

Resistance of sandflies to DDT in Kala-azar endemic districts of Bihar, India

Editor – Vector resistance to insecticides has been highlighted by Kasap et al. in their comparative study relating to malaria in Turkey (1). In India, phlebotomine sandflies used to be considered highly susceptible to all insecticides; but indiscriminate use poses a problem of resistance, for example, in Bihar, where spraying with DDT has continued since 1976 (2). Resistance to DDT in *Phlebotomus argentipes*, a proven vector of Kala-azar in India, was reported for the first time in a village of Samastipur district (3). We therefore undertook a study in seven villages in Patna, Darbhanga, Samastipur, and Vaishali districts to ascertain the present trend of susceptibility of sandflies to DDT spraying in endemic areas of Bihar.

Batches of 15–20 bloodfed and active half-gravid female adult sandflies were collected in the early morning by torchlight, with aspirators, and exposed to DDT 4% for one hour. Their susceptibility status was determined by the WHO test kit, and mortality was recorded after 24 hours. All the sandflies dead or alive were identified (4). The temperature and humidity were maintained at 24 ± 2 °C and 75%, respectively. A village in Patna district, where no DDT spraying had been carried out for 25 years, was selected as a control.

Effectiveness of the insecticide was found to be 100% in Patna and Samastipur districts. In Paswantola and Chakkatola villages (Darbhanga district) and Dakshinitola village (Vaishali district), vector mortality was 98.24%, 96.28% and 97.57%, respectively, whereas in another village in Vaishali (Ravidastola) susceptibility was 78.5% in 1998 and 71.42% in 1999.

Our observations indicate that the leishmaniasis vector *Ph. argentipes* has developed resistance to DDT in an area of Bihar, in line with findings reported from Samstipur district (3). Further study is therefore required in endemic areas. ■

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Conflicts of interest: none declared.

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The Mwanza trial: Dr Hudson replies

Editor – In January 2001 the *Bulletin* published my review of the Mwanza and Rakai community-based trials of sexually transmitted disease (STD) interventions, highlighting the role of behavioural factors in the effectiveness of STD treatment (1). My review was written as objectively as possible, though a literature search would show that I have been promoting behavioural intervention since 1988. In addition, my criticism of publications from Rakai has been more forthright than my comments on the Mwanza trial.

Now that the architects of the Mwanza trial have claimed that “most of [Hudson’s] assertions are inconsistent

with published data” (2), I feel free to express my true feelings about that trial. In my view the Mwanza trial was unethical in its timing, as it should have followed a similar trial of a behavioural intervention. The 1999 detailed publication of the trial makes the point that as late as 1993 “few schools were providing AIDS prevention education” (3).

Could it be that the Mwanza trial was designed to minimize the possibility that detractors would claim the effect was mediated by behavioural change? No sample size calculations were published to justify the interview of only one in eight participants about sexual behaviour. Why did the published papers not use baseline data on prevalence of STDs and HIV to assess bias?

Given that the delay in interviewing was as little as 4 months after the baseline survey in some communities (4), a remarkable 10% of the population had moved away from the area. The proportion was 11% (90/810) of women and 8% (53/688) of men (Yates $X=4.62$, $P=0.03$). Furthermore, only 16% (97/599) of females interviewed were aged 15–19 compared with 21% (110/518) of males (Yates $X=4.35$, $P=0.04$). In the baseline survey there were more young women than young men, and the strongest risk factor for HIV infection in women was travel outside of the district (5). Could it be that most high-risk women in Mwanza evaded the interviewers?

Furthermore, the significance levels of comparisons of behaviour between intervention and control communities were not given. It is possible that many were in the $P=0.05$ to $P=0.10$ range. Other important data were not published: given that a year elapsed between initiation of the first and last pairs in the trial (6), the authors could have looked for a time-dependent intervention effect for both follow-up STD prevalence and HIV incidence. More worrying is the failure of the team to report data from the cohort of 100 men sampled randomly from each community (6) or to present data on intervention effect on STDs by study pair. If their hypothesis holds that 80% of interven-

tion effect on HIV incidence is accounted for by effect on STD rates (2), the intervention effect for STD prevalence and incidence by study pair should have closely matched that of HIV incidence. ■

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Conflicts of interest: I have been trying to obtain funding for a randomized trial of behavioural intervention, but this is not the counselling trial suggested in my article.

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