Trypanosomiasis caused by *Trypanosoma evansi* (Surra) is endemic in SE Asia where it is a significant, but often underestimated cause of mortality in livestock. Recent estimates suggest that 22% of Indonesian buffalo infected with *T. evansi* either die or are sold for salvage slaughter. In the last 10-15 years a series of severe outbreaks of surra have occurred in the Philippines. What is most puzzling is that the epidemiology observed in these outbreaks differs from other parts of SE Asia such as Indonesia. These differences include the observation of fatal disease in small ruminants and cattle and the observation of different presenting signs. In addition, there have been reports of isolated cases of human trypanosomiasis in India. A variety of epidemiological tools are required in order to gain a better understanding of the biological basis of the epidemiological conundrum that we are faced with. In particular molecular tools can provide us with unique methods of providing us with insight but they must be used with caution to ensure that the benefit from the research reaches the key stakeholders – the farmer. This talk will describe a multidisciplinary approach to unraveling the epidemiology of surra in the Philippines.

(61) Human infection by *Trypanosoma evansi* in India: diagnosis, treatment, genetic and epidemiological investigations

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The first case of human trypanosomiasis in Asia was evidenced in 2004 in Seoni, India. A farmer presented a fluctuating trypanosome parasitaemia associated with febrile episodes for five months. Clinical and biological examinations, including parasitological, serological and molecular biological tests confirmed the infecting species as *T. evansi* (Joshi et al., 2005). Suramin was efficient to cure the patient (Joshi et al., 2006). The *SRA* gene (Xong *et al.*, 1998) was not detected by PCR in the *T. evansi* DNA, which had homogeneous kDNA minicircles of Type A (Truc et al., 2006). The parasite

appeared to be a typical *T. evansi*, suggesting that the explanation for this unusual infection may lie with the patient. Normally humans exhibit innate immunity against *T. evansi and T. brucei*, and in the latter case this immunity is known to involve apolipoprotein L-I (apoL-I, Vanhamme et al., 2003). The serum of the patient was found to be devoid of trypanolytic activity, and this was linked to the absence of apoL-I due to frameshift mutations in both *apoL-I* alleles (unpublished data). Therefore, the lack of apoL-I was sufficient to explain the human infection by *T. evansi*. Because of a mechanical transmission by insects was suspected, a serological investigation was conducted in the patient village in 2005. Out of 1806 individuals tested using the Card Agglutination Test for Trypanosomiasis/*Trypanosoma evansi* (Pathak et al., 1997), no trypanosomes were detected in the blood of 60 people who were positive at a significant serum dilution (1:4). The results indicate a frequent exposure of the human population to *T. evansi* in the study area, suggesting frequent vector transmission of parasites to humans (Shegokar et al., 2006). Further investigations are required to evaluate the importance of this phenomenon and the potential emergence of a new zoonotic disease (Brun, 2005).

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