### HIGHLIGHTS

### IN THE NEWS

### Losing your inhibition

Sorry, what was I supposed to be writing about again? Oh yes ... do you find that your memory increasingly fails you as you get older? It seems to happen to all of us, but why? It is generally assumed that the ageing brain becomes less efficient at remembering information, but, as The Guardian (UK, 9 August 2001) reports, Malcolm Macleod and his team from the University of St Andrews in Scotland want to challenge this assumption. With the help of a £30,000 grant from the Royal Society of Edinburgh, they hope to show that it is actually our ability to forget that becomes impaired as we get older.

Macleod argues that "forgetting is simply a problem of accessing relevant material". He suggests that, with time, the brain becomes full of competing information, making it harder to suppress unimportant details. To illustrate, he provides the following example: "You are trying to remember the telephone number of a friend. You might not simply access the friend's telephone number, but his previous telephone numbers as well. Now how does memory deal with that unwanted competition?".

According to Macleod, young brains seem to be much better at inhibiting this irrelevant information than older brains. However, it might not all be good news for the young, particularly those with a habit of leaving things until the last minute. As the article points out, "... younger students with better inhibition, who are indulging in a spot of last-minute swotting for exams, might run the risk of suppressing things they learned earlier".

To summarize, as Macleod puts it, "the process of remembering actually produces forgetting. But you can look at it the other way round. The process of forgetting results in remembering, if you see what I mean." Confused? Perhaps you're just getting old.

Heather Wood

### SENSORY SYSTEMS

# Gaps in the retina

The importance of electrical synapses for brain function has recently come into the limelight. Synaptic contacts of this type are formed between interneurons in several brain regions, prompting the suggestion that electrical coupling is a fundamental feature of local inhibitory circuits. But long before electrical synapses gained this notoriety, their existence in the retina had already captured the attention of many neuroscientists. Electrical synapses are formed between rod amacrine cells (the main output of rod bipolar cells) and cone bipolar cells of the ON pathway. Information from the rod pathway is fed to the cone pathway, partly through this electrical connection. But what is the precise role of electrical synapses in the early stages of visual processing? A recent study by Güldenagel et al. constitutes an important first step to answering this question.

Gap junctions, cellular specializations that bridge the cytoplasm of adjacent cells, are the main structural element of electrical synapses. Gap junctions are formed by a family of proteins called connexins. Only a few connexin types are expressed in the nervous system; in the retina, connexin36 (Cx36) is present in rod amacrine cells, where it forms gap junctions with a different connexin expressed by cone bipolar cells. Güldenagel et al. generated mice lacking Cx36, and explored the effect of this mutation on retinal structure and function. They found that the absence of Cx36 was not accompanied by anatomical abnormalities; all layers of the retina and its central projection seemed normal. In contrast, electroretinographic recordings showed that the so-called 'b-wave', which is related to depolarization of ON-type bipolar cells, was reduced in the

### NEURAL CODING

## Cracking the code

Our sensory systems are very good at adapting. For example, our eyesight rapidly adjusts to changes in the ambient light level, allowing us to see in both near darkness and bright daylight. This allows the sensory neurons to use their limited response range to the full, reassigning each specific response value to a different stimulus strength that is appropriate to the new conditions. But Fairhall et al. now show that there is much more to adaptation than meets the eye. Fairhall and colleagues recorded from motion-sensitive visual neurons in the blowfly Calliphora vicina to investigate the dynamics of adaptation to a randomly fluctuating motion stimulus, the variance of which changed over time. The average firing rate of the

neurons increased or decreased abruptly when the stimulus switched suddenly to a higher or lower variance. It then gradually reached a steady-state level, adapting relatively slowly. The timescale of this adaptation was related to the variable timescales of changes in the stimulus. The coding in terms of precise spike timing, however, adapted much more quickly - within tens of milliseconds. This adaptation time was limited only by how quickly the system could gather enough information to reliably recognize the change. In this way, the cell minimizes the amount of time for which its information transfer is suboptimal. So there are at least two independent timescales of adaptation, carried by different aspects of the response statistics.

To avoid ambiguity, the nervous system must also carry information about the context of the adapted signal — in this case, the stimulus variance. Here, this information is conveyed by the same neuron. Although the firing rate changes slowly in response to the variance, Fairhall *et al.* found that another firing statistic — the interspike interval distribution changed to reflect the new variance on similarly rapid timescales to the input–output relation.

So the coding scheme of this neuron, at least, seems to use several channels

mutant mice. Similarly, the latency of light-evoked field potentials recorded in the optic tectum was longer in mutant than in wild-type animals.

So gap junctions are important during early visual processing, as the absence of Cx36 impairs light perception. However, some loose ends must still be tied up. For example, the reduction of the b-wave was observed even if the rod pathway was saturated by light. Under these conditions, ONtype cone bipolar cells are solely driven by cones, and the influence of the rod amacrine cells should be irrelevant in both wild-type and mutant mice. Why, then, is the b-wave smaller under light saturation? A more detailed cellular analysis should give us the answer.

Juan Carlos López Concerning and the series of the series



Calliphora vicina, courtesy of Hein Leertouwer.

simultaneously to convey information about the stimulus — and these channels adapt independently, on different timescales, both to optimize efficient information coding and to prevent ambiguity.

#### Rachel Jones

### References and links

ORIGINAL RESEARCH PAPER Fairhall, A. L. Efficiency and ambiguity in an adaptive neural code. *Nature* **412**, 787–792 (2001)