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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