

# Funnel in the eye: Signal focusing increases photosensitivity

**In poor light the eyes of mice react like some digital cameras: they reduce their resolution while at the same time increasing their sensitivity. Specialists in the retina focus the information of several light sensor cells for this purpose. Scientists from the University of Bonn and their colleagues from Oldenburg, Bochum and Kobe (Japan), have now discovered how all this works. The study will be published on 3 November in the prestigious *Journal of Biological Chemistry*.**

This is something camera owners can only dream of: in poor light the eye becomes several million times more sensitive within half an hour. What are known as amacrine II cells in the retina play an important role in the process of adapting to darkness. They switch from the comparatively less sensitive retinal cones to the far more sensitive rods. Since the latter only record differences in brightness and no colours, in the dark all cats are grey, as the saying goes.

Grey and blurred: in faint light the amacrine II cells additionally focus the signals from different rods. This trick significantly boosts the sensitivity even more – one reason why people developing digital cameras are fond of using it. In poor light they frequently merge several pixels in one, thereby increasing the sensitivity of the CCD chip.

## Funnel in the eye

Until recently it was not known exactly how this merging of pixels in the eye occurs. A study under the overall control of the University of Bonn sheds light on the problem in the truest sense of the word. According to this study, in poor light more intercellular channels open in the amacrine II cells of the mouse. In doing so they combine temporarily to form an extended network. This network focuses the information from several photosensitive rods. The whole thing acts as a funnel which combines the individual electrical impulses from adjacent rods into a larger signal.

If there is sufficient light the signal focus is deactivated. This is triggered off by dopamine. As Klaus Willecke, a professor of genetics at the University of Bonn, explains, 'dopamine is formed by the retina when the retina is hit by light. Dopamine activates a cascade of signals which eventually results in the closure of a particular channel in the amacrine II cells called connexin36.' The research team also seems to have found the right cork for connexin36, abbreviated to 'Cx36'. Professor Willecke says that 'apparently, under the influence of dopamine, a phosphate group is bound to Cx36 and inhibits the channel.' It is the first time that direct proof has been available that this mechanism exists.

The importance of signal focusing is demonstrated by mice which cannot produce Cx36. Their amacrine II cells cannot combine to form networks when necessary. The consequences for these nocturnal rodents are devastating. Klaus Willecke says that their rod vision is extremely impeded, so that their visual perception is very limited in poor light.

Protein kinase A mediated phosphorylation of connexin36 in mouse retina results in decreased gap junctional communication between AII amacrine cells.

Source: University of Bonn

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