Abstract

Senescence is a state of a cell characterized by permanent cell cycle arrest, growth of the cell, and other phenotypic changes, including proinflammatory secretome. Along with the beneficial aspects of senescence, it has an unfortunate outcome – it is a cause of age-related pathologies and tumor progression. Senecence-associated secretory phenotype (SASP) is responsible for that, as it alters the tissue microenvironment by the secretion of the inflammatory agents – chemokines, cytokines and other factors. Cellular senescence is linked to the activation of the mTOR (mechanistic target of rapamycin) nutrient- and mitogen-sensing pathway. Therefore, mTOR inhibition is a promising therapeutic strategy for the SASP suppression and of various types of cancer and age-related diseases. In this thesis I am going to summarize the current understanding of the mTOR's role in the mediation of SASP.

Key words: Senescent cells, senescence-associated secretory phenotype, mTOR pathway, MTORC1