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Inheritance of DDT resistance in species A and B of the *Anopheles gambiae* complex

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It has been reported that Anopheles gambiae *species A is resistant to DDT in Upper Volta and Togo, and DDT resistance has been found in a Sudan strain of species B. The species A strain from Upper Volta was more resistant to DDT than the Togo strain, while the Sudan strain of species B was the least resistant. The expression of resistance is genetically determined, and studies on the mode of inheritance have shown that resistance is inherited as a single dominant gene in the Togo strain of species A and the Sudan strain of species B, but that it is inherited as a single incompletely dominant gene in the Upper Volta strain of species A.*

Anopheles gambiae species A and B, the principal malaria vectors in Africa, have developed resistance to dieldrin and the closely related compound gamma-HCH in most of West Africa and parts of East Africa (Davidson, 1956; Busvine, 1969). DDT has replaced dieldrin and HCH in the areas where resistance to these insecticides has developed and in fact it is the only insecticide for general application that can be used at present to control malaria in the African continent. However, DDT resistance has now been detected in *A. gambiae* species A and B, having been reported in species A in Upper Volta (Hamon et al., 1968) and Togo, and in species B in the Sudan and Senegal. This poses a serious problem for future efforts to control malaria in Africa.

This report describes laboratory investigations made to determine the way in which DDT resistance is inherited in colonies originating from field resistant populations of mosquitos.

MATERIALS AND METHODS

Altogether, 3 strains of *A. gambiae* species A and 2 strains of species B were employed in the present study. They included 2 DDT-resistant strains of species A and 1 DDT-resistant strain of species B.

Species A

Togo. A DDT-resistant colony (Tog), originating from Hlande-Wogba in Togo, was colonized in May 1969 from eggs obtained from DDT-resistant females in the field. The strain was selected for several generations in the laboratory until it attained complete homozygosity for stable DDT resistance. The strain was then maintained without further selection.

Upper Volta. A DDT-resistant colony (UV) was initiated from eggs received from Bobo-Dioulasso in Upper Volta in February 1969. After repeated selection with DDT the strain remained homozygous for DDT resistance and it was maintained without further selection.

Nigeria. A susceptible colony (IBAD) originating from Ibadan was colonized in 1966.

In view of the relatively stable resistance levels in both the Togo and the Upper Volta strains in the absence of selection, their genetic purity must be of a fairly high order.

Species B

Sudan. A DDT-resistant strain (SUD) originated from eggs obtained in January 1970 from a field population showing DDT resistance in the Elgunied locality in Central Sudan. The colony was maintained in the insectary after repeated selection with DDT in both larval and adult stages.

Southern Yemen. A susceptible strain (MAK SS) originated from adults collected from Makhzan, Aden, in 1962.

Rearing

Stocks were reared in an insectary at a temperature of approximately 27°C and a relative humidity of

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about 75%. Females were fed twice weekly on an anaesthetized guineapig. The oviposition interval was 3 days under laboratory conditions. Eggs were kept for 48 h in small bowls lined with filter paper to hatch. Larvae were reared in groups of 100–200 at a temperature of approximately 27°C, and young larvae were provided with a piece of turf on which to feed. Second stage and older larvae were also given small amounts of a finely ground proprietary cereal product twice a day. Once pupation occurred the pupae were strained off and placed in cages. The time for development from egg to adult was approximately 10 days.

Crossing

Crosses between resistant and susceptible strains were obtained either by mass matings or induced copulation (Baker et al., 1962). Studies on the inheritance of DDT resistance were carried out with progeny from single families. Gravid females were placed individually in labelled 7.5 × 2.5-cm glass vials. The vials were then covered with netting and a small amount of water was introduced. Only one egg batch was taken from each female and the eggs were counted and left for 48 h to hatch. The number of first-stage larvae from each family was counted and batches showing a hatch rate of less than 70% were discarded. Larvae of single families were reared separately in bowls as described above. Rearing procedures were standardized as far as possible in order to obtain adults of a reasonable size for testing. Only families that produced a high yield of adults from first-instar larvae were tested and included in the results; those showing a yield of less than 50% were discarded.

