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Sir,

Reply to Zayats *et al*

We thank Zayats *et al* for their comments and interests in our recently published article.[1] They were concerned with the different effect of PAX6 polymorphism in myopia between Taiwan and Europe, the reason of lack of association with high myopia, and the risk of a Type I error in our study. We would like to reply their comments as follows.

More and more evidences support that myopia is caused by both genetic and environmental factors and possibly their interactions.[2] Besides the interactions with environment, owing to multiple genes with small effects, genetic heterogeneity and phenotypic complexity, the study of the genetics of myopia poses a complex challenge and may obtain different results in different countries. Hence, the effects of PAX6 polymorphisms in myopia are likely to be different between Taiwan and Europe because of different environment and race.

Prolonged near visual tasks is an important environmental influence in myopia in Taiwan: individuals with higher education have a higher prevalence of myopia than people in the general population.[3,4] However, among the students in the same class of the same university, who were previously performing similar near visual tasks, their severity of myopia varied widely. For example, the first-year medical students in our China Medical University, although most of them are among mild-to-high myopia, there are extreme myopia. Because they did similar near visual tasks, we assume that their near works resulted in mild-to-high myopia, and there were genes predisposing some students to develop high-to-extreme myopia. Hence, the lack association of PAX6 with high myopia in our study may be due to the distinction in genetic risk factors for high and extreme myopia, or part of high myopia students are caused by their near works only, which is not related to PAX6 polymorphism, suggesting that high myopia can be caused by genetic or environmental factors separately or through their interactions.

The maximum chance of making a Type I error is denoted by alpha. Because our *P*-values are either 0.002 or less than 0.001, the probability of making a Type I error is low.

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Sir,

Bilateral *Candida* chorioretinitis following etanercept treatment for hidradenitis suppurativa

Hidradenitis suppurativa (HS) is an inflammatory disease with chronic acneiform infection of the cutaneous apocrine glands. Etanercept, an anti-tumor necrosis factor-α (TNF-α) agent, is effective in the management of HS.[1] Infectious complications have been described following treatment with etanercept,[2] including uveitis.[3]

Case report

A 48-year-old woman was referred to our department because of bilateral blurred vision and floaters for 2 days. She had been hospitalized 35 days before due to a secondary amyloidosis after 8 years of HS, which was being treated with prednisone 5 mg daily and subcutaneous 25-mg etanercept injections every 4 days for 3 months. During the hospitalization, she developed a superficial phlebitis in her left arm (where she had a catheter) followed by a septicemia, with positive cultures for *Candida albicans* in the catheter and in the hemocultives. She was treated with caspofungine and etanercept removal.

Baseline visual acuity was 20/60 in the right eye and 20/40 in the left eye. Ophthalmic exploration showed one yellow-white chorioretinal yuxtafoveal lesion with perilesional hemorrhage in both eyes and a similar parafoveal lesion in the left eye, with neither vitreous haze nor cells (Figure 1a). Chest X-ray, tuberculin skin test, and serologic tests were normal or negative. The association of these ocular and microbiologic findings drove us to the diagnosis of *Candida* chorioretinitis, which improved after systemic fluconazol, with no active

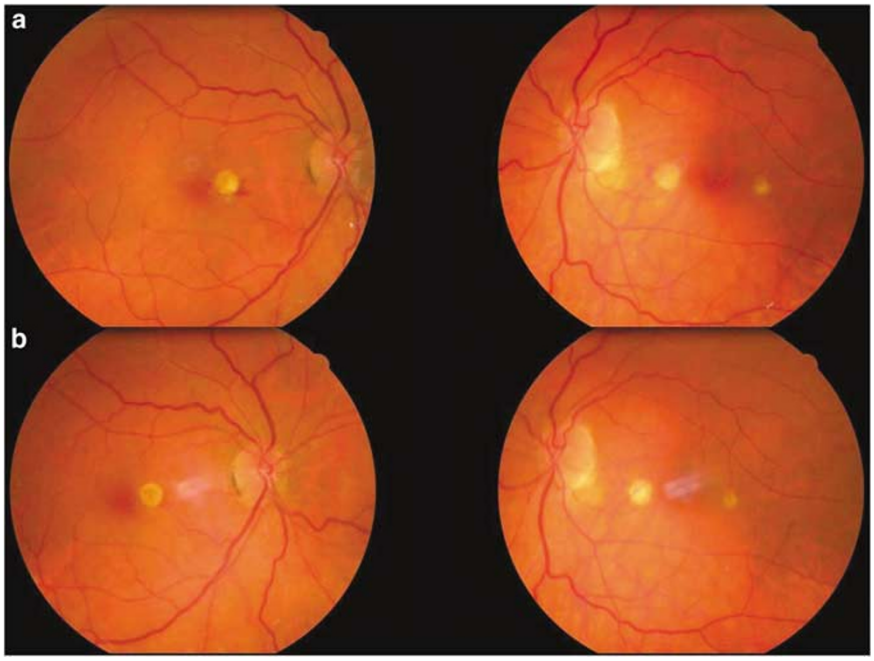

Figure 1 Both eye retinographies showing active chorioretinal lesions due to *Candida* on the baseline visit (a) and chorioretinal scars after treatment (b).

chorioretinal lesions after 3 weeks (Figure 1b). Final visual acuity was 20/30 in both eyes.

Comment

TNF-α is a proinflammatory cytokine, which plays an important role in the pathogenesis of inmune-mediated diseases and in the immune mechanisms against infection. The use of TNF-α inhibitors has been associated with an increased rate of intracellular infections.[4] Even so, there is little evidence about *Candida* infections among patients treated with etanercept. Wallis *et al*[4] cited a rate of 7.1 *Candida* infections (no reference to ocular affectation) per 100 000 patients who received etanercept. Its use, associated to chronic corticosteroid treatment and intravenous catheter, led to a fungal septicemia in our patient, which was followed by the bilateral chorioretinitis. In conclusion, we should consider *Candida* species as a possible etiology of chorioretinitis in patients taking etanercept.

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Sir,

Combined pharmacotherapy and thermotherapy for chronic central serous chorioretinopathy with anterior segment neovascularisation

Retinal vascular nonperfusion has long been recognised in chronic central serous chorioretinopathy (CCSC).[1] Occlusion of the peripheral vessels is often misdiagnosed as vasculitis, resulting in administration of corticosteroids.[1–3] We describe management of CCSC complicated by rubeosis in similar circumstances.

Case report

A 38-year-old healthy man presented with diminished vision in both eyes for many months. Best-corrected visual acuity was 6/24 in the right eye and 6/12 in the left eye. He was treated with oral corticosteroids elsewhere for presumed retinal vasculitis. He had no history of any previous ocular or systemic disease. Systemic examination and laboratory work-up by the