Platelet count and Interleukin 6 Gene polymorphism in healthy subjects

ABSTRACT

This is the first description, to our knowledge, of a genetical influence on basal platelet counts, which appears to be partially dependent on a polymorphism of the IL-6 gene, even in the absence of inflammation.

INTRODUCTION

Background Interleukin-6 (IL-6) is a pleiotropic cytokine involved in the regulation of the acute phase reaction, immune responses, and hematopoiesis. Earlier studies have found that IL-6 is a potent thrombopoietic factor in vivo in mice acting on maturational stages in megakaryocytopoiesis and promoting platelet production. Human IL-6 was also found as a direct promoter of maturation of megakaryocytes in vitro. In recent reports, circulating IL-6 appeared to be an active mediator involved in the regulation of thrombopoiesis during clonal and reactive thrombocytosis. However, at the present moment, there is no evidence that IL-6 is required for normal thrombopoiesis in humans. A polymorphism in the 5' flanking region of the IL-6 gene alters the transcriptional response to stimuli such as endotoxin and interleukin-1. This G/C polymorphism of the IL-6 gene at position -174 has been found to be associated to different plasma IL-6 levels in healthy volunteers. Given the role of IL-6 in megakaryocytopoiesis we aimed to study the G/C polymorphism of IL-6 in relation with the blood platelet count.

CONCLUSION

A polymorphism of the IL-6 gene influences the platelet count in healthy volunteers even in the absence of inflammation. Whether this polymorphism leads to differences in reactive thrombocytosis merits further research.