After many years of the “war on cancer”, progress in achieving long-lasting cures and treating disease is still unsatisfactory. This failure likely stems from our limited understanding of the true complexity of the disease. According to some theories, immortality of cancer cells is due to their peculiar life cycle. Very often cancer cells are resistant to any treatment as they can recover after therapy. This situation can be partially explained by the fact that upon drug or radiation treatment cancer cells do not die, but became giant, polyploid and senescent. Polyploidy means multiplification of the genetic material due to the failure of cell division. Mammalian cells are diploid, which means that they possess double set of chromosomes. Normal cells, before mitotic division into two daughter cells, duplicate their chromosomes to ensure their proper number in daughter cells. In many instances cancer cells duplicate their chromosomes even several times after treatment but they do not split into daughter cells and become polyploid. Sometimes polyploid cancer cells are also senescent, which means that they are alive but not able to divide any more. After anticancer therapy some cells, both in vivo and in vitro, become giant polyploid and senescent. Some researchers believe that this phenomenon is beneficial since it stops cancer cell replenishment and, moreover, cell polyploidy/senescence can be evoked by lower doses of drug than is usually needed for killing cancer cells, thus it is better for patients due to limitation of the side effects. On the other hand there are some data showing that these giant polyploid cells can divide, just like yeast, by budding. Moreover, the small progeny is more invasive and aggressive than the giant mother cell. This project aims to prove that indeed inducing senescence of cancer cells can be very dangerous as it may provoke senescence escape and repopulation of cancer cells. In our experiments performed in tissue culture we observed such giant polyploid senescent cancer cells which were able to divide by budding. We would like to identify genes which are possibly involved in polyploidization of cancer cells. We also aim to show that the progeny of these cells has altered genetic background and may contribute to the heterogeneity of cancer cells and increase their survival potential. We also observed that the giant polyploid cells had increased level of reactive oxygen species and that antioxidants could protect cells against polyploidization upon treatment. We would like to explore this issue by culturing cancer cells in low oxygen concentration to mimic conditions they encounter inside the tumor. It is also believed that the only aggressive cancer cells have specific genetic characteristics of stemness. Some researchers claim that polyploid cells, while others state that their small progeny, have the properties of stem cells. In our project we would like to elucidate this issue. Generally, we want to show that therapy-inducing senescence, which recently became very attractive, is not necessarily a good approach towards cancer elimination. Even more, it can be much more harmful than therapy causing cancer cell death. We believe that efficient anticancer therapy should be able to eliminate polyploid cells thus interrupting the cancer cell life cycle.