

PGE2 increases the excitability of CNS neurons so that even nonpainful stimuli are perceived as painful, explaining why inflamed tissue feels sensitive and tender. Furthermore, inhibiting COX-2 in the CNS also decreases pain and hypersensitivity. The researchers suggest that targeting the widespread CNS production of COX-2 might more-effectively relieve pain as well as improve secondary symptoms such as lethargy, depression and loss of appetite. The study is in the March 22 issue of *Nature*.

## Dyslexia as neurologic deficit

In the search for a universal neurologic basis for dyslexia despite differences in prevalence amongst countries, an

international research team found that English, French, and Italian individuals with demonstrated reading and phonological deficits also exhibit decreased left brain activity by PET scan. During reading and naming tasks, they show reduced activation in the left temporal lobe and middle occipital gyrus compared with normal readers. Why, then, is dyslexia so less prevalent in Italy compared with France, the UK or the US? The authors of the March 16 *Science* article argue that the same phonological deficits are more likely to cause literacy problems in languages with more irregular writing systems. Therefore, individuals with deficits would have more trouble with English, in which 40 sounds are represented by over a thousand different letter combinations and

some of them arbitrarily. By comparison, Italian has less ambiguity and uses only 33 combinations for 25 sounds.

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### Letters

## 'Saccadic suppression' – no need for an active extra-retinal mechanism

In their recent review article on intra-saccadic visual perception, Ross *et al.*<sup>1</sup> concluded that motion of the world during saccades is not perceived because motion sensitivity is selectively reduced by an extra-retinal signal – although they concede that visual masking might also play an important role. However, we believe that extant data favour a more parsimonious interpretation, namely that the phenomenon called 'saccadic suppression'<sup>2</sup> has no functional role and is not the result of an extra-retinal signal. Instead, we propose a new interpretation based only on visual information processing.

The first general reason to doubt that the decrease in contrast sensitivity observed during saccades<sup>2</sup> has a function is its lack of effectiveness. Three aspects of experimental data are worth considering. First, it has long been recognized that the threshold elevations reported in the literature are too small to account for the lack of motion perception when the scene contains high contrasts<sup>3</sup>. Second, 'saccadic suppression' does not seem very useful when considering its temporal course: it is maximal at the beginning of the saccade and becomes less-marked as the saccade

proceeds<sup>1</sup>. It seems unlikely that this interesting result is the consequence of a mechanism that prevents us from perceiving intra-saccadic motion. Ideally, we would expect such a mechanism to be efficient during the whole course of the saccade, especially for long amplitude saccades, whose durations outlast the reported duration of saccadic suppression. At least, if the postulated mechanism operates in a gradual way, it should be most efficient when retinal velocity is highest, that is, near the saccadic peak velocity rather than near the beginning of the saccade. Third, the most convincing evidence that 'saccadic suppression' does not have the function postulated by Ross *et al.* is that intra-saccadic motion can be easily experienced when visual factors are optimized. We recently showed that saccades made in the direction of rapidly moving gratings produced a vivid intra-saccadic motion percept<sup>4</sup>. This occurred when the retinal speed of the grating around the peak saccadic velocity was optimal for motion detection. Ross *et al.*<sup>1</sup> note that our study 'highlights the important point that centrally driven saccadic suppression attenuates visual motion but does not eliminate it'. If we all agree that 'saccadic suppression' is so inefficient, then regarding this phenomenon as a functional mechanism seems a highly controversial hypothesis. Finally, if suppressing the contrast of intra-saccadic images was really the mechanism (regardless of its origin) used

by the visual system to blunt our perception during saccades, our environment should be greyed out every time we make a saccade.

Altogether, the aforementioned points show that the interpretation of 'saccadic suppression' is controversial because this phenomenon does not appear to have a clear function. However, it is still possible in principle, as Ross *et al.*<sup>1</sup> believe, that 'saccadic suppression' although inefficient is caused by an extra-retinal signal. In our opinion, the only evidence, which up to now has supported this view, is based on striking results reported by Burr and his colleagues. The essence of their data is that 'saccadic suppression' specifically alters the magnocellular system leaving the parvocellular system unimpaired. Here we propose a new interpretation of this magno-specific sensitivity reduction that does not postulate any central signal but is based on visual information processing only. We first assume that the photoreceptors during the saccade are not aligned with the optical axis of the eye because the acceleration of the eyeball creates shearing forces near the vitreous–scleral boundary. This trailing of the photoreceptors, still present at the end of the saccade, has already been suggested by psychophysical data<sup>5</sup>. From studies of the Stiles–Crawford effect, it is known that rays of light that strike receptors at an angle are less efficient than those entering directly into the receptors. Therefore, the intra-saccadic tilt of the

