adjacent to the tourist area [9]. The increasing demand from tourist hotels and restaurants has led to a rapid increase in the number of small farms in which pigs are often fed swills containing raw pork scraps from restaurants and hotels, thereby providing a mechanism for the amplification of the *Trichinella* transmission cycle.

Finally, the use of 'ecological' or 'organic' pig farms, in which pigs are fed outdoors and pastured freely, might increase the risk of *Trichinella* transmission from wildlife and rodents to domestic swine [10]. Of the 19 infected pigs that were detected by microscopy in abattoirs in Nanning (Guangxi Zhuang Autonomous Region) between 2001 and 2002, 16 were from the mountainous areas in which animals are raised in the open and only three of the infected pigs came from large industrialized farms [11]. Thus, it seems that, although the organic and ecological farms rear fewer animals, the incidence of *Trichinella* infection among them is disproportionately high.

Human trichinellosis was extremely rare in the developing western areas of China, so local physicians are unfamiliar with its symptoms and signs [12], and misdiagnosis is likely to occur as a consequence. To prevent a major re-emergence of human trichinellosis in China, it is essential that the outbreaks of this infection be recognized and dealt with at a national level, using the effective control measures of health education, regulation of pig-feeding practices and mandatory inspection of all slaughtered animals under a standardized quality-assurance system.

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Doubts about *Trypanosoma equiperdum* strains classed as *Trypanosoma brucei* or *Trypanosoma evansi*

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We read with great interest the suggestion by Claes *et al*. [1] that some *Trypanosoma equiperdum* strains are, in fact, *Trypanosoma brucei* and that the remaining strains are *Trypanosoma evansi*. However, in our opinion, the classification of the *T. equiperdum* Onderstepoort Veterinary Institute (OVI) and Bordeaux *Trypanosoma* antigen type (BoTat) 1.1 strains as *T. brucei*, and the other *T. equiperdum* strains as *T. evansi* is premature.

Kinetoplast (k)DNA consists of two types of circle: thousands of minicircles bearing guide (g)RNA genes and dozens of maxicircles with (cryptic) protein- and rRNA-coding genes. Any differences in kDNA structure – such as the size of minicircles and maxicircles, the number of gRNAs per minicircle, and gene order and gene deletions in maxicircles – are considered to be species- or even genus-specific features [2]. In most cases, it is accepted that *T. brucei*, *T. evansi* and *T. equiperdum* belong to the subgenus *Trypanozoon* and that their bloodstream stages are often morphologically indistinguishable [3] (several biochemical and molecular methods fail to distinguish among them [4]).

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However, the mode of transmission, host range, pathogenicity and location of the parasite in its host are specific for each species [4–6]. Moreover, the presence of kDNA maxicircles in *T. brucei* and *T. equiperdum* distinguishes these species from *T. evansi*, which has only minicircles in its kDNA [7,8]. It has been suggested that maxicircles were partially and totally lost from *T. equiperdum* and *T. evansi*, respectively, rendering these flagellates unable to develop in insect vectors [8]. To our knowledge, a complete loss of maxicircle kDNA has been reported only for *T. evansi*, not *T. brucei* or *T. equiperdum*. Lun and Desser [9] proposed that the absence of maxicircle-encoded genes facilitated the use of a wide range of transmission mechanisms that enabled *T. evansi* to spread outside of Africa.

Our approach to distinguishing *T. evansi* from *T. equiperdum* is based on PCR amplification of the maxicircle kDNA fragment that encodes the NADH dehydrogenase subunit 5 gene. Amplified fragment was found in all nine of the *T. brucei* strains and five of the *T. equiperdum* strains [OVI, BoTat 1.1, Swiss Tropical Institute Basel (STIB) 818, STIB 841 and STIB 842] tested but the same fragment was not detected in any of the ten *T. evansi* strains analyzed. These results indicate that *T. equiperdum* STIB 818, STIB 841 and STIB 842 strains cannot be grouped together with *T. evansi* as suggested by Claes *et al.* [1] because the former species retains maxicircles and related genes such as NADH dehydrogenase subunit 5.

Furthermore, the proposed synonymization of *T. equiperdum* with *T. brucei* [1] does not take into consideration the marked differences in minicircle complexity between the species. Whereas *T. brucei* contains hundreds of minicircle sequence classes, both *T. equiperdum* and *T. evansi* have only a single – or at least an extremely predominant – minicircle sequence class (for review, see Refs [2,5]). Also, *T. equiperdum* OVI and BoTat 1.1 strains should not be assigned to *T. brucei* without examining minicircle complexity. It seems that kDNA is, to a varying degree, 'on the way out' from *T. evansi* and *T. equiperdum*, and dyskinetoplasty in these species can be induced by various factors. However, without detailed analyses of maxicircles and minicircles in individual strains, one cannot rule out that there is not a continuum of losses but, instead, two different degrees of kDNA loss that enable two specific lifestyles that characterize the two species. In our opinion, the available data do not justify abandoning the current species concept within the subgenus *Trypanozoon*.

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The pitfalls of assigning disease syndromes to pathogens and vice versa

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For many years, it was customary to assign parasite names in accordance with their associated disease syndrome. However, this practice has changed radically during the past two decades and there is now a tendency for disease names to be conserved, although the list of organisms attributed to them increases as levels of inter- and intra-specific variation are discovered: for example, cutaneous leishmaniasis. However, in some cases, differences in strain identification could be due to mistaken identities, 'mix-ups' or different identification methods.

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