Despite rapid progress in anticancer treatment, many types of cancers are still resistant to therapy. Thus, it is crucial to understand processes responsible for chemoresistance and to study the mechanisms behind them in order to prevent cancer recurrence. The main outcomes of anticancer therapy are cell death and senescence. Cellular senescence is associated with the permanent cessation of cell division and may be induced by lower doses of chemotherapeutics than cell death. Therefore, initially, cellular senescence was considered as a strategy of anticancer therapy. However, senescent cells do not die, they are metabolically active and secrete many factors, which may stimulate carcinogenesis of neighbouring cells. Moreover, it has been demonstrated that senescent cancer cells may resume proliferation, what, in consequence, resulted in cancer relapse. Thus, therapy-induced senescence was considered as a mechanism of cancer cell resistance for chemotherapy. Despite intensive studies of cancer cell escape from senescence, the mechanism of this process is still awaiting elucidation. Recent reports revealed that polyploidisation, the process of genetic material multiplication without cell division, plays an important role in the escape of cancer cells from senescence. Namely, senescent and, frequently, polyploid cancer cells escape senescence, resume proliferation and start the depolyploidisation process. It is believed that generation of small progeny by giant senescent polyploid cancer cells requires energy and remodelling of the cell interior. One of the cellular processes that is able to meet those demands is autophagy. Autophagy is a well-known cellular recycling system, responsible for removal of damaged organelles and providing metabolites to maintain energy supply and building blocks for cell reorganization. Therefore, it is postulated that autophagy may be involved in cancer cell escape from senescence. Indeed, increasing number of reports indicate stimulation of autophagy during senescence escape. However, the direct involvement of autophagy in senescent cancer cell regrowth was not confirmed. Thus, our project aims to analyse the role of autophagy in escape of cancer cells from senescence. We will determine the ability of senescent cancer cells to regrow after autophagy inhibition. Moreover, to unravel molecular mechanism of senescence escape, we will examine the involvement of fundamental cellular processes, such as cytoskeleton reorganisation, secretion and metabolism, in senescent cancer cells regrowth and determine their relation with autophagy. Understanding the autophagy role in the escape of cancer cells from senescence will allow us to elucidate the mechanism of senescent cancer cells regrowth and, in consequence, will support the development of a strategy for the prevention of cancer relapse.