IN THE NEWS

Fishy business

Apparently, we've been wrong to think for all these years that fish were dumb. The idea that fish have a three-second memory and little or no cognitive ability is obsolete, according to three experts writing in the journal *Fish and Fisheries*.

The researchers — Calum Brown, Keven Laland and Jens Krause — are from the universities of Edinburgh, St Andrew's and Leeds, in the UK. They claim that "Far from being instinct-driven dunces, held back by a three-second memory, fish are cunning, manipulative, cultured and socially aware" (*The Scotsman*, 1 September 2003). Evidence for the cognitive abilities of fish comes from observations that they can build complex nests, use tools and have impressive long-term memories. According to the *Sydney Morning Herald* (31 August 2003), "fish not only recognised individual shoal mates, but monitored the social prestige of others and tracked relationships". They also cooperate when faced with a predator or potential food source.

The scientists are quoted in *The Scotsman* as saying, "Although it may seem extraordinary to those comfortably used to pre-judging animal intelligence on the basis of brain volume, in some cognitive domains, fish can even be favourably compared to non-human primates". They go on to describe the "Machiavellian strategies of manipulation, punishment and reconciliation" that are used by fish in their social interactions.

Of course, none of this explains why my pet goldfish kept jumping out of its bowl…

Rachel Jones

DEVELOPMENT

A new source of RA

The vitamin A metabolite retinoic acid (RA) is known to be important for early nervous system patterning, but little is known about its involvement at later stages of brain development. As reported in the *Journal of Neuroscience*, Zhang *et al.* have identified a new source of RA that indicates a role in the patterning of anterior hindbrain derivatives, such as the nuclei of the precerebellar system.

The presence of RA-synthesizing enzymes, such as retinaldehyde dehydrogenase-2 (RALDH2), is a reliable indicator of a source of RA. Zhang *et al.* showed that the *Raldh2* gene is expressed in the meninges — a layer of tissue of mesenchymal origin that surrounds the brain — from around embryonic day 13 in the hindbrain region. The authors confirmed that the meninges can secrete RA by showing that isolated meningeal tissue could release RA into a tissue culture medium.

To find out where RA signalling was taking place, the authors examined transgenic mouse embryos in which a RA response element controlled the expression of a *lacZ* reporter gene. They showed that *lacZ* was expressed in cells that migrated beneath the meninges to colonize precerebellar nuclei. They also found that the RA catabolic enzyme CYP26B1 was expressed in the deeper layers of the hindbrain, perhaps indicating that other hindbrain tissues need to be protected from the effects of RA.

This is not the first time that the meninges has been shown to secrete growth and differentiation factors, but this case is unusual in that the synthetic enzyme is not evenly distributed. *Raldh2* is expressed at higher levels in the dorsal than the ventral meninges, raising the tantalizing possibility that the meninges

LEARNING AND MEMORY

Consolidating reconsolidation?

Reconsolidation — the idea that a memory, once retrieved, returns to a labile state that is susceptible to interference and must be reconsolidated into long-term storage — is controversial and surrounded by apparently conflicting data. New results from Eisenberg *et al.* might help to reconcile some of these findings.

The classic finding in support of reconsolidation is that, in some cases, a protein synthesis inhibitor given at or just after memory retrieval can cause subsequent loss of a previously stable memory. Presumably, the retrieved memory cannot be reconsolidated without protein synthesis. However, in other cases, a protein synthesis inhibitor can instead prevent extinction of a memory (which usually develops when a conditioned stimulus is presented without its associated unconditioned stimulus), so that the original memory persists when it would normally fade. Extinction is considered by many to be a process of learning, in which one memory trace replaces another, rather than simply loss of the conditioned memory, and it is this learning that is presumably prevented by the protein synthesis inhibitor.

Eisenberg *et al.* used two behavioural tests to investigate this apparent contradiction. In conditioned taste aversion in rats, a single training trial in which a taste is paired with administration of an agent that causes nausea is enough to cause aversion to the conditioned taste. Subsequent exposures to the taste without the nausea produce extinction, so that the aversion is lost — unless a protein synthesis inhibitor such as anisomycin is administered. However, if the animals are trained repeatedly on the aversion experiment, rather than just once, it takes many retrieval trials to produce extinction. In this case, the authors discovered that treatment with anisomycin during retrieval causes more rapid loss of the original association, rather than impairing extinction.

Another type of learning, fear conditioning in the medaka fish, showed similar results. In this case, a light is paired with a mild electric shock, so that the light will subsequently cause a fear reaction. If an anaesthetic is applied immediately after training, it blocks consolidation of the association into long-term memory. If it is applied after a single retrieval trial — which would not normally cause extinction — it reduces the fear response, indicating that it has interfered with reconsolidation of the original memory trace. By contrast, if the anaesthetic is applied after the tenth retrieval trial, when extinction would normally have occurred, it blocks extinction and spares the original memory.

Eisenberg and colleagues propose that these results arise from competition between two memory traces: one linking the conditioned and unconditioned stimuli, which produces a conditioned response, and a second linking the conditioned stimulus with absence of the