

DIFFERENTIAL VULNERABILITY TO AGE-ASSOCIATED NEURODEGENERATION IN HUMANS AND CHIMPANZEES

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With advancing age, humans appear to be uniquely vulnerable to neurodegenerative diseases such as Alzheimer's disease and Parkinson's disease. In Alzheimer's disease, the cardinal lesions - senile plaques and neurofibrillary tangles - consist primarily of the proteins A β and tau, respectively. These proteins are highly homologous in primates, especially in humans and chimpanzees, yet no nonhuman primate has ever shown the full behavioral and neuropathological spectrum that defines Alzheimer's disease. I will discuss the presence of Alzheimer-type lesions in human and nonhuman primates, and present pathological and biochemical evidence for important similarities and differences among species that may govern the species-specific susceptibility to senile plaques, cerebral β -amyloid angiopathy and neurofibrillary tangles in humans and chimpanzees. Supported by NIH RR-00165, PO1AG026423, P50AG025688 and the Woodruff Foundation.

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