Determination of resistance

The inheritance of DDT resistance was determined by the following procedure. First, reciprocal crosses between resistant and susceptible mosquitos were made to obtain an F_1 generation. The hybrids were tested by exposure to the discriminating dosage (4% DDT for 1 h) and mortality was calculated for each family. After selection, the survivors were inbred to give an F_2 generation while others were kept for backcrossing to the susceptible parent. The backcross progeny were exposed to the discriminating dosage and the survivors were kept for further backcrossing to the susceptible parent. This procedure was repeated for successive backcross generations. Adults from the last backcross progeny were selected after exposure to the discriminating dosage and the survivors were inbred. This was done in order to determine whether the genetic material responsible for DDT resistance would change after several backcrosses to the susceptible strain.

Adults were tested according to the WHO test for the susceptibility of adult mosquitos to organochlorine compounds.[1] The adults were exposed to 4% DDT for 1 h to discriminate between resistant and susceptible individuals. Prolonged exposure to 4% DDT for more than 1 h was used to determine the degree of resistance in the resistant parent and the hybrid adults.

RESULTS

Togo strain

Adults from the resistant parent colony tested with 4% DDT for 1 h showed an average of 16% mortality (Table 1). There was no significant difference in the susceptibility of male and female mosquitos. The log time–probit regression curve is a straight line, indicating homogeneity in resistance (Fig. 1). The level of resistance estimated from the graph is 5 times that of the susceptible IBAD strain. Hybrid adults from the cross between resistant and susceptible parents showed the same level of resistance as the resistant parent (Fig. 1). The average hybrid mortality on exposure to the discriminating dosage was 15% and there was no difference between the sexes. The hybrids from the reciprocal mating showed similar responses to DDT. Hence resistance is autosomal, and no extrachromosomal factors are involved. Resistance, as expressed in the F_1 heterozygotes, was dominant.

The F_2 progenies of single families were exposed to the discriminating dosage, which kills susceptible mosquitos. Most of the families segregated into resistant and susceptible individuals in a 3 : 1 ratio; for the pooled data the observed segregation closely approached a 3 : 1 ratio ($\chi^2 = 0.29$; $P = 0.5$). These results suggest that DDT resistance in the Togo strain is probably due to a single gene. Further evidence for monofactorial inheritance can be obtained by following a method originated by Wright (1952) that involves the repeated backcrossing of the heterozygote to the susceptible parent. If resistance is determined by one major factor that is not entirely recessive, half the progeny of each successive backcross will be resistant and half will be susceptible. If several major factors determine resistance, the level of resistance will decrease with each successive back-

[1] *Wld Hlth Org. techn. Rep. Ser.*, No. 443, p. 47.

Table 1. Rates of mortality in *A. gambiae* adults following exposure of DDT-resistant species A strains Tog and UV, DDT-resistant species B strain SUD, and the hybrids from the reciprocal crosses between the resistant and susceptible parents to 4 % DDT for 1 h.

Strain	Cross	No. of females tested	Percentage mortality	No. of males tested	Percentage mortality	Total no. tested	Percentage mortality
Tog	R ♀ × R ♂	312	15.6	297	16	609	16
	R ♀ × S ♂	473	15	621	15	1 094	15
	R ♂ × S ♀	223	13	223	18	446	15.5
UV	R ♀ × R ♂	202	5	181	6	383	5.5
	R ♀ × S ♂	470	19	529	11	999	16
	R ♂ × S ♀	263	16	336	17	599	17
SUD	R ♀ × R ♂	496	26	496	42	992	34
	R ♀ × S ♂	458	20	514	38	972	29
	R ♂ × S ♀	168	23	160	46	328	34

