Acute lymphoblastic leukaemia is the most common malignancy in childhood. Various acquired and congenital factors are involved in leukemogenesis including aberrant cell signaling. Transmembrane adaptor molecules could play an important role in development and propagation of leukemia. In a first part of our study, we analyzed an expression of adaptor molecules PAG, LAT and NTAL in physiological lymphocyte precursors and in diagnostic samples of different subtypes of childhood acute lymphoblastic leukemia (ALL). In
physiological lymphocyte development the expression of adaptor molecules has significant dynamics (increase of LAT and decrease of NTAL in T-lymphocyte development; decrease of PAG in B-lymphocyte development). Similarly, in subtypes of childhood ALL the expression of adaptor molecules is very different. Especially, TAL/AML1 positive acute lymphoblastic leukemia has a unique expression profile of adaptor molecules (high expression of PAG and LAT, low expression of molecule NTAL). In T-cell acute lymphoblastic leukemia the expression of NTAL molecule identifies two groups of patients – those, who respond favourably to initial prednisone treatment, have higher level of NTAL comparing to patients, who respond to prednisone unfavourably. Those patients have low level of NTAL molecule expression. In a second part of our study, we examined the role of NTAL molecule to glucocorticoid induced cell death in in-vitro experiments. We used T-cell acute lymphoblastic leukemia cell line Jurkat (Jurkat/wt) and we derived Jurkat cell line with stable NTAL expression (Jurkat/NTAL+). Cell signalling and cell death after methylprednisolone treatment and after T-cell receptor stimulation were analysed using flow cytometry, Western blot and quantitative polymerase chain reaction. Jurkat/NTAL+cell line was more sensitive to glucocorticoid treatment than Jurkat/wt cell line.