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## Aberrantly Regulated Molecular Signaling Pathways and Targets in MPNs

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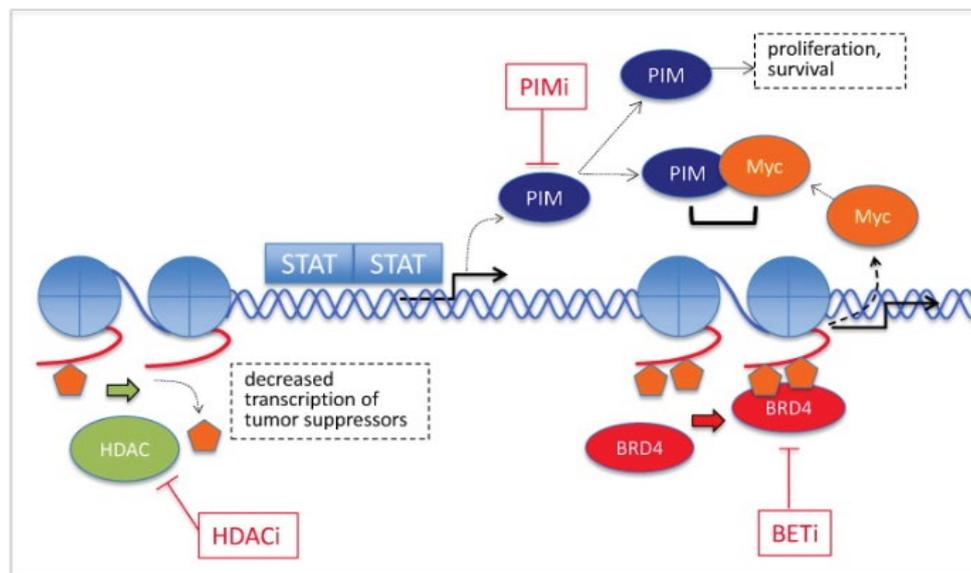
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**Figure 1** Examples of Aberrantly regulated molecular signaling pathways and targets in MPNs. From the abundance of studies, reveal that by targeting epigenetic processes in epigenetic deregulation is involved in MPN pathogenesis, is of significant therapeutic interest. Four HDAC inhibitors (HDACis) are FDA-approved for use in multiple myeloma or T-cell lymphomas, along with the DNA methyltransferase inhibitors can be used in epigenetic therapy for the treatment of different malignancies. To perform normal cellular functions and various transcription factors to maintain guided by Histone modification patterns, regulated by HDACs and histone acetyltransferases. Dysregulation of the epigenetic process results in suppression of transcription of tumour suppressor and cell differentiation genes, which leads to MPN pathogenesis. Chromatin condensation and transcriptional silencing of tumour suppressor genes are caused by HDAC-mediated deacetylation of the lysine residues of histone tails. Induce in JAK-STAT signaling leads to PIM kinase expression, involved in a number of pro-survival functions such as stabilization of Myc and phosphorylation. The BET family of BRD proteins includes BRD4, which has a region for acetylated lysine residues of the histone tail. BET: Bromodomain and Extra Terminal family of Bromodomain-containing proteins; BRD4: Bromodomain-containing protein 4; HDAC: Histone Deacetylase; PIM: Proviral Integration of Moloney Virus; STAT: Signal Transducer and Activator of Transcription-i inhibitor.