million people are infected with trachoma. Five million people are infected with its late stages and two million are blind because of it.

The trachoma biovar of *C. trachomatis* can be subdivided into 15 serovars which are designated by the letters A–K based on the polymorphism in the sequence of the major outer membrane protein.^{2,3} Serovars A–C can usually be isolated from patients with clinical trachoma in regions where trachoma is endemic.

Oculogenital infection caused by serovars D–K is common in developed countries and leads to inclusion conjunctivitis, also called paratrachoma. Up to 90% have concurrent urogenital infections.

Although the different groups of serovars display unique tissue tropisms, they are not tissue selective, and serovars D–F, J and K have all been isolated from conjunctival swabs taken from individuals with typical clinical signs of active trachoma.^{4–7} However, all these reports are from areas where trachoma is endemic.

Therefore, the traditional distinction between ocular and genital strains may have to be reconsidered.

Clinically, it is difficult to diagnose the beginning of trachoma or inclusion body conjunctivitis, and it is only by laboratory testing that the diagnosis can be confirmed. Among the available assays, nucleic acid amplification tests have proven superior in detecting, quantifying and genotyping *C. trachomatis*.⁸ Because chlamydia is an intracellular organism, the correct swab technique is very important to obtain a positive test result. This includes firm rubbing with the swab in the fornix, which is unpleasant for the patient. Because chlamydial infection is frequently oligosymptomatic and routine laboratory screening is seldom not readily available for the ophthalmologist practitioner, the diagnosis of the disease is often delayed or even missed. Additionally, repeated reinfection occurs when the sexual partner is not treated as well.

These difficulties lead to a prolonged course of the infection and possibly to scarring due to chronic inflammation and repeated reinfections. Unfortunately, we were not able to differentiate the serovar of the *C. trachomatis* isolate in our case, as serotyping is not routinely performed in Switzerland. However, the advanced clinical findings leading to entropion necessitating surgery emphasise not only the need for early diagnosis and treatment of this disease but also the need for thorough clinical examination including eversion of the upper lid and inspection of the tarsal conjunctiva in any patient with conjunctivitis.

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CORRECTIONS

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Several errors occurred in the paper titled, Linezolid-induced optic neuropathy: a mitochondrial disorder? (*Br J Ophthalmol* 2007;**91**:111–5). A fully corrected pdf is available online at <http://bjo.bmj.com/>. The journal apologises for these errors.

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In the paper titled, Plasma apolipoproteins and risk for age-related maculopathy (*Br J Ophthalmol* 2006;**90**:1028–33) previous literature has been incorrectly cited within the text.

In second paragraph of the Discussion section (page 1030, right column), the authors refer to "large associations between neovascularisation and plasma cholesterol (OR = 4.1) in the Eye Disease Case Control Study (EDCCS)⁴⁸ and neovascularisation and apoB (OR = 9.2) in the Beaver Dam Eye Study (BDES).⁴⁹" Further on in the same paragraph, we state, "It is not clear why the EDCCS and BDES obtained the strong effects they did".

In both cases these results were incorrectly attributed to the BDES. These two sentences should have instead cited Reference 79, which pertains to data from the NHANES III study. Citations to References 49 (BDES) and 79 (NHANES II) are correct in the 3 tables.