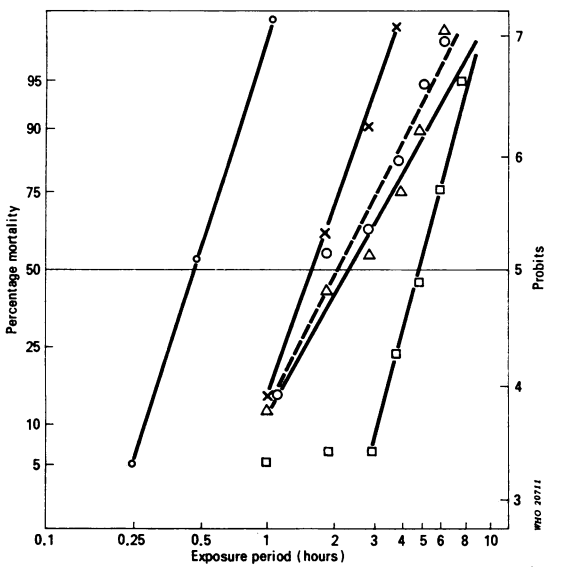

Fig. 1. Log time–probit curves for DDT-resistant strains Tog and UV, susceptible strain IBAD, and for the F_1 generation of DDT-resistant strains (females) × susceptible strain IBAD (males) exposed to 4.0% DDT-impregnated papers for various lengths of time. Small circles, susceptible strain IBAD; X, F_1 (UV×IBAD); triangles, resistant strain Tog; large circles, F_1 (Tog× IBAD); squares, resistant strain UV.

cross; as a result, mortality will increase progressively.

The progenies of single families from the first backcross to the susceptible parent segregated into resistant and susceptible phenotypes in a 1 : 1 ratio. The total segregation of 35 families showed an insignificant departure from the 1 : 1 ratio (χ^2 = 0.53; P > 0.3), the average mortality being 49% ± 1.25% (Table 2). Families in the second backcross were heterogeneous for the 1 : 1 ratio. In only 17 of 25 families did the segregation agree with the expected ratio. There was a significant shortage of the resistant phenotype in the total segregation of all the families, as indicated by the χ^2 test (χ^2=34.1; P<0.01). The average mortality rate was 56%± 1.9%. The third backcross also revealed great variation in the response to DDT among the families; altogether, 6 of 13 families segregated according to the 1 : 1 ratio. There was a loss of the resistant phenotype as indicated by the χ^2 test for the pooled data of all the families (χ^2=14.83; P<0.01); the average mortality rate was 55%±4.2%. In the fourth backcross generation, segregation in only 7 of 16 families agreed with the 1 : 1 ratio of resistant to susceptible phenotypes. Again, there was a significant loss of resistant individuals (χ^2=66.0; P<0.01), the average mortality rate being 60%

Table 2. Segregation of DDT resistance in the pooled data of F_2 generations and successive backcrosses to the susceptible parent in strains Tog and UV of DDT-resistant populations of species A

Strain	Generation	No. of families	No. of eggs	No. of larvae	Total no. of adults tested	Percentage mortality	P
Tog	F_2	9	1 450	1 360	1 010	26	0.5 [a]
	1st backcross	35	4 669	4 374	3 928	49 ± 1.25	0.3
	2nd backcross	25	2 726	2 543	2 285	56 ± 1.9	0.01
	3rd backcross	13	1 756	1 653	1 431	55 ± 4.2	0.01
	4th backcross	16	2 013	1 853	1 667	60 ± 4.6	0.01
	5th backcross	10	1 290	1 174	898	52 ± 2.6	0.02
	5th backcross hybrids inbred	11	1 575	1 451	1 095	26	0.2 [a]
UV	F_2	6	890	752	639	30	0.01 [a]
	1st backcross	20	2 423	2 218	2 002	50 ± 2	0.7
	2nd backcross	12	1 976	1 828	1 512	50 ± 2.5	0.7
	3rd backcross	12	1 716	1 631	1 364	51 ± 2.5	0.2
	3rd backcross hybrids inbred	11	1 540	1 427	1 102	26	0.5 [a]

[a] P value to test for a 3 : 1 ratio. For backcrosses P is testing for 1 : 1 ratio.

±4.6%. Families in the fifth backcross generation showed a closer fit to the 1 : 1 ratio of resistant to susceptible mosquitos ($\chi^2 = 3.0$; $P > 0.02$), with less heterogeneity than in the previous backcross generations. The average mortality rate was 52%±2.6%.

