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The influence of the *y* locus on DDT resistance in the mosquito *Aedes aegypti* L.

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SUMMARY

The influence of the linkage group II locus *y* on DDT resistance in *Aedes aegypti* has been studied in crosses between the Trinidad resistant strain and QS susceptible strain. The *y* locus influences DDT resistance in both R/R and $R/+$ larvae. The effect of *y* may be interpreted as reducing the penetrance of *R* (R^{DDT1}), which is also located on linkage group II. y^+ is partially dominant and incompletely penetrant in its resistance-enhancing role (although in its pleiotropic effect on larval colour it is dominant and fully penetrant). Penetrance of y^+ is influenced by an environmental factor, probably associated with the larval diet.

The effect of *y* on resistance is evaluated in relation to other genetic influences on the expression of R^{DDT1}.

The significance of polymorphism at the *y* locus is discussed.

1. INTRODUCTION

Larvae of the mosquito *Aedes aegypti* derive their DDT resistance mainly from a gene, R^{DDT1}, on linkage group II (Brown & Abedi, 1962; Klassen & Brown, 1964; Wood, 1967*b*). This gene shows no dominance in the progenies of mass crosses, but in single pair progenies it varies in dominance (Wood, 1965). The locus *y* on linkage group II, which influences larval colour (Craig & Gillham, 1959), also affects resistance to DDT (Wood, 1965, 1968). The y^+ allele, causing a grey-brown coloration due to crystals of uric acid in the fat body (Wigglesworth, 1942), is associated with a higher level of resistance than is found in yellow (y/y) larvae, in which these crystals are absent. Consequently larvae of genetic constitution $R+/+y$ or $R+/++$ are more tolerant to DDT than those of constitution $Ry/+y$; and $R+/R-$ larvae are more tolerant than Ry/Ry.

The present study looks more closely at the influence of *y* and of other components of the genetic background on the expression of R^{DDT1}.

2. MATERIALS AND METHODS

Two laboratory populations have been used: (1) a DDT-susceptible strain (QS), never exposed to insecticides, which is homozygous for *y*; (2) a highly DDT-resistant Trinidad strain which is polymorphic for *y* (frequency of y/y = 5–12 %, Wood, 1962). The investigation was divided into two parts.

1. Reciprocal single pair crosses were made between the QS and TRINIDAD populations to investigate differences in resistance between the progenies (F_1's) of single pairs, and between the segregants $R+/+y$ and $Ry/+y$, where these occurred in the same progeny (i.e. from matings $R+/Ry \times +y/+y$).

