should be considered in patients with unexplained loss of foveal architecture.

Conflict of interest
The authors declare no conflict of interest.

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Sir,

Response to 'Inhibitory effects of maternal smoking on the development of severe retinopathy of prematurity'

The article by Hirabayashi *et al*[1] is an interesting report on the inhibitory effects of maternal smoking on the development of severe retinopathy of prematurity (ROP). However, I do not believe that the conclusion derived (that maternal smoking leads to a reduction in the incidence of severe ROP) is at all supported by the results reported. There were 27 infants that developed severe ROP, of whom only a single mother smoked (and the other 26 mothers were non-smokers). The authors' conclusion that maternal smoking reduced the incidence of severe ROP is based on a single smoker, as they ignored the 26 other non-smoking mothers. In fact, using the reported events rates for development of severe ROP (1/27 maternal smokers versus 26/27 non-smokers), one obtains a relative risk (RR) of 0.04 and 95% CI of 0.01–0.26 ($P = 0.0009$, see Figure 1). This clearly shows that non-smoking provides protection against the development of severe ROP, with a reduction in risk of 96% compared with maternal smoking. Strangely enough, the authors reported these data using odds ratios, especially as the event rate in the maternal smoking group is low and their reported 95% CI (Table 2, p 1026) includes '0' in the interval, making the result statistically non-significant. Therefore, one can only conclude that maternal smoking does not reduce the incidence of severe ROP.

The authors have erroneously concluded that maternal smoking reduced the incidence of severe ROP, when in fact only 1/27 (or 4%) reported maternal smoking and 26/27 (or 96%) did not report any maternal smoking. *Lack of evidence does not equate to evidence of an effect (or association in this case)*. In the non-severe ROP group, 15/59 (or 25%) mothers reported maternal smoking and the authors did not report a reduction in the incidence of non-severe ROP. Re-analysis of the reported data (development of non-severe ROP; smokers 15/59 versus non-smokers 44/59) provides the following: RR 0.12, 95% CI 0.05–0.27 ($P < 0.00001$), favouring non-smokers with a reduction in the incidence of non-severe ROP of 88%.

The correct and only conclusion from this report should read as follows: No maternal smoking provides protection against the development of both severe and non-severe ROP. There is no evidence to support that maternal smoking offers any protection against the development of ROP (Figure 1).

Conflict of interest
The authors declare no conflict of interest.

Reference
1 Hirabayashi H, Honda S, Morioka I, Yokoyama N, Sugiyama D, Nishimura K *et al*. Inhibitory effects of maternal

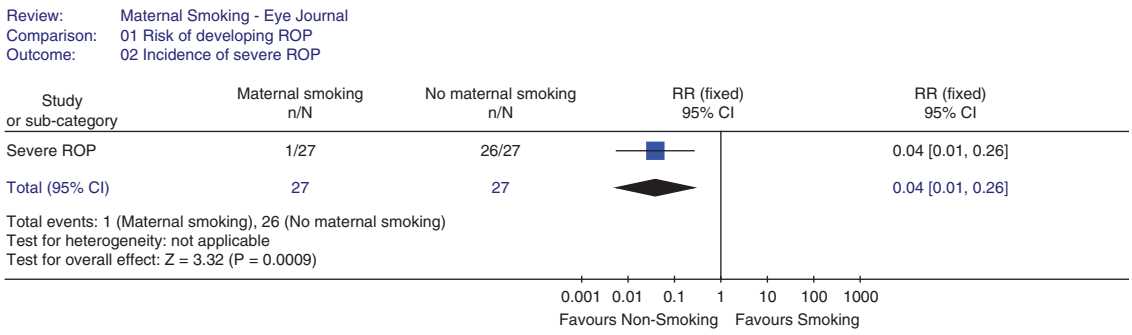

Figure 1 Risk of developing severe retinopathy of prematurity during maternal smoking.

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.