sickle cell disease. This is unusual because the heterozygous states of sickle cell disease have generally been considered as benign with regard to retinal lesions unless associated with other systemic diseases.[2]

Sickling of the red blood cells within the retinal vasculature has long been considered the primary process responsible for the retinal signs of sickle cell disease. In their paper, Hingorani *et al.* argue that 'since deoxygenated haemoglobin C . . . does not cause sickling . . . decreased plasticity and increased blood viscosity of HbCC and HbAC erythrocytes . . . due to intracellular poorly soluble precrystallin haemoglobin' may be at work, and sickling may not be required for such retinopathy to occur.

We have previously hypothesised that denser red blood cells due to high intracellular HbS polymer concentration are less able to pass through the precapillary arteriole and this results in secondary microvascular obstruction.[3] The heterogeneity of the red blood cell population density can be assessed by using the calibrated phthalate ester technique. The middle 60% density range (R60 values) serves as an indicator of the heterogeneity of the density of red blood cells. In sickle cell patients, R60 values are significantly and positively associated with reticulocyte counts and significantly and negatively associated with fetal haemoglobin levels.[4]

We have shown that severity of the conjunctival signs in sickle cell disease is significantly and positively associated with R60 values.[5] We have also shown that acute retinal arteriolar occlusion seen in sickle cell patients is significantly and positively associated with higher reticulocyte counts, while the presence of proliferative sickle cell retinopathy is significantly and negatively associated with fetal haemoglobin levels.[6] These data support our hypothesis that HbS polymer concentration within red blood cells may be far more important than sickling in relation to the ocular manifestations of the various states of sickle cell disease. We are glad to read that the observation of Hingorani *et al.* is providing more evidence for our hypothesis.

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Sir,

I was interested to read the experiment performed by Dua *et al.*[1] on the growth of corneal epithelial cells in the presence of a magnetic field. I was disappointed, however, to find no estimation of the magnitude of the magnetic field generated by the eye nor to find any rationale for the strength of the fields used.

The magnetic field of the Earth is approximately 1 gauss. Any field produced by an eye is likely to be considerably smaller than this. To estimate the field one needs to know the current flowing from cornea to posterior pole. This requires an estimate of the resistance of the eye. The electrical conductance will occur via the electrolytic solutions which make up the majority of the eye. The electrical resistance of five separate aliquots of 7 ml of balanced salt solution (BSS) and subsequently normal saline in an approximately spherical container of diameter 24 mm was measured with a multimeter. The mean value for BSS was 51.9 kΩ (SD 3.89) and for normal saline was 48.3 kΩ (SD 9.79). Thus a reasonable estimate for ocular resistance would be 50 kΩ. As the potential difference across the eye is 6 mV, the current flowing anteroposteriorly is thus estimated to be 1.2×10^{-7} A, using Ohm's law.

The easiest way to estimate the field is to take this current as running along a wire of length 24 mm lying along the geometric axis of the eye. Referring to Lorrain and Corson[2] the field a distance ρ from an infinite wire carrying a current I is given by the formula $B = \mu_0 I / 2\pi\rho$, where μ_0 is the permeability of free space ($4\pi \times 10^{-7}$ H/m). The result is in units of tesla, where 1 tesla = 10^4 gauss. It can be shown that the field strength a distance ρ perpendicular to the end of a wire of length L is given by the formula $B = (\mu_0 I / 4\pi\rho) \sin(\tan^{-1} L/\rho))$. Taking $L = 24$ mm, $\rho = 5$ mm (i.e. in the position of the mid-cornea) and the current calculated above, gives an estimate of the

field strength in the mid-cornea to be 2.349×10^{-8} gauss. This agrees with measurements made during the magneto-oculogram.[3]

This field is around 42.5 million times smaller than the Earth's magnetic field. In comparison with the field strengths used in the experiment, the estimated ocular magnetic field is 638 million times smaller than a 15 gauss field, 851 million times smaller than a 20 gauss field and 6.4×10^{10} times smaller than a field of 1500 gauss.

It seems that the field strengths used in the experiment are far higher than any that would be encountered in an eye under normal conditions. In fact, by far the largest field strength in any eye will be that of the Earth, and as these are vector quantities this would significantly disturb any concentric pattern of field lines across the cornea. Perhaps a better test of the hypothesis would be to have an electrical wire running vertically through a tissue culture plate. This could then carry a known current and generate a magnetic field with concentric field lines. The current in the wire and thus the field generated could be altered to test many field strengths, bearing in mind the magnitudes estimated above. The demonstration of epithelial whorling around such a wire generating a much smaller magnetic field would be much better evidence of the validity of the original hypothesis.

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Sir,

I concur entirely with the comments made by Davies on our paper cited above. Davies has put in quantitative terms what we have already said in the last paragraph of the paper: 'The hypothesis that we originally set out to test is not totally substantiated by the above experiment. The response of corneal epithelial cells to magnetic fields *in vitro* does not prove that the same occurs on the ocular surface. The electromagnetic field of the eye is, theoretically, several orders of magnitude smaller than that used in the above experiments. This study does however, reveal a unique behaviour of cultured human corneal epithelial cells in response to static magnetic fields.'

Our original study was designed to demonstrate whether corneal epithelial cells exhibited magnetotaxis or magnetotrophism, whatever the strength of the field. As to the rationale of the strength of fields used, we were guided by the only previous publication by Galaktionova[1] in this regard, who had used magnetic field strengths of 0.4–1.6 T to induce changes in mitotic index of murine corneal epithelial cells. The appearance of 'whorls' was, to us, peculiar, unusual, unexpected and interesting. We were aware of the vast differences in order of magnitude of the electromagnetic fields of the eye and those used in the study and, as also indicated by Davies, are at present conducting experiments using a Helmholtz coil to subject corneal cells to finite and measurable quantities of current. We thank Davies for his formulae and calculations, which will certainly help us augment our thoughts in this regard.

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Sir,

Fleck *et al.*[1] report in their audit on screening for retinopathy of prematurity (ROP) that no cases of threshold ROP developed in infants with birth weights >1250 g. They question the need to screen infants over 1250 g. Current Royal College of Ophthalmologists (RCO) guidelines recommend that all neonates with a birth weight ⩽1500 g and gestational age ⩽32 weeks should be screened.[2]

A recent audit carried out at St James's Hospital, Leeds, looked at all cases of neonates screened between July 1993 and May 1996. One hundred and eighty-nine patients were screened and a total of 288 screenings were carried out. Only 5 patients developed threshold disease (1.7%) as defined by RCO guidelines for screening of ROP.[2] Birth weights of these individuals ranged from 495 to 780 g (average 810 g).

These findings are consistent with other studies which have also found no cases of cicatricial or threshold ROP among infants with a birth weight >1250 g.[3–6] We agree that the current RCO guide-