Although a large population was evaluated in this study, the diagnosis of AD was gathered exclusively through clinical information and neuropathological features typical of vascular and Lewy bodies dementia were included in the analysis. The objective of the current study was to examine whether the association between neuropathological features related to AD and dementia varies in the very-old group (age = 80 years) compared to the young-old group (age = 79 years). **Methods:** A post-mortem study evaluating individuals = 60 years, included in the Brazilian Aging Brain Study. Cognitive evaluation was gathered with a semi-structured interview with the next of kin informant using the CDR and the IQCODE. Individuals were classified as cognitively normal if the CDR = 0 and the IQCODE < 3.20 and as demented if the CDR = 2 and the IQCODE > 3.80. Neuropathological examinations were carried out based on accepted criteria, using immunohistochemistry. All subjects were classified according to Braak & Braak staging, CERAD and the NIA-RI criteria. ROC analysis were preformed to evaluate the predictive value of each neuropathological feature in both intervals of age. **Results:** We examined 360 individuals. Of the participants 52.5% were female, 66.4% were classified as CDR = 0, 12.5% as CDR = 2 and other 21.1% as CDR = 3 and 41,1% were = 80 years. The relationship between AD-type neuropathological features and dementia were not different among the Young-old compared to the very-old group. Braak & Braak staging was considered the most predictive feature in both groups (ROC area under the curve 0.75, 0.61-0.89, $p < 0.01$ for the very-old and 0.73, 0.52-0.93, $p = 0.04$ for the young-old group). The NIA-RI (ROC area 0.59, 0.43-0.73 and 0.67, 0.46-0.87 for the very-old and young-old, respectively) and the CERAD criteria (ROC area 0.53, 0.36-0.70 and 0.66, 0.45-0.87 for the very-old and young-old, respectively) did not reach statistical significance as predictors. **Conclusions:** Neruopathological features related to AD demonstrate the same predictive value for dementia in the very-old compared to the young-old. Braak & Braak staging is the most predictive neuropathological feature in both groups of age.

P4-008 LIVING LONGER WITHOUT ALZHEIMER'S DISEASE: A NEUROPATHOLOGICAL STUDY OF THE INDIVIDUALS AGED ≥ 90 YEARS.

Jose Farfel[1], Lea Grinberg[2], Renata Ferretti[1], Claudia Suemoto[1], Renata Leite[1], Carlos Pasqualucci[1], Ricardo Nitrini[1], Wilson Jacob-Filho[1], *[1]University of Sao Paulo, Sao Paulo, Brazil; [2]University of California San Francisco, San Francisco, California, United States.*

Background: The oldest old (≥90 years) are the fastest growing demographical population. However there are few neuropathological studies in this group and many clinicians keep the concept that Alzheimer's disease (AD) is a paradigm of longevity. Studies including oldest old individuals focus on demented individuals but fail to characterize the healthy individuals who spare clinical and neuropathological features of AD. The objective of the current study is to characterize the oldest old free of cognitive impairment and of neurofribrillary tangles and neuritic plaques and to compare demographical, clinical, neuropathologic data and apoE genotype between nonagenarians and octogenarians. **Methods:** A post-mortem study evaluating individuals, aged 80 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) and the Informant Questionnaire on Cognitive Decline in the Elderly (IQCODE). Subjects were classified as cognitively normal if the CDR = 0 and the IQCODE < 3.20 and as demented if the CDR ≥ 2 and the IQCODE > 3.80. Neuropathological examinations were carried out based on accepted criteria, using immunohistochemistry. Alzheimer's disease was diagnosed if the subject presented moderate or high likelihood for AD base in the NIA-RI criteria. ApoE genotyping was performed in subgroup of patients (10 nonagenarians and 46 octogenarians randomly selected). **Results:** Twenty-five individuals ≥ 90 years were included, mean age 93.3 years (90-102 years), being 64% females. From this sample, 44.0% were free of clinical and neuropathological features of AD. A total of 121 individuals of 80 to 90 year were included, mean age 83.7 (80-89 years), being 64.2% females and 45.5% were totally free of AD There were no differences between the two groups regarding gender ($p = 0.98$), education ($p = 0.37$), systemic hypertension ($p = 0.97$), diabetes ($p = 0.27$), presence of microinfarcts ($p = 0.23$), hyaline atherosclerosis ($p = 0.31$), prevalence of AD-related neuropathological features through the fulfillment of moderate and high likelihood criteria of the NIA-RI ($p = 0.85$). There was significant differencea between the groups regarding the presence of apolipoprotein e4 (37% in octogenarians and 0% in nonagenarians, $p = 0.03$). **Conclusions:** There is no difference in the prevalence of AD between octogenarians and nonagenarians. Nonagenarians may present a favorable genetic profile protecting them against dementia and balancing the effects of aging.

