eligible. Diagnoses were verified through use of more than a single ophthalmologist. Owing to the large sample size ($N = 962$), these results would be more representative of the study population than in the current (inner city) study.

Finally, the authors need to be commended for presenting not just 'point prevalence values' but also confidence intervals (CI) for this parameter. This obviously helps the reader to see that virtually all of the values have wide CI raising questions on the precision of these estimates. For example, nonstandardized prevalence of visual disability was 500 per 10 000 (95% CI, 242–900 per 10 000), and nonstandardized prevalence of low vision and blindness 400 per 100 000 (95% CI, 174–770).

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Sir,

Reply to Dr Muula

We would like to thank Dr Muula for his comments regarding our recent publication.[1]

The purpose of this paper was not to precisely define the prevalence of disability in a population, but to identify a vision crisis that has not previously been recognized and to put a best estimate on the prevalence of this problem in a very marginalized community.

The first issue Dr Muula raises concerns how representative our study sample was of the general inner-city population in Vancouver's downtown eastside (VDES). We agree that our data may underrepresent individuals who do not attend medical care and have noted this in the second-last paragraph of the paper. However, we must emphasize that our subjects were not attending the Vancouver Native Health Society (VNHS) for eye examinations as Dr Muula suggests. Instead, these individuals were there for general, nonophthalmic care (paragraph 2 of the Methods section). As a result, we believe, there is no selection bias towards eye disease in our sample. Moreover, the dates and times of each intake clinic were varied over the course of the 2-year study period, and were not conducted at the same time of day or on the same day of the week. As such, we believe that we achieved as representative a sample of clinic attendees as possible. We also know that demographic data from the VNHS clinic has been found to correspond quite closely to the larger VDES community.

Dr Muula also has concerns regarding the use of a single ophthalmologist for the eye examinations in our study. We do not believe this is a valid criticism. First, our study did not require specific patient diagnoses, only a simple categorization of the aetiology of vision loss—a routine practice for ophthalmologists. Second, although it would have been interesting to have more than one physician confirm our ocular classification, such an approach was not practical from a physician availability standpoint and would not necessarily have improved our categorizations. Third, contrary to Dr Muula's comments, all of the ophthalmic diagnoses in our prior study of a medium-sized Canadian city (Prince George) were also made by a single ophthalmologist.[2] This latter study was a chart review and, as such, the patients' ophthalmologists were occasionally consulted if there was diagnostic uncertainty for the physician performing the data abstraction.

We agree that there are methodological differences between our VDES and Prince George studies. These differences were unavoidable given the dissimilarities of the medical and social environments in these communities. Our intergroup comparisons are not intended to be unqualified; however, the prevalence figures for our VDES population (even taking into

account the lower bound of the confidence intervals) do suggest that this is a distinctly different cohort of individuals with alarmingly high rates of vision loss (up to 10 times higher) compared with the general Canadian population.

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Sir,

Nodular non-necrotising anterior scleritis due to *Nocardia nova* infection

The genus *Nocardia* encompasses saprophytic, aerobic, Gram-positive, nonmotile, weakly acid-fast, and branching filamentous bacteria. Ocular manifestations of *Nocardia* infection vary. Isolated scleritis due to *Nocardia* species are rare.[1] Scleritis usually spreads from corneal infection involving the limbus. The identification of the organism to species level is important as antimicrobial susceptibility patterns may vary widely between *Nocardia* species. Reported predisposing factors for scleritis due to *Nocardia* are trauma, cataract surgery, exposed scleral buckle, and contact lens wear.[2–6] We are unaware of a previous report on *Nocardia nova* infection in the *Eye*.

Case report

A 40-year-old woman presented with a 10-week history of excruciating pain and redness in the left eye. She was using topical steroid and oral nonsteroidal anti-inflammatory drugs (NSAIDs) for the previous 4 weeks. There was a history of mud splashed into her face 1 week before symptom onset, although she did not feel any splash into the eyes. She had a history of hepatitis C and past intravenous drug use, but denied injecting intravenous drugs for the past 10 years. Her only regular medication was methadone. At initial examination, her visual acuity in both eyes was 6/6. Her right eye examination revealed no abnormality.

Slit-lamp examination of the left eye showed a single focal superonasal elevated (size 6 × 6 mm in diameter and 2 mm elevation) subconjunctival-congested nodule (Figure 1a). It was firm. The cornea was clear and anterior chamber was quiet. The posterior segment was normal. Oral prednisolone (60 mg) was started, with immediate resolution of pain. However, the nodule persisted and 3 weeks later the apex of the nodule became yellow. The appearance and the lack of response to therapy suggested an infective aetiology. Therefore,

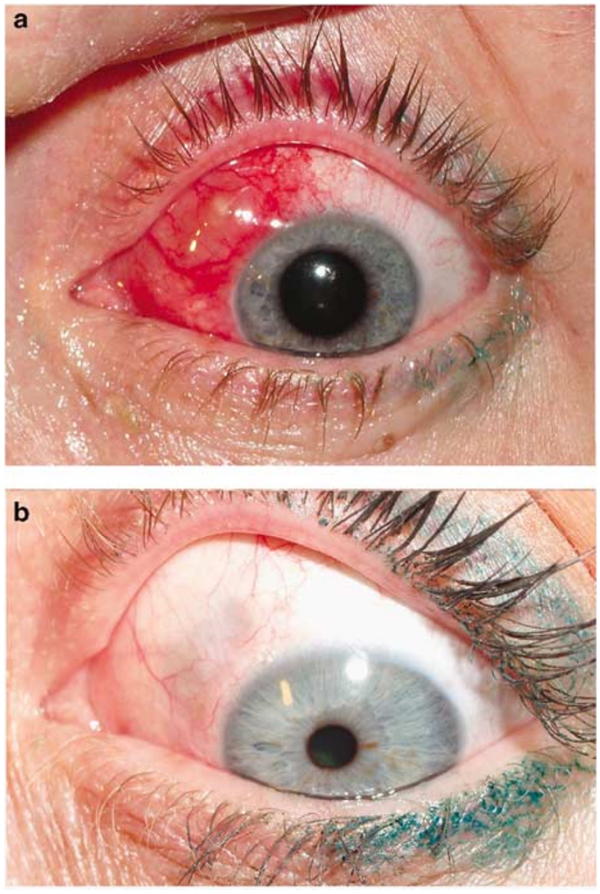

Figure 1 (a) Clinical photograph of superonasal scleral nodule before biopsy. (b) Clinical photograph after resolution of nodule showing scleral thinning.