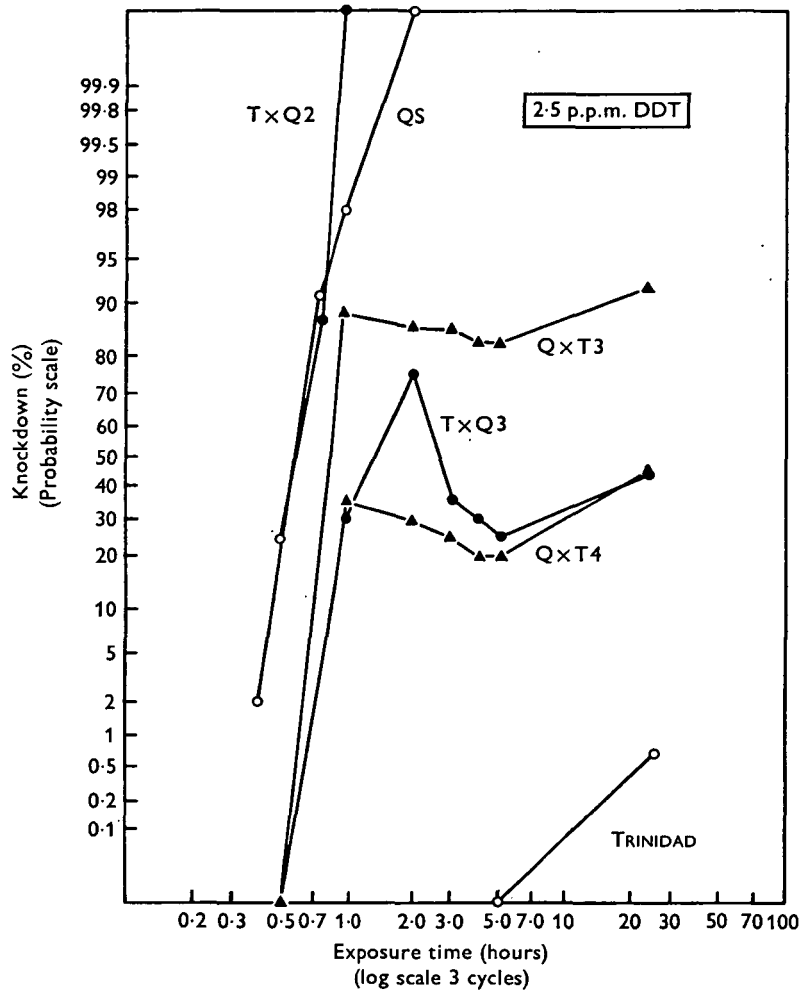

Fig. 1. Variation in response to 2·5 ppm DDT in F_1's from single pair crosses QS♀ × TRINIDAD ♂ (Q × T) and TRINIDAD ♀ × QS ♂ (T × Q) tested at the fourth larval stage.

2. One such F_1 progeny (QS♀ × TRINIDAD ♂ 6), in which resistance was manifested as a recessive, was backcrossed to the TRINIDAD parent to investigate the influence of y on the R/R homozygote. This has allowed a comparison to be made between Ry/Ry, $R+/Ry$, and $R+/R+$ genotypes as well as between $Ry/+y$, $R+/+y$, and $R+/++$.

Resistance was assessed under standard conditions, the larvae at early fourth stage being exposed to DDT in aqueous suspension at 23 ± 2 °C. The criterion of

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'mortality' was the inability of an individual to leave the floor of the test container and swim to the surface. This method of assessing mortality was found to be satisfactory at concentrations at 5 ppm DDT and above. At 2·5 ppm some larvae initially 'knocked down' would recover (Fig. 1). Further details of this method of testing are given by Wood (1967*a*, 1968).

