T Zayats[1], JA Guggenheim[1], CJ Hammond[2] and TL Young[3]

[1]School of Optometry and Vision Institute, Cardiff University, Cardiff, Wales, UK
[2]Twin Research and Genetic Epidemiology Unit, St Thomas' Hospital, London, UK
[3]Duke University Center for Human Genetics, Durham, NC, USA
E-mail: guggenheim@Cardiff.ac.uk

Eye (2008) **22,** 598–599; doi:10.1038/sj.eye.6703096; published online 25 January 2008

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.

References

1 Tsai YY, Chiang CC, Lin HJ, Lin JM, Wan L, Tsai FJ. A PAX6 gene polymorphism is associated with genetic predisposition to extreme myopia. *Eye* 2007; e-pub ahead of print 19 October 2007; doi:10.1038/sj.eye.6702982.
2 Lyhne N, Sj_lie AK, Kyvik KO, Green A. The importance of genes and environment for ocular refraction and its determiners: a population based study among 20–45 year old twins. *Br J Ophthalmol* 2001; **85**: 1470–1476.
3 Lin LL, Shih YF, Hsiao CK, Chen CJ, Lee LA, Hung PT. Epidemiologic study of the prevalence and severity of myopia among schoolchildren in Taiwan in 2000. *J Formos Med Assoc* 2001; **100**: 684–691.
4 Wong TY, Foster PJ, Hee J, Ng TP, Tielsch JM, Chew SJ *et al.* Prevalence and risk factors for refractive errors in adult Chinese in Singapore. *Invest Ophthalmol Vis Sci* 2000; **41**: 2486–2494.

Y-Y Tsai, C-C Chiang, L Wan and F-J Tsai

Department of Ophthalmology, China Medical University Hospital, Taichung, Taiwan, China
E-mail: elsa10019@yahoo.com.tw

Eye (2008) **22,** 599; doi:10.1038/sj.eye.6703097; published online 25 January 2008

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