Rheumatoid arthritis (RA) is a chronic autoimmune disease characterized by progressive joint inflammation, as well as cartilage and bone damage leading to disability. The disease is characterized by increased inflammation that causes swelling and excessive synovial fluid accumulation.

The functional role of natural killer (NK) cells in inflammation in RA patients has been poorly described, so the aim of this project is to analyse NK cell populations isolated from patients' peripheral blood in the context of their diverse repertoire of cell surface receptors at different times after the implementation of biological treatment with TNF-alpha inhibitors. The kinetics of NK cell receptor changes may be altered after treatment compared to the NK cell profile before therapy.

In addition to the characterization of NK cells of RA patients, we will analyse extracellular vesicles (EVs) isolated from patients' serum and synovial fluid. EVs are structures surrounded by a lipid bilayer membrane that contain a number of nucleic acids and proteins, and are essential for mutual communication between target cells through a specific interaction. EVs initiate these intercellular interactions and generate new intracellular signalling pathways essential for both physiological and inflammatory processes. Therefore, it is important to understand all the molecular interactions occurring between EVs and the NK cells involved in inflammation that may be the target cells for EVs.

The mechanism of action of EVs is related to their ability to be selectively absorbed by target cells, their potential for intercellular communication and diverse interactions, and their potential to induce phenotypic changes involving the profile of specific surface markers on target cells.

The mechanisms involved in the effect of EVs in RA patients are not fully understood. Investigating differences in the surface components of extracellular vesicles isolated from the serum or synovial fluid of RA patients may contribute to understanding how target NK cells change their specificity, and what specific interactions occur between the surface components of EVs and receptors in the cell membrane of target NK cells.

The study of EVs and NK cells will help define the role of EVs in inflammation induction in RA and in the activation of receptors on NK target cells, which may contribute to initiation of a new intracellular signalling pathway.