Congress IUBMB-FEBS Sevilla 2012

P13-26

Isoenzymatic expression of Na,K-ATPase in islets of Langerhans from rats with normal and impaired glucose tolerance

Ana R. Costa[1], Fernando Capela e Silva[1], Célia M. Antunes[2], Júlio Cruz-Morais[1]

[1] Institute of Mediterranean Agricultural and Environmental Sciences (ICAAM), ÉVORA, PT, [2] Center for Neuroscience and Cell Biology (CNC), Coimbra, PT

Na,K-ATPase is ubiquitously expressed in all eukaryotic cells where it acts as a regulator of Na+ and K+ homeostasis. The Na,K-ATPase pump consists of (αβ)2. The α-subunit contains the catalytic and ligand binding sites. The β-subunit is a glycosylated protein, and its role in Na,K-ATPase enzyme function remains somewhat obscure. Four α- and three β-isoforms have been described in mammals. The isoenzymatic expression pattern is tissue- or cell line-specific and is a factor accounting for its regulation. Isoenzymatic distribution in islets of Langerhans is unknown however previous work has shown altered Na,K-ATPase activity in pancreatic β-cells from glucose intolerant subjects and alterations in isoenzymatic expression in other tissues has been described in type 2 diabetes. The aim of this work was to determine isoenzymatic expression of Na,K-ATPase in the islets of Langerhans from rats with normal and impaired glucose tolerance. Pancreata from control (Wistar) and glucose intolerant rats (GIR) were excised and fixed and prepared for immunohistochemistry analysis of α1, α2, α3 and β2 isoforms. Isoforms α1, α2, α3 and β2 were found in islets of Langerhans suggesting that the isoenzymes α1β, α2β and α3β of Na,K-ATPase are expressed. The isoenzymes α1β and α3β were found throughout the whole islet whereas $\alpha_2\beta$ isoenzymes were predominantly expressed at the periphery. Compared to controls, an increased expression of isoenzymes α2β and a decreased expression of isoenzymes α1β of Na,K-ATPase was found in GIR islets of Langerhans. In conclusion, since isoenzymatic expression may contribute to the differential sensitivity of Na,K-ATPase to substrates, these alterations may contribute to abnormal regulation of Na,K-ATPase activity in pancreatic β-cells from GIR.