The JAK2V617F Mutation in Polycythemia Vera and Other Myeloproliferative Disorders: One Mutation for Three Diseases?

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Abstract

The discovery of the JAK2V617F mutation has made the diagnosis of polycythemia vera (PV) much easier, but the pathogenesis of PV is still incompletely understood. In particular, it is not yet elucidated how a single mutation can be found in multiple myeloproliferative disorders (MPD) and myelodysplastic syndromes with ring sideroblasts and whether the sole JAK2V617F is sufficient to induce a MPD in humans. Several hypotheses are under investigation such as differences in the targeted hematopoietic stem cells (HSC), host modifier polymorphisms, intensity of JAK2V617F signaling, presence of other somatic mutations, or the presence of a pre-JAK2 event that may vary according to the MPD phenotype. Multiple studies have provided some evidence for and against each hypothesis, but it now seems possible to reconcile these hypotheses into a model that will need to be tested using newly developed tools. Recent investigations have also led to new treatment modalities that could benefit patients with PV.

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