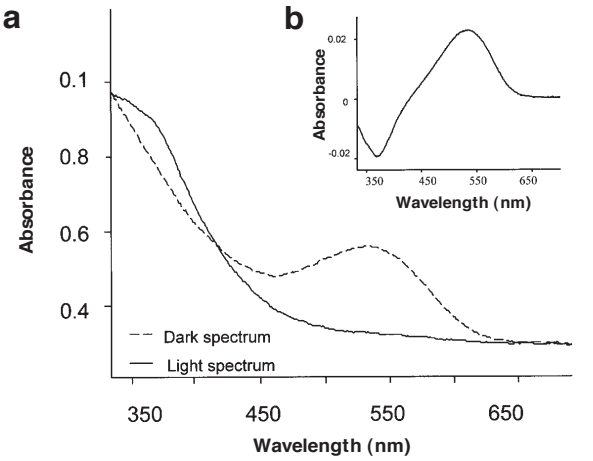

Fig. 2. Absorbance spectrum of the expressed LWS photopigment of *Spalax*. **(a)** Spectral properties of partially purified *Spalax* green cone pigment. The pigment cDNA was extended with a 6x his-tag, expressed using recombinant baculovirus, incubated with 11-*cis* retinal and partially purified by nickel-affinity chromatography[4]. The pigment shows a peak absorbance at 534 ± 4 nm. Upon illumination in the presence of hydroxylamine, this absorbance disappears and is replaced by absorbance at 365 nm, which represents the oxime (stabilized form of the bleached photopigment chromophore) of the liberated retinal. These data demonstrate that the retina of *Spalax* contains a fully functional green cone photopigment. **(b)** Inset shows the corresponding difference spectrum (dark minus light).

tified[5,6]. In *Spalax*, this red shift may be associated with the subcutaneous, and hence hemoglobin-dominated, light environment of the eye.

Despite the dramatic loss of the neurological components associated with image-forming vision, *Spalax* has maintained a functional LWS photopigment. The phenotypic degeneration of the eye has clearly not been accompanied by a parallel loss of opsin function. These results are surprising, given the expected loss of functional constraints in an animal that, for 25 million years, has inhabited a subterranean environment where visual cues are redundant. Evolutionary conservation suggests functional importance, and we propose that the positive selection pressures that have maintained this photopigment in *Spalax* are associated with the non-image-forming, light-detection task of photoentrainment.

The photoreceptors that mediate the effects of light on the mammalian circadian system are unknown[7]. Our results in *Spalax*, coupled with action spectrum studies in mice[8], suggest that cone photopigments may be involved. However, we would not want to exclude the possibility that unidentified ocular photopigments also contribute to the process of photoentrainment. In several respects, the degenerate eye of *Spalax* resembles the photosensory pineal organs of non-mammals. Both are screened by overlaying tissues and are obligate irradiance detectors, both contain receptors with cone-like characteristics[9,10], and both mediate the effects of light on circadian physiology.

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1. Crandall, K. A. & Hillis, D. M. *Nature* 387, 667–668 (1997).
2. Cooper, H. M., Herbin, M. & Nevo, E. *Nature* 361, 156–159 (1993).
3. Rado, R. & Terkel, J. in *Environmental Quality and Ecosystem Stability* (eds Spanier, E., Steinberger, Y. & Luria, M.) (ISEEQS, Jerusalem, 1989).
4. Vissers, P. M. A. M. (Ph.D. Thesis, University of Nijmegen, 1996).
5. Jacobs, G. H. & Deegan, J. F. *Behav. Neurosci.* 108, 993–1004 (1994).
6. Jacobs, G. H. *Biol. Rev.* 68, 413–471 (1993).
7. Foster, R. G. *Neuron* 20, 829–832 (1998).
8. Provencio, I. & Foster, R. G. *Brain Res.* 694, 183–190 (1995).
9. Sanyal, S., Jansen, H. G., De Grip, W. J., Nevo, E. & De Jong, W. W. *Invest. Ophthalmol. Vis. Sci.* 31, 1398–1404 (1990).
10. Vigh, B., Röhlich, P., Vigh-Teichmann, I. & Aros, B. *Z. Mikrosk. Anat. Forsch.*94, 623–640 (1980).
11. Nevo, E. in *Evolutionary Biology*, Vol. 25 (eds Hecht, M. K., Wallace, B. & MacIntyre, R. J.) (Plenum, New York, 1991).
12. Foster, R. G. *et al. J. Comp. Physiol. A* 169, 39–50 (1991).

