smoking on the development of severe retinopathy of prematurity. *Eye* 2010; **24**: 1024–1027.

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Sir,

Response to Ram

We appreciate the letter from Ram and McDonald (2010) regarding our manuscript.[1]
We understand that this letter questions our statistics. We discussed this issue with our statisticians and would like to argue against the letter.

(1) A relative risk can be obtained only by cohort study, not by case–control study as performed in our manuscript. Only 'odds ratio' can be obtained by case–control study. If Ram does want to work out a relative risk ratio using our data set, probably it will be better to subdivide the group into 'smokers' and 'non-smokers'. Dividing into severe ROP and non-severe ROP groups is not appropriate, as they are just the outcome of the observation. Anyhow, it does not make sense in such a case–control study.
(2) We would like to explain the meaning of 95% CI. We reported the odds 'ratio' for possible risk factors of severe ROP. If the 95% CI of odds ratio includes '1' (not 0) in the interval, it makes the result statistically non-significant. However, the 95% CI for maternal smoking was lower than '1' in our result, which clearly showed statistical significance.

Hence, we would like to say that our statistical analysis was correct. However, we never recommend maternal smoking, owing to a number of smoking-related systemic adverse events in mothers and infants. We reported our results only because they may give insight into some aspects of complicated ROP pathogenesis.

Conflict of interest
The authors declare no conflict of interest.

Reference

1 Ram FSF, McDonald EM. Response to 'Inhibitory effects of maternal smoking on the development of severe retinopathy of prematurity'. *Eye* 2010; **25**: 123–124.

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Sir,

Serous retinal detachment induced by topical bimatoprost in a patient with Sturge–Weber syndrome

We report a patient with Sturge–Weber syndrome-associated diffuse choroidal haemangioma who developed serous retinal detachment shortly after starting topical bimatoprost. Cessation of bimatoprost led to complete resolution of the subretinal fluid. This adverse effect of bimatoprost has not been previously reported.

Case report

A 16-year-old girl with Sturge–Weber syndrome-associated bilateral diffuse choroidal haemangiomas was referred with a 6-month history of blurred vision in her left eye. She had been treated for ocular hypertension for 11 years and was using timolol/dorzolamide (Cosopt), brimonidine, and bimatoprost for both eyes.

The blurred vision began shortly after switching from latanaprost to bimatoprost in both eyes. Visual acuities were 6/5 in the right and 6/9 in the left eye. Bilateral diffuse choroidal haemangiomas were confirmed on angiography (fluoroscein and indocyanaine green) and ultrasound. OCT confirmed the presence of significant subretinal fluid inferiorly, which extended to the left fovea, where the changes looked chronic and there was fibrinous deposit (Figures 1a and b).

An adverse effect of bimatoprost was suspected due to its recent introduction and the fact that a similar case related to travoprost had previously been reported.[1] Bimatoprost was therefore stopped and the patient was reviewed 6 weeks later, during which time she noticed subjective visual improvement. Visual acuity had improved to 6/5 in either eye and OCT confirmed complete resolution of the subretinal fluid (Figures 1c and d).

Visual acuity has remained 6/5 in either eye with no subretinal fluid through 1 year of follow-up. Intraocular pressures are controlled on g.Cosopt tds, g.Apraclonidine 0.5% tds, and g.Pilocarpine 2% tds to both eyes.

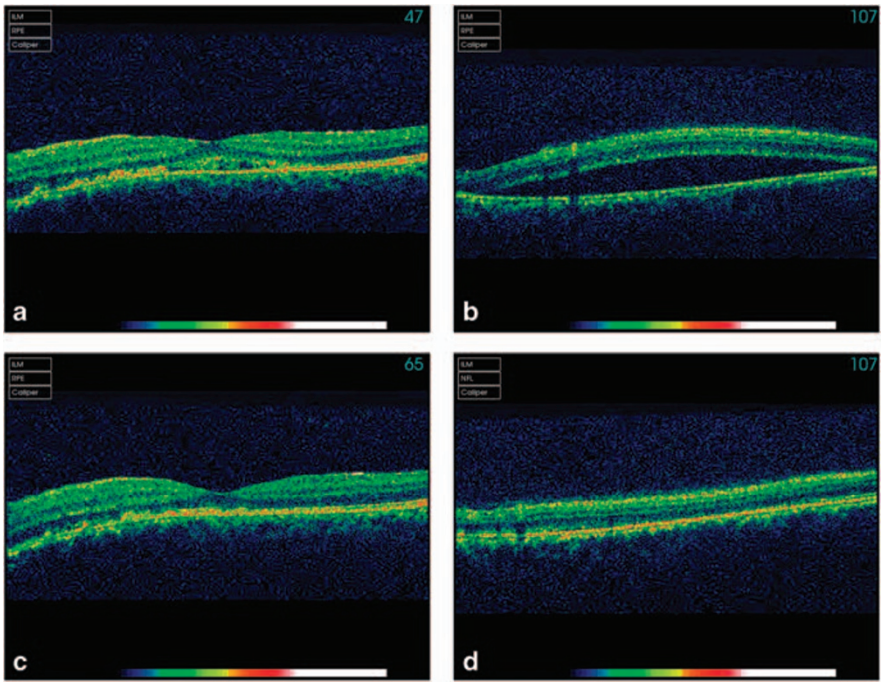

Figure 1 OCTs of the left eye showing fovea on presentation (a), inferior retina on presentation (b), fovea 6 weeks after stopping bimatoprost (c), inferior retina 6 weeks after stopping bimatoprost (d).

Comment

Sturge–Weber syndrome is a sporadic neurocutaneous disorder characterised by facial capillary malformation (port-wine stain), leptomeningeal angioma, and vascular ocular abnormalities.[2,] The ocular manifestations consist of glaucoma in 71%, conjunctival or episcleral haemangiomas in 69%, and diffuse choroidal haemangiomas in 55% of patients.[3] There has been one report of a patient with Sturge–Weber syndrome who developed uveal effusion shortly after starting topical travoprost, which resolved approximately 3 weeks after stopping the travaprost.[1] Our case report suggests that bimatoprost, as well as travoprost, should be used with caution in eyes with Sturge–Weber syndrome-associated diffuse choroidal haemangioma.

Conflict of interest

The authors declare no conflict of interest.

References

1 Gambrelle J, Denis P, Kocaba V, Grange JD. Uveal effusion induced by topical travoprost in a patient with Sturge-Weber-Krabbe syndrome. *J Fr Ophtalmol* 2008; **31**(9): e19.
2 Baselga E. Sturge-Weber syndrome. *Semin Cutan Med Surg* 2004; **23**(2): 87–98.
3 Sullivan TJ, Clarke MP, Morrin JD. The ocular manifestations of the Stugre-Weber syndrome. *J Paediatr Ophthalmol Strabismus* 1992; **29**(6): 349–356.

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