In comparing the average mortality rates of the 5 backcross generations, it can be seen that there was a loss of resistance in the second to fourth backcross generations. Mortality increased from 49% in the first generation to 56%, 55%, and 60% in the second, third, and fourth generations, respectively. However, this steady increase in mortality did not persist to the fifth generation. The mortality rate in the fifth generation was 52%; i.e., lower than in the previous generations. The results also revealed considerable variation within each generation. Phenotypic variation was high among the families of the third and fourth generations, and mortality rates between 30% and 80% were found. This indicates that heterogeneity was not due to the loss of the resistant phenotype alone, but resulted from the loss of the resistant phenotype in some families and of the susceptible phenotype in others. A comparative estimate of the variation, as shown by the coefficient of variation around the average mortality rate, was 15%, 17%, 27%, 30%, and 16% for the successive generations. Since variation was observed within each generation as well as between generations, an analysis of variance was performed (Table 3). If the variance between generations was found to be significantly greater than the variance within generations, it could be considered that the average mortality rates between the 5 generations were statistically different. In fact, the variance within generations was nearly equal to the variance between generations ($P > 0.05$). Therefore, the amount of variation observed between generations was not significantly different from that expected to arise from the normal biological variation exhibited by families in these crosses.

The results of the 5 backcrosses described above clearly support a hypothesis of monofactorial inheritance. The evidence is based largely on the progenies in each of the 5 generations falling into 2 equal categories of resistant and susceptible individuals, and mortality in the fifth backcross being less than that found in the previous crosses. If DDT resistance were the result of more than one factor a progressive increase in mortality, and not a decrease, would be expected.

Heterozygous adults of the fifth backcross generation that survived the discriminating dosage of DDT

Table 3. Mortality rates and analysis of variance in 5 successive backcross generations of DDT-resistant strain Tog of species A with the susceptible strain IBAD

Generation	No. of families	No. of individuals	Average mortality (%)	Variance (S^2)	Coefficient of variation %	Mean square between generations	Mean square within generations
F_1	35	3 928	49	57	15		
F_2	25	2 285	56	95.6	17		
F_3	13	1 431	55	231	27	243	253
F_4	16	1 667	60	337	30		
F_5	10	898	52	73	16		

[a] For 4 and 94 degrees of freedom, P = 0.05.

were allowed to mate. If a single dominant factor is involved, the progeny of this cross should be like that of the F_2 generation, with a segregation in the ratio of 3 : 1 of resistant and susceptible phenotypes. Altogether, 11 families from such a cross were tested (Table 2). The total segregation in the pooled data of all the families fitted very well to the expected 3 : 1 ratio ($\chi^2 = 1.28$; $P > 0.2$). These results clearly show that the expression of the major gene responsible for resistance was not reduced as a result of repeated outcrossing to the susceptible parent. Thus the existence of a major gene for DDT resistance has been clearly demonstrated in strain Tog of species A, and resistance is dominant.

Upper Volta strain

Homozygous individuals from the parent colony tested with 4% DDT for 1 h showed an average mortality of only 6%, and there was no significant difference in the susceptibility levels of the sexes. The degree of resistance in strain UV of species A was higher than that of strain Tog. The probit line of strain UV was to the right of the strain Tog regression (Fig. 1). The degree of resistance in strain UV was 10 times that of the susceptible strain and twice that found in strain Tog.

Hybrid adults from the reciprocal crosses between the resistant strain UV and the susceptible strain IBAD had an average mortality rate of 16% on exposure to the discriminating dosage of DDT, no clear difference between the reciprocal crosses being observed. The probit regression line of the F_1 hybrid lies between the probit lines of the resistant and susceptible parents. Resistance, as expressed in the F_1 hybrids, is incompletely dominant.

