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# Genetic diversity in *Trypanosoma (Trypanozoon) brucei* isolates from mainland and Lake Victoria island populations in south-eastern Uganda: epidemiological and control implications

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## Genetic diversity in *Trypanosoma (Trypanozoon) brucei* isolates from mainland and Lake Victoria island populations in south-eastern Uganda: epidemiological and control implications

The Buvuma Islands, a collection of approximately 48 islands and islets, are situated off the northern shores of Lake Victoria, in Mukono District, Uganda. A mass evacuation of the islands' inhabitants was undertaken by the authorities in the 1940s because of an epidemic of sleeping sickness, the causative parasites being transmitted by *Glossina palpalis* (Mackiehan, 1944). Later, however, the mid 1980s. As part of this programme, isoenzyme data for 79, previously uncharacterized, *Trypanozoon* isolates collected between 1988 and 1993 (53 from humans, nine from cattle and 17 from *G. f. fuscipes*), including eight stocks from the Buvuma Islands, were analysed. [Full details of the previously uncharacterized primary isolates included in the current study are available from the senior author, J. C. K. Enyaru, on request.]

that beginning in 1976 in south-eastern Uganda (Abaru, 1985; Mbulamberi, 1989), affected these islands. This epidemic was caused by *Trypanosoma brucei rhodesiense* and began after the trypanosomiasis spread from its endemic areas along the shores of Lake Victoria in Busoga to the districts of Tororo, Mukono and Mbale (Enyaru *et al.*, 1992). It can probably be linked to the breakdown of tsetse control and the suspension of medical surveillance following the political turbulence of 1971 onwards.

Tsetse-control operations, initially involving ground and aerial spraying and subsequently the use of pyramidal traps and live-bait technology, were put in place on mainland, south-eastern Uganda from the late 1980s and these successfully curbed the epidemic in the area. However, as these control operations did not include the Buvuma Islands, the tsetse populations on these islands remained largely unaffected. A survey in 1988 (Ogwal and Kangwagye, unpubl. obs.) revealed that *G. fuscipes fuscipes* was the principal vector of trypanosomiasis on the islands.

A long-term programme to monitor sleeping sickness in south-eastern Uganda, organized by the Livestock Health Research Institute (LIRI) [formerly the Uganda Trypanosomiasis Research Organization] began in Stevens *et al.* (1992) was employed, to exclude several highly polymorphic enzymes used in a number of previous studies of *Trypanozoon* (Gibson *et al.*, 1980; Godfrey *et al.*, 1990). To allow a meaningful comparison of the results of this study with those of earlier, related investigations, the data for the 79 isolates were combined with those for the 151 isolates studied by Enyaru *et al.* (1993) and all were then analysed by the same numerical methods. Additional zymograms (for TDH, SOD<sub>A</sub> and SOD<sub>B</sub>) were prepared for the 151 stocks that had been studied before and this led to some re-classification. The 230 stocks compared (135 from humans, 47 from cattle, 17 from pigs, two from dogs and 29 from *G. f. fuscipes*; see Table) were isolated in Tororo, Mukono including the Buvuma Islands (only in 1993) and in the three districts of Busoga (Iganga, Kamuli and Jinja). A full genetic analysis was not undertaken because of the primarily epidemiological and control aims of the study; Hide *et al.* (1994, 1996) give a detailed review of this aspect of trypanosomiasis in Uganda. Estimates of tsetse infection rates within the study area indicate that only 0.14%–0.78% of flies are infected with *T. brucei*, even during epidemic periods (Okoth and Kapaata, 1986; Maudlin *et al.*, 1990).

The 230 stocks were separated into 73 zy-

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