

Homocysteine Decreases Extracellular ATP and ADP Hydrolysis in Rat Platelets *In vitro* and *Ex vivo*

Zanin, R.F.¹, Braganhol, E.¹, Campesato, L.F.¹, Schetinger, M.R.C.², Wyse, A.T.S.¹
and Battastini, A.M.O.¹

¹Departamento de Bioquímica, Instituto de Ciências Básicas da Saúde, Universidade Federal do Rio Grande do Sul, Porto Alegre, RS, Brazil

²Departamento de Química, Centro de Ciências Naturais e Exatas, Universidade Federal de Santa Maria, RS, Brazil

Introduction: Elevated homocysteine (Hcy) concentration is associated with increased risk of coronary artery disease, stroke, and venous thromboembolism. The possible mechanisms by which Hcy must be contributing to atherogenesis and thrombosis include increased oxidative stress, stimulation of low-density lipoprotein oxidation, induction of endothelial dysfunction and platelet activation. Platelets develop an important role on the regulating thrombus formation, mainly by the release of active substances such as ADP. ENTPDases enzymes are the main responsible by ATP and ADP hydrolysis and are essential on the regulation of blood flow and thrombogenesis by ADP catabolism. **Objective:** Considering the importance of adenine nucleotides to the physiological homeostasis, we investigated the Hcy influence in the ATP and ADP hydrolysis. **Results:** The results demonstrated that ATP and ADP hydrolysis of rat platelets exposed to Hcy *in vitro* and *ex vivo* were decreased in approximately 40%, when compared with the control group. The hydrolysis activity returned to control levels when the treatments *in vitro* and *ex vivo* received antioxidants agents. **Conclusion:** Our data suggest that inhibition of ecto-ATPDase activity must be an indirect action of Hcy and the elevated ADP around of the platelets by inactivating of ectonucleotidase may be contributing to increase thrombotic risk described in hyperhomocysteinaemia.

Key Words: ADP, Homocysteine, NTPDase and Platelet

Supported by: CNPq