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THE VECTORIAL CAPACITY OF AN INSECTICIDE-RESISTANT
AND A SUSCEPTIBLE STRAIN OF A. GAMBIAE IN NORTHERN NIGERIA

by

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Introduction

Busvine (1957), in concluding the discussion on his paper on insecticide-resistant strains of insects of public health importance, pointed out that while resistant Anopheles saccharovi in Greece were known to transmit malaria, relative vector efficiencies of resistant as compared with susceptible strains were in general not known. It has commonly been observed that the insecticide-resistant strains of Anopheles which have appeared in different parts of the world have been responsible for the maintenance of malaria transmission, but few figures on the subject are available. The present communication is an attempt to supply this deficiency as regards the strain of A. gambiae which was detected showing resistance to dieldrin and BHC in Western Sokoto in 1955 (Elliott & Ramakrishna, 1956). The history of malaria control activity in the province is given by Ramakrishna & Elliott (1959); the zone concerned here is the 300 square miles of the original pilot project which were treated by residual house-spraying as follows:

July 1954 to May 1955: Dieldrin at 0.25 g/m²;
June 1955: Dieldrin dosage doubled over half the area;
May 1956: BHC at 0.4 g/m² introduced to replace the higher dieldrin dosage;
August 1956: Dieldrin dosage doubled in area of previously 0.25 g/m²;
April 1957: DDT at 2.0 g/m² introduced to replace both the other insecticides;
September 1957: DDT completely replaced other insecticides.¹

Observations

The monthly average Anopheles densities, sporozoite rates and average infective densities of A. gambiae in areas under the four regimes and in an unsprayed area are shown in Tables 1 to 3. The terms are defined as follows:

Average Anopheles density: Number of female A. gambiae per room per day, as determined by pyrethrum spray catches;

¹ It might be recalled here that in 1955 a strain of A. gambiae resistant to dieldrin with an LC₅₀ value about 800 times that of the susceptible strain was isolated in the dieldrin-treated zone of the Western Sokoto Project. The investigation of the distribution of resistance to dieldrin in this area was carried out in 1956 and it was found that in the centre of the dieldrin-treated zone the A. gambiae population consisted of 89% homozygous resistant, 9% homozygous susceptible and only 2% heterozygous. In a zone which was treated with BHC only a similar composition of the population was found. The dieldrin and BHC resistance were found to be linked. The LC₅₀ of adult A. gambiae in the pilot project, assessed by the WHO method of susceptibility testing, was found to be between 0.13 and 0.52 for BHC (relevant figures for susceptible strains in unsprayed areas were 0.0032-0.004). LC₅₀ for dieldrin have not been obtained but the percentage surviving exposure to 4% impregnated papers varied between 78 and 100. Susceptibility to DDT was only slightly above the normal. Further investigation on the distribution of resistance in Western Sokoto was carried out using the larvae susceptibility test. It was found that where BHC had been used for two-and-a-half years the A. gambiae population was homozygous for the cross-resistance gene. Where dieldrin had been used a mixed population was found consisting of homozygous and heterozygous resistant material. A marked spread of the resistance factor, at least 14 miles from the limit of the sprayed area, was detected. No sign of resistance to DDT was found.

The reader will find the relevant references in the paper by Ramakrishna & Elliott (1959). A report by Armstrong et al. was issued in WHO/Mal/182 of this series. (Editor's remark)

Sporozoite rate: Percentage with salivary gland infections;

Average infective density: $\frac{AAD \times SR}{100}$ (Davey & Gordon, 1933).

The tables and the figure show that in general malaria transmission in the area is detectable in the insect mainly in the third and fourth quarters of the year; average infective density figures are nil throughout in the first quarter, and only the unsprayed area shows a low figure for the second quarter of 1956. The lower dosage of dieldrin appears to have produced an initial reduction in the average infective density from which it rapidly recovered. The drop recorded in the third quarter of 1956 refers to a reduced area. The doubled dose of dieldrin had no effect on the recovery of average infective density figures, which by 1956 were of the same order as those of the unsprayed area.

The effect of the introduction of BHC was shown mainly in a reduction in the Anopheles population, although the last figure for sporozoite rate is a low one. BHC was, therefore, able to reduce the intensity of malaria transmission as measured by average infective density. There are reasons for supposing that BHC exerts a degree of control on the heterozygous resistant fraction of an A. gambiae population; this is deduced by Ramakrishna & Elliott (1959) from the fact that populations from areas where BHC only was used become homozygous for the resistance factor. Davidson & Pollard (1958) have shown experimentally that a simulated field dosage of 0.5 g/m² gamma isomer of BHC kills a high percentage of heterozygotes up to eight weeks after spraying, while the same dosage of dieldrin was virtually ineffective in a much shorter time.

Finally, DDT so reduced the mosquito population that a sporozoite rate was unobtainable and the average infective density was recorded as nil.

