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Article

FoxOs Are Critical Mediators of Hematopoietic Stem Cell Resistance toÂ Physiologic Oxidative Stress

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Summary

To understand the role of FoxO family members in hematopoiesis, we conditionally deleted *FoxO1*, *FoxO3*, and *FoxO4* in the adult hematopoietic system. *FoxO*-deficient mice exhibited myeloid lineage expansion, lymphoid developmental abnormalities, and a marked decrease of the lineage-negative Sca-1$^+$, c-Kit$^+$ (LSK) compartment that contains the short- and long-term hematopoietic stem cell (HSC) populations. *FoxO*-deficient bone marrow had defective long-term repopulating activity that correlated with increased cell cycling and apoptosis of HSC. Notably, there was a marked context-dependent increase in reactive oxygen species (ROS) in *FoxO*-deficient HSC compared with wild-type HSC that correlated with changes in expression of genes that regulate

ROS. Furthermore, in vivo treatment with theÂ antioxidative agent N-acetyl-L-cysteine resulted in reversion of the *FoxO*-deficient HSC phenotype. Thus, FoxO proteins play essential roles in the response to physiologic oxidative stress and thereby mediate quiescence and enhanced survival in the HSC compartment, a function that is required for its long-term regenerative potential.

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