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MUTATIONAL ANALYSIS OF THE EGF RECEPTOR GENE IN BCR-ABL1 NEGATIVE AND JAK2V617F NEGATIVE CHRONIC MYELOPROLIFERATIVE NEOPLASMS

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Background: *BCR-ABL1* negative chronic myeloproliferative neoplasms (CMPNs) are a heterogeneous group of clonal hematological malignancies. In last years, some genetic alterations have been described to cause these diseases, most of them activating tyrosine kinase (TK) genes. Tyrosine kinases proteins have an important role in cell growth and oncogenesis. Gain-of-function mutations in TK genes can produce a constitutive activation of several signaling pathways.

Aims: In this study, we study the *EGFR* gene that codes for a tyrosine kinase receptor (RTK) involved in signaling pathways relevant in hematological cells. Mutations in this gene have been found involved in lung cancer, and it could have an important role in the pathogenesis of hematological disorders.

Methods: We have analyzed the transmembrane and TK-coding domains of *EGFR* by dHPLC to detect mutations on samples from 44 *BCR-ABL1* negative / V617F*JAK2* negative CMPN patients and 20 control samples from healthy individuals.

Results: Our results show that this gene is not frequently mutated in CMPNs.

Conclusion: The EGF Receptor gene does not appear to be involved in the pathogenesis of the myeloproliferative neoplasms.

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