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# “C1ORF150”: A Novel Regulator of JAK2 Kinase, and Candidate Tumor Suppressor in Human Blood Cell Progenitors

Tyler M. Wade

## Abstract

Erythropoietin (EPO) is an essential growth factor for red blood cell (RBC) production. In response to anemia, hypoxia-sensing cells in the kidney express and release EPO. EPO then acts in bone marrow to drive RBC formation from erythroid progenitors. Upon binding to its cell surface receptor, EPO triggers a JAK2 kinase signaling cascade for progenitor cell growth. Our lab has discovered a novel regulator of JAK2, “C1ORF150” (“150”). “150” is conserved in H sapiens and primates and is a new orthologue of the B-cell receptor adaptor protein and tumor marker, HGAL. Using a shRNA knockdown approach, I investigated the actions of “150” in both a UT7epo-E cell line model, and in primary human hematopoietic progenitor cells. I first used a clonal colony forming assay approach and discovered that the knockdown of “150” increased the proliferation of erythroid colonies without affecting the development of non-erythroid myeloid cells. Using cell fractionation and Western blot approaches, I further observed that the knockdown of 150 markedly escalates the activation of not only ERK1/2, AKT and STAT5 but also JAK2. Activated, phosphorylated JAK2 (p-JAK2) also aberrantly relocalizes to the cytoplasm. “150” therefore plays roles in compartmentalizing and anchoring JAK2 signaling at the plasma membrane. In conclusion, I have discovered that “150” functions as a new governor of JAK2 activation, and importantly prevents the over-expansion of hematopoietic progenitor cells.