Sir,

**Authors' reply: 'Optical coherence tomography in a patient with tobacco-alcohol amblyopia'**

We thank Dr Grzybowski for his interest in our article,[1] and his comment that the term, 'tobacco-alcohol amblyopia' is inappropriate, because 'it is in fact just 'nutritional optic neuropathy' related to deficiencies of vitamin B and foliate acid'. In addition, Dr Grzybowski suggested that Leber's hereditary optic neuropathy (LHON) should be considered.

We completely agree that the use of amblyopia is inaccurate, and also that there is a nutritional component to tobacco–alcohol amblyopia.[2] Nevertheless, many studies have confirmed the toxic effects of tobacco smoke and ethanol, and the pathogenesis of tobacco–alcohol amblyopia has yet to be elucidated.

Tobacco smoke contains a range of toxins, such as carbon monoxide and cyanide, that are capable of damaging the optic nerve and retina.[3] Cyanide in cigarette smoke is not properly metabolized in patients with tobacco–alcohol amblyopia because of alcohol-related hepatic dysfunction and a lack of vitamin $B_{12}$.[4] Moreover, cyanide blood levels are elevated,[5] and smoking is known to impair vitamin $B_{12}$ absorption in tobacco–alcohol amblyopia patients.[6]

Alcohol consumption can cause electroretinographic abnormalities,[7,8] and prolonged exposure to ethanol during fetal development is known to have adverse effects on the retina that may persist into adulthood.[9] Moreover, by-products generated during the metabolism of ethanol affect metabolic processes in neural tissues.[10] Several underlying mechanisms, associated with the toxic effects of tobacco smoke and alcohol or related to nutritional deficiency, may determine the development of tobacco–alcohol amblyopia.[11] Thus, in our opinion, tobacco–alcohol amblyopia cannot be straightforwardly classified as a nutritional optic neuropathy.

Regarding the possibility of LHON, four mutations at nucleotides 11778, 14484, 3460, and 4171[12] previously found in Koreans were not found in our patient. Moreover, although there is a possibility that some other LHON mutation may have been present, we emphasize that the clinical features of our patient were incompatible with a diagnosis of LHON. We would have preferred to have added details of our mitochondrial DNA mutation analysis results, but unfortunately, could not do so owing to the word count restriction.

Regarding blood levels of $B_{12}$ and folic acid, when the patient was referred, he was in good nutritional condition, and therefore these levels were not determined. However, as we described in the article, our patient admitted not eating when he had been drinking 4–5 bottles of Korean gin per day for 10–14 days, once or twice a year for the past 4 years. We appreciate Dr Grzybowski for having given us another opportunity to clear these issues.

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Sir,

**Tobacco amblyopia: does it really exist?**

With reference to Kee and Hwang's article entitled 'Optical coherence tomography in a patient with tobacco-amblyopia', I doubt whether the presented case of optic neuropathy could be appropriately named 'tobacco amblyopia'.

In the last two centuries it has been believed that tobacco consumption might result in toxic neuropathy called 'tobacco amblyopia'.[1] Tobacco neuropathy has been supposed to originate from excessive cigar and pipe smoking (or tobacco chewing or snuffing) rather than from cigarette smoking.[1] Presently, this disorder is less frequent. Some even argue that 'tobacco amblyopia' does not exist. Nevertheless, some data collected during the