# CORRESPONDENCE

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results should the crops be attacked by more than one pest. Chemical pesticides are still the main component of most methods of integrated control and several progressive companies have now developed insecticides (chlorvinphos) that are particularly suitable for this purpose.

Integrated pest management is now accepted and promoted by WHO and FAO wherever scientific agriculture and progressive health programmes exist. No doubt wider use of this approach is desirable but this depends largely on the availability of well trained specialists and on the understanding by farmers of the need for restraint in the use of chemicals. In this respect the paper by Chapin and Wasserstrom is of some value, although its sensational presentation (with sub-titles such as "Deadly link") undermines the credibility of the authors involved.

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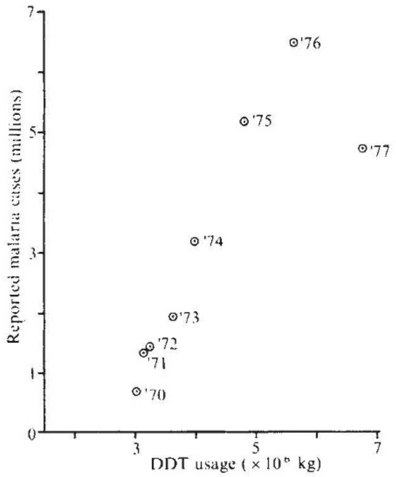
1. WHO *Official Records of the WHO* **176**, 106–124 (1969).
2. Akhtar, R. & Learmonth, A. *Malaria* (Research Paper No.3, Science Faculty, Open University, 1979).
3. Ray, A.P. *J. Communicable Dis.* **9**, 145–171 (1977).
4. Rao, T.R. *Sisir Kumar Mitra meml Lect.* (Indian National Science Academy, New Delhi, 1978).
5. Agency for International Development *Audit Report* **76-348**, 37–40 (1976).
6. *WHO tech. Rep. Ser.* No.549 (1974).
7. *WHO tech. Rep. Ser.* No.585 (1976).

Sir — It is generally agreed among malariologists that agricultural insecticides have made a contribution to selection for insecticide resistance in mosquitoes and that such resistance has made a contribution to the resurgence of malaria in Central America and South Asia. It is also generally agreed that one should be very careful before inferring a causal relationship from the discovery of a correlation between two sets of measurements.

No such care was exercised by Chapin and Wasserstrom (*Nature* 17 September, p.181–185). They infer that in El Salvador




"each kilo of insecticide added to the environment will generate 105 new cases of malaria". Taken literally and from their own data this would imply 168 million cases of the disease in a country with a population of 4.3 million.

Chapin and Wasserstrom present three figures with apparently calculated points and lines but no actual data points. Each graph has a correlation coefficient marked on it of 0.96 or 0.99, but, especially in the case of the curvilinear relationships, the reader has no means of assessing how these coefficients were calculated. Their Fig. 1 is entitled "Effect of DDT on rice production in India, 1970–77", as if the correlation between these two parameters were a simple causal one. In fact, however, it is well known that other independent factors, notably the introduction of high yielding varieties, have both boosted rice yield and allowed and/or required more insecticide usage.

The basis of Chapin and Wasserstrom's curves (Figs 2 and 3) purporting to relate malaria incidence in India during 1969–77 to DDT usage and rice production, is unclear. In the figure (see below) I have plotted the data on malaria incidence issued by the Indian National Malaria Eradication Programme (NMEP) against the data shown by Chapin and Wasserstrom (Fig.4) for DDT usage. Certainly both quantities tended to rise over those years but so did other probably relevant factors such as irrigation. My graph shows a much less startling relationship than Chapin and Wasserstrom's because, according to the NMEP data, the minimum number of cases was higher, the maximum was lower and the malaria resurgence peaked in 1976 (and is reported to have continued to fall in subsequent years). No doubt the NMEP figures greatly under-report the true incidence of the disease, but there seems no reason to suppose that this under-reporting was greater in the later years than in the earlier: Chapin and Wasserstrom give no information about any "correction factors" which they may have applied to the available data. I conclude that Chapin and Wasserstrom's graphs give a grossly misleading impression that there is a simple causal relationship between agricultural insecticides and malaria.

That the relationship is actually more complex is indicated by the following facts:

(1) Spraying of cotton crops has the, at least short term, beneficial side effect of suppressing mosquito populations.

(2) The large tonnage of insecticides used in anti-malaria spraying has certainly contributed to the selection for insecticide resistance in mosquitoes, as shown by the fact that withdrawal of this spraying has been found to lead to a levelling out or decline in the frequency of resistance genes.

(3) In the Gezira area of intensive agriculture in Sudan, resistance in *Anopheles arabiensis* is to malathion which is used in anti-malaria spraying and not to the other organophosphates used for spraying the cotton crop.

(4) Sri Lanka has very wisely banned the use of DDT and malathion in agriculture and reserved them for the anti-malaria campaign but has still had a hard struggle to contain and reverse its resurgent malaria problem.

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## A reply —

Sir — We are sorry that Professor Bruce-Chwatt disagrees so radically with our interpretation of the facts surrounding malaria resurgence in India. In our own defence, however, we would suggest that the military conflict with Pakistan, the sharp fall in the flow of American aid, the temporary food shortages that took place there between 1973 and 1975, etc., do not explain the abundant entomological reports of *Anopheles* resistance which we cited in our article — even from such prosperous agricultural regions as Maharashtra and Gujarat. These reports, together with official accounts of the deliberations within WHO and FAO, constitute the major source of evidence upon which we have based our analysis.

Finally, like Professor Bruce-Chwatt, we appreciate the difficulties of implementing effective systems of integrated pest management in tropical areas. It was during the successful development of one such system in southern Mexico that the idea of writing this article first occurred to us.

In reply to Dr Curtis, it is unfortunate that in the process of reproducing our illustrations, many of the data points have become difficult to discern. Disregarding for the moment the 1979 figure on malaria incidence in India, however, Dr Curtis's information suggests an even higher correlation between DDT usage and the spread of disease than we have calculated. As for the question of decreased transmission, it may well be true that malaria in India peaked in 1976, but the 1977 figure he cites has been questioned by numerous specialists and in any case does not contradict our argument about pesticide abuse.

As for the four points raised in his letter, we offer the following response:

(1) It is precisely the short-term beneficial effect of insecticides that encourages cotton growers and public health officials to apply them. What we have argued, however, is that the disadvantages of insect resistance soon come to outweigh these rather ephemeral benefits.

(2) It is difficult to separate the effects of insecticides used for malaria control and those used in agriculture. What is clear, however, is that the decline in resistance after anti-malaria programmes are discontinued is a limited and unfortunately rare phenomenon.

(3) Although it may be true that *Anopheles* mosquitoes in Sudan are not resistant to the organophosphates used on cotton, most countries have not been so lucky. How does Dr Curtis explain the almost complete and apparently irreversible resistance among malaria vectors in India, South-East Asia and Central America?

(4) As the case of Sri Lanka indicates, restricting the use of a particular chemical does not guarantee that mosquitoes will remain susceptible to it: application of related (and even unrelated) compounds is often sufficient to stimulate resistance. Moreover, if initial success in combating malaria leads to the reduction of screening and treatment procedures (as commonly occurs), epidemic resurgence will indeed be exceedingly difficult to control.

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