IN THE NEWS

Juggling boosts the brain

When people spend three months learning to juggle, according to a paper published in *Nature*, parts of their brains grow. "Researchers in Germany split 24 students into two groups, one of which was given three months to learn a classic three-ball cascade juggling routine. Brain scans were then carried out on both sets of volunteers." (*The Scotsman*, 22 January 2004). The brains of the jugglers and non-jugglers were scanned before and after the three-month learning period.

According to *BBC News Online* (22 January 2004), "Jugglers had more grey matter — which consists largely of the nerve cells — in the mid-temporal area and the left posterior intraparietal sulcus, which both process visual motion information."

Arne May, of the University of Regensburg, Germany, led the group that carried out the research. May said, "Our results challenge our view of the human central nervous system. Human brains probably must be viewed as dynamic, changing with development and normal learning." (*CNN*, 22 January 2004).

When the same groups were scanned again after another three months, the increase in grey matter had reduced. Talking to *BBC News Online*, Vanessa Sluming of the University of Liverpool, UK said, "It would be interesting to know at what point this acquired grey matter can be retained. Does it mean you need to continuously practise the acquired skill to retain it, or at some point have you done enough to retain it?"

Rachel Jones

SENSORY SYSTEMS

The flavour of long life

In the nematode *Caernorhabditis elegans*, mutations in genes that are required for sensory transduction can dramatically extend lifespan. Writing in *Neuron*, Alcedo and Kenyon use laser ablation of individual neurons to identify a subset of sensory neurons that interact to regulate longevity, and find that the sensory control of lifespan is surprisingly complex.

Mutations that inhibit sensory function in *C. elegans* are thought to extend lifespan in adults by decreasing signalling through a pathway that involves DAF-2, a homologue of the insulin/IGF-1 receptor. Lifespan can also be increased by mutations in *daf-2*, and this effect depends on the transcription factor DAF-16. Early in life, this pathway also mediates the formation of a specific larval state called the dauer that is specialized for survival in unfavourable conditions.

To investigate the relationship between sensory input, DAF-2 signalling and longevity, the authors killed specific pairs of sensory neurons by laser ablation. Three pairs of gustatory neurons, called ADF, ASI and ASG, inhibit dauer formation — ablation of these neurons causes the worms to form dauers. Alcedo and Kenyon found that ablation of either the ASI or the ASG neurons significantly extended lifespan, but that ablation of the ADF neurons did not. There are also neurons that promote dauer formation, called the ASJ and ASK neurons, but ablating these did not reduce lifespan. However, if either of these pairs of neurons was killed, it suppressed the longevity induced by ASI ablation. So, some neurons promote longevity, whereas others inhibit it.

Although it seems that regulation of lifespan and dauer formation might have overlapping mechanisms, the authors also found that lifespan can be regulated independently. If they ablated a pair of olfactory neurons, called AWA, lifespan was extended, and this effect was amplified if another pair of olfactory neurons (the AWC neurons) were also killed. But ablation of these neurons has no effect on dauer formation. A mutation in the *odr-7* gene, which is specifically required for AWA function, also induced longevity.

The gustatory neurons seem to act through the DAF-2 pathway to regulate longevity, as the effect of ablating these neurons depends on DAF-16. However, the lifespan extension that is caused by ablating the olfactory neuron AWA depended only partly on DAF-16, indicating that the olfactory and gustatory neurons act through partially independent pathways to regulate longevity.

So how do these neurons interact to regulate longevity? Among the gustatory neurons, it seems that some inhibit longevity — such as ASI and ASG — whereas others, including ASK, promote it. Given that ablation of ASK alone had no effect on lifespan, it is most likely that ASI and ASG inhibit some

longevity-promoting effect of ASK. In this model, ablation of ASI or ASG lifts the inhibition, allowing ASK to promote longevity. However, when ASK is also ablated, the effect is reversed.

Presumably, specific environmental cues that are detected by these gustatory and olfactory neurons can influence longevity. One attractive model had been that the pheromone that induces dauer formation in juvenile animals induces longevity in adults. However, the authors showed that this was not the case. It is possible that the signals include food-related substances.

The AWC and ASI neurons express a putative chemosensory G-protein-coupled receptor, STR-2, and the study showed that reductions in levels of this receptor also extended lifespan, supporting the idea that specific cues are involved in longevity.

Insulin/IGF-1 signalling pathways can also influence lifespan in flies and mammals, and there is evidence that food-related sensory input can alter insulin levels in humans. So it is possible that sensory inputs can work through neuroendocrine mechanisms to alter longevity in organisms other than *C. elegans*. Further work should investigate this intriguing possibility.

Rachel Jones

References and links

ORIGINAL RESEARCH PAPER Alcedo, J. & Kenyon, C. Regulation of *C. elegans* longevity by specific gustatory and olfactory neurons. *Neuron* **41**, 45–55 (2004)

WEB SITE

Kenyon laboratory: http://wormworld.ucsf.edu/labhomepage.html