Further studies using selective vasopressin receptor antagonists, perhaps targeted to specific regions such as the amygdala, septum or hippocampus, should help to clarify the role of AVP in anxiety and stress.

Rachel Jones

References and links

ORIGINAL RESEARCH PAPER Griebel, G. *et al.* Anxiolytic- and antidepressant-like effects of the non-peptide vasopressin V_{1b} receptor antagonist, SSR149415, suggest an innovative approach for the treatment of stress-related disorders. *Proc. Natl Acad. Sci. USA* **99**, 6370–6375 (2002)

FURTHER READING Wong, M.-L. & Licinio, J. Research and treatment approaches to depression. *Nature Rev. Neurosci.* **2**, 343–351 (2001)

ectodermal markers. Inhibition of BMP signalling also causes expansion of the neuroectoderm, and the authors found that this phenotype could be rescued by forcing the expression of ΔNp63. Overexpression of ΔNp63 in wild-type embryos, by contrast, led to a reduction in the amount of neural tissue.

So, ΔNp63 acts downstream of BMP signalling and it seems to be involved in ectodermal cell-fate specification, but how does it work? Bakkers *et al.* found that if the DNA-binding domain of ΔNp63 was linked to the repressor domain of a different protein, overexpression of the resulting chimeric protein could produce the same phenotype as ΔNp63 overexpression. This implies that ΔNp63 normally functions as a transcriptional repressor, with its DNA-binding domain conferring target specificity.

Taken together, these lines of evidence point towards ΔNp63 acting in the ventral ectoderm, downstream of BMP signalling, to repress genes that promote neural cell-fate specification. To complete the picture, it will be necessary to identify the target genes of ΔNp63, and to find out more about the mechanisms by which it achieves this repression.

Heather Wood

References and links

ORIGINAL RESEARCH PAPER Bakkers, J. *et al.* Zebrafish *ΔNp63* is a direct target of Bmp signaling and encodes a transcriptional repressor blocking neural specification in the ventral ectoderm. *Dev. Cell* **1**, 617–627 (2002)

FURTHER READING Muñoz-Sanjuán, I. & Brivanlou, A. H. Neural induction, the default model and embryonic stem cells. *Nature Rev. Neurosci.* **3**, 271–280 (2002) | Yang, A. & McKeon, F. P63 and P73: P53 mimics, menaces and more. *Nature Rev. Mol. Cell Biol.* **1**, 199–207 (2000)

WEB SITES

Encyclopedia of Life Sciences:
http://www.els.net/
BMP antagonists and neural induction

IN BRIEF

BEHAVIOURAL GENETICS

Influence of gene action across different time scales on behavior.

Ben-Shahar, Y. *et al*. *Science* **296**, 741–744 (2002)

Different alleles of the *foraging* gene (*for*) in *Drosophila* cause flies to be either 'rovers' (foraging over a wide area) or 'sitters' (feeding more locally). Ben-Shahar *et al.* show that increased expression of the same gene is associated with the age-related transition from hive work to foraging in honeybees. The *for* gene encodes a cyclic-GMP-dependent protein kinase (PKG), and the authors found that increasing PKG activity could increase precocious foraging.

ADDICTION

Psychostimulant-induced behavioral sensitization depends on nicotinic receptor activation.

Schoffelmeer, A. N. M. *et al. J. Neurosci.* **22**, 3269–3276 (2002)

The authors found that repeated exposure to nicotine enhanced the psychomotor effects of amphetamine, and that nicotinic antagonists prevented the development of amphetamine- and cocaine-induced behavioural sensitization. Nicotinic antagonists also prevented the increase in dopamine release that was found in the nucleus accumbens after treatment with the psychostimulant drugs. The results indicate that nicotine might enhance the long-term effects of amphetamine and cocaine, and alter their addictive properties.

NEUROLOGICAL DISEASES

Gene-microarray analysis of multiple sclerosis lesions yields new targets validated in autoimmune encephalomyelitis.

Lock, C. *et al*. *Nature Med.* **8**, 500–508 (2002)

Lock *et al.* compared the gene expression profiles of 'active' and 'silent' multiple sclerosis (MS) lesions to identify genes that are expressed at different stages of the disease. Several genes that were differentially expressed had not previously been associated with MS. By modulating the expression of two of these genes, the authors reversed the symptoms of EAE (experimental allergic encephalomyelitis), a mouse model of MS. This approach might help in the development of therapies to treat specific aspects of MS.

PSYCHIATRIC DISORDERS

Small intestinal enteropathy with epithelial IgG and complement deposition in children with regressive autism.

Torrente, F. *et al. Mol. Psychiatry* **7**, 375–382 (2002)

The authors describe a systematic study of lymphocytic colitis in children with regressive autism, comparing duodenal biopsies from these individuals with those from both normal and disease control groups. Their findings confirm the presence of a new form of enteropathy in autistic children, including increases in mucosal lymphocyte density, crypt cell proliferation, and epithelial deposition of IgG with complement C1q. They point to a possible autoimmune lesion in regressive autism.