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JAK2-Mutant Hematopoietic Cells Display Metabolic Alterations

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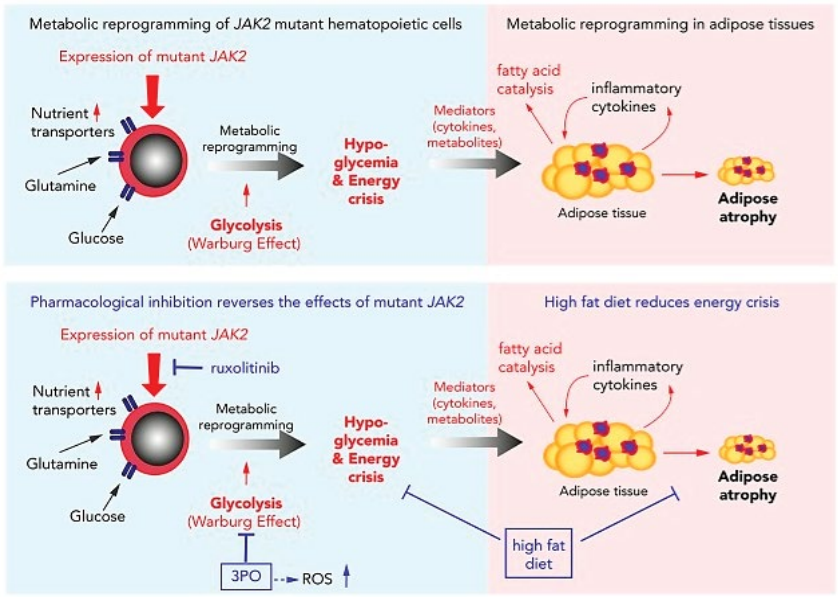

Figure 1 Myeloproliferative Neoplasms (MPNs) are clonal disorders of hematopoietic stem cells driven by gain-of-function mutations in *JAK2, MPL,* or *CALR* genes. There are 3 phenotypic manifestations of MPNs can be distinguished such as Essential Thrombocythemia (ET), Polycythemia Vera (PV), and primary myelofibrosis. For the pathogenesis of mutant JAK2-driven Myeloproliferative Neoplasms (MPNs), metabolic alterations in hematopoietic cells are fundamental. The expression of mutant JAK2 increased and destabilized metabolic activity of Myeloproliferative Neoplasms (MPN) cells, resulting in systemic metabolic changes *in vivo*, including adipose tissue atrophy, hypoglycaemia and early mortality rate. Hypoglycemia or energy crisis in Myeloproliferative Neoplasms (MPN) correlated with hyperactive erythropoiesis due to a combination of increased oxidative phosphorylation and elevated glycolysis. The improvement in survival depends upon the modulating supply of nutrients through a high-fat diet, whereas MPN phenotype is augmented through a high glucose diet. Redox homeostasis altered by inhibition of glycolysis by 3PO, which leads to the accumulation of reactive oxygen molecule and increases the rate of apoptosis. It has been observed, that the allowance of metabolic alteration to the MPNs pathogenesis reveal that mutant cells metabolic dependencies constitute vulnerabilities that can be considered for the treatment of MPNs.