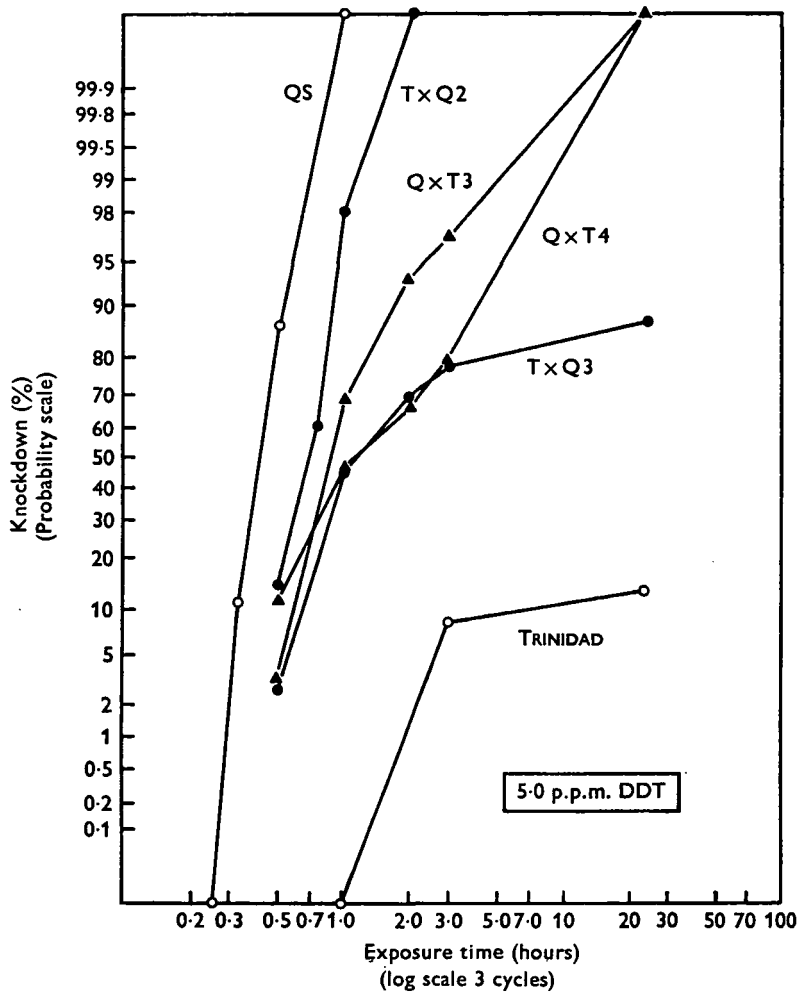

Fig. 2. Variation in response to 5·0 ppm DDT in F_1 progenies from single pair crosses QS ♀ × TRINIDAD ♂ (Q × T) and TRINIDAD ♀ × QS ♂ (T × Q) tested at the fourth larval stage.

3. SINGLE PAIR RECIPROCAL CROSSES BETWEEN QS AND TRINIDAD

F_1 progenies from nine pair crosses were tested at 2·5 and 5·0 ppm of DDT. Three F_1's showed approximately the same level of tolerance as the QS parent, i.e. resistance appeared recessive; the other six were intermediate between the parents; no F_1 resembled the TRINIDAD strain, i.e. in no progeny was resistance dominant.

The range of variation between F_1 progenies at 2·5 and 5·0 ppm DDT is represented in Figs. 1 and 2, compared with the parent strains. No account is taken in these figures of segregation at the y locus; each line represents total F_1 larvae. Larvae tested at 2·5 ppm were derived from the first egg batches; those tested at 5·0 ppm from the second egg batches.

Progenies showed the same order of resistance from one series of tests to the next, despite the fact that these were made on different days using larvae derived from different egg batches, sometimes exposed to different concentrations of DDT (compare Figs. 1 and 2). This supports previous evidence (Wood, 1965, 1968) of a genetic component to inter-progeny variation. No overall difference was observed between the reciprocal crosses.

Table 1. *Knockdown in QS♀ × Trinidad♂ and Trinidad♀ × QS♂ F_1 progenies derived from single pair matings*

(First batches were tested at 2·5 ppm DDT (5 h exposure) and are compared with second batches tested at 5·0 ppm DDT (1 h exposure). Tests at the two concentrations were made 1 week apart.)

	Batch			
	1		2	
Concn. of DDT (ppm)	2·5		5·0	
Exposure (h)	5		1	
Genotype	*Ry*/+*y*	*R*+/+*y*	*Ry*/+*y*	*R*+/+*y*
F_1 (QS × Trinidad) pair matings				
1	.	84·0 ± 5·2	.	.
2	.	.	96·0 ± 3·9	92·0 ± 5·4
3	93·3 ± 3·7	73·3 ± 6·6	75·6 ± 6·4	62·2 ± 7·2
4	.	20·0 ± 8·9	.	47·1 ± 8·6
5	100	42·7 ± 7·4	.	.
6	.	.	100	100
F_1 (Trinidad × QS) pair matings				
2	100	100	100	96·0 ± 3·9
3	20·0 ± 8·0	28·0 ± 9·0	66·7 ± 7·9	27·8 ± 8·5
5	.	72·0 ± 9·0	.	.