F_2 families segregated into resistant and susceptible phenotypes in accordance with a 3 : 1 ratio. The average mortality for the total number of mosquitos tested was 30% (Table 2). It is probable that DDT resistance in strain UV is monofactorial. To provide further evidence to support the hypothesis of single gene inheritance, repeated backcrossing of the hybrid to the susceptible parent was carried out. As shown in Table 2, the offspring of the first backcross screened with the discriminating dosage of DDT segregated into susceptible and resistant phenotypes in a 1 : 1 ratio ($\chi^2 = 0.09$; $P > 0.7$). The average mortality rate was 50%±2%, and the coefficient of variation was only 14%. The second backcross also segregated according to the 1 : 1 ratio ($\chi^2 = 0.09$; $P > 0.7$). The average mortality rate was 50%±2.5% and the coefficient of variation was 20%. The third backcross again sorted out into 2 groups of resistant and susceptible mosquitos statistically equal in size ($\chi^2 = 1.21$; $P > 0.2$). The average mortality rate was 51%±2.5% and the coefficient of variation was 20%. There was no obvious increase in mortality in the three successive generations. Variation within generations was nearly the same as that shown by the coefficient of variation.

Heterozygous adults from the third backcross generation were inbred. If a single gene were operating we would once again expect the progenies of this cross to segregate in the ratio 3 resistant to 1 susceptible mosquitos and in fact the total segregation of 11 families fitted closely to the 3 : 1 ratio ($\chi^2 = 0.29$; $P > 0.5$).

In all the crosses described above, segregation of resistance fitted closely to that expected from the hypothesis of monofactorial inheritance. The results therefore provide strong evidence that there is a single incompletely dominant gene controlling the inheritance of DDT resistance in strain UV of species A.

Sudan strain

Adults of species B from the colony of resistant strain SUD showed highly variable responses to exposure to 4% DDT for 1 h. Mortality in some families was approximately 10% while in others it was about 50%, the average mortality rate being 34%. Such variation was not observed in DDT-resistant species A. Mortality in strain SUD males was 42%, significantly higher than that in the females, which was 26% (Table 1). This difference in tolerance between the sexes was observed consistently. The level of resistance estimated graphically was only 4 times that of the susceptible strain MAK SS (Fig. 2).

Families from reciprocal crosses between the resistant and the susceptible parent showed various levels of tolerance; mortality rates after exposure to 4% DDT for 1 h were between 0 and 68%. The average mortality rate among all mosquitos tested was 29%. Mosquitos from the reciprocal matings showed similar responses to DDT; thus sex linkage and maternal effects were not indicated. Heterozygous males from both reciprocal crosses showed a lower level of tolerance to DDT than did females,

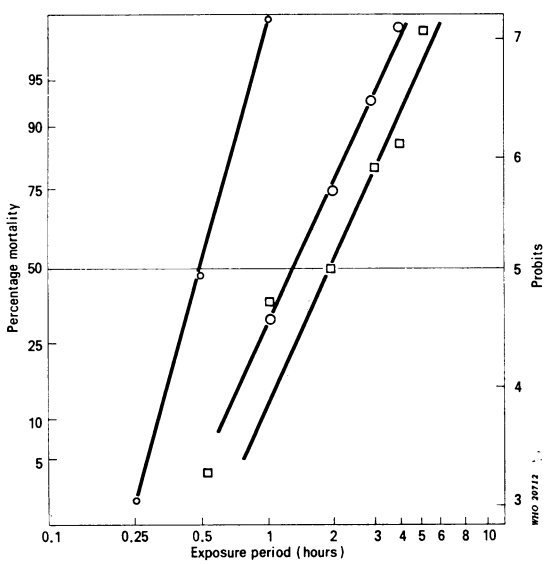

Fig. 2. Log time–probit curves for DDT-resistant strain SUD, susceptible strain MAK SS, and the F_1 generation of resistant strain SUD (females) × susceptible strain MAK SS (males) exposed to 4.0% DDT-impregnated papers for various lengths of time. Small circles, susceptible strain MAK SS; squares, resistant strain SUD; large circles, F_1 (SUD × MAK SS).

the average mortality of hybrid males being about 40%, i.e., almost twice that of the hybrid females. DDT resistance in the F_1 heterozygotes was essentially similar to that of the homozygous resistant parent. The regression line for the heterozygotes is close to that for the homozygous resistant parent. Resistance, as expressed in the F_1 generation, was dominant, but there was considerable interprogeny variation in the expression of dominance.

After selection by means of 4% DDT for 1 h, the surviving F_1 hybrids were inbred to obtain the F_2 generation. Single F_2 families were then exposed to the discriminating dosage of DDT. If it was assumed that a single dominant gene was operating, then the calculated expected mortality would be 47.5%. As shown in Table 4, the average observed mortality rate was 48%, which is in excellent agreement with the expected value.

