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Erythrocyte nitric oxide in glaucoma patients

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Erythrocyte or red blood cells (RBCs) are influents in the blood flow velocity and hemorheology. RBCs also participate in hemostasis systems and in body tissues oxygenation through the vessel endothelium. Endothelial cells and lymphocytes are able to synthesize acetylcholine (ACh) which is to release plasma. Depending on the endothelium integrity degree, the circulating ACh induce vasodilation or vasoconstriction according to the amount of nitric oxide (NO) synthesized and is released into smooth muscle cells (SMC) or to plasma. The NO released is scavenged by erythrocytes and blood cell free hemoglobin. NO enter into RBCs through its membrane protein band 3 and binds to oxyhemoglobin generating S-nitrosohemoglobin (SNO-Hb) and to glutathione originating nitrosoglutathione (GSNO). The NO efflux from erythrocyte is under dependence of mechanical or chemical stimuli bound to membrane receptors. Those affect the NO reservoir molecules inside erythrocyte in dependence of protein phosphorylation degree and redox thiol status. Timolol maleate is a compound used in treatment of patients with open angle glaucoma (OAG).Timolol is a weak inhibitor or erythrocyte membrane acetylcholinesterase (AChE) which behaves as an enzyme and a receptor of ACh. The erythrocytes obtained from blood samples of OAG patient's present higher AChE enzyme activity and high NO efflux than those obtained from healthy persons. When blood samples taken from OAG patients were incubated in the presence of Ach, no changes in NO efflux in GSNO were verified. At variance, in presence of timolol, instead of ACh, both NO efflux levels and GSNO concentration increase. These data evidences show that the erythrocyte membrane of OAG patients have different molecular properties than healthy subjects which corroborate the increasing tendency of RBCs to aggregate as observed in previous studies. The NO efflux signal transduction pathway associated to AChE-ACh and AChE-timolol will be described at the conference.

Recent Publications

1. C Saldanha (2017) Human erythrocyte acetylcholinesterase in health and disease. Molecules 22(9): E1499.
2. Saldanha C and Silva-Herdade A S (2017) Physiological properties of erythrocytes in inflammation. J Cel Biotec. 3:15–20.
3. Esteves R, Freitas T, Teixeira P, Napoleão P, Neves C, et al. (2016) Erythrocyte nitric oxide in glaucoma patients–*ex vivo* study. Clinical Hemorheology and Microcirculation 64:989–994.
4. Ana S Silva-Herdade, T Freitas, J P Almeida and C Saldanha (2016) Fibrinogen signaling in erythrocyte nitric oxide mobilization in presence of PI3-K and adenylyl cyclase inhibitors. Eur J of Biom and Pharm Science 3:28–34.
5. Ana S Silva-Herdade, G Andolina, C Faggio, A Calado and C Saldanha (2016) Erythrocyte deformability—A partner of the inflammatory response. Microvasc Res. 107:34–38.

Biography

Carlota Saldanha is the Professor of Biochemistry and Head of Unit of Institute of Molecular Medicine João Lobo Antunes, Faculty of Medicine, University of Lisbon. She is a Member of the European Society for *Clinical Hemorheology and Microcirculation* (ESCHM); Coordinating Committee and Editorial Board of the Clinical Hemorheology and Microcirculation; Strategic Group of the European Society for Microcirculation; President of Sociedade Portuguesa de Hemorreologia e Microcirculação and Collaborator of CEMAT–Center for Computational and Stochastic Mathematics of FCT. She has done basic and clinical research on Biochemistry: membrane properties, cell function, enzymology, metabolism, signal transduction. Her research interests include applied hemorheology and microcirculation, inflammation, erythrocyte signal transduction mechanism, fibrinogen binding, nitric oxide metabolism and signal transduction.

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