Leukemia accounts for about 1/3 of all cancers in children. Treatment of acute lymphoblastic leukemia (ALL) has made great advance in recent years, whereby 98% of all treated children are currently in complete remission and more than 80% of them are fully treated. Unfortunately remaining 15-20% of children still relapse. The specific group of ALL consists of patients with the TEL/AML1 fusion gene resulting from translocation t (12; 21) (p13; q22) and is present in approximately 25% of patients with B-precursor ALL. This translocation is considered to be a favorable prognostic feature, a rapid response to the initial treatment and increased sensitivity to the cytotoxic agent commonly used in therapy, L-Asparaginase (L-Asp). In the dissertation thesis we focused on the study of current therapy and potential targeted approach. In the first part we tried to clarify the mechanism of L-Asp effect in this genotypically defined ALL subgroup. The main effect of L-Asp lies in the depletion of asparagine and glutamine in the serum of patients. The anti-leukemic effect is associated with a lower level of asparagine synthetase enzyme (ASNS) specifically in leukemic cells. Previous work has shown the relationship between lower sensitivity of L-Asp and higher ASNS in leukemic cells. However, our results of monitoring the AsnS expression dynamics in different cell lines have not confirmed this relationship. Furthermore, we demonstrated the prolonged effect of L-Asp on TEL/AML1 positive leukemia cells with G1 / G0 phase arrest and increased expression of the ASNS gene as opposed to TEL/AML1 negative leukemia in which cells were able to pass S phase and ASNS gene expression returned to basal level after administration of L-Asp. These observations show that the nutritional stress caused by the depletion of asparagine and glutamine cannot be circumvented by TEL/AML1 positive cells whereas TEL/AML1 negative cells are capable of it. In the next part of the project, ALL patients showed a significantly lower level of glutamate dehydrogenase (GDH) expression in TEL/AML1 positive vs. TEL/AML1 negative patients when analyzing the expression data. We assume that insufficient GDH function (due to low expression) leads to insufficient glutamine production in the cell, which leads to a weakening of cellular metabolism to respond to the limitation of asparagine and glutamine external delivery. These results revealed the effect of the regulatory mechanisms of cellular metabolism of asparagine and glutamine in leukemic cells. In the second part of the project we focused on new approaches in the treatment of ALL. The role of the TEL/AML1 fusion gene stems from its binding to a corepressor complex that contains histone deacetylase 3. Based on this binding, the transcription of the original AML1 transcription factor target genes is silenced. In this work we studied the effect of histone deacetylase inhibitors - valproate and trichostatin A on cell survival, differentiation and transcriptional regulation of selected genes. We have demonstrated that the release of the repressive effect caused a shift in differentiation and an increase in the number of apoptotic cells. We also described new target genes of the TEL/AML1 fusion protein, which may play a role in leukemogenesis. These results demonstrate the importance of using target therapy in the treatment of ALL patients non-responsive to current therapy.