

CTLA-4 A49G polymorphism and autoimmune blood disease: A comment

CTLA-4 A49G polimorfizmi ve otoimmün kan hastalığı: Bir yorum

Viroj Wiwanitkit

Wiwanitkit House, Bangkhae, Bangkok, Thailand

To the Editor,

I read the recent publication on cytotoxic T lymphocyte antigen-4 (CTLA-4) A49G polymorphism with a great interest [1]. Aktürk et al. concluded that, “these data suggest that CTLA-4 A49G polymorphism does not contribute to the pathogenesis of lymphoproliferative diseases itself, nor does it increase the risk of autoimmune complications in patients with lymphoproliferative disease [1].” Aktürk et al. tried to determine allele frequencies and genotype distributions for some autoimmune blood diseases. There are some problems with their conclusion. First, the study included only a few patients and no controls. Second, not all autoimmune blood diseases were analyzed; therefore, they cannot conclude that their finding supports or

refutes the contribution of the studied polymorphism to the pathogenesis or risk of disease. Third, when investigating a single polymorphism the possibility of other polymorphisms that were not investigated, must be considered.

Conflict of interest statement

The authors of this paper have no conflicts of interest, including specific financial interests, relationships, and/or affiliations relevant to the subject matter or materials included.

References

1. Aktürk F, Hançer VS, Küçükkaya R. Cytotoxic T lymphocyte antigen-4 (CTLA-4) A49G polymorphism and autoimmune blood diseases. *Turk J Hematol.* 2010;27:78-81.