TABLE 1. AVERAGE ANOPHELES DENSITY OF A. GAMBIAE
 IN WESTERN SOKOTO (NORTHERN NIGERIA)

Quarter and year	Unsprayed area	Dieldrin 0.25 g/m ²	Dieldrin 0.5 g/m ²	BHC 0.4 g/m ²	DDT 2.0 g/m ²
III 1954	90.6	38.4	-	-	-
IV 1954	72.1	2.6	-	-	-
I 1955	11.2	1.0	-	-	-
II 1955	31.3	3.5	0.06	-	-
III 1955	143.2	25.7	11.9	-	-
IV 1955	28.2	20.4	13.2	-	-
I 1956	2.2	5.0	8.7	-	-
II 1956	10.4	7.4	6.3	3.1	-
III 1956	147.5	19.0	112.9	46.9	-
IV 1956	36.5	-	20.4	8.8	-
I 1957	1.2	-	-	4.6	-
II 1957	8.7	-	-	8.7	0.8
III 1957	66.4	-	-	14.8	0.9
IV 1957	34.8	-	-	-	0.2

TABLE 2. SPOROZOITE RATE IN A. GAMBIAE
 IN WESTERN SOKOTO (NORTHERN NIGERIA)

Quarter and year	Unsprayed area	Dieldrin 0.25 g/m ²	Dieldrin 0.5 g/m ²	BHC 0.4 g/m ²	DDT 2.0 g/m ²
III 1954	2.5	0.0	-	-	-
IV 1954	3.6	0.5	-	-	-
I 1955	0.0	0.0	-	-	-
II 1955	0.0	0.0	0.0	-	-
III 1955	2.2	0.6	1.7	-	-
IV 1955	3.1	3.5	1.6	-	-
I 1956	0.0	0.0	0.0	-	-
II 1956	0.1	0.0	0.0	0.0	-
III 1956	1.5	0.7	1.2	1.2	-
IV 1956	1.7	-	8.7	1.7	-
I 1957	0.0	-	-	0.0	-
II 1957	0.0	-	-	0.0	0.0
III 1957	3.2	-	-	0.66	0.0
IV 1957	0.5	-	-	-	0.0

TABLE 3. AVERAGE INFECTIVE DENSITIES OF A. GAMBIAE
 IN WESTERN SOKOTO (NORTHERN NIGERIA)

Quarter and year	Unsprayed area	Dieldrin 0.25 g/m ²	Dieldrin 0.5 g/m ²	BHC 0.4 g/m ²
III 1954	2.265	0.0	-	-
IV 1955	2.595	0.013	-	-
III 1955	3.150	0.154	0.187	-
IV 1955	0.874	0.714	0.211	-
III 1956	2.212	0.133	1.355	0.563
IV 1956	0.620	-	1.775	0.150
III 1957	2.12	-	-	0.098
IV 1957	0.17	-	-	-

Discussion

It appears that the changes induced by the successive treatments with dieldrin and BHC in the A. gambiae population are in the case of dieldrin the appearance of a predominantly heterozygous resistant population, while BHC replaces this with a homozygous resistant population. The evidence shows that the vectorial capacity of a mainly heterozygous population is fully as great as that of a susceptible population.

The period following the introduction of BHC shows a reduction in both mosquito population and in sporozoite rates, so that the vectorial capacity of the now predominantly homozygous resistant population is low. Malaria transmission can still be shown, however, and the causes of the reduction in its intensity seem more likely to be the reduction in numbers and in length of life of the individual mosquito rather than in a change in its vectorial capacity. This point can only be cleared up by laboratory trials of the capacity of the different genetic types for acquiring malaria infection, and on their longevity.

Finally the effect of DDT was so to reduce both the A. gambiae population and its sporozoite rate as to render malaria transmission undetectable in the mosquito. In the human population a reduced level of transmission could still be detected, but in the mosquito its measurement must await more refined methods of collection capable of producing adequate samples of the reduced population.

Conclusions

It is concluded therefore that:

1. A population of A. gambiae consisting mainly of heterozygous resistant material is as capable of transmitting malaria as a susceptible population, and that the vectorial capacity of the individual of heterozygous genotype is probably equal to that of the homozygous susceptible.
2. The vectorial capacity of the homozygous genotype may possibly be less than that of the heterozygous, but this is not proved by the short period of observation recorded here, since the population level had been considerably reduced by the use of BHC.
3. The introduction of DDT so reduced the remaining population that malaria transmission could not be detected by the methods in use, although it was known to be taking place.

Summary

Observations on the average Anopheles density, sporozoite rate and average infective density of populations of A. gambiae in Western Sokoto show that the transmission of malaria by populations subjected to selection by both dieldrin and BHC may be reduced on account of reduction in their numbers under different insecticidal regimes, but that the vectorial capacity of the species is probably unchanged by the alteration in the character of the population. The introduction of DDT so reduced Anopheles densities that the sporozoite rate became unobtainable and average infective density was recorded as nil.

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REFERENCES

- Busvine, J. R. (1957) Trans. Roy. Soc. trop. Med. Hyg. 51, 1. p. 11
- Davey, T. H. & Gordon, R. M. (1933) Ann. trop. Med. Parasit. 27, 27
- Davidson, G. & Pollard, D. G. (1958) Nature, 182, 439
- Elliott, R. & Ramakrishna, V. (1956) Nature, 177, 532
- Ramakrishna, V. & Elliott, R. (1959) Trans. Roy. Soc. trop. Med. Hyg. 53, 1.
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