**Deregulated expression of microRNAs miR-29 and miR-210 in peripheral T cells of patients with Hashimoto's thyroiditis**

Stana Tokić1,3,4, Mario Štefanić2,3, Ljubica Glavaš-Obrovac3, Amit Kishore4, Zdenka Navratilova4, Martin Petrek4

1Department of Molecular Diagnostics and Tissue Typing, Institute of Clinical Laboratory Diagnostics, Osijek University Hospital, Josipa Huttlera 4, HR-31000 Osijek, Croatia

2Clinical Institute of Nuclear Medicine and Radiation Protection, Osijek University Hospital, Josipa Huttlera 4, HR-31000 Osijek, Croatia 3Faculty of Medicine, University of Osijek, Cara Hadrijana 10E, HR-31000 Osijek, Croatia 4Laboratory of Immunogenomics, Institute of Molecular and Translational Medicine, Palacky University, 775 15 Olomouc, Czech Republic

Hashimoto’s thyroiditis (HT) is a common autoimmune thyroid disorder frequently evolving from asymptomatic, T-cell mediated chronic inflammation towards overt hypothyroidism. MicroRNAs, non-coding RNAs serving as transcription regulators, have been implicated in the generation of HT autoreactive T cells. Because their precise role across the spectrum of HT clinical presentations is unknown, we characterized the expression of three candidate immunoregulatory miRNA (miR-9, miR-29 and miR210) in peripheral CD4+T cells of 10 hypothyroid, untreated patients (hypoHT), 10 hypothyroid cases rendered euthyroid by L-thyroxine therapy (substHT), 11 spontaneously euthyroid HT subjects (euHT) and 10 healthy controls (ctrl) by qRT-PCR. Data were normalized to RNU48 and fold difference in expression was calculated by DDCt method. Down-regulated expression of miR-29 [median expresssion levels (IQR), HT vs ctrl, 0.48 (0.31-0.85) vs 0.59 (0.47-2.27), P=0.038] and miR-210 [0.33 (0.25-.07) vs 0.69 (0.42-3.11), P=0.025] was observed in peripheral CD4+T cells of HT patients compared to controls. Subgroup analysis demonstrated reduced levels of miR-29 in euthyroid [0.35 (0.23-0.5), P=0.019], but not in patients with burned out phase of HT, both hypothyroid [0.33 (0.24-0.88), P>0.05] or rendered euthyroid by L-thyroxine replacement therapy [0.64 (0.41-1.16), P>0.05]. No change in miR-9 transcript levels was observed across the studied groups [hypoHT, substHT, euHT, ctrl, P>0.05 for all]. In conclusion, T cell miR-29 and miR-210 are down-regulated in HT patients and should be explored further as biomarkers or plausible targets for therapeutic interventions in HT. The differential expression of miR-29 at different clinical endpoints of HT suggests that different disease entities may be associated with specific miRNA profiles.