Leukemia. 2002 Apr;16(4):708-15.

Nitric oxide induces the apoptosis of human BCR-ABL-positive myeloid leukemia cells: evidence for the chelation of intracellular iron.

Ferry-Dumazet H, Mamani-Matsuda M, Dupouy M, Belloc F, Thiolat D, Marit G, Arock M, Reiffers J, Mossalayi MD.

Bone Marrow Transplantation, CNRS UMR5540 and EA482, Bordeaux-2 University, Bordeaux, France.

Abstract

Anti-leukemia activity of human macrophages involves the generation of nitric oxide (NO) derivatives. However, leukemic transformation may involve mechanisms that rescue cells from NO-mediated apoptosis. In the present work, we analyzed the effects of exogenous NO on the proliferation of BCR-ABL(+) chronic myelogenous leukemia (CML) cells. As normal leukocytes, the proliferation of leukemia cells was inhibited by SNAP (S-nitroso-N-acetyl-penicillamine), GEA (Oxatriazolium amino-chloride), and SIN-1 (Morpholino-sydnonimine), whereas SNP (sodium nitroprusside) had no effect on leukemia cell growth. SIN-1 induced higher anti-proliferation activity in BCR-ABL(+) cells, compared to normal hemopoietic cells. Inhibition of leukemia cell proliferation correlated with increased apoptosis and DEVDase activity. The simultaneous addition of exogenous iron reversed NO-mediated inhibition of cell growth, caspase activation and apoptosis in all BCR-ABL(+) cells tested. The quantification of intracellular iron levels in leukemia cells indicated that NO induced an early, dose-dependent decrease in ferric iron levels. Accordingly, elevation of intracellular iron protected leukemia cells from NO-mediated apoptosis. Together, the present work reveals the presence of an iron-dependant mechanism for leukemia cell rescue from NO-induced growth inhibition and apoptosis.

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