Sir,

Response to Bunce *et al*

We read with interest the study of Causes of blind and partial sight certifications in England and Wales: April 2007—March 2008.[1] It was stated that 'The numbers of certificates are also lower than the figures reported as registered at Social Services'. This finding was interesting to us as we had noted a similar problem while performing a cross-sectional study of all Certificates of Vision Impairment (CVI) for Bristol and North Somerset between September 2005 and September 2008. We found that 54 patients were registered separately at both Bristol and North Somerset Social Services. All these 54 patients had been referred from North Somerset into Bristol for sub-specialty ophthalmology assessment and had a CVI completed in Bristol Eye Hospital. The patient was entered onto the Bristol Social Services register at that visit. The CVI was later forwarded to North Somerset Social Services and entered onto their register. The patient was not deleted from the Bristol register and therefore these patients, representing 5.3% of all patients in our study, would be counted twice when the individual Social Services figures were forwarded to The Health and Social Care Information Centre. However, due to the way the CVI forms are processed locally, only one copy of the CVI form would be forwarded to the Certifications Office in London. If this problem was duplicated around other regions in England and Wales, then it may explain a proportion of the discrepancy noted by Bunce *et al*.[1]

Conflict of interest

The authors declare no conflict of interest.

Reference

1 Bunce C, Xing W, Wormald R. Causes of blind and partial sight certifications in England and Wales: April 2007–March 2008. *Eye* 2010; **24**: 1692–1699.

MJ Wakefield, DM Tole, CC Bailey, MV Mundasad and JM Sparrow

Department of Ophthalmology, Bristol Eye Hospital, Bristol, UK
E-mail: mjwakefield_uk@hotmail.com

Eye (2011) **25,** 821; doi:10.1038/eye.2011.20; published online 25 February 2011

Sir,

Response to Wakefield *et al*

We thank Wakefield *et al*[1] for their interest in our paper[2] and for a possible explanation regarding the discrepancy between certification and registration figures. An additional reason is that, despite much publicity, some Eye units are not using the correct address for the Certifications Office. It is important to note that we would not expect an exact match between certification and registration figures for the same time period, as there can be some delay between certification and registration.

We believe, however, that increasing use of an electronic alternative will resolve these issues and lead to high-quality data being readily available on the numbers newly certified by cause.

Conflict of interest

The authors declare no conflict of interest.

References

1 Wakefield MJ, Tole DM, Bailey CC, Mundasad MV, Sparrow JM. Response to Bunce *et al*. *Eye* 2011; **25**: 821.
2 Bunce C, Xing W, Wormald R. Causes of blind and partial sight certifications in England and Wales: April 2007–March 2008. *Eye* 2010; **24**: 1692–1699.

C Bunce, W Xing and R Wormald

Research & Development, Moorfields Eye Hospital, London, UK
E-mail: c.bunce@ucl.ac.uk

Eye (2011) **25,** 821; doi:10.1038/eye.2011.21; published online 25 February 2011

Sir,

Cystoid macular oedema successfully treated by cryotherapy in retinitis pigmentosa with Coats'-like retinal exudation

The association between RP and exudative retinopathy was first reported by G. Zamorani in 1956.[1] Coats'-like retinal changes are seen in 1–3% of patients affected by RP.[2] The aetiology is unknown. Mutations in the Crumbs homologue 1 (*CRB1*) gene have been reported[3] as a risk factor for developing Coats'-like changes in patients with autosomal recessive RP.

We present a case of a young patient with Coats'-like autosomal dominant retinitis pigmentosa (ADRP), exudative retinal detachment (E-RD), and cystoid macular oedema (CME), successfully treated with cryotherapy.

Case report

A 10-year-old boy presented at the Retina Clinic in Southampton Eye Unit with marked deterioration in visual acuity (VA) in his right eye. VAs were 0.67 logMAR OD and 0.14 logMAR OS. There was a family history of ADRP (Figure 1). RP was diagnosed in the proband by retinal findings (Figure 2) and by electrophysiology performed 7 years before presentation (ERG, VEP). Mixed cone/rod ERGs as well as independent testing of the cone and rod-mediated responses were of borderline amplitude and binocular occipital pattern VEPs were present to the smallest checks. No funding for genetic testing for RP was available for any of the patient's affected relatives. In the right eye fundoscopy demonstrated mild vitreous haze, 'bone spicule like' pigment accumulation in the periphery bilaterally, Coats'-like E-RD, CME, teleangectatic vessels, and preretinal haemorrhages in the infero-temporal quadrant.