Summary

In our works we aimed to study the role of rh Hsp60, *M. bovis* Hsp65 and stress-inducible rh Hsp70 in pathogenesis of juvenile idiopathic arthritis (JIA) and rheumatoid arthritis (RA). In the study of humoral response against rh Hsp60, *M. bovis* Hsp65 and rh Hsp70 were found significant elevated levels of antibodies against -inducible Hsp70 in a total cohort of patients with JIA when compared with healthy individuals. The prevalence of anti-Hsp70 antibodies is much higher in JIA patients when compared with healthy controls suggesting their possible role in pathological mechanism of the disease.

In the study of membrane expression of inducible Hsp70 we detected high membrane expression of Hsp70 on fibroblast-like synovial cells derived from synovia affected by autoimmune inflammation. We screened the presence of Hsp receptors like TLR2 and TLR4, CD14, CD36, CD40 and CD91 as well as their association with inducible Hsp70 on RA derived fibroblast-like synovial cells. Both synovial cells and skin fibroblasts expressed high levels of cell surface CD91, however, no or low levels of other receptors We speculated that inducible Hsp70 released from inflamed synovial tissue might be captured onto the cell surface of synovial cells from the extracellular space via CD91 receptor.

In the study of humoral responses against the heat shock proteins in relation to transplant-related complications in sera of paediatric patients undergoing allogeneic haematopoietic stem cell transplantation (HSCT) we found, that antibodies against Hsp60, Hsp65 and inducible Hsp70 including total immunoglobulins (Ig), IgG and IgM were detected before conditioning, over the course of conditioning and all the time posttransplant. We found no correlation between anti-Hsp-60, -65 and -70 antibodies and the occurrence and severity of GvHD and/or other transplant-related complications. However, elevated anti-Hsp antibodies involving IgM and IgG isotypes were found to be associated with bacterial and fungal infection depending on aetiological agents. We demonstrated *de novo* humoral response to Hsps in a cohort of patients with actual infection caused by *Klebsiella pneumoniae* (anti-Hsp60, anti-Hsp65 and anti-Hsp70), *Pseudomonas aeruginosa* (anti-Hsp60, anti-Hsp70) and Aspergillus fumigatus (anti-Hsp65).

We conclude that anti-Hsp antibodies might be produced after SCT in relation to infection depending on aetiological agents; however transplant-related complications by themselves had a little impact.