IN BRIEF

NEUROPHARMACOLOGY

Pain, pain, go away

Placebo responses to painful stimuli are mediated by both opioid and non-opioid mechanisms, and the latter are poorly understood. Here, Benedetti and colleagues examined the role of the endocannabinoid system in such responses. The authors conditioned individuals, who were undergoing daily pain tolerance tests, to expect analgesia from either an opioid drug (morphine) or a non-opioid drug (ketorolac). They then administered rimonabant — an antagonist of cannabinoid receptor 1 — to a subset of these individuals, to assess the impact of this compound on the placebo analgesic effects. Interestingly, rimonabant blocked the placebo effect in people who had undergone non-opioid preconditioning but not those who had been subjected to opioid preconditioning. This finding suggests that the endocannabinoid system plays an important part in non-opioid-mediated placebo effects.

ORIGINAL RESEARCH PAPER Benedetti, F. *et al.* Nonopioid placebo analgesia is mediated by CB1 cannabinoid receptors. *Nature Med.* 2 Oct 2011 (doi:10.1038/nm.2435)

OLFACTORY CODING

Smell gets organized!

The receptive surfaces for senses such as hearing (cochlea) and vision (retina) are organized along axes of sensory perception, but whether a similar type of organization is present in the olfactory epithelium is unknown. The authors measured the response of sensory receptors in the adult human olfactory epithelium to different odours. They found that the receptors that responded to pleasant odours tended to be grouped together in patches, as were the receptors that responded to unpleasant odours. These results suggest that like the cochlea and retina, the olfactory epithelium is organized along its primary axis of perception.

ORIGINAL RESEARCH PAPER Lapid, H. *et al.* Neural activity at the human olfactory epithelium reflects olfactory perception. *Nature Neurosci.* 25 Sep 2011 (doi:10.1038/nn.2926)

NEUROGENETICS

Expanding ALS and FTD genetics

Amyotrophic lateral sclerosis (ALS) and frontal temporal dementia (FTD) can occur in the same family with an autosomal-dominant pattern of heritance that is linked to chromosome 9p21. Now, two studies independently report that a hexanucleotide repeat expansion in the non-coding region of *C9ORF72* causes 9p21-linked ALS and FTD. Both studies initially identified this genetic expansion in families with an established history of 9p21-linked ALS, FTD and/or FTD–ALS (a phenotype with characteristics of both diseases). Subsequently, DeJesus-Hernandez *et al.* showed that in a clinical series of patients, this expansion was the most common genetic cause of familial and sporadic ALS, and a major cause of familial FTD. Meanwhile, Renton *et al.* showed that the hexanucleotide expansion underlay ~21% of sporadic cases and 46% of familial cases of ALS in a Finnish cohort of patients. The mechanism by which this expansion causes disease is unclear — although, together, the studies show that it may have an effect on C9ORF72 expression and lead to the formation of nuclear RNA foci.

ORIGINAL RESEARCH PAPERS DeJesus-Hernandez, M. *et al.* Expanded GGGGCC hexanucleotide repeat in noncoding region of *C9ORF72* causes chromosome 9p-linked FTD and ALS. *Neuron* 21 Sep 2011 (doi:10.1016/j.neuron.2011.09.011) | Renton, A. E. *et al.* A hexanucleotide repeat expansion in *C9ORF72* is the cause of chromosome 9p21-linked ALS–FTD. *Neuron* 21 Sep 2011 (doi:10.1016/j.neuron.2011.09.010)