Streszczenie w języku angielskim

Regulated Cell Death (RCD) plays a significant role in the homeostasis of the organism, both in physiological and pathological states. Excessive or insufficient RCD can lead to diseases such as neurodegenerative or cancerous conditions. Cell death is a necessary process in many physiological processes, including embryogenesis, immune system development, and the elimination of damaged cells. Regulated cell death can take on various forms, such as apoptosis, necroptosis, or ferroptosis. Understanding and knowledge of these death pathways, as well as their regulation, is important for both scientific and medical applications.

The issues presented in the doctoral dissertation focus on the study of ferroptosis progression in different *in vitro* cell lines, as well as processes related to the regulation of programmed and non-programmed cell death. So far, the work has examined the impact of various factors that induce or inhibit death pathways in cells. The literature describes cases of inhibiting or inducing cell death by adding specific compounds, called regulators (inducers or inhibitors of signaling pathways). The assumptions allow for testing the relationships between various factors, both chemical and physical, such as UV or ionizing radiation (IR), in combinations of regulator-regulator; regulator-UV; regulator-IR, to determine their influence on a selected death pathway, as well as to observe the effects and side effects on neighboring cells (bystander effect). **The aim of the conducted work was to characterize different cell death pathways and attempt to create a decision-making system for regulating their induction/suppression.** Cells can exhibit different sensitivity to a specific type of death, with some being more sensitive and others more resistant. This dependency is crucial in cancer treatment. Among the tested cell lines, two have shown resistance to death induction, which was confirmed by the expression profile of molecular markers, lipid oxidation levels, database analysis, and literature. Normal keratinocyte cells, HaCaT, are resistant to ferroptosis induction, as well as chronic myeloid leukemia cells, K562. However, melanoma cells from the primary tumor, 1205Lu, showed the highest sensitivity to ferroptosis.