Six matings were $+y/+y \times R+/Ry$. Consequently six F_1 progenies were composed of $R+/+y$ and $Ry/+y$ larvae in approximately equal proportions, the DDT resistance of which could be compared. In three such progenies (Q × T2; Q × T6; T × Q2) the colour classes were equal in resistance (both very low); see Table 1. In Q × T5 (first test) and T × Q3 (second test) $R+/+y$ larvae were significantly more resistant than $Ry/+y$ ($P < 0{\cdot}02$). In no progeny were $Ry/+y$ larvae significantly superior in tolerance to $R+/+y$. This indicates that the resistance-enhancing effect of y^+ (or a closely linked locus) occurs in some single pair progenies but not in others.

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One pair (T × Q 3) produced four egg batches at weekly intervals. These were hatched and tested separately. The result of testing batch 1 at 2·5 ppm DDT and batches 2–4 at 5 ppm is shown in Table 2. Note that whereas in batches 2 and 3 a clear difference occurred between the colour classes (batch 2, $P < 0{\cdot}02$; batch 3, $P < 0{\cdot}05$), this difference was not found in the first and fourth batch ($P > 0{\cdot}5$). The values of y/y agree in batches 2, 3 and 4 ($P > 0{\cdot}1$) whereas the values of $y/+$ do not ($P < 0{\cdot}01$). We may conclude that in T × Q 3 batches 1 and 4, y^+ failed to operate as a modifier of resistance, i.e. the penetrance of y^+ is incomplete. This assumes that the batches represent random samples of this progeny. The batches were otherwise very similar.

Table 2. *Percentage knockdown in tests with DDT of larvae derived from egg batches 1–4 from pair cross* TRINIDAD♀ × *QS* ♂ *3, in which the two phenotypes,* $R+/+y$ *and* Ry/+y *are compared*

(Batch 1 was tested at 2·5 ppm DDT, batches 2–4 at 5·0 ppm DDT.)

Exposure	Batch 1		Batch 2		Batch 3		Batch 4	
(h)	$R+/+y$	$Ry/+y$	$R+/+y$	$Ry/+y$	$R+/+y$	$Ry/+y$	$R+/+y$	$Ry/+y$
0·5	0	0	0	5·6 ± 3·8	0	0	0	0
1·0	30·0 ± 9·2	30·0 ± 9·2	27·8 ± 7·5	66·7 ± 7·9	24·0 ± 8·5	64·0 ± 9·6	83·3 ± 7·5	80·0 ± 8·0
3·0	72·0 ± 8·7	80·0 ± 8·0	52·8 ± 8·3	86·1 ± 5·8	40·0 ± 9·8	80·0 ± 8·0	87·5 ± 6·6	92·0 ± 5·4
24·0	36·6 ± 9·6	48·0 ± 10·0	80·6 ± 6·6	94·4 ± 3·8	88·0 ± 6·5	100	100	96·0 ± 3·9
No. tested	25	25	36	36	25	25	25	25

Backcross (*QS* × *TRINIDAD 6*) × *TRINIDAD*

In F_1 Q × T 6 resistance was recessive in both $Ry/+y$ and $R+/+y$ classes (Table 1). Each colour class was backcrossed by single pair mating to the TRINIDAD resistant strain. Examination of backcross progenies indicated that the following matings had been made:

$$Ry/+y \times R+/Ry; \quad Ry/+y \times R+/R+; \quad R+/+y \times R+/R+.$$

(1) $Ry/+y \times R+/Ry \rightarrow R+/Ry : Ry/Ry : R+/+y : Ry/+y.$

Mortality (knockdown) at 5 ppm DDT was recorded up to 24 h (Table 3 (1)). When scored after 1 h, an exposure time which kills 100% $Ry/+y$ or $R+/+y$ (Table 1), the yellow phenotype ($Ry/Ry : Ry/+y$) and the grey-brown phenotype ($R+/Ry : R+/+y$) were significantly different ($P < 0{\cdot}001$).

