specific concentration range neuroprotective effectiveness in ex vivo tissue model. **Objective:** To apply computational methodologies for rapid screening of potential neuroprotective therapeutic targets induced in response to IL-1α signaling. **Methods:** We have performed computational modeling of IL-1/NF-kB pathway by initiating functional diagram of interactions based on cumulative literature data and their subsequent transformations into modular subgroups for application using MatLab software. We have subsequently analyzed functional output diagrams showing connectivity points based on synchrony in time-course oscillations among individual network components, such as NF-κB, IKK, IκB. While IKK appears as major controller module after neuroinflammatory activation of IL-1/IL-1R/IRAK/TRAF pathway, number of other molecular time-responses are observed. Analysis of dynamics in NF-κB oscillations and response of interlinking adaptor and effector proteins showed profound effect on TRAF and IKK complexes, and most particularly, rapid downregulating inhibitory effect on cellular expression of MyD88. **Conclusion:** Our data demonstrate general beneficial and limiting usefulness of computational approaches in studies of signal transduction in AD. Using IL-1 pathway, we present sequential time-dependent analysis for individual components activated in response to neuroinflammatory stimulus. We identify MyD88 as novel target with therapeutic relevance. Since complete inhibition of IL-1α by immunosuppressive, anti-inflammatory NSAID treatments does not appear beneficial, likely due to disruption of neuroprotective IL-1 regulating effect on microglial chemotaxis, our study implies that fine mapping of individual factors in IL-1α signaling, such as MyD88, may provide, by targeting beneficial targets in inflammatory cascades, an improvement to the existing treatments, including Aβ antibody based immunization. Acknowledgment: Alzheimer's Association (O.M.).

P4-312 LIFETIME DIET AND COGNITIVE PERFORMANCE IN AN OLDER COMMUNITY-DWELLING POPULATION

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Background: Research has identified the possible role of diet in age-related cognitive decline and cognitive impairment. The aetiology of cognitive decline suggests past dietary intake may be relevant to cognitive status in later life. **Objective:** To investigate possible long-term influences of diet on cognitive status in old age. **Methods:** A sample of 352 community-dwelling older adults (Females = 189) completed a non-quantitative food frequency questionnaire designed to assess lifetime diet. Ages ranged from 65 to 91 years (M = 73.15, SD = 5.48) with a mean of 13 years of education (SD = 5.7). Participants also undertook an extensive battery of computerised and pencil and paper cognitive tasks to assess reasoning, knowledge, perceptual speed, memory, choice-reaction time and inhibition. Principle components analysis was performed on each of the lifetime periods to determine possible dietary patterns within those periods. Relationships were then examined between dietary patterns and performance on the cognitive tasks. Factor scores derived from confirmatory factor analytic models of the cognitive domains were the dependant variables in these analyses. Co-variates included current diet, physical activity, smoking status, and relevant health and demographic variables. **Conclusion:** Common dietary patterns emerged within each life-period; they were labelled 'Traditional Australian' 'European/Mediterranean' and 'Sweets and Processed foods'. Associations between dietary patterns across different life-periods, cognitive outcomes, and demographic influences will be discussed. Dietary intake across the lifetime, as measured by this retrospective food frequency questionnaire, may be related to cognitive status in later life.

P4-313 SUB-CLINICAL ZINC DEFICIENCY FOUND IN ALZHEIMER'S DISEASE

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Background: Copper/zinc dyshomeostasis and chronic copper exposure have been linked to the progression and pathogenesis of AD. Sub-clinical zinc deficiency in another chronic neurodegenerative disease, age-related macular degeneration (AMD) , was discovered in the 1980s (Newsome, et al., Arch Ophthalmol, 1988); now zinc supplementation is a critical part of the standard of care for AMD. There are no well-controlled reports comparing serum zinc concentrations between AD and normal subjects. Squitti, et al., (Neurology,2009) recently reported a negative correlation trend between serum zinc concentrations and cognitive decline in AD patients in longitudinal observations. **Objective:** A prospective, blinded, observational clinical study mounted at the Alzheimer's Center of Albany Medical Center, involved the collection of serum. **Methods:** After IRB approval, 90 subjects, 30 AD, 30 Parkinson's Disease (PD) and 30 normal subjects were recruited through referrals and radio advertising. AD was diagnosed by clinical, functional and NINDA-ADRDA criteria. PD patients met typical diagnostic criteria. All subjects either did not take or had not taken for 30 days zinc/copper containing supplements. Fasting blood and spot urine samples were collected in the morning. Protocols were followed to avoid environmental zinc/copper contamination of samples. Blood was immediately spun to obtain serum; serum and urinary aliquot samples were immediately frozen at -80^{o} for later analysis by atomic absorption. **Conclusion:** Results: AD subjects had 14% lower zinc concentrations as a group than normals (64.6 microgm/dL vs. 76.1 microgm/dL; $p < 0.021$), as well as compared to PD subjects (74.9 microgm/dL; ; $p < 0.020$). **Conclusions:** Serum zinc concentrations in AD subjects demonstrated data consistent with a sub-clinical zinc deficiency. These findings indicate a zinc dyshomeostasis in AD deserving further study.

P4-314 INHIBITORS OF CATALASE-AMYLOID INTERACTIONS PROTECT CELLS FROM OXIDATIVE STRESS AND TOXICITY INDUCED BY AGGREGATED ALZHEIMER'S-RELATED BETA-AMYLOID PEPTIDES

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Background: While oxidative stress is commonly associated with aging, increased oxidative damage mediated by oxidative stress (such as increased oxidation of proteins, lipids and nucleic acids) and subsequent neuronal loss in