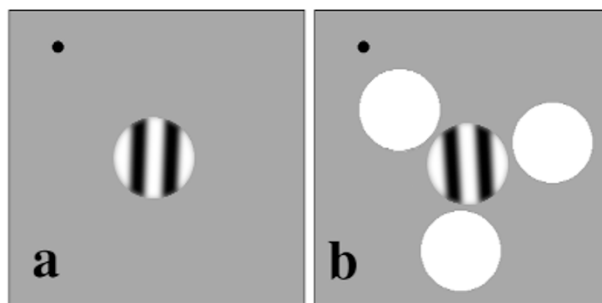


Fig. 2. Two important stimulus configurations used by De Weerd and colleagues. **(a)** Stimulus configuration for orientation discrimination with no distractor disks present. **(b)** Stimulus configuration with distracting disks present. To reproduce the conditions under which the monkeys viewed these stimuli, the figure should be held at a distance of 8.5 inches from the eye while fixating the round fixation dot in the upper left of each figure. While viewing each figure this way, the reader should try to determine if the target grating is vertically oriented or is rotated slightly clockwise or counterclockwise.



always appeared in the same location with respect to the fixation point from trial to trial. Consequently, the monkey was not tested for its ability to shift attention from one trial to the next. Second, the distractor stimuli appear in different locations from trial to trial, with some closer to and some farther from fixation than the oriented gratings. It will be interesting in future studies to see if this aspect of the task is critical to the observed result. Finally, the disruptive effect of the distractors is very marked at low contrasts (<20%) and then does not increase much further at contrasts between 20% and 50% (Fig. 3 of the paper). This can be seen most readily for the more disrupted visual field locations (where V4 and TEO lesions overlap), especially when ignoring the two points at 50% at which monkey M2 could not do the discrimination. In future studies, it will be interesting to determine how the contrast dependence of this effect changes as the distractors are made less or more similar to the test stimuli.

The attentional deficits observed in this study are surprisingly profound. One might have anticipated that spatial attention could also be controlled by the dorsal pathway, whose neurons also show strong attentional modulation¹⁵, yet these results indicate that the dorsal pathway cannot compensate for the loss of V4 or TEO. This raises the question of whether the role of V4 and TEO in spatial attention is general or task specific. If it turns out that orientation discrimination is performed in the ventral pathway (which remains to be shown), the deficits seen here might reflect a task specific role, which could explain why the dorsal pathway cannot provide compensation. If, on the other hand, V4 or TEO lesions also affect tasks that are known to be performed in the dorsal pathway (for

instance, discrimination of motion direction), this would argue for a more global role in attention, perhaps through top-down influences on earlier cortical areas.

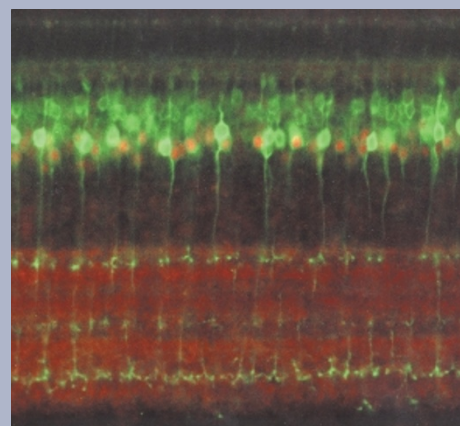
There are several further complications to the interpretation of these findings. One possibility, suggested by psychophysical experiments in my own laboratory, is that the loss of sensitivity may reflect a retinotopic but nonspecific increase in neural noise, presumably in lower areas that are closely connected to the lesioned area. Another uninteresting possibility is that the deficits might reflect a disruption in the monkey's ability to judge orientation relative to nearby visual cues (for example, the edge of the display),

with the result that the animal uses the distractors as an (unreliable) reference for judging orientation. It will therefore be important to determine in future experiments whether the powerful effects reported in this study can be generalized to a wider range of attention-demanding tasks.

1. Distler, C., Boussaoud, D., Desimone, R. & Ungerleider, L. G. *J. Comp. Neurol.* 334, 125–150 (1993).
2. Felleman, D. J. & Van Essen, D. C. *Cereb. Cortex* 1, 1–47 (1991).
3. Kaas, J. H. *Curr. Biol.* 5, 1126–1128 (1995).
4. De Weerd, P., Peralta, M. R., Desimone, R. & Ungerleider, L. G. *Nat. Neurosci.* 2, 753–758 (1999).
5. Zeki, S. M. *Brain Res.* 53, 422–427 (1973).
6. Merigan, W. *Curr. Biol.* 3, 226–229 (1993).
7. Merigan, W. H. *Vis. Neurosci.* 13, 51–60 (1996).
8. Gallant, J. L., Braun, J. & Van Essen, D. C. *Science* 259, 100–103 (1993).
9. Desimone, R. & Schein, S. J. *Neurophysiol.* 57, 835–868 (1987).
10. Walsh, V., Butler, S. R., Carden, D. & Kulikowski, J. J. *Behav. Brain Res.* 50, 115–126 (1992).
11. Iwai, E. & Mishkin, M. *Exp. Neurol.* 25, 585–594 (1969).
12. Schiller, P. H. & Lee, K. *Science* 251, 1251–1253 (1991).
13. Moran, J. & Desimone, R. *Science* 229, 782–784 (1985).
14. Braun, J. *J. Neurosci.* 14, 554–567 (1994).
15. Treue, S. & Maunsell, J. H. *Nature* 382, 539–541 (1996).

Signaling myopia

Clear vision requires that the cornea and the lens focus light onto the retina. If the shape of the eye produces a focal plane behind the retina, the eye is myopic, and the image appears blurred. In many developing animals, the sharpness of retinal images regulates the growth of the sclera (the outer connective tissue sheath of the eye), so that axial eye length is matched to the refractive power of the lens and cornea. In this issue (pages 706–712), Andy Fischer and colleagues suggest a candidate for this growth-regulating signal. They found that expression of the immediate-early gene ZENK (red) in a particular class of chick retinal amacrine cells (containing glucagon) was regulated by image defocus. ZENK expression was suppressed by several experimental conditions that enhance eye growth (such as a lens that blurs vision), but increased under conditions that suppress it, such as lens removal. This suggests that these amacrine cells could be involved in focus-induced changes in ocular growth and refraction.



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