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JAK-STAT Signaling Contributes to the Pathogenesis of MPNs

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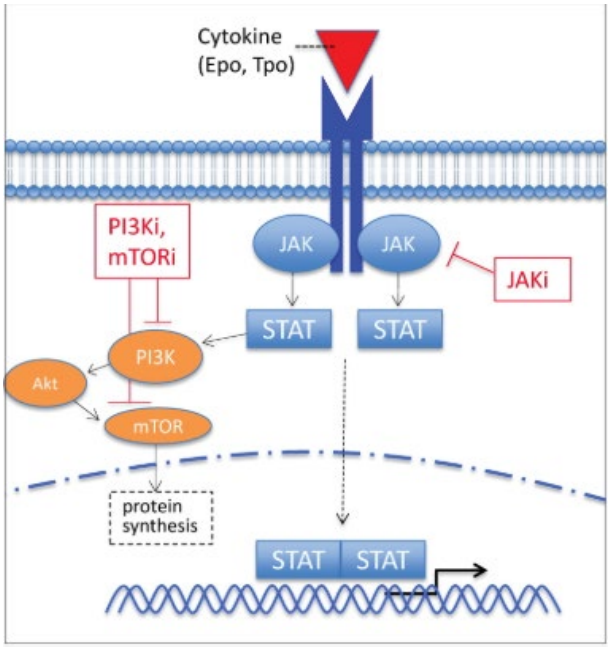

Figure 1 Activation of the PI3K/Akt/mTOR and Unregulated JAK-STAT signaling, leading to STAT-mediated hematopoiesis result from a number of aberrations including point mutations JAK2 V617F, leading to constitutive activation of, MPL W515L, an activating mutation of the thrombopoietin receptor and JAK2 kinase. There is a number of small-molecule inhibitors of these pathways are in clinical development such as JAK, PI3K, and mTOR inhibitors. There are some inhibitors present while JAK-STAT signalling to the pathogenesis of MPNs such as EPO, erythropoietin; JAK, Janus kinase; mTOR, mammalian target of rapamycin; PI3K, phosphoinositide-3 kinase; STAT, signal transducer and activator of transcription; Tpo, thrombopoietin; -i, inhibitor in hematopoiesis, a mutation leads to constitutive activation of Janus Kinase 2 (JAK2), a member of the Janus family of kinases, phosphorylated/activated by various cytokine receptors to Drive Signal Transducer and Activator of Transcription (STAT) pathways. The thrombopoietin receptor mutation MPL W515L, discovered after the discovery of JAK2 V617F, is another operator of mutation which leads to activation of the JAK-STAT pathway and present in a minority JAK2 V617F-negative cases of MF and ET.