heterozygote			♀ Heterozygote		
Genes tested	Parentals	Recombinants	% Recombination ± S.E.	Parentals	Recombinants	% Recombination ± S.E.
$R^{DDT1}-y$	454	59	11·5 ± 1·4	608	90	12·9 ± 1·3
$R^{DDT1}-s$	204	8	3·8 ± 1·3	314	8	2·5 ± 0·9
$y-s$	411	30	6·8 ± 1·2	1471	132	8·2 ± 0·7
$R^{DDT2}-blt$	73	11	13·1 ± 3·7	404	95	19·0 ± 1·8

7. DISCUSSION

The experiments confirm that a gene R^{DDT1} on linkage group II exerts a major influence on DDT resistance in larvae of the T strain of *A. aegypti*. Adult DDT resistance, however, is due to a different gene R^{DDT2} on linkage group III. This conflicts with studies of Brown & Abedi (1962), Klassen & Brown (1964) and Coker (1966) who found DDT resistance to be associated solely with linkage group II in both larvae and adults, except when DDT resistance resulted from selection with the DDT substitute insecticide WARF (Pillai & Brown, 1965). Then, in adults, linkage groups II and III contributed equally to DDT resistance.

The disparity between the present and previous studies finds no immediate explanation. It may result from the very high level of resistance in the T strain. But differences also occurred in genetic procedure (single-pair mating *versus* mass mating) and in testing procedure (time in one concentration *versus* a range of concentrations for standard time).

Recombination between larval resistance and the adult marker *blt* on linkage group III was unexpectedly low at 39·5 ± 2·1 %, suggesting an influence of linkage group III on larval resistance. However, there were two potential sources of error in the method of estimation: (*a*) Only *survivors* of larval DDT tests, R^{DDT1} *blt*/+ *blt* and R^{DDT1} +/+ *blt*, could be scored; + *blt*/+ *blt* and + *blt*/++ could not be separately identified. (*b*) Post-treatment mortality (between fourth-stage larvae and adults) was 19·2 % compared with 8 % in untreated stocks (Wood, 1961, 1962). Because of

potential inaccuracy intrinsic to the method of estimation, the apparent linkage could be an artefact. This conclusion is supported by the identity of adult resistance in backcrosses, whether selected at the larval stage to remove +/+ segregants or not (Table 3). Certainly the linkage group III effect on larval resistance, if it exists, has no obvious connexion with the major gene R^{DDT2} conferring resistance in adults.

From two-point test data the linkage relationship of R^{DDT1}, y and s suggests the order on chromosome II of $R^{DDT1}-s-y$

$$\underset{R^{DDT1}}{\quad}\overset{3{\cdot}0\pm0{\cdot}7}{s}\qquad\overset{7{\cdot}9\pm0{\cdot}6}{y}$$
$$\underset{\quad}{\quad}\overset{12{\cdot}3\pm0{\cdot}9}{\quad}\underset{\quad}{\quad}$$

This is supported by a modest three-point test (150 larvae) which gave the same order.

$$\underset{R^{DDT1}}{\quad}\overset{1{\cdot}5+1{\cdot}5}{s}\qquad\overset{4{\cdot}4\pm2{\cdot}5}{y}$$
$$\underset{\quad}{\quad}\overset{5{\cdot}9\pm2{\cdot}9}{\quad}\underset{\quad}{\quad}$$

The order $R^{DDT1}-s-y$ agrees with Coker (1966) for *adult* resistance but not with the study on larval resistance of Klassen & Brown (1964) who found the order to be $R^{DDT}-y-s$. Recombination values $R^{DDT1}-y$ and $R^{DDT1}-s$ are lower in the present study than in previous ones, although the value $s-y$ is slightly higher. Because it is possible to score only survivors of larval tests the linkage distance $R^{DDT1}-s$ is approximate (probably an underestimate).

The TRINIDAD resistant strain has been obtained by repeated larval selection for DDT resistance. There is, therefore, the possibility that it contains more than one larval resistance gene. Modifiers of resistance are known (Wood, 1965, 1967). Moreover it is possible that the present resistance gene R^{DDT1} may be different from the gene DDT of Klassen & Brown (1964) because of its different linkage relationships. Nevertheless the explanation which most nearly fits the present results is a single gene of major effect.

F_1 (T × 64) showed a significantly greater larval resistance than F_1 (64 × T), thereby modifying the expression of $R^{DDT1}/+$. Such a difference was not evident in earlier mass crosses between T and the susceptible strain Q (Wood, 1967) but it has been observed in mass crosses of their parent stocks TRINIDAD × QUEENSLANDENSIS (Wood, 1965). The four possible combinations in backcrosses to strain 64 did not differ significantly.

In backcrosses to strain 64, where $R^{DDT1}/+$ and +/+ phenotypes were expected to segregate 1:1, the +/+ phenotype was found in only 38% of larvae. Because of the influence of this distortion on a second locus y, it is thought to have been due to differential mortality of +/+ eggs and/or larvae. Mortality is normally 10–15% between the egg and fourth larval stage (Wood, 1961). If this is assumed to have been the level of mortality in the $R^{DDT1}/+$ phenotype, the +/+ phenotype must have suffered a mortality of 40–50%. In the absence of direct measurements of egg and larval mortality this figure is provisional. But it implies a superiority of the hybrid ($R^{DDT1}/+$) phenotype which has not been observed before.

