Cellular senescence is a process,in which cells lose the ability to divide themselves. What is interesting, cancer cells cannot lose their unrestrained ability to divide on their own (they cannot undergo spontaneous senescence) but they can still be forced to enter senescent state by treating them with DNA-damaging agent, for example chemo- and radiotherapy. Cellular senescence is therefore an important outcome of anticancer treatment. Despite the fact that senescent cancer cells are unable to divide themselves and cannot directly increase tumor volume, the presence of these cells is not neutral for the organism. Their impact appears to be largely deleterious due to the fact that they secrete an array of different proteins, which are able to influence the microenvironment. Among theses proteins there are such factors which cause inflammation, rebuild extraxellular matrix or promote vasculature growth. All of them can stimulate tumor growth and metastasis formation. Therefore discovering the molecular mechanisms which determine the appearance of SASP is so important. It can result in discovery of such protein, whose inhibition will downregulate SASP. By this, the efficiency of anticancer treatment could be increased. In the proposed project I would like to investigate the involvement of AP-1 protein complex in the process of cellular senescence, especially in the expression of SASP factors. To this end, I will treat colon cancer cells and breast cancer cells with chemiotherapeutic drugs (doxorubicin and/or bleomycin) to induce senescence. Then, I will focus on AP-1: I will strive to answer the question, whether AP-1 becomes activated in cells undergoing senescence. Moreover, as AP-1 is a dimer, consisting of many different possible proteins, I want to know, which of these proteins are especially active in senescent cells. The main and final goal of the project is to assess how AP-1 activity affects SASP (its composition as well as the level of particular SASP components) and what impact it has on the cells motility, which - if increased - can favour metastasis.