P4-009 DECREASE OF PTEN EXPRESSION LEVELS AMONG NORMAL, SYMPTOMATIC AND ASYMPTOMATIC ALZHEIMER'S DISEASE (AD) SUBJECTS, MEASURED IN HIPPOCAMPUS, TEMPORAL AND ENTORHINAL CORTICES.

Ana Cecília Feio dos Santos[1], Aderbal Silva[1], Lea Grinberg[2], Jose Farfel[3], Renata Ferretti[4], Maria Dirlei Ferreira Begnami[1], Rafael Rocha[1], Fernando Soares[1], Helena Brentani[4], *[1]A C Camargo Hospital, São Paulo, Brazil; [2]UCSF, San Francisco, California, United States; [3]University of Sao Paulo, São Paulo, Brazil; [4]University of São Paulo Medical School, São Paulo, Brazil.*

Background: PTEN negatively regulates intracellular levels of PIP3 and antagonizes the PI3K signaling pathway important for tumor suppressor, modulating cell apoptosis. In hippocampus, PTEN deficiency causes defects in synaptic structure and plasticity. In AD brain, recent studies showed decreased levels and altered distribution of PTEN along with, suggesting that a loss of PTEN contributes to neurodegeneration in AD. **Methods:** This study measured distribution of PTEN in postmortem human brain tissue (hippocampus - H, entorhinal - EC andtemporal cortices - TC) through the tissue microarray technique. We compared three groups: symptomatic AD (sAD) -with dementia and AD pathology; asymptomatic AD (asAD) - with AD pathology but without dementia; and normal elderly subjects (N) - without dementia and AD pathology, accounting 61 subjects. Asemi-quantitative analysis was employed. Statistical analysis was performed by chi-square. **Results:** Statistically significant decrease was found when we analyzed samples by absence/presence of AD pathology, in all brain regions together (Normal versus sAD+asAD)($x^2 = 29.97$ and p-value <0.0001), and analyzing the regions separately (EC - $x^2 = 14.00$ and p-value = 0.003; and H - $x^2 = 13.13$ and p-value = 0.004). However, nostatistically difference in PTEN levels was found at TC ($x^2 = 5.911$ and p-value = 0.116). When we analyzed separated by absence/presence of dementia (aAD versus sAD) statistical difference was found ($x^2 = 16.62$ and p-value = 0.0008) at all regions, and at TC ($x^2 = 10.81$ and p-value = 0.012), but no at EC ($x^2 = 6.881$ and p-value = 0.075) and H ($x^2 = 3.39$ and p-value = 0,334). At 42.5% of TMA cores showed prominent cytoplasmatic staining, but there was not statistical difference. **Conclusions:** Publications on the role of PTENin AD are still controversial. Our results corroborates some papers in the literature that showed decrease in PTEN levels when we analyzed samples by absence/presence of AD pathology (Normal vs sAD+aAD). Literature have no previous data that have measured and compared PTEN levels in asymptomatic ADindividuals, and the differences found when individuals were separated by the absence/presence of dementia (aAD versus sAD) is an unpublish 2nd data. Others authors showed decreasing PTEN levels in EC, TC and H. Regarding the cytoplasmatic immunoreactivity, this localization is in agreement with data from other studies reporting that PTEN is ubiquitously expressed in CNS and is both nuclear and cytoplasmatic.