IN BRIEF

DEVELOPMENT

PHOX2A regulation of oculomotor complex nucleogenesis

Hasan, K. B. *et al. Development* **137**, 1205–1213 (2010)

In humans, mutations in the homeodomain transcription factor PHOX2A are linked to severe defects in extra-ocular muscle movement and in pupillary light reflexes. The authors show that in chick embryos PHOX2A regulates the development of the oculomotor complex, inducing the production of visceral neurons that control pupil dilation and somatic motor neurons that innervate the eye's external muscles, in a spatially organized fashion. Thus, a single transcription factor can determine both the organization and cell fates of a brain nucleus.

NEURODEGENERATIVE DISEASE

Diabetes-accelerated memory dysfuntion via cerebrovascular inflammation and Aβ deposition in an Alzheimer mouse model with diabetes

Takeda, S. *et al. Proc. Natl Acad. Sci. USA* 15 Mar 2010 (doi:10.1073/pnas.1000645107)

Diabetes mellitus has been associated with the development of Alzheimer's disease. To examine this association, the authors crossed a mouse model of Alzheimer's disease with two mouse models of diabetes. Diabetes accelerated Alzheimer's disease-like cognitive dysfunction without increasing the deposition of amyloid-β and caused cerebrovascular inflammation. Furthermore, the diabetic phenotypes were more severe in the cross-bred mice, suggesting that Alzheimer's disease can aggravate diabetes.

MEMORY

β-adrenergic blockade during memory retrieval in humans evokes a sustained reduction of declarative emotional memory enhancement

Kroes, M. C. W. *et al. J. Neurosci.* **30**, 3959–3963 (2010)

Remembering emotional events depends on amygdala activation. Noradrenaline is involved in the acquisition of emotional memories, but its role in retrieval is debated. Here, the authors tested the effects of the β-adrenergic receptor antagonist propranolol on cued recall of emotionally aversive verbal stimuli. Propranolol reduced emotional-memory recall performance, even 24 hours after administration, indicating that β-adrenergic antagonists could be useful for attenuating unwanted emotional memories.

NEUROPROTECTION

Polycomb group proteins as epigenetic mediators of neuroprotection in ischemic tolerance

Stapels, M. *et al. Sci. Signal.* **3**, ra15 (2010)

A brief period of ischaemia can induce ischaemic tolerance. This is associated with a general suppression of gene expression and protects against subsequent, longer interruptions of blood flow. Proteomic analyses revealed that ischaemia-tolerant mouse brains express high levels of transcriptional repressors, particularly polycomb group proteins. Overexpression of the polycomb proteins SCMH1 and BMI1, which reduced K^+ channel abundance and activity in cultured neurons, induced ischaemic tolerance without preconditioning. These findings shed new light on the mechanisms underlying ischaemic tolerance.