Repeated backcrossing of the heterozygotes to the susceptible parent was continued through three successive generations. The expected mortality rate for the backcross, calculated on the basis of monofactorial inheritance, was 65%. The average mortality rate in the first backcross was 69%, a value fairly close to the expected value (Table 4). In the second backcross the average mortality rate from the pooled data of 8 families was 70%; again this is rather close to the expected value of 65%. The average mortality rate in the third backcross was 52%, i.e., less than the expected rate of mortality. Thus segregation in the third backcross progeny, unlike that in the first and second backcross generations, was close to the 1 : 1 ratio.

Interprogeny variation was a common feature in all the crosses made with strain SUD, and this was particularly obvious in the three backcross generations. Mortality rates close to the 50% level were found in some families and rates near the 100% level in others. An analysis of variance was made to compare the variance within and between the three backcross generations (Table 5); variation between generations was found to be significantly greater than that within generations ($F = 4.18$; $0.01 < P < 0.05$). The average mortality rates of the three generations were thus unequal, the difference being the result of a decrease in mortality in the last backcross generation.

Heterozygotes from the third backcross were inbred. On the basis of monofactorial inheritance, the expected maximum corrected mortality would be about 47.5%, but the average observed mortality was 32%; i.e., lower than the expected maximum

Table 4. Rates of mortality in adult mosquitos after exposure to 4 % DDT for 1 h in the F_2 generation, backcrosses to the resistant parent, and successive backcrosses to the susceptible parent in DDT-resistant strain SUD of species B

Generation	No. of families	No. of females tested	Percentage mortality		No. of males tested	Percentage mortality		Total mosquitos tested	Percentage mortality	
			expected	actual		expected	actual		expected	actual
F_2	17	919	40	38	923	55	54	1 842	47.5	48
Backcross to RR	6	115	26	6	141	42	27	256	34	17
1st backcross to SS	16	1 125	60	72	943	70	70	2 068	65	69
2nd backcross to SS	12	842	60	71	812	70	69	1 654	65	70
3rd backcross to SS	12	708	60	50	665	70	55	1 373	65	52
3rd backcross hybrids inbred	7	357	40	31	385	55	32	742	47.5	32

Table 5. Mortality rates and analysis of variance in 3 backcross generations of SUD DDT-resistant strain SUD of species B with the susceptible MAK SS strain

Generation	No. of families	No. of individuals	Average mortality (%)	Variance (S^2)	Coefficient of variation (%)	Mean square between generations	Mean square within generations	Variance ratio (F)
F_1	16	2 068	69	248	23			
F_2	12	1 654	70	381	28	657	157	4.18[a]
F_3	12	1 373	52	148	28			

[a] For 2 and 37 degrees of freedom $0.01 < P < 0.05$.

and less than that found in the F_2 generation obtained by inbreeding F_1 heterozygotes.

The results described above suggest that a single genetic factor is responsible for DDT resistance in strain SUD of species B, and that resistance is dominant. The existence of monofactorial inheritance is corroborated by the observations that mortality rates in the F_2 generation and in the first and second backcross generations were close to those expected; that mortality rates did not rise in the third backcross generation, but actually decreased; and that progenies derived from crosses between the third backcross heterozygotes segregated in the ratio of approximately 1 susceptible to 3 resistant mosquitos.

DISCUSSION

The mode of inheritance of DDT resistance in *A. gambiae* species A and B was determined, by the use of a single discriminating dose of DDT, on the offspring of repeated backcrosses between the resistant hybrid and the susceptible parent. The procedure of repeatedly backcrossing to the susceptible parent was originally suggested by Wright (1952) as a means of isolating a major gene, and was recommended by Crow (1957) as a technique for studying the inheritance of insecticide resistance. By the use of this method, DDT resistance was found to be monofactorial in two species A populations from two localities in West Africa, and in a species B population from Sudan. The evidence for monofactorial inheritance is based largely on the close agreement of observed segregation in the F_2 and backcross generations with that expected on a hypothesis of monofactorial inheritance, and on the capacity of mosquitos to recover the original level of resistance after a series of backcrosses with selection to a susceptible strain.

