Effect of Intraocular Pressure on the Bayesian Estimation of Rates of Visual Field Progression in Glaucoma

I read, with great interest, the study by Anderson and Johnson on the use of population information to modify estimates of rates of visual field progression in glaucoma through Bayesian analysis.1 I was surprised, however, to verify that the authors omitted previous work on this subject, which was actually the first to investigate this issue.2–3 Using real data from a large cohort of 352 eyes of 250 glaucoma patients, my colleagues and myself showed that incorporating risk factors into the estimation of rates of visual field change using Bayesian analysis resulted in significant improvement over the ordinary least squares approach.2 We showed that incorporating information about IOP during follow up, along with corneal thickness and presence/absence of progressive disc damage, resulted in more accurate and precise slopes, with better prediction of future standard automated perimetry mean deviation (MD) values.

In contrast to our results, Anderson and Johnson concluded that failure to consider information on IOP did not alter the performance of a Bayesian estimator of visual field progression.1 They also suggest that because of a lack of significant influence of IOP, efforts of considering other risk factors would have even smaller significance.1 However, there are simple explanations for the lack of significance of the results presented by Anderson and Johnson and they are mostly related to the poor definition and characterization of the priors used in their study. The authors evaluated whether two different priors, representing the distributions of slopes of change in treated versus untreated glaucomatous populations, would differently influence the estimates of rates of visual field progression.1 The priors used in their study, however, were derived from previously published data from two widely different populations followed as part of the Canadian Glaucoma Study (the treated group) and Early Manifest Glaucoma Trial (EMGT, the untreated group).2–3 This approach, however, has major limitations. It would have been more appropriate to obtain the two distributions from a homogenous population randomized to treatment versus no treatment. By using widely different populations from different geographic areas and clinical settings, the authors are ignoring potentially confounding factors that could be important in determining the distribution of rates of change in these populations. Even more importantly, to evaluate the effect of IOP by simply using two prior distributions of rates of change categorized as “treated” versus “untreated” is largely inappropriate. This essentially ignores the continuous effect of IOP on the risk of glaucoma progression, as shown by several major clinical trials.4–6 For example, while the distribution of rates of change for a population of treated glaucomatous eyes with IOP of 12 mm Hg will be largely different than that of an untreated population with IOP of 30 mm Hg, such differences will be largely missed by simply considering these eyes as part of broad groups of treated versus untreated eyes. In the treated group from the Canadian Glaucoma Study there were many eyes with IOPs that overlapped with those of the untreated group from the EMGT. Additionally, within each one of these two groups there are major differences in the IOPs of the included eyes. As another example, one should not expect that the distribution of rates of change for eyes with a mean IOP of 10 mm Hg would be the same as that for eyes with a mean IOP of 20 mm Hg, even if these eyes were both treated over time.6 In essence, the methodology used by Anderson and Johnson was not able to capture the effect of IOP on estimation of rates of change.1 In contrast to their analysis, our method allowed prior distributions that would vary according to each level of IOP providing a much better way of assessing the impact of IOP on the estimation of rates of change using Bayesian analysis.2

In conclusion, I congratulate the authors for their effort in evaluating the complex issue of estimating rates of change in glaucoma. However, the conclusions of their work are the result of improper prior design and do not seem to be justifiable. I hope that their results will not adversely impact the promising use of Bayesian techniques to incorporate risk factor information into the assessment of rates of progression in glaucoma.

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References


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