concomitant AD pathology. Findings that TL is shorter even in substantially non-mitotic brain tissue suggests that telomere shortening might in part be an innate feature of individuals, either owing to congenitally shortened telomeres, or an inherited tendency for reduction in telomere length. Additional experiments examining longitudinal decline in blood leukocyte DNA may be able to further clarify the degree to which telomere length is environmental or acquired versus innate.

P1-214 ELEVATED IRON AND ZINC IN ALZHEIMER HIPPOCAMPUS: A POSTMORTEM STUDY USING NEUTRON ACTIVATION ANALYSIS

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Background: Iron and zinc are essential for the normal brain physiology. However an imbalance of these metals has been postulated to play a role in the pathogenesis of AD. We aimed to correlate levels of iron and zinc with the cognitive status and beta amyloid burden in postmortem well-characterized cases. **Methods:** Subjects with AD (n = 13) and normal cognition (n = 19) of the Brain Brain of Brazilian Aging Brain Study Group were classified according to the clinical and neuropathological evaluations. The clinical diagnosis was established through a postmortem interview with an informant including validated scales and questionnaries The neuropathological examinations were carried out based on accepted criteria, using immunohistochemistry. Neuropathologically, AD was defined by a CERAD [3] B and a Braak and Braak [3] IV. Levels of Fe and Zn were measured in the hippocampus using instrumental neutron activation analysis (INAA). Certified reference materials were analysed for assuring the quality of the analitycal results. Protocols were approved by the local ethics committee. The statistical analysis was performed using the IBM PAWS Statistics Package v.18 and Minitab Statistical Software v.15. **Results:** Significantly higher ($p < 0.05$) concentrations for Fe and Zn were found in the hippocampus of AD cases. The level of these elements was correlated to the amyloid plaques burden. **Conclusions:** Brain iron and zinc accumulation are a prominent feature of advanced Alzheimer disease. Recent evidence suggests that beta amyloid precipitation and toxicity by formation of H2O2 and increase of oxidative stress in AD are caused by abnormal interactions with metal ions, especially Zn and Fe. Our findings corroborate this hypothesis. However the exactly role of the iron and zinc alterations in brain in the pathogenesis of AD is yet to be clarifyed.

P1-215 ETIOLOGY OF DEMENTIA IN THE DIFFERENT STAGES OF DISEASE: RESULTS FROM A POST-MORTEM STUDY

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Background: Both vascular dementia and Alzheimer disease are chronic and progressive conditions resulting in cognitive decline. Sometimes it is difficult to distinguish between these two conditions and differences in clinical evolution could help establish an accurate etiological diagnosis. This clinicopathological study aims to analyze the etiology of dementia in individuals who died at different stages of disease. **Methods:** A post-mortem study evaluating individuals, aged 50 years or older, included in the Brain Bank of the Brazilian Aging Brain Study from University of Sao Paulo. Cognitive evaluation was gathered with a semi-structured interview with the next of kin informant using the Clinical Dementia Scale (CDR). Cases were classified as moderate dementia if the CDR = 2 and advanced dementia if the CDR = 3. Neuropathological examinations were carried out based on accepted criteria, using immunohistochemistry. An Alzheimer's disease diagnosis required a Braak stage ≥ IV and moderate or frequent cortical neuritic plaques according to the Consortium to Establish a Registry for Alzheimers Disease (CERAD) criteria. The presence of hyaline atherosclerosis and microinfarcts were registered. Demographical data and cardiovascular risk factors were recorded. **Results:** From the 122 individuals analyzed, 46 (37.7%) were classified as CDR = 2 and the other 76 individuals (62.3%) had a CDR = 3. The occurrence of a Braak stage ≥ IV (21.7% and 62.7% in CDR 2 and 3, respectively, $p < 0{,}001$) and moderate or frequent neuritic plaques according to CERAD (30.2% and 68.1% in CDR 2 and 3, respectively, $p < 0{,}001$) was higher in individuals with CDR = 3 and suggested that Alzheimer's disease predominated in patients dying at this stage. A higher prevalence of systemic hypertension (71.7% and 46.1 for CDR 2 and 3, respectively, $p = 0{,}01$), heart failure (26.1% and 11.8% for CDR 2 and 3, respectively, $p = 0{,}04$) and a trend toward higher prevalence of stroke (45.7% and 30.3% for CDR 2 and 3, respectively, $p = 0{,}08$), suggested that vascular disease was more prevalent among individuals dying with a CDR = 2 stage. **Conclusions:** Individuals suffering from Alzheimer's disease are prone to die at advanced stage of dementia while those who suffer form vascular dementia die at moderate stage of disease.

P1-216 CEREBRAL AMYLOID ANGIOPATHY, NEURODEGENERATION, AND DEMENTIA IN A POPULATION-BASED STUDY ON THE VERY ELDERLY FINNS (VANTAA 85+)

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Background: Alzheimer's disease (AD) has traditionally been neuropathologically characterized by the accumulation of neuritic plaques and neurofibrillary tangles. Recently, it has been claimed that cerebral amyloid angiopathy (CAA), deposition of β-amyloid in the walls of cerebral cortical and leptomeningeal blood vessels, plays a central role in the development of clinical dementia in AD. Although there are several types of hereditary dementias with a purely genetic background, sporadic AD is considered a multifactorial disease. However, it is unclear, how neuropathological and genetic factors and other diseases interact to result in clinical dementia. This study, as a part of the prospective population-based Vantaa 85+ human autopsy study, aims to investigate 1) the role of CAA in AD and other dementias and 2) the relationships between CAA and other neuropathologies such as cerebral infarctions, hemorrhage and inflammation, and to 3) identify novel genetic risk factors for CAA. **Methods:** The study is based on a cohort of 307 autopsied individuals aged 85 years or over,