Cisplatin, the basis of head and neck cancer chemotherapy regimens, is not effective in many patients, and cisplatin-based regimens may result in suboptimal disease control. Due to the frequent resistance to current methods of treatment, extensive research has been carried out in order to develop molecularly targeted therapies, that is, directly targeting cells with a specific genetic change. However, the targeted drugs approved so far have only limited efficacy in patients in the advanced stages of the disease. Since both standard chemotherapy and existing targeted therapies have a high treatment failure rate, new supportive therapies are urgently needed to sensitize patients to available drugs and improve their prognosis. One of the possible causes of therapy failure is the presence of slow-dividing, self-renewing, and therapy-resistant cancer stem cells among the cancer cell population. While traditional chemotherapy kills the rapidly dividing cells that constitute most of the tumor mass, cancer cells, displaying some features of stem cells, can remain intact and cause the cancer to relapse after treatment. Eliminating this group of cells to improve response to therapy is not a new idea, and the scientific community has been exploring this option for decades, but it remains a challenge to this day. A promising strategy in these efforts is to target cancer epigenetics. Epigenetics is the study of changes in gene expression that are not related to changes in DNA sequence. The epigenetic machinery of the cell consists of various enzymes whose role is to attach and detach certain chemical moieties from DNA and from the proteins onto which the nuclear DNA is wound, called histones. The presence of these chemical groups affects the availability of DNA for enzymes involved in gene expression. Importantly, some histone demethylases, which catalyze the detachment of methyl groups from histone lysine residues, are crucial for the unveiling of stem cell characteristics in cancer cells. The aim of this project is to verify the hypothesis that modulation of epigenetic information with inhibitors of selected histone demethylases may sensitize cancer cells from head and neck lesions to cisplatin and / or erlotinib. We hypothesize that these inhibitors will enhance the activity of cisplatin and erlotinib by targeting a subpopulation of cancer cells displaying stem cell features. The research will be conducted with the use of a three-dimensional model of cell culture based on the formation of spheroids. Spheroids are clusters of cells that resemble small tumor nodules. Such a model better reflects the conditions in the body and ensures a better translation of the obtained results into clinical practice.