receptors should produce an overall decrease of luminance. We suggest that this luminance decrease, which cannot itself account for reduced contrast sensitivity, causes a sudden decrease in the visual adaptation level. Indeed, recent psychophysical studies with static eyes<sup>6</sup> show that abrupt changes in the adaptation state produce an immediate reduction of sensitivity. This would explain why there is a peak of intra-saccadic suppression near the beginning of the saccade, that is, when acceleration is high. Most importantly, this hypothesis would account for the magno-specific loss of sensitivity. It is well known that retinal contrast gain control is a key characteristic of the magnocellular pathway, whereas it is almost absent in the parvocellular pathway. The temporal contrast created by rapid changes in the adaptation level should therefore saturate magnocellular neurons much more than parvocellular neurons. This would explain why intra-saccadic sensitivity of the magnocellular system is specifically altered and also why 'saccadic suppression' seems to act at a very early level<sup>1</sup>.

In summary, current data seem to indicate that the so-called 'saccadic suppression' is an epiphenomenon probably occurring in the retina without any external influence. The mechanism preventing us from perceiving intra-saccadic motion is of a different nature and is still to be found.

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## Response: 'Saccadic suppression' – no need for an active extra-retinal mechanism

Castet *et al.* pursue their claim that vision is not actively suppressed at the time of saccades – a claim initially made on the basis of observations showing that saccades made in the direction of moving stimuli can improve the detectability of those stimuli by reducing retinal speed<sup>1</sup>. This can clearly occur, and indeed the results can be well-modelled from the spatio-temporal sensitivity functions of human vision<sup>2</sup>. However, the fact that the suppression does not occur for saccades simulated by mirror-motion<sup>3</sup> suggests a central origin of the suppression. In their letter, Castet *et al.* now consider an alternative explanation, revisiting the old idea<sup>4</sup> that the suppression might be a by-product of mechanical shearing forces during saccades. They suggest that these forces cause the photoreceptors to bend away from the pupil on each saccadic eye movement, resulting in less-efficient wave-guiding of light (the Stiles–Crawford effect<sup>5</sup>), transiently changing the adaptation state of the retina and therefore lowering sensitivity.

This is undoubtedly an interesting idea, but unfortunately encounters some difficulties with much of the existing data on saccadic suppression. For example, it is well known that the Stiles–Crawford effect is unique to cones being virtually absent in rods<sup>5</sup>: yet strong saccadic suppression has been reported in dark-adapted conditions as low as  $4 \times 10^{-4}$  cd/m<sup>2</sup> (Refs 3,6,7). Even more problematic is the observation that in total darkness electrically produced visual phosphenes are strongly suppressed by saccades<sup>8</sup>, which cannot be readily attributable to the optical wave-guide properties of photoreceptors.

The theory of Castet *et al.* also encounters difficulties with the specificity of saccadic suppression, which is restricted to low-frequency stimuli<sup>7</sup> that are modulated in luminance<sup>9</sup>. Changes in retinal adaptation would adversely affect all classes of neurones, and therefore affect both the luminance and chromatic

response at all spatial frequencies. However, one could consider the effects of a sudden dimming of the whole visual field, an effective luminance flash that might selectively mask luminance-modulated stimuli of low spatial frequencies by acting on the contrast gain control of detectors for these stimuli. Such a flash could account for the specificity of suppression, the fact that it occurs early<sup>9</sup> and its dependency on saccadic size<sup>3</sup>. So the question becomes quantitative: can the bending of photoreceptors create a flash strong enough to produce one log-unit of masking at low spatial frequencies? Presumably the masking flash must be at least one log-unit itself (probably greater) in order to do so. The Stiles–Crawford effect is usually described by a Gaussian function, with a squared space constant (*rho*) of 0.05 (Ref. 10). One log-unit of attenuation would require the receptors to bend by a massive 12.5°, a difficult feat within their tight mosaic packing. The only available measurements of which we are aware<sup>4</sup> suggest that cones might bend by 2° after a 5° saccade, producing only 0.025 log-units of attenuation. Furthermore, although it is true that the maximum shear forces occur at the beginning and at the end of each saccade (when acceleration and deceleration are maximal), the viscous intra-ocular medium will dampen these effects, so the maximal tilt of the photoreceptors will not occur at saccadic onset – where suppression is maximal<sup>3</sup> – but some considerable time later. In short, the suppression predicted by shear forces is too little, too late.

As highlighted by Castet *et al.*, one of the more puzzling aspects of saccadic suppression is that physiological evidence for suppression has been scant and often not observed at all in early visual centres. So one has to ask why this massive luminance signal (from the Stiles–Crawford effect) does not produce a measurable change in neural activity? However, recent evidence suggests that in monkey middle temporal area (MT), the effects are complicated: some cells respond less, others more, whereas some even reverse their direction selectivity around the time of saccades<sup>11</sup>. Such effects are difficult to explain by misaligned photoreceptors.

Finally, a mechanism of this type would introduce a new mystery: if the bending of