The lungs are organs exposed to numerous pathogens, including toxicological factors causing cancer and viral factors responsible for infectious diseases.

The area of interest of this PhD thesis pertains to the synthesis of organometallic erlotinib (anticancer drug) conjugates and the examination of these bioconjugates as anticancer agents and SARS-CoV-1/2 entry inhibitors to human cells.

Chemically, the goal of my thesis was achieved through the use of Cu(I)-catalyzed or Ru-catalyzed azide-alkyne 1,3-dipolar cycloaddition reactions (CuAAC and RuAAC). These reactions are widely used in medicinal chemistry and molecular biology, which convinced me to choose them as the basic synthetic tools in my research.

Erlotinib, a first-generation EGFR inhibitor, can become ineffective due to therapeutically unfavorable mutations in the EGFR receptor gene. To circumvent this problem, subsequent generations of EGFR inhibitors were developed and introduced into clinical practice. The idea behind the synthesis of organometallic conjugates of erlotinib was to introduce a second mechanism of anticancer activity into the molecule, which would be independent of EGFR mutations. This mechanism relies on the organometallic entity and is based on generating oxidative stress in lung cancer cells. I successfully achieved this goal, as some of my compounds exhibited greater activity against erlotinib-resistant lung cancer cells (specifically, cell lines H1650 and H1975) compared to erlotinib itself.

Additionally, using click chemistry, I obtained a diferrocenyl derivative of AZT with high activity against lung cancer cells. As in the case of active erlotinib derivatives, the mechanism of its activity was based on the generation of reactive oxygen species and oxidative stress.

Another important achievement of my work is the discovery of a ruthenocenyl erlotinib conjugate, which acts as a SARS-CoV-1/2 virus entry inhibitor to human HEK293T cells.