Senescence

Senescence or biological aging (also spelled biological ageing) is the gradual deterioration of function characteristic of most complex lifeforms, arguably found in all biological kingdoms, that on the level of the organism increases mortality after maturation. The word “senescence” can refer either to cellular senescence or to senescence of the whole organism. It is commonly believed that cellular senescence underlies organismal senescence. The science of biological aging is biogerontology.

Primary and secondary traumatic brain injury (TBI) can cause tissue damage by inducing cell death pathways including apoptosis, necroptosis, and autophagy. However, similar pathways can also lead to senescence. Senescent cells secrete senescence-associated secretory phenotype proteins following persistent DNA damage response signaling, leading to cell disorders. TBI initially activates the cell cycle followed by the subsequent triggering of senescence. This study aims to clarify how the mRNA and protein expression of different markers of cell cycle and senescence are modulated and switched over time after TBI. We performed senescence-associated-β-galactosidase (SA-β-gal) staining, immunohistochemical analysis, and real-time PCR to examine the time-dependent changes in expression levels of proteins and mRNA, related to cell cycle and cellular senescence markers, in the cerebrum during the initial 14 days after TBI using a mouse model of controlled cortical impact (CCI). Within the area adjacent to the cerebral contusion after TBI, the protein and/or mRNA expression levels of cell cycle markers were increased significantly until 4 days after injury and senescence markers were significantly increased at 4, 7, and 14 days after injury. Our findings suggested that TBI initially activated the cell cycle in neurons, astrocytes, and microglia within the area adjacent to the hemicerebrum contusion in TBI, whereas after 4 days, such cells could undergo senescence in a cell-type-dependent manner [1)].

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Tominaga T, Shimada R, Okada Y, Kawamata T, Kibayashi K. Senescence-associated-β-galactosidase staining following traumatic brain injury in the mouse cerebrum. PLoS One. 2019 Mar 11;14(3):e0213673. doi: 10.1371/journal.pone.0213673. eCollection 2019. PubMed PMID: 30856215.