(2) $Ry/+y \times R+/R+ \rightarrow R+/Ry : R+/+y.$

Mortality after exposure for 1 h to 5 ppm DDT (Table 3 (2)) did not differ significantly ($P > 0{\cdot}3$) from the mortality in the same genotypes in (1) (see above).

(3) $R+/+y \times R+/R+ \rightarrow R+/R+ : R+/+y$ ($R+/Ry : R+/++$).

Mortality after exposure to 5 ppm DDT for 1 h (Table 3 (3)) indicated that $R+/R+$ and the crossover recombinant $R+/++$ are considerably more tolerant to DDT ($P < 0{\cdot}00001$) than $R+/Ry$ or $R+/+y$.

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Table 3. *Percentage knockdown in tests with 5·0 ppm DDT on larvae of backcrosses* $(QS \times TRINIDAD\ 6) \times TRINIDAD$

(1) $Ry/+y \times R+/Ry$; (2) $Ry/+y \times R+/R+$; (3) $R+/+y \times R+/R+$. (The numbers tested are given in parentheses.)

Backcross	Genotypes in backcross progeny	Exposure (h) 0·5	1·0	2·0	3·0	5·0	24·0
(1) $Ry/+y \times R+/Ry$	$Ry/Ry : Ry/+y$	11·0 ± 3·3 (91)	90·1 ± 3·1 (91)	92·3 ± 3·0 (91)	91·2 ± 3·0 (91)	93·4 ± 2·6 (91)	96·0 ± 3·9 (25)
	$R+/Ry : R+/+y$	3·9 ± 1·7 (128)	63·3 ± 4·3 (128)	68·8 ± 4·1 (128)	71·1 ± 4·8 (90)	74·2 ± 3·9 (128)	70·0 ± 7·3 (40)
(2) $Ry/+y \times R+/R+$	$R+/Ry : R+/+y$	6·7 ± 2·9 (75)	54·7 ± 5·7 (75)	78·7 ± 4·7 (75)	72·0 ± 9·0 (25)	90·0 ± 4·2 (50)	88·0 ± 3·8 (75)
(3) $R+/+y \times R+/R+$	$R+/R+ : R+/+y$ $[R+/Ry : R+/++]$*	1·5 ± 0·6 (463)	16·0 ± 2·5 (413)	31·7 ± 2·5 (413)	—	45·4 ± 2·9 (293)	54·6 ± 2·3 (463)

* Crossovers.

Table 4. *Observed and calculated values for the percentage knockdown at 5 ppm DDT in the various genotypes derived from the backcross* $(QS \times TRINIDAD\ 6) \times TRINIDAD$ *compared with the parent strains*

Genotypes	Source of data	Exposure (h) 0·5	1·0	2·0	3·0	5·0	24
1. $Ry/+y$	Table 1	12	100	100	100	100	100
2. $Ry/Ry : Ry/+y$	Table 3(1)	11	90	92	91	93	96
3. Ry/Ry	Calculated	10	80	84	82	86	92
4. $R+/+y$	Table 1	0	100	100	100	100	100
5. $R+/Ry : R+/+y$	Table 3(1) and (2) combined	5	60	72	71	79	82
6. $R+/Ry$	Calculated	10	20	44	42	58	64
7. $R+/R+ : R+/+y$ $[R+/Ry : R+/++]$*	Table 3(3)	2	16	32	—	45	55
8. 6 % $R+/Ry$: 44 % $R+/+y$							
Contribution of $R+/Ry$	Calculated	1	1	3	3	3	4
Contribution of $R+/+y$	Calculated	0	44	44	44	44	44
Total for mixture	Calculated	1	45	47	47	47	48
9. 44 % $R+/R+$: 6 % $R+/++$	Calculated	1	−31	−15	—	−2	7
10. 12·5 % $R+/Ry$: 37·5 % $R+/+y$							
Contribution of $R+/Ry$	Calculated	1	3	6	5	7	8
Contribution of $R+/+y$	Calculated	0	38	38	38	38	38
Total for mixture	Calculated	1	41	44	43	45	46
11 37·5 % $R+/R+$: 12·5 % $R+/++$	Calculated	1	−24	−12	—	0	9
TRINIDAD strain (R/R, polymorphic for y)	Fig. 2	0	0	0	7	—	13
QS strain ($+y/+y$)	Fig. 2	85	100	100	100	100	100

* Crossovers in parentheses.

