#### DRRESPONI DEN E

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cells to be tested in humans without the whole range of present toxicity study requirements. This may perhaps be part of Weatherall's "unattractive message that the experts must tell the public" and does fall into the category of "desperate remedies for desperate ills": it has also served to reactivate industrial interest in this therapeutic area.

An even more delicate area was not discussed, Proposals<sup>9</sup> for attaining a population of whom 95 per cent may use synthetic mood-modifying drugs regularly by the 1990s may be becoming more tangible with the latest developments in our biochemical understanding of the already overprescribed 1,4-benzodiazepines: identification <sup>10</sup>of a specific receptor for them, modifications of the endogenous ligand(s)11 of the benzodiazepines and the subclassification of those receptors13 with their own agonist/antagonist complement such as the type I-selective triazolopyridazines, 14 exposes a whole new area for exploitation into social psychotropic drugs. The experts ought to be talking about some of these aspects also, accepting Sakharov's charge15 that we the scientists should take responsibility for the applications of science and technology to life. C. UPTON

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# **Return of malaria**

SIR - Chapin and Wasserstrom<sup>1</sup> suggest that resurgence of malaria in Central America and India was the result of introduction of high yielding varieties (HYV) of rice and cotton. We feel that this conclusion is incorrect and that the basic data used by Chapin and Wasserstrom were defective. What follows is

our view of how malarial resurgence came about in India.

(1) During the years of resurgence cotton production remained at a roughly constant level. There was a marginal increase in rice production but the area under HYV was low. Use of DDT in 1960-64 was 73,313 tonnes in public health as against 3,000 tonnes in the agricultural sector (see table). Even in subsequent years, use of DDT in agriculture was far less than for public health. However, it may also be pointed out that malathion insecticide has been in continuous use in agriculture since 1958 and malaria vectors have remained susceptible to it wherever it has not been used by the National Malaria Eradication Programme (NMEP). It was used in Gujarat to control DDT and HCH resistant Anopheles culicifacies and between 1970 and 1973, susceptible A. culicifacies developed 4-fold resistance<sup>2</sup>

(2) The origins of the setback to the Indian anti-malaria programme can be traced to 1963 when approximately 2 million of the population were involved in scattered focal outbreaks in the consolidation phase. Focal outbreaks during subsequent years became more serious. In areas which could not be tackled an estimated population of 12, 17 and 32 million respectively were temporarily reverted to attack phase during 1965, 1966 and 1967. During 1968, out of a total of 393.25 units, 71.385 units involving a population of 91 million were reverted to spraying, 51.785 from the consolidation and 19.60 from the maintenance phase. In addition, there were 41.60 units under NMEP which never moved out of the attack phase. These units were located in hyperendemic areas of 20 states. union territories and the coalfields3,4

(3) Whilst the incidence of malaria in rural areas went down, there was a resurgence in the cities of India. In Tamil Nadu in 1963, 95 per cent of the cases came from urban areas. In 1964-67 urban malaria constituted 80 per cent of the problem, and malaria was found to be diffusing towards the rural areas<sup>3</sup>. A. stephensi, the urban malaria vector, was found to be resistant to DDT and HCH.

(4) There are 7 major vectors of malaria in India. Insecticide resistance has been detected in Anopheles culicifacies and A. stephensi with isolated reports in A. fluviatilis<sup>6,7</sup>. The first report of increased tolerance in A. stephensi came from Erode, Tamil Nadu in 19568 and that in A. culicifacies from Gujarat in 1957<sup>9</sup>. Between 1959 and 1967 DDT resistance in A. stephensi and A. culicifacies became widespread. During 1967-69 there were 96 units under persistent attack phase and malaria incidence increased in 56 units. In 44

units tested, A. culicifacies was found resistant to DDT in 35 units and to DDT + HCH in 5 units. A. stephensi was also tested against DDT in 8 units and was found to be resistant in four (ref. 10). In fact withdrawal of spraying has been reported to have resulted in reversion towards susceptibility. Thus resistance was the result of spraying under NMEP and it preceded the introduction of HYV in India.

We believe that resurgence of malaria in India was largely due to administrative, social, economic and financial reasons11, and its diffusion was facilitated by the widespread occurrence of the parasite and anophelism in the country. The resistant populations of these mosquitoes found additional breeding grounds due to development activities. In order to stabilize and increase agricultural production, the gross area under irrigation was increased from 29.05 million hectares in 1960-61 to 37.10 in 1968-69, steadily rising to 57.02 million hectares in 1979-8012. Similarly, unplanned growth in urban and rural housing and lack of adequate water disposal facilities made the surroundings more mosquitogenic and receptive to malaria. Large areas of the country (such as the Punjab) which in the past were prone to malaria epidemics in years of high precipitation turned endemic. Irrigation increased the average humidity of the atmosphere and made the regions more conducive for mosquitoes' survival. This had the most profound affect on the basic reproduction rate. Thus there was a resurgence of malaria in areas of the country at one time freed from the disease.

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|  | Incidence of malaria, DDT used and agricultural production in India |       |       |       |       |       |       |       |       |        |        |        |        |             |               |
|--|---|-------|-------|-------|-------|-------|-------|-------|-------|--------|--------|--------|--------|-------------|---------------|
|  | 1960  | 1965  | 1966  | 1967  | 1968  | 1969  | 1970  | 1971  | 1972  | 1973   | 1974   | 1975   | 1976   | <b>1977</b> | 1978          |
| Malaria incidence<br>(millions)              |   | 0.10  | 0.15  | 0.28  | 0.27  | 0.35  | 0.69  | 1.31  | 1.43  | 1.93   | 3.17   | 5.16   | 6.46   | 4.74        | 4.14          |
| DDT (tech) used in public<br>health (tonnes) | 21,007  | 6,671 | 2,762 | 3,045 | 5,821 | 6,401 | 6,205 | 7,350 | 7,034 | 6,821  | 6,700  | 7,250  | 7,250  | 9,051       | 6,800         |
| DDT (tech) used in<br>agriculture (tonnes)   | 600   | 2,400 | 2,400 | 2,400 | 2,400 | 2,400 | 2,400 | 2,400 | 2,400 | 2,880  | 2,934  | 2,450  | 3,000  | 2,450       | 4,720         |
| Rice production<br>(million tonnes)          | 34.50   | 30.59 | 30.44 | 37.61 | 39.61 | 40.43 | 42.23 | 43.07 | 39.25 | 44.05  | 39.58  | 48.74  | 44.91  | 52.67       | 53.77         |
| Area under rice in million                   | 34.1  | 35.5  | 35.3  | 35.4  | 37.0  | 37.6  | 37.6  | 37.8  | 36.7  | 38.3   | 37.9   | 39.5   | 38.5   | 40.3        | 40.5          |
| hectares (in parentheses,<br>under HYV)      | (0)   | (0)   | (0.9) | (1.8) | (2.7) | (4.3) | (5.6) | (7.4) | (8.2) | (10.0) | (11.2) | (12.4) | (13.3) | (16.1)      | (16.9)        |
| Cotton production<br>(million bales)         | 5.60  | 4.85  | 5.27  | 5.78  | 5.45  | 5.56  | 4.76  | 6.95  | 5.74  | 6.31   | 7.16   | 5.95   | 5.84   | 7.24        | 7 <b>.9</b> 6 |

Source: NMEP, IARI & Fertilizer Statistics, Fertilizer Association of India (1980-81). Accurate figures for DDT used in agriculture (1960-72) are not available.