Latency difference, not spatial extrapolation

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The inevitable neural delays involved in processing visual information should cause the perceived location of a moving stimulus to lag significantly behind its actual location. However, Nijhawan[1–3] has proposed that the visual system corrects the perceived location of the moving stimulus by extrapolating it along the trajectory of motion, so that the stimulus is perceived at its expected actual location. We provide new evidence to the contrary, demonstrating that the visual system does not compensate for neural delays but simply shows a reduced delay for moving stimuli.

The spatial extrapolation model[1–3] is suggested by the finding that a continuously moving bar is perceived as being ahead of a stimulus briefly flashed in alignment with it. According to this model, the perceived location of the moving bar is spatially extrapolated to its physical location based on its past trajectory and velocity, whereas the flash is accurately perceived where it was presented, thus resulting in the apparent offset of the bar relative to the flash. For subjects to perceive the flash as aligned with the moving bar, the flash must actually be presented ahead of the bar.

To test the extrapolation model, we investigated the effect of a sudden reversal of motion on the perceived location of the moving bar relative to the flash. The extrapolation model predicts that the bar's perceived location will continue to be extrapolated forward in space, overshooting the reversal point. In this experiment, a linearly translating bar reversed direction at a random time and location while an observer fixated on a central point. (Subsequent analysis found that there were no effects of eccentricity or duration). The physical location of a flash that was perceived as aligned with the continuously moving bar was measured at various times before and after the motion reversal. Contrary to the prediction of the extrapolation model, the perceived position of the moving bar (as revealed by the flash) never overshot the reversal point.

