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Red Blood Cell (RBC) oxidative stress contributes to reduced RBC deformability and oxygen delivery leading to the occurrence of anemia

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Abstract

Red blood cells (RBCs), while passing through the circulatory system, are continuously exposed to exogenous reactive oxygen species (ROS) as well as ROS generated from the large pool of oxygen carried by them. However, RBCs have an extensive antioxidant system including glutathione, Vitamin E, ascorbate, Ergothioneine, superoxide dismutase (SOD1), catalase, glutathione peroxidase and peroxiredoxin (PRDX2), which are supposed to eliminate ROS. Nevertheless, we have been able to detect an un-neutralized pool of ROS that is generated due to hemoglobin auto-oxidation leading to the production of heme-degradation products (marker of RBC oxidative stress). These are thought to be formed under hypoxic conditions in the microcirculation. A contribution of this RBC oxidative stress to many pathological conditions and aging are indicated by increases in the in-vivo levels of heme degradation products. Our recent mouse model studies showed that deficiencies in PRDX2 and SOD2 genes caused anemia with significant reduction in RBC deformability and increase in heme degradation. It is important to note that even though mature RBCs do not have SOD2, its absence still contributed significantly to the RBC oxidative stress and function. We have also shown that under hypoxic conditions, the generated RBC oxidative stress allows partially oxygenated hemoglobin to bind to membranes (thus escaping RBC antioxidant system) that perhaps contributes to heme degradation and the damage of the RBC membrane affecting its deformability. This reduced deformability due to RBC oxidative stress eventually contributes to the loss of RBCs (anemia) and decrease in oxygen delivery to tissues leading to pathological conditions.

Biography

Joy Mohanty has completed his Ph.D in Chemistry in 1977. Following his Ph.D., he has been involved in biomedical research at various institutions like, NIH, Northeast Ohio Universities College of Medicine, University of Montreal, McGill University and Hahnemann University Medical School (presently Drexel University College of Medicine) prior to joining NIH as a researcher in 2003. Since then, he has been involved in studies on hemoglobin oxidation mechanisms, red blood cell (RBC) deformability changes, RBC properties in mouse model involving oxidative stress, RBC properties in Alzheimer's disease patients and RBC properties in human subjects having anemia. He has published several papers in the field with more than 40 papers including other research areas in reputed journals and has reviewed several research manuscripts of different journals.