Correspondence to the Editor

Nomenclature of trypanosomes of the subgenus *Trypanozoon*

It is probably widely accepted by now that the trypanosomes causing surra, *Trypanosoma evansi* (Steel, 1885), and dourine, *T. equiperdum* Doflein, 1901, have been derived from the African tsetse-transmitted species *T. brucei* Plimmer & Bradford, 1899, the causative agent of nagana and human sleeping sickness. The arguments for this theory, so convincingly summarized by Hoare (1972: *The Trypanosomes of Mammals*. Oxford: Blackwell) make a strong case indeed.

Hoare (1966: *Ergebnisse der Mikrobiologie, Immunitätsforschung und experimentellen Therapie*, **39**, 43) suggested that the trypanosomes of African sleeping sickness, surra and dourine should be considered as subspecies of the original African parasite and that their correct designation would be: *T. brucei brucei* (nagana), *T. brucei gambiense* and *T. brucei rhodesiense* (sleeping sickness), *T. brucei evansi* (surra) and *T. brucei equiperdum* (dourine). Although most authors have followed Hoare's trinomial nomenclature for the tsetse-transmitted subspecies, this has not generally been the case for the surra and dourine trypanosomes. Gibson *et al.* (1980: *Advances in Parasitology*, **18**, 175), on the basis of their isoenzyme work, proposed uniting *T. evansi* with the tsetse-transmitted parasites under the name *T. brucei*, but only in more recent years have some authors started to use the name *T. brucei evansi* for the surra parasite (Zweygarth *et al.*, 1984: *Annales de la Société Belge de Médécine Tropicale*, **64**, 309 and 1986: *Tropical Medicine and Parasitology*, **37**, 105). However, attractive as it may be to unite the various parasites under the name first given to the original African species, *T. brucei*, there can be no doubt that this approach clashes with the law of priority of the International Code of Zoological Nomenclature (1985). The name *T. evansi* predates that of *T. brucei* by 14 years. Unless therefore it were to be shown that *T. evansi* is not a valid name, the parasites discussed here should be united under the species name of *T. evansi*. Using a trinomial nomenclature, the various parasites would be designated as follows: *T. evansi evansi* (formerly *T. evansi*), *T. evansi brucei* (formerly *T. brucei*), and *T. evansi equiperdum* (formerly *T. equiperdum*).

Under the name *T. evansi brucei* would be included nosodemes which are not infective to man (formerly *T. b. brucei*) and others causing Rhodesian sleeping sickness (formerly *T. brucei rhodesiense*). A special case might be made for retaining the former *T. gambiense* as a separate subspecies, *T. evansi gambiense*, on the basis of isoenzyme and antigen studies (summarized by Tait *et al.*, 1984: *Parasitology*, **89**, 311).

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Editorial comment

Professor Uilenberg makes an interesting point, valid if one accepts the synonymy of *Trypanosoma evansi* and *T. brucei*. However, adopting the change would lead to an alarming amount of confusion. There seem to be two possible solutions to the dilemma: (1) to petition the International Commission on Zoological Nomenclature to retain the name *T. brucei* on the grounds of stability, or (2) to accept that the great difference in life cycle between *T. brucei* and *T. evansi* — the presence or absence of cyclical development in a vector — is sufficient to warrant retaining their specific differentiation. The latter possibility has the merit of simplicity.

Use of difluoromethylornithine (DFMO, eflornithine) for late-stage African trypanosomiasis

With regard to the recent article by RASEROKA & ORMEROD (1986: *Transactions*, **80**, 634) on the effects of trypanosomicidal drugs in different parts of the brain, we feel that the authors made a significant omission. While Raseroka & Ormerod were able to suggest that one of the combinations they tested might be evaluated in man, they failed to point out that a relatively new compound, difluoromethylornithine (DFMO, eflornithine hydrochloride), included in their tests, has already been clinically proven to be highly effective against brain infections in man when used as a single agent. Overwhelming evidence from both the laboratory and the clinic show that DFMO is an effective drug against late-stage trypanosomiasis involving the central nervous system. BACCHI *et al.* (1987: *American Journal of Tropical Medicine and Hygiene*, **36**, 46) have shown that monotherapy results in complete cures of infected mice with CNS involvement. Furthermore, clinical cures by DFMO of a total of 109 cases of late-stage *Trypanosoma brucei gambiense* trypanosomiasis (100 refractory to arsenicals) without significant toxicity are now well documented (SCHECHTER & SJOERDSMA, 1986: *Parasitology Today*, **2**, 223; MCCANN *et al.*, 1986: *American Journal of Tropical Medicine and Hygiene*, **35**, 1153).

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Use of difluoromethylornithine (DFMO, eflornithine) for late-stage African trypanosomiasis: a reply

Dr Peter McCann and colleagues complain that our paper (1986: *Transactions*, **80**, 634) failed to mention