Introduction of tyrosine kinase inhibitors (TKI) like imatinib has radically improved treatment results and prognosis of chronic myeloid leukemia (CML). However, long-term imatinib treatment lead to the constant exposure for adverse effects and increase the cost of therapy. Recent clinical trials demonstrated almost 40% of CML patients with 2-year remain molecular response after imatinib discontinuation do not need further treatment. Furthermore, nearly all relapses occur in the first 6 months after imatinib withdrawal, suggests that restoration of immune surveillance cells might contribute to maintaining long-term remission status.

CML is characterized by completely deregulation of mechanisms responsible for the immune response directed against leukemic cells. The aim of our project is to characterize alterations in the subpopulation of the immune system cells that occur in CML patients during imatinib discontinuation. We plan to evaluate immune biomarkers defining the group of patients who may safely discontinue imatinib treatment as well as characterize immune responses to leukemia-associated antigens in patients who sustain molecular remission after stopping the therapy. In addition, we will measure CMV reactivation status, which was found to have a favorable effect on curative effect. Finally, it is planned to characterize so-called “TKI withdrawal syndrome” which affects approximately 20-25% possibly as a result of unblocking tyrosine kinases.

Altogether the project realization may help to optimize modes of using molecular biology methods, to get a better understanding of the immune mechanisms involved in the treatment process, which can improve its outcome and contribute to increase the percentage of patients with successful outcome of treatment complete discontinuation trial.