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The calculations which follow and which are summarized in Table 4 rest on the assumptions: (1) that R^{DDT1} is segregating as a major gene; (2) that R^{DDT1} continues to be recessive in the backcross, as it was in the F_1; (3) that the different genotypes are equally viable to non-insecticidal influences.

On the basis of the measured resistance of $Ry/+y$ and $(Ry/Ry : Ry/+y)$, the resistance of Ry/Ry has been calculated (Table 4 (1–3)).

On the basis of the measured resistance of $R+/+y$ and $(R+/Ry : R+/+y)$ the resistance of $R+/Ry$ has been calculated (using combined results of backcrosses (1) and (2) (Table 4: 4–6).

Using the measured resistance of $R+/+y$ (Table 4 (4)) and the calculated resistance of $R+/Ry$ (Table 4 (6)) it has been possible to calculate the resistance of mixtures of $R+/Ry$ and $R+/+y$ (Table 4 (8, 10)). On the basis of the measured resistance of $R+/R+ : R+/+y$ $(R+/Ry : R+/++)$, the genotypes in parentheses being the crossovers, it has been possible to calculate the resistance of mixtures of $R+/R+$ and $R+/++$ (Table 4 (9, 11)).

Assuming 12% recombination between R^{DDT1} and y (as observed by Wood, 1967*b*), values have been derived to represent the tolerance of a mixture of 44% $R+/R+$ and 6% $R+/++$. This is shown in Table 4 (9) together with a line for 37·5% $R+/R+$: 12·5% $R+/++$ (Table 4 (11)), taking into account the observation of Klassen & Brown (1964) of 25% crossing over between R^{DDT1} and y.

From the results of backcrossing Q × T 6 we may conclude that:

(1) y modifies resistance in the homozygote (R/R) (Table 4 (3, 6, 9, 11)) as well as the heterozygote $(R/+)$ (Table 4 (1, 4, 9, 11)).

(2) y is incompletely dominant in its modifying role on the homozygote (R/R), i.e. y/y, $y/+$ and $+/+$ depress resistance to different degrees (Table 4 (3, 6, 9, 11)). (N.B. Q × T 6 is exceptional compared with most other progenies (see above and Wood, 1965) in that y^+ is fully *recessive* in its effect on the heterozygote $(R/+)$ (Table 4: 1, 4).

(3) Resistance in $R+/++$ (no mortality after 5 h at 5 ppm DDT; Table 4 (9, 11) is *greater* than resistance in either $R+/Ry$ (58% mortality) (Table 4 (6)) or Ry/Ry (86% mortality) (Table 4 (3)), i.e. the genotypes in ascending order of resistance are: $(Ry/+y, R+/+y)$; Ry/Ry; $R+/Ry$; $(R+/++, R+/R+)$.

(4) The negative values in Table 4 (9, 11) derive from the fact that the $+/y$ genotypes in the backcross were more tolerant than expected. In particular the $R+/+y$ genotype was not fully susceptible; i.e. resistance, which was fully recessive in the F_1, did not remain so in the backcross. Resistance in $R+/R+$ and $R+/++$ has therefore been overestimated. $R+/Ry$ (Table 4 (6)) was also probably less tolerant than has been estimated.

