Background: The discovery of the JAK2V617F mutation has triggered the development of agents that target the activity of this mutant enzyme. It has been recently shown that both wild-type JAK2 and JAK2V617F kinases translocate to the nucleus of various human leukemia cell lines, which may be relevant for the function of JAK-STAT signaling. Recent studies have implicated JAK2 in hematopoietic progenitors and phospho-histone H3, resulting in loss of function of the histone deacetylase (HDAC) gene, which is associated with cell cycle arrest. A new approach to targeting JAK2 involves the use of pracinostat (SB939), a histone deacetylase inhibitor, which has been shown to decrease JAK2V617F mutant allele burden and increase hemoglobin levels, or symptoms. The aim of this study was to assess...