The lower survival of +/+ rather than $R^{DDT1}/+$ is at first sight surprising since it is not usual to expect a selected mutation to increase viability. The most likely explanation is perhaps that strain 64, which is strongly inbred, has in consequence a low viability because of its homozygosity, so that the heterozygote with R^{DDT1} gives some heterosis.

19·2% of the survivors of larval tests ($R^{DDT1}/+$) did not survive to the adult stage, more than double the mortality in untreated larvae and pupae. 'Post-treatment mortality' with DDT has been observed before in the TRINIDAD strain (Wood, unpublished results) although delayed insecticidal action is more characteristic of dieldrin and related compounds.

The adult resistance gene, R^{DDT2} is 18·2 ± 2·1 units from *blt* on linkage group III. Since this gene is independent of the linkage group II gene R^{DDT1} it is interesting to speculate how it has arisen. Resistance in TRINIDAD was detected in a natural population after 1–2 years of perifocal spraying with DDT, directed mainly at the larval stage. Larval resistance was fairly high, adult resistance significantly higher than normal, although low by comparison with larval resistance. Since colonization of this population in 1956, larval selection has been imposed on a succession of occasions as the strain has passed through various laboratories. Larval resistance has increased to a very high level. Adult resistance has also increased although (so far as the author is aware) adults have never been directly exposed to DDT. Could it be that the chromosome III gene has been selected as a modifier of larval resistance but acts as a major gene in adults? This is considered to be the most likely hypothesis on present evidence.

SUMMARY

Inheritance of DDT resistance has been studied in crosses between the highly resistant 'T' strain of *A. aegypti* (constituted by inbreeding from the TRINIDAD DDT-resistant stock) and the '64' susceptible strain.

Larval DDT resistance derives from a major gene R^{DDT1} on linkage group II, the order being $R^{DDT1} - s - y$. Linkage group III may also contribute to larval resistance. Linkage group I makes no contribution.

Adult DDT resistance derives from a major gene R^{DDT2}, 18·2 ± 2·1 units from the market *blt* on linkage group III. Linkage group II has no influence on adult resistance.

Selection with DDT to retain only $R^{DDT1}/+$ segregants in larvae of backcrosses $R^{DDT1}/+ \times +/+$ did not increase resistance in resulting adults, confirming the difference in genetic mechanism at the two stages.

The F_1 progenies from reciprocal crosses between 'T' and '64' differed slightly but significantly in larval resistance, modifying the influence of the major gene R^{DDT1} in the heterozygote.

The early developmental stages of the $R^{DDT1}/+$ phenotype (up to the fourth larval stage) were more viable than the +/+ phenotype in backcross segregation. The difference in mortality probably exceeded 30%.

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REFERENCES

Abedi, Z. H., Duffy, J. R. & Brown, A. W. A. (1963). Dehydrochlorination and DDT resistance in *Aedes aegypti*. *J. Econ. Ent.* **56**, 511–517.

Bailey, N. T. J. (1961). *Mathematical Theory of Genetic Linkage*. London: Oxford University Press.

Brown, A. W. A. & Abedi, Z. H. (1962). Genetics of DDT-resistance in several strains of *Aedes aegypti*. *Can. J. Genet. Cytol.* **4**, 319–332.

Chattoraj, A. N. & Brown, A. W. A. (1960). Internal DDE production by normal and DDT-resistant larvae of *Aedes aegypti*. *J. econ. Ent.* **53**, 1049–1051.

Coker, W. Z. (1966). Linkage of the DDT-resistance gene in some strains of *Aedes aegypti* (L.). *Ann. trop. Med. Parasit.* **50**, 347–356.

Hickey, W. A. & Craig, G. B., Jr. (1966). Genetic distortion of sex ratio in a mosquito, *Aedes aegypti*. *Genetics*, **53**, 1177–1196.

Kimura, T. & Brown, A. W. A. (1964). DDT-dehydrochlorinase in *Aedes aegypti*. *J. econ. Ent.* **57**, 710–716.

Klassen, W. & Brown, A. W. A. (1964). Genetics of insecticide resistance and several visible mutants in *Aedes aegypti*. *Can. J. Genet. Cytol.* **6**, 61–73.

Pillai, M. K. K. & Brown, A. W. A. (1965). Physiological and genetical studies on resistance to DDT substitutes in *Aedes aegypti*. *J. econ. Ent.* **58**, 255–266.

Wood, R. J. (1961). Biological and genetical studies on sex ratio in DDT-resistant and susceptible strains of *Aedes aegypti* (L.). *Genet. agr.* **13**, 287–307.

Wood, R. J. (1962). DDT-resistance and larval development in *Aedes aegypti* (Linnaeus). The length of larval development and adult resistance in the Trinidad strain. *Ann. trop. Med. Parasit.* **56**, 450–456.

Wood, R. J. (1965). A genetical study on DDT-resistance in the Trinidad strain of *Aedes aegypti* (L.). *Bull. Wld Hlth Org.* **32**, 563–574.

Wood, R. J. (1967). The use of discriminating concentrations in genetical studies on DDT-resistance in larval *Aedes aegypti* (L.). *Bull. Wld Hlth Org.* (in press).

World Health Organization (1963). Insecticide resistance and vector control: 13th Rep. Expert Comm. Insecticides. *Tech. Rep. Ser. Wld Hlth Org.* **265**, 41–56.