(5) Mortality in Ry/Ry (Table 4 (3)) and $R+/Ry$ (Table 4 (6)) does not increase between 2 and 3 h. This could be an artifact but it could also indicate genetic heterogeneity, i.e. reflect a qualitative distinction between susceptible and resistant larvae. This is the period of the test during which such a distinction would be expected to show up, if at all (compare the pattern of knockdown in QS and Trinidad, Table 4). Since $R+/R+$ shows no mortality at 3 h we may tentatively

interpret the influence of y as reducing the penetrance of R^{DDT1}. On this interpretation the penetrance of R^{DDT1} is 16–18% in Ry/Ry and 56–58% in $R+/Ry$ (assuming penetrance to be complete in $R+/R+$). Calculated values of penetrance will be lower if resistance is not fully recessive. There is some evidence for this in $R+/Ry$ (see note 4 above).

4. DISCUSSION

Investigation of single pair crosses between the QS DDT-susceptible and TRINIDAD DDT-resistant strains and subsequent backcrosses has shown y to depress larval resistance in both the homozygote (R/R) and the heterozygote ($R/+$).

The effect of y on the resistance heterozygote has been shown to vary between pair crosses. Studies on the homozygote have been made on the progeny of one pair cross. Therefore it is possible that the influence of y on the homozygote is more variable than is indicated by these experiments.

The analysis of F_1 (Q × T 6) in backcrosses to TRINIDAD assumes that R remains recessive in the changed genetic background. If this is not so, then the values of resistance calculated for Ry/Ry, $R+/Ry$ and $R+/R+$ are overestimates. However, this should not affect the conclusion that the three genotypes differ in resistance.

Considering the penetrance of R in the homozygote, it appears that the effect of two y^+ alleles on resistance is approximately twice that of one y^+ allele. This may relate to an observation by Craig & Gillham (1959) that $+/+$ larvae appear darker in colour than $y/+$ larvae (although since they cannot be accurately separated on this basis, y^+ is technically dominant). Craig & Gillham (1959) suggested an additive effect of the alleles for uric acid production. The same may be postulated for their effect on DDT resistance, although the connexion between uric acid production and DDT resistance remains unknown. Brown & Abedi (1963) comment on the smaller size of y/y larvae in some strains but they did not establish whether these larvae were less tolerant to DDT.

Investigation of the four egg batches from the cross T × Q 3 indicates that y^+ itself varies in penetrance. Craig & Gillham (1959) point out that under conditions of 'heavy overcrowding or underfeeding' the penetrance of y^+, as it affects colour, may be reduced in the heterozygote ($y/+$), a proportion of which appear yellow; in consequence of which the segregation ratio is distorted. In the present experiments larvae were reared in small numbers under optimum conditions and the ratio of $y/y : y/+$ did not differ significantly from 1 : 1.

Thus while y^+ is almost completely dominant and normally 100% penetrant in its effect on larval colour (Craig & Gillham, 1959), it is partially dominant and incompletely penetrant in its role in modifying DDT resistance.

Penetrance of y^+ as it varies in F_1 T × Q 3 is environmentally dependent (assuming that batches represent random samples of progenies). We may suspect that the environmental influence on penetrance comes from some nutritional variant in the larval diet, this being less easily standardized than the conditions of testing and also likely to be closely involved with nitrogen metabolism.

Penetrance of y^+ may be assessed by considering all batches in which it segregated. Here we have the possibility of genetic as well as environmental influences. The proportion of batches showing a significant difference between $R+/+y$ and $Ry/+y$ larvae (at 2·5 ppm DDT after 5 h or 5·0 ppm after 1 h) was $4/12 = 0{\cdot}33$. There is some indirect evidence suggesting a genetic influence on the penetrance of y^+: in two experiments Wood (unpublished) has observed the modifying effect to disappear after inbreeding by brother–sister mating for one or two generations.

Batches from the same F_1 progeny tested on different days were closely similar (apart from variation in the penetrance of y^+). By contrast, individual progenies could be markedly different from one other. This constitutes further evidence (see also Wood, 1965, 1968) that R^{DDT1} is modified by differences in genetic background. The genetic influence deriving from segregation at the y locus may be considered as one component of this background, a substantial one in fact.

