learning mutants, to establish a similar kind of mechanism in *Drosophila*. One mutant, *dunce*, has a defective phosphodiesterase and thus presumably abnormal cyclic AMP metabolism[6]; a second, *rutabaga*, isolated by Patricia Szieber in his laboratory, has been shown by Margaret Livingstone to be deficient in adenylate cyclase activity. Quinn is now hoping to identify the neurotransmitter whose receptor is (presumably) coupled to the crucial adenylate cyclase, so as to explore further the possible parallel with *Aplysia*. Preliminary evidence implicates either dopamine or serotonin, which is particularly interesting in view of recent evidence that the distribution of dopamine is correlated in rat brain with that of a membrane protein whose phosphorylation can be shown *in vitro* to depend on the dopamine-activated synthesis of cyclic AMP[7].

Thus catecholamine-activated protein phosphorylation may be part of a general mechanism of memory. But how far can the phosphorylation of ionic channels explain the phenomenological properties of memory? Stevens, in his summary discussion, pointed out that it cannot on its own account for any memory lasting more than two or three weeks, which is the maximum lifetime of a membrane protein. If learning depends on the phosphorylation of membrane proteins, then neurones must be re-programmed to keep phosphorylating them. It was this kind of consideration that led him to suggest that memory can be seen as the last step in neural differentiation: a final change in gene expression that is reserved until after birth and is contingent on experience. Just as during development, a morphogenetic signal changes the state of differentiation of a cell and thus its responses to subsequent signals, so the early changes in neural activity that underlie short-term memory may render the neurone susceptible to later signals that induce a change in gene expression and long-term storage. There is in fact evidence that in some systems, neural morphogenesis is profoundly influenced by neural activity: learning may reflect the same (unknown) mechanism. Indeed changes in dendritic or synaptic morphology may be the basis for long-term memory.

An understanding of memory at this level will not, in Stevens's view, provide a complete understanding of the encoding of memories — any more than understanding the behaviour of transistors would help with the understanding of a computer program. But it could establish, for example, whether the process is reversible or not. It is possible that memories are for ever, and forgetting is just loss of access. Certainly this is to be expected from the developmental parallel, since differentiation is generally extremely stable, if not completely irreversible. There is, however, one notable case in which terminal differentiation entails the editing of the DNA and irrevocably limits the potential of the cell for further change. This is the case of the antibody-producing lymphocyte, in which recombination of the DNA is part of the basis for antibody diversity. Stevens, echoing Nottebohm's speculations, suggested that some similarly irreversible step might be made by the song cells of the canary HVc — so that the only way to change the repertoire would be by the loss of the old neurones and the addition of new ones.

It is however more likely (as he went on to emphasize) that versatility in memory depends not on the generation of diverse proteins by DNA recombination, but on the versatile deployment of a limited number of related families of receptor and channel proteins whose expression in different combinations confers on nerve cells the specialized properties of which so far we have such a limited understanding. Now that, with the cloning of the genes encoding the acetylcholine receptor[8], the first members of these protein families have yielded to recombinant DNA technology, dramatic advances in understanding their relationships and expression may not be far away.

1. Cohen, N.J. & Squire, L.R. *Science* **210**, 207 (1980).
2. DeVoogd, T.J. & Nottebohm, F. *J. comp. Neurol.* 196, 309 (1981).
3. Nottebohm, F. *Brain Res.* **189**, 429 (1980).
4. Klein, M., Camardo, J. & Kandel, E.R. *Proc. natn. Acad. Sci. U.S.A.* **79**, 5713 (1982).
5. Siegelbaum, S., Camardo, J. & Kandel, E.R. *Nature* **299**, 413 (1982).
6. Byers, D., Davis, R.K. & Kiger, J.A. *Nature* **289**, 79 (1980).
7. Greengard, P. *et al. Nature* (in the press).
8. Stevens, C.F. *Nature* **299**, 776 (1982).

Seeing in colour

THE showpiece of the First McDonnell Conference on Higher Brain Function was the spectacular re-entry into research on colour vision of David Hubel (Harvard University), who, in a kingfisher shirt and scarlet tie, and with the assistance of Margaret Livingstone manipulating coloured lights, gave a personal demonstration of the importance of context in hue perception. The demonstration is simple (and in principle well known): bathe the outrageous shirt and tie in red light and the outrage vanishes: the shirt becomes black while the tie assumes the same pallid appearance as the speaker's face; similarly in blue light the vividness of the shirt is drained. This illustrates that human colour vision is not a simple function of the wavelength of light reaching the retina — as any reader who has a copy of *Nature* for 3 April 1980 (and has not lost the colour filters supplied with it) can check for himself.

Among the best explanations so far offered for the relative indifference of the visual system to the absolute wavelength of the stimulus is Land's retinex theory (discussed in the same issue of *Nature*[1]), according to which the sensation of colour is the outcome of interactions between neurones that respond selectively to lights of different wavelengths. Specifically, the theory predicts the existence of cells whose receptive fields are so organized that they respond maximally when the centre of the field is illuminated by light of a wavelength complementary to that illuminating the surround. A highly effective stimulus would thus be a red dot in a blue surround. Neurones responding to such stimuli are known as double-opponent colour cells and have been detected in very small numbers in the lateral geniculate nucleus of the cat[2], which has relatively poor colour vision, and in the visual cortex of the monkey[3], whose colour vision is comparable with man's. This anatomical difference between cat and monkey reflects the general rule that sophisticated sensory computations tend to be postponed to increasingly central sites with ascent up the phylogenetic tree.

Hubel and Livingstone have now illuminated a further species difference by locating the cortical double-opponent cells precisely in the patches, or 'blobs', of exceptionally high metabolic activity recently revealed by cytochrome oxidase staining in layers II and III[4,5]. These patches have two curious features (discussed in *Nature* by Swindale[6]). First, they contain cells that, unlike most visual cortical neurones, are not selective for stimulus orientation[7]; and second, they are absent from the visual cortex of the cat.

The absence of orientation selectivity could be explained if the patches represented areas of convergence of orientation columns[6] — and thus containing neurones responding to all orientations. But then why none in the cat? A possible answer to this question now suggests itself: the patches may, instead of representing converging orientation columns, be colour columns; and their absence in cats probably reflects the relative crudeness of feline colour vision.

1. Zeki, S. *Nature* **284**, 412 (1980).
2. Perlman, A.L. & Daw, N.W. *Science* **167**, 84 (1970).
3. Michael, C.R. *J. Neurophysiol.* **41**, 572, 1233, 1251 (1978).
4. Hendrickson, A.E., Hunt, S.P. & Wu, J.-Y. *Nature* **292**, 605 (1981).
5. Horton, H. & Hubel, D.H. *Nature* **292**, 762 (1981).
6. Swindale, N. *Nature* **293**, 509 (1981).
7. Hubel, D.H. & Livingstone, M. *Soc. Neurosci. Abstr.* 357 (1981).

0028-0836/82/460220-01$01.00