ALBUMIN AND ITS POTENTIAL APPLICATION IN FUTURE THERAPEUTIC PROTOCOLS IN THE TREATMENT OF LEPTOSPIROSIS

Introduction and objectives: Leptospirosis is a bacterial zoonosis and affects wild, domestic animals and humans assuming worldwide distribution. Our group hypothesized that changes in clinical and histopathological aspects of leptospirosis infection are due an endotoxin released during bacterial lysis, as a consequence of host immune response and Na⁺, K⁺-ATPase (NKA) inhibition. Accordingly, the glycolipoprotein fraction of *Leptospira* (GLP) was found in lesion sites at affect organs. GLP has a specific biological target: the inhibition of NKA, this inhibition is associated with its lipid portion, particularly the non-esterified fatty acids, including oleic acid. The GLP and oleic acid-induced lung injury and inflammation through NKA inhibition, suggesting their role in kidney and liver failure and acute respiratory distress syndrome observed in severe leptospirosis. The NKA function as ion transporter, cell signaling transduction and it is important to lung edema clearance. Thus, this work aims to study the direct effect of GLP on the NKA and the influence of human albumin on GLP in type II human pneumocytes. Materials and methods: NKA activity was measured by non-radioactive Rb⁺ incorporation by cells (Rb⁺ is a K⁺ substitute). The phosphorylation of p38 protein was assayed by western blot and the cytokine production was measured by ELISA. Results and conclusions: GLP inhibited NKA and human albumin prevented its inhibition. GLP induced P38 activation and stimulated the production of cytokine IL-6 and IL-8, also prevented by human albumin. Human albumin has two beneficial effects prevents NKA inhibition and avoids intracellular pathway activation with cytokine production. Therefore, we propose that albumin administration may be used as a potential adjuvant therapeutic strategy for patients with leptospirosis. Acknowledgements: The financial support of CNPq, FAPERJ and PROPPPI-UFF. Key Words: Leptospirosis, GLP, Na⁺, K⁺-ATPase.