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Interleukin-1 gene cluster polymorphisms associated with coronary slow flow phenomenon

To the Editor,

We found the publication "Association of Interleukin-1 Gene cluster polymorphisms with coronary slow flow phenomenon (CSFP)" (1) very interesting. Mutluer et al. (1) concluded that "IL-1ß+3954 SNP mutations are significantly more common in patients with CSFP" and "It may suggest that the tendency for inflammation may contribute to the presence of this phenomenon." In fact, based on the present study, a conclusion can be made only regarding genetic frequency. It is not possible to propose any pathophysiology regarding the inflammation process since no inflammatory parameter was assessed. In fact, if there is a direct pathological process as a result of the polymorphism, similar findings should be observed for both IL-1ß+3954 SNP and IL-1ß+3954 SNP. Finally, other SNPs of IL-1ß, which were not investigated by Mutluer et al. (1), such as IL-1ß -634SNP (2), can also have the same effect on CSFP.

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Author`s Reply

To the Editor,

We would like to thank authors for their valuable comments on our recently published study titled "Association of Interleukin-1 Gene cluster polymorphisms with coronary slow flow phenomenon (CSFP)" (1). We cannot disagree on their comment on the association between inflammation tendency and IL-1 gene polymorphisms. We would like to clarify that there is a thin line between drawing conclusions and suggesting hypotheses, and we stay on the side of just suggesting hypotheses. The main weakness of small-sized genetic case-control studies is their lack of power to draw conclusions from the results. This is the reason why the methodology of genetic studies is moving toward genome-wide association studies (2). It would have been better if serum interleukin-1ß and interleukin-1RA levels were evaluated in our study. This is among the limitations of our study. However, it should be noted that the effects of mutations on inflammatory mechanisms might as well be simply beyond increasing and decreasing the synthesis of the gene product. Conflicting results testing the same hypothesis that these mutations have effects on the course of diseases associated with inflammation also underline this complexity. Additionally, we should emphasize that the co-occurrence of single nucleotide polymorphisms is not a rule. Associations might vary between different polymorphisms in the same gene as a result (3). Finally, screening for all defined mutations and even describing new mutations is possible with next-generation sequencing. However, with conventional methodologies, how many different mutations can be studied is a matter of time and resources (4).

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