ultraviolet light.[2] It is certainly worthwhile to identify high-risk groups and offer screening, but it would also be helpful to identify the relevant environmental factor(s) as well.

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Sir,

The purpose of our article[1] was to highlight the increasing evidence that some cases of uveal melanoma arise as a result of a genetic predisposition. We do not think that we overstated the role of constitutional gene mutations in the development of uveal melanoma. Although as Foss points out only 2% of cases are familial as defined by having a relative affected with uveal melanoma, indirect evidence suggests that a greater number, around 7% of cases, are likely to be caused by mutations in genes with pleiotropic effects such as *BRCA2* and those causing the atypical naevus syndrome.

To assess the contribution of germline mutations to the development of uveal melanoma we have made a systematic collection of family histories, blood samples and tumour material from over 400 patients attending the Ocular Oncology Service in Liverpool. Using this resource we are currently investigating the contribution of mutations in *BRCA2* and *CDKN2A* to uveal melonoma by screening both these genes in this series of blood samples from these patients.

Clearly the identification of genetic factors does not detract from the potential role of specific environmental factors in the aetiology of uveal melanoma, and in this respect we concur with the view of Foss.

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Sir,

I have read with interest the paper by Zambarakji *et al.*,[1] and subsequent correspondence,[2] concerning capsulorhexis phymosis following phacoemulsification. My own experience illustrates another possible parameter in the estimation of capsulorhexis phymosis.

A healthy 70-year-old man underwent phacoemulsification via a 5.0 mm capsulorhexis. The procedure was uncomplicated apart from a small (2 clock-hours) zonular dehiscence. A 23.0 D acrylic intraocular lens (Acrygel, Corneal Laboratoire) was implanted into the capsular bag.

Post-operative acuity was 6/9, and further changes were as shown in Table 1 (periods of subnormal best corrected visual acuity were assumed to be due to cystoid macular oedema, not proven by fluorescein angiography, but which improved with the use of oral acetazolamide and topical betamethasone). At this point capsulorhexis phymosis was noted, and nine radial relaxing incisions were made in the anterior capsular ring with a YAG laser. The posterior capsule was left intact. The refraction and best corrected acuities following YAG laser are shown in Table 2.

Table 1. *Post-operative refraction and best corrected acuity*

Post-operative time (weeks)	Refraction	Best corrected acuity
4	+2.00/—	6/18
8	0.00/–1.50 × 90°	6/9
20	+7.00/–2.00 × 150°	6/18

The only logical explanation for this remarkable variation in post-operative refraction is posterior displacement, or posterior bowing, of the flexible intraocular lens caused by capsulorhexis phymosis and relieved by YAG laser relaxing incisions. Previous literature has alluded to the fact that capsulorhexis phymosis can alter refraction,[3] and Shammas[4] has measured such changes in refraction. In one case, he reports +1.25 D, and in another, +0.75 D of induced hyperopia.

My experience indicates that accurate refraction may help in monitoring some cases of capsulorhexis phymosis, especially if a foldable intraocular lens is used.

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Table 2. *Post-YAG laser refraction and best corrected acuity*

Post-YAG laser time (weeks)	Refraction	Best corrected acuity
2	+4.00/–1.50 × 180°	6/18
7	+1.75/–0.25 × 180°	6/6
26	+1.00/—	6/6