Rheumatoid arthritis is a chronic inflammatory autoimmune disease leading to progressive joint destruction and the development of disability. The key element of modern rheumatoid arthritis therapy are biological drugs, among which the most commonly used are so-called anti-TNF-alpha blockers. They reduce clinical symptoms and prevent disease progression. Unfortunately, some patients due to unknown reasons do not respond to anti-TNF alpha drugs. The causes of rheumatoid arthritis remain unclear, however genetic predisposition, including genetic variabilities in NCF1, NCF2 and NCF4 genes, is presumed to play an important role. These genes encode proteins that form NADPH oxidase enzyme complex, which is responsible for the so-called “respiratory burst" in immune cells. It has been shown that TNF alpha influences NADPH oxidase activity what led us to hypothesize that differential expression of NCF genes may be connected with difference in response to anti-TNF-alpha blockers. The aim of the project is to evaluate the relationship between NCF genes expression levels, disease aggressivity and benefit from anti-TNF alpha drugs. We believe that our project may identify novel immune factors that influence clinical presentation of rheumatoid arthritis and provide a rationale for better selection of patients to treatment with anti-TNF alpha drugs.