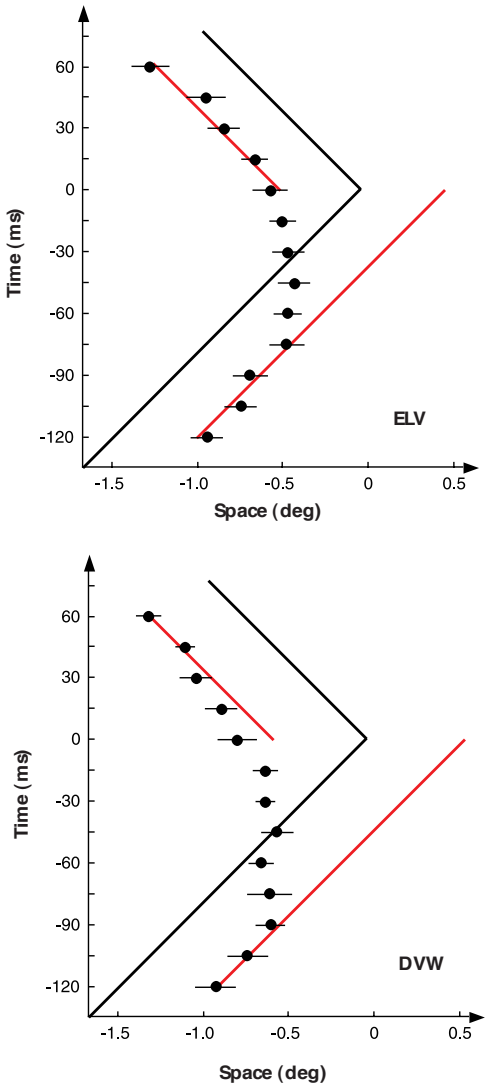

Fig. 1. Locations of flashes that are perceived as aligned with a moving bar. Separate panels show data for a naïve observer and one of the authors. (A third observer had similar results). For example, at –120 ms the flash would appear to lag behind the moving bar if it were presented in alignment, so to cancel the apparent lag, the flash must be presented ahead of the moving bar (data point). Lines through data points are 95% confidence intervals. The rounding of the curve may result from neural delay variability or a biologically plausible spatiotemporal averaging filter[6]. Black line, physical motion of the bar (the initial direction of the moving bar was determined randomly from trial to trial as either from the left or from the right). The duration of bar movement before the reversal, and therefore the eccentricity of the reversal point, were randomized to control for effects of eccentricity and predictability. Red line, predicted location of flash for it to be perceptually aligned with the bar according to the spatial extrapolation model.

In addition, to fit our data, the extrapolation model would require that the perceived position of the moving bar begin to be affected by the reversal before the bar actually had reversed direction. According to the extrapolation model, the perceived location of the moving bar should match its physical location (during continuous motion). Therefore, the temporal misalignment between the flash and the bar that is required to create an apparent spatial alignment neatly measures the neural delay being corrected by extrapolation. (The flash and bar have the same neural delay in the extrapolation model). During the continuous motion segments in Fig. 1, the delay between the presentation of the flash and the presentation of the moving bar with which it appears to be aligned (the vertical offset between the data points and the black line marking the physical trajectory) is about 45 ms. Therefore, 45 ms is the neural delay according to the extrapolation model, and this places an absolute constraint on the moment at which the data can start to reflect the reversal of motion. Any deviation from the expected trajectory during continuous motion (red line in Fig. 1) that occurs at –45 ms or earlier refutes the extrapolation model because the model would then imply that the reversal affects perception before the reversal has actually occurred.

Between –120 and –90 ms, the flash was perceived to be aligned with the bar when presented along a roughly continuous trajectory ahead of the bar in time (consistent with the extrapolation model's prediction; red line in Fig. 1). However, the data at –60 and –45 ms for ELV, and at –75, –60 and –45 ms for DVW and a third subject (not shown) deviated significantly from this path. (The least significant of these points was $t_{(6)} = 3.69$, $p < 0.02$ at –75 ms for the third subject whose data are not shown.) These results show that, if the extrapolation model were correct, the bar's reversal would start to affect perception before the physical reversal took place. This, of course, is impossible and reveals that the extrapolation model cannot account for the perceived misalignment of flashed and moving stimuli.

What then could cause the perceived offset between the moving bar and an aligned flash? The simplest explanation is that the neural delays for the flash and the moving bar are different. Indeed, rather than reflecting the actual neural delay for the bar and the flash, the vertical distance between the physical motion curve and the data curve (approximately 45 ms; Fig. 1) represents the difference between the latencies for moving and flashed stimuli. Specifically, the delay for the moving bar is shorter[4], perhaps because responses of motion detectors at one location facilitate the response of other detectors along the expected path of motion[5].

This experiment shows that there is no spatial extrapolation process allowing us to see moving objects where they really are. Rather, we perceive objects, moving or stationary, where they were before neural delays, but those delays can be shorter for objects in motion.

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1. Nijhawan, R. *Nature* 370, 256–257 (1994).
2. Khurana, B. & Nijhawan, R. *Nature* 378, 565–566 (1995).
3. Nijhawan, R. *Nature* 386, 66–69 (1997).
4. Allik, J. & Kreegipuu, K. *Psychol. Sci.* 9, 135–138 (1998).
5. Grzywacz, N. M. & Amthor, F. R. *J. Neurophysiol.* 69, 2188–2199 (1993).
6. Snowden, R. J. & Braddick, O. J. *Vision Res.* 29, 1621–1630 (1989).