IN BRIEF

HOMEOSTASIS

The molecular and cellular identity of peripheral osmoreceptors

Lechner, S. G. *et al. Neuron* **69**, 332–344 (2011)

Central osmoreceptors play a part in tightly regulating the osmolality of the extracellular fluid in mammals, but the role of peripheral osmoreceptors is less clear. The authors identified a population of sensory afferents in the mouse liver, with cell bodies in thoracic dorsal root ganglia, that respond to physiologically relevant hypo-osmotic stimuli. The osmosensitive currents in these neurons were absent in mice lacking transient receptor potential channel V4 (TRPV4), suggesting a possible molecular correlate of these hepatic osmoreceptors.

DEVELOPMENT

Phosphorylation of E3 ligase Smurf1 switches its substrate preference in support of axon development

Cheng, P. L. *et al. Neuron* **69**, 231–243 (2011)

Ubiquitin E3 ligases are involved in targeting proteins for degradation by the ubiquitin–proteasome system (UPS). The authors showed that growth factor stimulation of neurons *in vitro* led to protein kinase A-dependent phosphorylation of the E3 ubiquitin ligase SMURF1 (SMAD ubiquitination regulatory factor 1). This caused SMURF1 to preferentially target its growth-inhibiting substrate, RHOA, for UPS-mediated degradation, rather than targeting its growth-promoting substrate, PAR6. These pathways might contribute to the establishment of axon and dendrite polarity in response to local signals during development.

SENSORY SYSTEMS

The anterior piriform cortex is sufficient for detecting depletion of an indispensable amino acid, showing independent cortical sensory function

Rudell, J. B. *et al. J. Neurosci.* **31**, 1583–1590 (2011)

Animals can detect a lack of essential amino acids (EAAs) in food, not through smell or taste but through a process that requires the anterior piriform cortex (APC). In this study, isolated APC slices responded to the absence of an EAA with the same neuronal activation and molecular processes as those seen *in vivo* after dietary EAA depletion. This suggests a novel, direct sensory function for cortex that does not require peripheral neural input.

RETINA

DICER1 deficit induces *Alu* RNA toxicity in age-related macular degeneration

Kaneko, H. *et al. Nature* 6 Feb 2011 (doi 10.1038/nature09830)

Degeneration of the retinal pigmented epithelium (RPE) leads to a form of age-related macular degeneration (AMD). The authors show that levels of the RNase DICER1 are reduced in the RPE of patients with AMD, and that mice lacking DICER1 develop RPE degeneration. DICER1 knockdown and overexpression in RPE cells respectively increased and decreased RNA levels of the retrotransposon *Alu*. Increasing *Alu* RNA levels induced RPE degeneration in mice, and *Alu* RNA inhibition blocked RPE degeneration in mice lacking DICER1. Thus, *Alu* RNAs cause RPE degeneration and could be targeted for treatment of AMD.