

**P005** Proteolytic activities are altered in the brain of senescence accelerated mice at 10 months old

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Disturbances of the normal enzymatic activities are the first insult in brain aging and age-related diseases, losing of defence and repair mechanisms in aged cells. Alterations in proteolytic activities such as cathepsin D, caspase-3, calpains were observed in older senescence accelerated mice (SAM), being more pronounced in SAMP8 mice, a model of pathological aging, senile dementia and Alzheimer disease in contrast to SAMR1 mice, a model of physiological aging. Moreover, autophagy as form specialized of lysosomal proteolysis, has been reported in neurodegenerative pathologies and normal aging as mechanism of cell death or to removal of toxic components from inside the cell. However, high levels of Beclin-1 autophagic protein together a lack of LC-3 II protein, may be associated with an alteration of macroautophagy in brain of older SAMR1 and SAMP8 mice contributing to malfunctioning of brain for aging. Moreover, aging-related increments of Lamp2, overall in older SAMP8 mice, can be consequence of chaperone-mediated autophagy as response mechanism against oxidative stress.