Resistance in the three strains of species A and species B was a dominant factor, but there was variation in the expression of dominance between the progenies of single-pair matings in some of the crosses. Variation was less pronounced in strain UV and the results were generally closer to expectation than in the other two strains. In crosses involving strain Tog interprogeny variation was observed after the first backcross and was particularly high in the third and fourth backcross generations. It is noteworthy that

families of Tog fifth backcross generation of strain Tog were more stable than the previous crosses, and displayed comparatively less variation in resistance.

An even greater variation was observed between families of species B. In contrast to strains Tog and UV, the expression of resistance was variable in the heterozygous and homozygous resistant genotypes. Significantly high discrepancies with greater than expected mortalities were observed in some families of the F_2 generation of strain SUD and in the first two backcross generations. However, there was less heterogeneity in the progenies of the third backcross, and the results strongly support the hypothesis of monofactorial inheritance. If the low levels of resistance manifested by some families in the early backcross generations of strains Tog and SUD were the result of the absence of major genetic factors, an even lower level of resistance in the later backcrosses could have been expected. This was not observed, suggesting that there were environmental and minor genetic differences, rather than a major genetic influence. One of the major environmental influences on resistance comes from variation in the rearing of larvae; overcrowding or underfeeding may result in small weak adults that are generally more susceptible to insecticides. Procedures for rearing larvae are always difficult to standardize.

An important factor that could be responsible for some of the observed heterogeneity is differential mortality. A shortage of one of the genotypes could lead to a significant deviation from the expected mortality, depending on the magnitude of the deficiency. In strain SUD there was a significant sexual difference in DDT resistance that was not exhibited by species A strains and this may account for a great many of the discrepancies observed in the SUD crosses. Moreover, minor genetic factors are probably involved, particularly modifiers of expressivity that have a great effect on the final expression of an oligogene of DDT resistance. It was perhaps the loss of modifying genes in the later backcross generations that caused an increase in mortality in some families. The influence of modifying genes on insecticide resistance was fully discussed by Spiller (1958), who pointed out that modifying genes seem to be particularly essential for the expression of an oligogene of DDT resistance. The influence of modifying genes was also evident in the offspring of single pair matings in a Trinidad strain of *Aedes aegypti* (Wood, 1965).

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RÉSUMÉ

TRANSMISSION HÉRÉDITAIRE DE LA RÉSISTANCE AU DDT DANS LES ESPÈCES A ET B DU COMPLEXE *ANOPHELES GAMBIAE*

On a étudié les modalités de la transmission héréditaire de la résistance au DDT chez trois souches résistantes du complexe *Anopheles gambiae*, deux appartenant à l'espèce A et originaires de la Haute-Volta et du Togo et la troisième appartenant à l'espèce B et en provenance du Soudan. On a utilisé la technique des rétrocroisements répétés entre adultes hybrides résistants et adultes de la souche parente sensible.

Les recherches ont montré que la résistance au DDT est sous la dépendance d'un gène unique à caractère dominant chez la souche de l'espèce A originaire du Togo et chez la souche de l'espèce B du Soudan. Chez la souche de l'espèce A en provenance de la Haute-Volta, la transmission de la résistance au DDT est aussi contrôlée par un gène unique mais dont la dominance est incomplète.

REFERENCES

Baker, R. H. et al. (1962) *Mosquito News*, **22**, 1, 16-17
Busvine, J. R. (1969) *Trans. roy. Soc. trop. Med. Hyg.*, Suppl. No. 4, pp. 19-30
Crow, J. F. (1957) *Ann. Rev. Ent.*, **2**, 227-246
Davidson, G. (1956) *Nature (Lond.)*, **178**, 861-863
Hamon, J. et al. (1968) *Méd. trop.*, **28**, 222-228
Spiller, D. (1958) *N. Z. Entomol.*, **2**, 1-18
Wood, R. J. (1965) *Bull. Wld Hlth Org.*, **32**, 563-594
Wright, S. (1952) *Quantitative inheritance*, London, Agricultural Research Council, pp. 5-24