

## WEB WATCH

Kid's stuff

If you are a parent or a teacher, you have a great excuse to visit the 'Neuroscience for kids' web site. If not, you will just have to hope that nobody looks over your shoulder while you browse.

Run by Eric Chudler of the University of Washington, the site provides a wealth of resources on all parts of the nervous system. The colourful homepage invites you to explore the nervous system, after which you can select a specific subject. For example, if you select sensory systems, you can find a description of the visual pathway, with links to related subjects — the retina and the eye — and to games and activities related to vision. (These are, of course, my favourite bits.)

But there's more. Visitors to the site can send an e-mail to Dr Chudler, and ask a question related to neuroscience. Questions are answered by 'The neuroscientist network', an international group of neuroscientists who answer queries on everything from the number of neurons in the spinal cord to the effects of temperature on the shape of the action potential. There are graded lesson plans and resources, short articles on neuroscience in the news, and a helpful list of neuroscience links. And did you know that a giraffe sleeps for only around two hours each day? Facts like this can be at your fingertips if you follow the link to the treasure trove of brain trivia.

'Neuroscience for kids' is a lot of fun for those of us who never really grew up. But it is also a great way of getting younger students to think about how they think.

Rachel Jones



SLEEP

## Out for the count

Around 1 person in 2,000 suffers from narcolepsy — a debilitating disorder that causes extreme sleepiness and is associated with obesity. Recent work has implicated a pair of neuropeptides, the orexins (also known as the hypocretins), in narcolepsy, but their exact involvement has remained unclear. Now, Hara *et al.* have addressed this question by knocking

out the hypothalamic neurons that contain orexin.

Previous work had shown that mice lacking the gene for orexins appear to be narcoleptic, and a single human patient has been identified in whom a mutation in this gene led to early-onset narcolepsy. In most sufferers, though, the disease is not monogenic, and the

symptoms develop around adolescence. These patients show decreased orexin levels and apparent loss of the orexin-containing neurons of the lateral hypothalamic area.

In an attempt to reproduce this phenotype, Hara *et al.* generated mice carrying a transgene coupled to the *prepro-orexin* promoter so that the gene product, a truncated form of ataxin 3 with 77 polyglutamine repeats that causes apoptosis, would be expressed only in the orexin neurons. When the transgenic mice were born they appeared to be normal, but over the subsequent weeks they showed a gradual loss of orexin neurons, which was almost complete by 15 weeks.

At six weeks of age, when the loss of neurons was between 75% and 90%, the mice began to show symptoms of narcolepsy. When filmed at night (when mice are most active) using infrared video, they showed frequent episodes of 'behavioural arrest' (periods of inactivity) like those seen in *prepro-orexin* knockout mice. The mice also showed increased REM sleep and a highly fragmented sleep/wake pattern during the dark period, and disturbed sleep patterns during the light period.

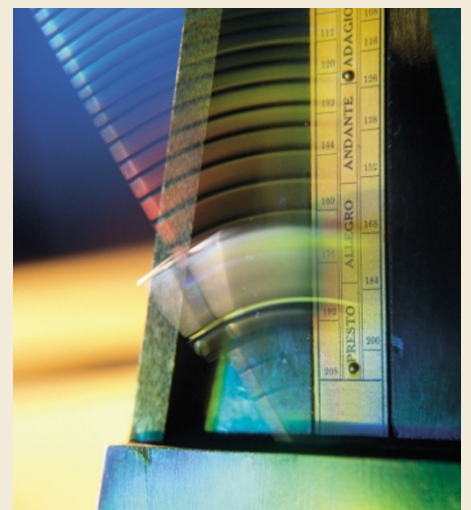
AUTONOMIC NERVOUS SYSTEM

## Rhythms of the periphery

An old colleague of mine used to liken the autonomic nervous system to the suburbs of a city in a condescending way: "why bother going there if there's never anything going on?". Of course, this patronizing view is quite inaccurate; we may not know a lot about what happens in the periphery, but this should actually encourage us to find out more about it. Take, for example, the physiology of the sympathetic neurons that control vasomotor tone. These postganglionic cells show bursts of activity, with a periodicity that is related to the cardiac and respiratory cycles — a coordination that might help to optimize blood supply to every organ. How is this bursting activity controlled? One leading

idea is that an oscillatory network in the brainstem entrains the sympathetic neurons, causing them to fire synchronously. In fact, there seem to be not one, but several oscillatory networks, as there is variability in the rhythmic patterns of activity measured in the vascular systems of different organs. And now, a recent paper in *The Journal of Physiology* reports that afferent somatic activity can reset the oscillatory networks and transiently synchronize sympathetic neuron firing, adding an additional complication to this system.

Staras and his colleagues investigated the effect of radial nerve stimulation on the burst firing of the postganglionic



neurons that innervate the caudal ventral artery of the rat tail. They found that, following the stimulus, the activity of sympathetic neurons was first reduced, and subsequently synchronized, in a transient manner. The authors interpreted these findings as evidence that stimulation of the