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Beyond JAK Inhibition: Molecular Pathogenesis and Novel Therapeutic Strategies for the Treatment of Myeloproliferative Neoplasms (MPNs)

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Message from the Guest Editor

Because of the shared presence of aberrant JAK/STAT signaling, MPN driver mutations have long been thought of as overlapping in their mechanisms of hematopoietic transformation. However, in recent years, evidence of their distinct molecular and cellular effects has begun to emerge. Understanding the mechanisms underlying the differential molecular pathogenesis of each MPN driver, and exploiting this knowledge to identify unique, targetable dependencies for each mutation, will help to move the needle towards the development of novel, highly specific and potentially curative treatment strategies for MPNs.

This Special Issue will highlight work that sheds light on the molecular mechanisms underlying the pathogenesis of mutant *JAK2*, *CALR*, and *MPL*-driven MPNs, and the targetable dependencies unique to each that may represent new therapeutic avenues. This work will provide invaluable insight into the future of MPN treatment, in which the elucidation of basic molecular mechanisms will give way to rationally designed therapies and precision medicine to cure patients without HSCT.



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Special Issue



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Message from the Editor-in-Chief

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