Altered gene expression in myeloproliferative disorders correlates with activation of signaling by the V617F mutation of Jak2

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We identified 13 new gene expression markers that were elevated and one marker, ANKRD15, that was down-regulated in patients with polycythemia vera (PV). These 14 markers, as well as the previously described PRV1 and NF-E2, exhibited the same gene expression alterations also in patients with exogenously activated granulocytes due to sepsis or granulocyte colony-stimulating factor (G-CSF) treatment. The recently described V617F mutation in the Janus kinase 2 (JAK2) gene allows defining subclasses of patients with myeloproliferative disorders based on the JAK2 genotype. Patients with PV who were homozygous or heterozygous for JAK2-V617F exhibited higher levels of expression of the 13 new markers, PRV1, and NF-E2 than patients without JAK2-V617F, whereas ANKRD15 was down-regulated in these patients. Our results suggest that the alterations in expression of the markers studied are due to the activation of the Jak/signal transducer and activator of transcription (STAT) pathway through exogenous stimuli (sepsis or G-CSF treatment), or endogenously through the JAK2-V617F mutation.