Monkeys can recall

Using a novel method for testing recall, researchers have shown that rhesus monkeys can recall and reproduce simple shapes (*Curr. Biol.* doi:10.1016/j.cub.2011.03.044; published online 28 April 2011). The monkeys appeared to use a procedure for recall that is similar to that used by humans.

In this study, Benjamin Basile and Robert Hampton of Emory University in Atlanta, GA, devised a recall test for monkeys that is modeled on the Rey-Osterrieth Complex Figure Test. When taking the Rey-Osterrieth Complex Figure Test, humans reproduce a complex line drawing from memory. For their version of the test, Basile and Hampton trained five adult male rhesus macaques to use a computer touchscreen to reproduce a simple figure from memory. In the study phase, a monkey saw a shape consisting of two colored boxes on a 5 × 5 grid on the touchscreen. During the test phase, the monkey saw one of the boxes on the screen (this box appeared in a new location). The monkey could then touch the screen to reproduce the absent box. If a monkey correctly reproduced the shape, he received a food reward. If a monkey made an error, he was put in 'time out' and received no reward.

The monkeys learned to reproduce two-box shapes more accurately than expected by chance. The researchers then tested whether the monkeys could reproduce new three-box shapes. The monkeys completed the novel three-box shapes with much higher accuracy than would be expected by chance, suggesting that the monkeys had learned a general reproduction rule rather than inflexible stimulus-response rules.

The authors note that virtually all memory tests used with nonhumans are recognition tests, in which animals must recognize something that is familiar. In contrast, to solve this memory test, the monkeys needed to use recall, or retrieve information about something (the shape) that was not present. Recognition and recall solve different problems. Recognizing food as familiar is quick and resistant to distraction. Recollection, such as recalling the location of distant food, is slower and vulnerable to distraction but supports a more detailed memory.

Nico Smit

"[W]e've shown that for simple shapes, monkeys have a pattern of performance for recognition and recall that mirrors that of humans," said Basile in a press release. "And their ability to immediately transfer their performance to new shapes suggests we're tapping into some general cognitive capacity. With this type of information, we are moving closer to better diagnosing and developing treatments for memory impairments in humans."

Kirsten Dorans

CLEARER VIEW OF 'ACUTE' GLAUCOMA

Glaucoma is a common eye disease and leading cause of blindness. In glaucoma, intraocular pressure (IOP; fluid pressure within the eye) increases, damaging the optic nerve and causing vision loss. The familiar 'chronic' subtype of glaucoma develops slowly and may have few symptoms; many of those affected do not know they have the condition until it is detected during a clinical eye examination. Treatment for chronic glaucoma normally includes medication and laser surgery, and the prognosis is good.

In contrast, angle-closure glaucoma (ACG) is a severe subtype of glaucoma. Acute ACG attacks are emergencies: debilitating symptoms, including severe eye pain, headache, blurred vision, nausea and vomiting, occur suddenly. If IOP is not reduced promptly, rapid vision loss may ensue. Roughly 16 million people worldwide have ACG; 4 million people are blind in both eyes as a result.

Despite its high incidence and impact, little is known about the molecular basis of ACG. People with ACG have some common characteristics: slightly smaller than normal eyes (microphthalmia), relatively large lenses and unusually short axial lengths (distance from the front to the back of the eye). These traits increase the likelihood that the eye's drainage system, called the angle, may become blocked, leading to a build-up of fluid and high IOP. Previous work has shown that IOP elevation in ACG is not caused simply by blocked drainage, however; other physiologic mechanisms must be involved.

To learn more about the potential causes of ACG, scientists led by Simon W.M. John, a Howard Hughes Medical Institute Investigator at the Jackson Laboratory (Bar Harbor, ME) sought to identify genes associated with IOP elevation in mice. They carried out a mutagenesis screen, producing a mutation that resulted in elevated IOP in the mice. Mice carrying the mutation also had a phenotype that resembled certain features of humans with ACG (*Nat. Genet.* doi:10.1038/ng.813; published online 1 May 2011). The team then mapped and sequenced the mutation and found that it was located in a gene, called *Prss56*, that encodes a previously unidentified serine protease. Then, with collaborators Mounira Hmani-Aifa and colleagues (Université de Sfax, Tunisia), the group identified mutations in the human ortholog (*PRSS56*) in people with posterior microphthalmia.

The results suggest that alterations of this serine protease may be associated with a range of ocular conditions and provide a new mouse model for future research on such disorders, including ACG.

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