Abstract:

The risk of developing many pathological conditions and ageing-releated diseases increases persistently throughout a lifetime. A dramatic increase in the number of people suffering from one of these diseases, such as atherosclerosis, Alzheimer´s disease or Parkinson´s disease, is caused by constant elevation of human life´s length due to advancements in modern medicine and changes in life style. Several recent studies have demonstrated that senescent cells accumulate in aged and ill tissues. Senescent cells are metabolically active, but unable of proliferation and unlike the terminally differented cells, they secrete many factors that contribute to the transformation of the tissue microenviroment. The role of senescence as anticancer barier is known for a long time, but its importance in physiological processes and aging is mainly a matter of a recent time. While there is also a lot of studies focusing on cellular senescence in peripheral tissues, their involvement in or contribution to cognitive decline with aging of the central nervous system (CNS) remains relatively unknown. Recent data of many laboratories suggest that senescence-associated secretory phenotype of the non-neuronal senescent cells in brain can cause chronic level of inflammation and thus accompany aging and ageing-related diseases and contribute to their progression. Thus, senescent cells in brain could be a new therapeutic target for aging-related neuropathologies of the CNS. The aim of this thesis is a compilation of a current knowledge about the role of senescence in the CNS with focus on cancer and neurodegenerative diseases of CNS.

Key words: cellular senescence, neuredegenerative diseases, aging, senescent secretome, central nervous system