I thank Sterling Poultry Products, Ltd., for their co-operation in carrying out the laying trial, and Professor W. W. Fletcher and Mr J. F. Dunachie for telling me of their results before publication.

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Received May 10, 1967.

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Distribution of *Biomphalaria* Species in Sudan

INTESTINAL bilharzia (caused by the trematode *Schistosoma mansoni*) is transmitted by snails of the genus *Biomphalaria*. A survey has recently been conducted to determine the distribution of these snails in the Khartoum and Blue Nile provinces of Sudan.

The occurrence of *Biomphalaria alexandrina* (Ehrenberg) has until now been recorded only from Lower Egypt[1] but the present survey shows that this species is frequently present in Sudan. The distribution in the White Nile appears to extend from the dam at Jebel Aulia (50 km south of Khartoum) to at least as far south as Kosti (300 km south of Khartoum). High populations were found particularly among *Eichornia* (water hyacinth) plants and to a lesser extent in pump irrigation schemes. This species was also found on the west of the Gezira irrigation scheme (taking water from the Blue Nile) but its distribution east of this is uncertain.

Biomphalaria sudanica (Martens) also appears to have a wider distribution than formerly recognized. High populations of this species were found at Shambat, a pump scheme 8 km north of Khartoum on the main Nile, and at various points in the White Nile between Kosti and Khartoum. Previously[2], it had been found no farther north than Kosti. *Biomphalaria pfeifferi* (Krauss), while common throughout the Gezira and pump schemes from the Blue Nile, was not found in the White Nile or its associated irrigation schemes.

It can be concluded from these observations that the distribution of the snails is changing rapidly. Presumably the northwards extension of the distribution of *B. sudanica* has been influenced by a similar extension of *Eichornia* since 1958 (ref. 3) but the reasons for the southwards movement of *B. alexandrina* are obscure. Measures to control bilharzia in Sudan will need to take the present complex distribution of snails into account and recognize that snails will rapidly move back into controlled areas.

We thank Dr G. A. Mandahl-Barth for identifying specimens and the Rockefeller Foundation for financial assistance.

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Received August 18, 1967.

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Effect of Carbon Monoxide on Metabolism of Insecticides *in vivo*

THERE have recently been several reports which indicate that the oxidative metabolism of drugs by liver microsomes involves a cytochrome-like pigment usually referred to as *P*-450 (refs. 1 and 2). This pigment, which is characterized by the difference spectrum observed when microsomal suspensions reduced either with hydrosulphite or

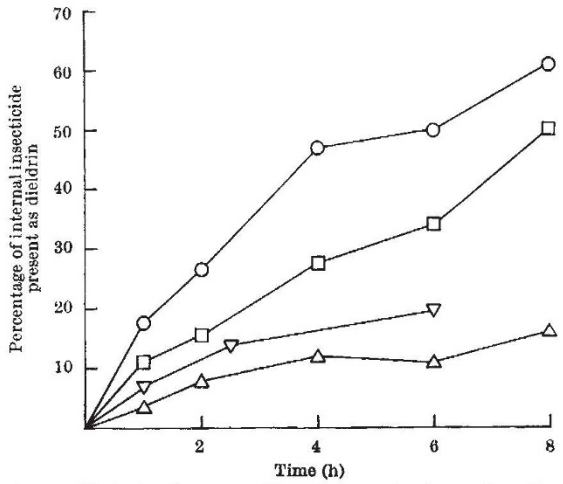

Fig. 1. Effect of carbon monoxide and oxygen tension on formation of dieldrin in houseflies treated with aldrin. ○, Flies kept in air; ▽, flies kept in 40 per cent carbon monoxide, 20 per cent oxygen, 40 per cent nitrogen; □, flies kept in 90 per cent nitrogen, 10 per cent oxygen; △, flies kept in 90 per cent carbon monoxide, 10 per cent oxygen.

with NADPH are saturated with carbon monoxide, has recently been found in microsomes from the housefly *Musca domestica* (*vicina* strain)[3]. Furthermore, the inhibition by carbon monoxide of the epoxidation of aldrin and the partial reversal of this inhibition by light supports the view that *P*-450 is involved in epoxidation by fly microsomes *in vitro*. Similar evidence has been produced to support the view that this pigment is involved in the metabolism of drugs by microsomes from rat liver[2]. Evidence for the role of a reaction in the metabolism of insecticides *in vivo* which is sensitive to carbon monoxide has not, however, been reported.

The relative affinities of carbon monoxide and oxygen to haemoglobin, *P*-450 and cytochrome oxidase are 200 : 1, 1 : 1 and 1 : 10, respectively[4]. The housefly, in common with other insects, does not rely on haemoglobin for the transport of oxygen and so it is likely to survive in a mixture of carbon monoxide and oxygen which would inhibit reactions involving *P*-450. Thus it would seem that insects may be particularly suitable animals in which to examine the effect of carbon monoxide on the metabolism of foreign compounds *in vivo*. The epoxidation of the insecticide aldrin is a reaction which is well suited for this purpose because in the housefly there is little evidence of further metabolism of the epoxide (dieldrin)[5].

Four-day-old female houseflies *Musca domestica* (*vicina* strain), resistant to dieldrin, were treated individually with 2 μg of aldrin in 2 μl. of acetone. Groups of ten flies were confined in glass cylinders of 1 l. capacity which had previously been filled with an appropriate mixture of carbon monoxide, oxygen and nitrogen. They were supplied with sucrose and water in glass tubes plugged with cotton wool and held at a temperature of 25° C. When exposed to a gas mixture consisting of 90 per cent carbon monoxide and 10 per cent oxygen the flies were unable to fly but were not completely immobilized. Separate experiments had shown that after being exposed to this gas mixture for 16 h flies recovered completely when the gas mixture was replaced by air. In a gas mixture consisting of 20 per cent oxygen, 40 per cent carbon monoxide and 40 per cent nitrogen flies, behaved in a normal manner for at least 48 h.

At appropriate times after treatment with aldrin the flies were removed from the cylinders and immediately rinsed with two 3 ml. volumes of acetone in order to remove external aldrin. They were then homogenized and extracted twice, first with 4 ml. and then with 3 ml. of acetone. Four ml. of water and sodium sulphate was added to the acetone extract which was then extracted with two 4 ml. volumes of petroleum ether. The aldrin