# ABSTRACTS

## IS SLEEPING SICKNESS SPREADING?

Human African trypanosomiasis, or sleeping sickness, causes around 100 000 deaths every year. There are two pathogens involved: *Trypanosoma brucei rhodesiense*, which causes an acute form of the disease, and *T. b. gambiense*, which causes a chronic form. *T. b. rhodesiense* is found in East Africa and *T. b. gambiense* in Central and West Africa. However, although traditionally the 2 parasite species, and so the two diseases, are thought of as geographically separate, Uganda is a region in which there could be potential overlap, and where two disease foci are expanding towards each other.

The history of sleeping sickness in this region is interesting. It was first recognised in southeast Uganda in 1898 and in the north-west of the country in 1902. However, modern refugee movements have spread *T. b. gambiense* so that it forms a contiguous focus with south Sudan. This raises the possibility that refugees may carry this particular form into areas that are endemic for *T. b. rhodesiense*. In the 1940s there was an epidemic in southeast Uganda, probably spread by animals. The disease re-emerged in this area between 1976 and 1983, when nearly 20 000 people were diagnosed with sleeping sickness. An outbreak of *T. b. rhodesiense* disease in 2000 was associated with cattle restocking and 18% of cattle were found to be carrying the human pathogen. Since then, the disease has spread to two adjacent districts and the two foci are predicted to merge, which could complicate diagnosis and treatment.

It was this potential overlap of foci that prompted Kim Picozzi and her team to investigate, using molecular diagnostic tools, whether the two species of parasite that cause sleeping sickness have started to overlap ranges. They took blood from 231 available patients with sleeping sickness who presented between June 2001 and June 2005 in central Uganda and between July and September 2003 in northwest Uganda. They also analysed sleeping sickness records in Uganda between 1985 and 2005. Their patients were drawn from sleeping sickness treatment centres in central and northwest Uganda and in south Sudan.

They found that, at the time of sampling, the two foci of sleeping sickness remained discrete, but also that the area of Uganda affected by the acute form of the disease had increased by 2.5-fold since 1985. The acute form has spread to three new districts within the past 5 years through movement of infected livestock. The authors suggest that without preventive action that is targeted at the livestock reservoirs of this zoonotic disease, it is likely that the two disease foci will converge. Sleeping sickness is already a neglected disease, affecting only the developing world. Any overlap in the ranges of the acute and chronic form is likely to have a major impact on the diagnosis and treatment of the disease. They recommend real-time monitoring, using molecular diagnostic tools targeted at both livestock and human patients.

Picozzi K, *et al. BMJ* 2005; **331:** 1238-1241.



## DOLPHINS AND DEPRESSION

There has been much research suggesting that animals and nature can be therapeutic for sick and disabled people. There is also an increasing demand among the public for alternative treatments in psychiatry and a lack of adequately controlled and designed research studies to look at alternative treatment approaches. Christian Anatonioli and Michael Reveley, the authors of this recent paper in the *British Medical Journal*, place their study within the context of the concept of biophilia. This term was first used by psychologist Erich Fromm to underline the need for 'cultivating the capacity for love as a basis for our mental health and emotional wellbeing'. The authors interpret this to show how

human health and wellbeing are strictly dependent on our relationships with the natural environment.

Anatonioli and Reveley evaluated the effectiveness of animal-facilitated therapy with dolphins in the treatment of mild to moderate depression, in the context of the biophilia hypothesis. They recruited 30 patients from the USA and the Honduras and the study was carried out in the Honduras. Patients were randomised to either a treatment group or a control group. Treatment was interesting. All patients in the treatment arm were assigned to an animal care programme and all trials were conducted in the presence of dolphins. Participants were asked to play with, swim with and take care of the dolphins. They all had an introductory session at which they learned about dolphin behaviour and playing safely with the animals. Initial interactions were in the presence of the dolphin's trainers. The interaction also involved touching the dolphins and included half an hour of free swimming and playing with the dolphins. Those in the control group were assigned to an outdoor nature programme, in which they swam and snorkelled in a barrier coral reef for an hour a day, but without the presence of dolphins. The participants who had been randomised to the programme without the dolphins had an opportunity to swim with the dolphins for an hour at the end of the 2-week period of study.

Using measures of depression, including the Beck depression inventory, Anatonioli and Reveley found that the group who were randomised to interact with the dolphins had fewer symptoms of depression than those who had simply been swimming around the coral reef. From this, they concluded that animal therapy was effective in alleviating mild to moderate symptoms of depression. Intuitively, the idea of contact with other animals being effective in the treatment of depression is intriguing. But to me, the paper seems to conclude rather too much from the presence of dolphins alone. The patients in the treatment arm of the programme had a lot more to do than those who simply swam around the coral reef. That, along with increased contact with people through the dolphins' trainers, could have had a major impact on depression, which is an isolating illness. However, it is an idea that is worth following up.

Anatonioli C, Reveley MA. *BMJ* 2005; **331:** 1231–1233.

**Bridget Farham**

## SINGLE SUTURE

### PARACETAMOL AND LIVER FAILURE

Paracetamol, known as acetaminophen in the USA, is generally thought to be a safe and effective pain killer. But, between 1998 and 2003, the number of cases of liver failure caused by the drug nearly doubled in the USA and many of these cases were caused by accidental poisoning. William Lee and colleagues from the University of Texas Southwestern Medical Center followed up patients in acute liver failure who were in a coma. Of the 275 people who had paracetamol poisoning, 8% received a liver transplant, 65% survived without one and 27% died. Researchers found that people who had intentionally overdosed tended to be identified and treated faster, but had similar liver damage to those who had overdosed accidentally. Many of the people who had overdosed accidentally had taken only 10 g of the medication each day for about 3 days. This is the equivalent of 20 pills per day instead of the recommended 8. Other people had unknowingly taken two products that contained paracetamol.

*Hepatology* 2005; **42:** 1364.