

Publication**Age-related increase of oxidative stress-induced apoptosis in mice prevention by Ginkgo biloba extract (EGb761)****JournalArticle (Originalarbeit in einer wissenschaftlichen Zeitschrift)****ID** 107218**Author(s)** Schindowski, K; Leutner, S; Kressmann, S; Eckert, A; Müller, W E**Author(s) at UniBasel** [Eckert, Anne](#) ;**Year** 2001**Title** Age-related increase of oxidative stress-induced apoptosis in mice prevention by Ginkgo biloba extract (EGb761)**Journal** Journal of neural transmission**Volume** 108**Number** 8-9**Pages / Article-Number** 969-78

Keywords reactive oxygen species, aging, lymphocyte, programmed cell death, 2-deoxy-D-ribose

Enhanced apoptosis and elevated levels of reactive oxygen species (ROS) play a major role in aging. In addition, several neurodegenerative diseases are associated with increased oxidative stress and apoptosis in neuronal tissue. Antioxidative treatment has neuro-protective effects. The aim of the present study was to evaluate changes of susceptibility to apoptotic cell death by oxidative stress in aging and its inhibition by the antioxidant Ginkgo biloba extract EGb761. We investigated basal and ROS-induced levels of apoptotic lymphocytes derived from the spleen in young (3 months) and old (24 months) mice. ROS were induced by 2-deoxy-D-ribose (dRib) that depletes the intracellular pool of reduced glutathione. Lymphocytes from aged mice accumulate apoptotic cells to a significantly higher extent under basal conditions compared to cells from young mice. Treatment with dRib enhanced this difference, implicating a higher sensitivity to ROS in aging. Apoptosis can be reduced in vitro by treatment with EGb761. In addition, mice were treated daily with 100 mg/kg EGb761 per os over a period of two weeks. ROS-induced apoptosis was significantly reduced in the EGb761 group. Interestingly, this effect seemed to be more pronounced in old mice.

Publisher Springer**ISSN/ISBN** 0300-9564**edoc-URL** <http://edoc.unibas.ch/dok/A5253490>**Full Text on edoc** No;**Digital Object Identifier DOI** 10.1007/s007020170016**PubMed ID** <http://www.ncbi.nlm.nih.gov/pubmed/11716149>**ISI-Number** WOS:000171099600006**Document type (ISI)** Journal Article