The dominance of resistance varied between progenies. However, in no progeny was R^{DDT1} fully dominant. Moreover a fully recessive heterozygote was found only in the presence of at least one y gene. These findings may be contrasted with those of Wood (1965), in which dominant resistance was observed in some progenies and recessive resistance occurred in the absence of y. However, the TRINIDAD population used in the former study was 100 times less resistant to DDT than that used in the present one. Thus the difference from the QS population was also 100 times less and the likelihood of some F_1 progenies overlapping with the parents by chance, correspondingly greater.

It could be argued that 'non-penetrance' of y in some progenies might be due to crossing-over, i.e. the modifier is not at the y locus but is linked to it. However, an hypothesis based on linkage must be reconciled with the following evidence.

1. That when larvae are taken from the TRINIDAD DDT-resistant strain, which is polymorphic for y, $R+/R-$ larvae are more tolerant than Ry/Ry, despite many generations of laboratory culture in the TRINIDAD strain, giving ample opportunity for crossing-over between y and the hypothetical modifier.

2. That when $R+/Ry$ larvae from the TRINIDAD strain are outcrossed to $+y/+y$, the phenotypes $R+/+y$ and $Ry/+y$ differ significantly in resistance.

To allow for linkage we must assume a specific mechanism maintaining y^+ and the modifier in coupling in the resistant (TRINIDAD) strain for which there is no evidence.

Klassen (1966) observed a higher resistance associated with y^+ and interpreted this in terms of linkage with a modifier (M). However, Klassen's data are also compatible with an interpretation based on pleiotropism. The weight of evidence would seem to be on the side of pleiotropism although the possibility that the modifier crosses over with y^+ cannot yet be ruled out.

Craig & Gillham (1959) demonstrated that selection with DDT to a high level of resistance did not change the frequency of y (0·25) in a naturally polymorphic population. Yet the modifying effect of y on resistance was the same before and after selection (Wood, 1965, 1968). The stability of y under DDT selection is surprising considering its influence on DDT resistance.

It is known that several factors are potentially able to influence the frequency of *y*: *y*/*y* eggs live longer under unfavourable conditions and are more successful at suboptimal temperatures (Adhami, 1963, 1964); *y*/*y* male adults are more active than +/+ males and show a superior ability to inseminate either *y*/*y*, *y*/+ or +/+ females (Adhami & Craig, 1965); *y*/*y* males mate more rapidly and are probably more likely to fill the female spermathecae. On the other hand, *y*/*y* larvae when given excess food, are less able to inhibit the formation of a bacterial scum on the surface of the medium, and die (Craig & Gillham, 1959; Wood, 1959), and the growth-rate of *y*/*y* is slower (Craig & Gillham, 1959); selection for slow growth-rate (Wood, 1962) in a polymorphic population increased the frequency of *y* from 0·34 to fixation in 12 generations.

These various observations do not explain how the *y* polymorphism is maintained. Nor do they indicate why DDT selection should have no apparent effect on it. However, they do suggest that the control of *y* polymorphism may be complex, with several factors interacting to produce a stable frequency. That this stability is maintained despite strong DDT selection suggests that the fact of carrying the *y* gene is more vital to the insect than high DDT resistance. But nevertheless there should be an immediate effect of the selection. The DDT should initially wipe out many *y*'s and the frequency of *y* should only climb later. The frequency of *y* has not been followed throughout the course of selection (only before and after).

In conclusion we may note that it has yet to be determined (1) by what mechanism *y* influences resistance; (2) whether this effect of *y* is found in other populations besides Trinidad; (3) whether *y* influences resistance in the adult mosquito as well as in the larva, the major DDT-resistance loci being different at the two developmental stages (Wood, 1967*b*).

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