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41

**Antioxidant regulation of oxidative stress in normal and thalassemic erythrocytes**

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Erythrocytes are known to produce oxygen radicals when induced *in vitro* by prooxidants like quinones, phenylhydrazine, hydroperoxides and transition metal ions. Red blood cells also release active oxygen species during *in vivo* oxidative stress exemplified by hematological disorders such as thalassemia. In this experiment, the effect of two types of free radical inhibitors, Bio-Normalizer and 1-allyl-2-methyl-3-hydroxy-4-pyridinone (AMHP, an oral chelator) on superoxide release by normal and thalassemic erythrocytes, as well as on hemoglobin oxidation were analyzed. Oxidative stress was induced by the addition of primaquine (PQ), menadione, 1,4-naphthoquinone-2-methyl-3-sulfonate (HQMS), doxorubicin and benzoquinone (BQ). Bio-Normalizer and AMHP suppressed the damaging effect of the prooxidants used. With cytochrome c reduction test, it was found that both Bio-Normalizer and AMHP inhibited superoxide release. BN also decreased hemoglobin oxidation to methemoglobin. The findings imply the usefulness of Bio-Normalizer and AMHP in oxidative stress-related pathologies.