Cancer is currently one of the greatest challenges of modern medicine. Attempts to obtain such drugs that could effectively cure a patient diagnosed with cancer, extend his/her life, or at least improve the quality of his/her life do not stop. It can be noted that research is conducted on a large scale in many different areas of medicine, chemistry, and biology.

One of the methods proposed by scientists to fight cancer is a meticulous analysis of currently used drugs and an attempt to learn the mechanisms of their actions in the cell thoroughly. This approach allows the development of more effective medicines in the future.

The discovery of a new drug and its use for treating patients is only sometimes accompanied by the simultaneous learning of its mechanism. It turns out that many drugs that have long been used in hospitals do not have fully understood mechanisms of action. An example is oxaliplatin - an anticancer drug used to treat colorectal cancer since the 1970s. Its mechanism of action is based on its binding to DNA and blocking its activity. In recent months, however, it has been shown that the toxicity of this drug is not due to its ability to bind DNA, as previously described, but with the ability to inhibit the maturation of ribosomes, organelles responsible for the synthesis of protein in the cell.

The presented project aims to explain mitoxantrone's new mechanism of action, its derivatives, and other anticancer drugs as drugs blocking protein biosynthesis. This is a new mechanism, distinct from previously known ones, and includes blocking topoisomerase II by this drug, an enzyme responsible for unraveling DNA strands. Previously, it was indicated that this drug's blockage of topoisomerase II led to the arrest of cell division and the reduction of gene expression in the cell. However, the preliminary studies conducted by our team have shown that mitoxantrone blocks the mechanism of ribosome action, which was previously unknown. Limiting or stopping the synthesis of new proteins poses a threat to the cells, including cancer cells, because they cannot synthesize protective proteins, reducing the stress caused by other drugs or other adverse factors activated in the patient's body to eradicate cancer cells.

Understanding the mechanism of interaction of mitoxantrone, its derivatives, and other anticancer drugs with the ribosome at the molecular level will allow in the future to design such derivatives of this drug that their cytotoxicity will be significantly increased compared to the initial molecule. Equally important, understanding the mechanism of action of mitoxantrone will reduce its cytotoxicity in healthy cells of the body.