WEB WATCH

Blind prejudice

- Colour Blindness (Andrew Oakley) http://www.delamare.unr.edu/cb/
- Firelily Designs http://www.firelily.com/opinions/color.html
- Vischeck http://www.vischeck.com/index.php

Most of us take colour vision for granted, but there are still many people who are unable to enjoy its benefits. One in 20 European Caucasian males has a red/green colour vision deficiency, and the internet provides plenty of useful information about this and other types of colour blindness.

Many colour blind people feel that they are discriminated against. Andrew Oakley, who is red/green colour blind, says "we have instructions telling us to 'press the red button', machines which have a green safety light and a red warning light, even colour-coded pills!" He also vents his spleen on light bulbs that change from red to green, which he calls "the work of Satan".

It seems that we need a better understanding of the needs of people with colour vision deficiencies. For example, Diane Wilson from the web design company Firelily Designs offers advice on how to make your web site more user-friendly (handy hint — see how it looks in black-and-white!). Wilson herself is an 'anomalous trichromat'; that is, her red and green cone cells do not function as effectively as they should. If you want to see how your own web site looks to someone with red/green colour blindness, try running it through Vischeck.

Although colour blind people are prevented from doing certain jobs, it's worth noting that they might have certain advantages. Oakley points out that "we look for outlines, not colours, so we don't get easily confused by camouflage", which is why "colour blind people were used in World War II spy planes to spot camouflaged German camps".

Heather Wood

DEVELOPMENT

Nature vs nurture

When reading about neural development in a textbook, chances are that we will find a clear division between activity-dependent processes and those that do not require neural activity. Are there developmental mechanisms that bridge this divide? Although this question has profound implications, this issue has not, so far, been a mainstream area of research. But now Takasu *et al.* have provided a breakthrough in this field by showing that Eph receptors can modulate the function of NMDA (*N*-methyl-D-aspartate) receptor ion channels.

Eph receptors and their ligands, the ephrins, participate in several developmental phenomena, such as segmentation and axon guidance. Previous observations had shown that EphB2 receptors interact with and cluster NMDA receptors at synaptic sites. Takasu *et al.* explored the functional meaning of such an interaction and found that EphB2 potentiates the influx of calcium through NMDA channels in cortical neurons, an effect that depended on phosphorylation of the NMDA receptor. Although Eph receptors are themselves kinases, this NMDA receptor phosphorylation involves a member of the Src family of kinases. As this increase in NMDA receptor function was also accompanied by an increase in gene expression, it is possible that this new signalling pathway participates in the long-term structural remodelling of the synapse. Indeed, two additional papers point in this direction by showing that the absence of EphB2 impairs long-term synaptic plasticity.

Henderson *et al.* found that mice lacking EphB2 showed a reduction in hippocampal long-term potentiation (LTP) in both the CA1 region and the dentate gyrus. Grunwald *et al.* found a similar reduction in CA1 LTP, particularly during its late phase, and marked impairments of both long-term depression (LTD) and a reversal of LTP known as depotentiation. Moreover, Grunwald *et al.* showed that spatial memory, which depends on hippocampal function, was defective in EphB2 knockout mice.

SENSORY SYSTEMS

Keeping an ear out for modifiers

Although the ability of some mouse genetic backgrounds to modify mutant phenotypes is well known, only a handful of the responsible modifier genes have been found. Now Ikeda *et al.* report a new mouse modifier gene — *Mtap1a* — that rescues the hearing defects of C56BL/6J (B6) tubby (*tub*) mutant mice. Importantly, their findings strongly indicate that Mtap1a interacts with proteins that establish and maintain synapses, and that in the B6 mouse strain, this association is disrupted by *Mtap1a* alleles that carry polymorphisms in functionally important regions of the gene.

The *tub* modifier (*moth1*) locus was mapped to chromosome 2 in 1999, after it was discovered that *tub* mice retain their hearing on some genetic backgrounds, such as on the AKR/J (AKR), CAST/Ei and 129P2/OlaHsd (129P2) mouse strains, while still developing other *tub*-associated phenotypes. So, to