

The JAK/STAT Pathway as a Molecular Driver of Endometriosis: Therapeutic Potential of JAK Inhibition

Endometriosis is a chronic inflammatory disease in which endometrial cells—the cells that line the inside of the uterus—are found outside their normal location, such as on the ovaries, fallopian tubes, or peritoneal cavity. This condition leads to chronic pain, inflammation, infertility, and a reduced quality of life in women. Despite its prevalence, the mechanisms responsible for the development of endometriosis are still not fully understood, and the available treatment options are often inadequate or associated with significant side effects.

The aim of our project is to investigate the role of the JAK/STAT pathway—a key signaling mechanism for inflammatory processes—in the development and persistence of endometrial lesions, as well as to evaluate the therapeutic potential of blocking this pathway using JAK inhibitors.

JAK inhibitors are modern small-molecule drugs that block the activity of Janus kinases (JAKs)—enzymes essential for transmitting signals from various cytokines and growth factors. These drugs are already used in the treatment of autoimmune diseases such as rheumatoid arthritis, psoriasis, and ulcerative colitis. Their potential use in endometriosis could provide a new, targeted, and less burdensome therapeutic alternative.

In the first stage of the study, we will analyze the concentrations of selected proteins—components of the JAK/STAT pathway and pro-inflammatory cytokines—in endometrial tissue samples collected from women with and without endometriosis. This will allow us to identify the most active and relevant components of the pathway in the disease pathogenesis.

In the second stage, we will conduct experiments on commercially available endometriotic cell lines that model the behavior of endometrial cells. On these models, we will test different JAK inhibitors to examine their effects on cell growth and survival, helping us determine which compounds show the greatest therapeutic potential.

In the final stage of the project, we will evaluate gene expression changes in endometrial cells before and after treatment with the most promising inhibitors. By analyzing genes involved in inflammation, angiogenesis (formation of blood vessels), and cell growth, we aim to better understand the mechanisms through which these drugs work and the molecular pathways they influence.

Our research may provide new insights into the biology of endometriosis. A better understanding of the mechanisms driving inflammation, angiogenesis, and survival of ectopic endometrial cells could help identify key control points in these processes. We believe this will contribute to the development of new, more effective targeted therapies that improve patient quality of life and reduce the side effects of current treatment strategies.