IN BRIEF

SOCIAL NEUROSCIENCE

Intact rapid detection of fearful faces in the absence of the amygdala

Tsuchiya, N. *et al. Nature Neurosci.* 30 Aug 2009 (doi:10.1038/nn2380)

Personal space regulation by the human amygdala

Kennedy, D. P. *et al. Nature Neurosci.* 30 Aug 2009 (doi:10.1038/nn2381)

The amygdala is thought to be essential for rapid, non-conscious fear processing, but previous data contradict this idea. Two new studies find that patient S. M., who has complete bilateral amygdala lesions and cannot recognize fear in faces, could rapidly distinguish fearful faces from other faces and rapidly categorize faces as being fearful or neutral. S. M. also had no sense of personal space, feeling no discomfort standing chin-to-chin with the experimenter. These studies suggest that the amygdala is not essential for early fear processing but instead might be involved in the appraisal of social stimuli.

SYSTEMS NEUROSCIENCE

A serotonin-dependent mechanism explains the leptin regulation of bone mass, appetite, and energy expenditure

Yadav, V. K. *et al. Cell* **138**, 976–989 (2009)

The hypothalamus is required for the effects of leptin on appetite, energy expenditure and bone mass. However, hypothalamic leptin receptors are not essential for these effects, indicating that leptin acts elsewhere in the brain. This paper shows that leptin inhibits the activity of, and serotonin (5-hydroxytryptamine (5HT)) synthesis in, brainstem raphe nucleus neurons. Moreover, the authors showed that these neurons project to the hypothalamic arcuate nucleus, to regulate appetite and energy expenditure through $5HT_{1A}$ and $5HT_{2B}$ receptors, and to the ventromedial nucleus, to regulate bone mass through $5HT_{2C}$ receptors.

NEURODEGENERATIVE DISEASE

Genome-wide association study identifies variants at CLU and PICALM associated with Alzheimer's disease

Harold, D. *et al. Nature Genet.* 6 Sep 2009 (doi:10.1038/ng.440)

Genome-wide association study identifies variants at CLU and CR1 associated with Alzheimer's disease

Lambert, J.C. *et al. Nature Genet.* 6 Sep 2009 (doi:10.1038/ng.439)

Two independent studies published in *Nature Genetics* report three new and robust genetic associations for late-onset Alzheimer's disease. In addition to replicating the well-established association of apolipoprotein E (*APOE*) with the disease, these two-stage genome-wide association studies showed that variants at the clusterin (*CLU*), complement component (3b/4b) receptor 1 (*CR1*) and phosphatidylinositol-binding clathrin assembly protein (*PICALM*) loci are associated with susceptibility to late-onset Alzheimer's disease. Although it is known that CLU interacts with β-amyloid, that CLU together with CR1 could have a role in the clearance of β-amyloid, and that PICALM is involved in clathrin-mediated endocytosis and in directing the trafficking of VAMP2, the precise contribution of these genes to pathogenesis remains to be established.