Abstract: **Introduction:** Alzheimer's disease (AD) has been identified as a protein-misfolding disease, caused by accumulation of abnormally folded amyloid beta (Aβ) and tau proteins in the brain. Aβ<sub>1-40</sub> and Aβ<sub>1-42</sub> are the main components of amyloid plaques that are found in the brain of patients with AD. Identification of an AD-biomarker that can be derived from blood samples has been a goal of researchers for many years. Here, we have tried to determine the Aβ<sub>1-40</sub> levels in the peripheral blood samples obtained from AD patients. Our preliminary results indicated that the levels of Aβ<sub>1-40</sub> in the peripheral blood could be of considerable value in the differentiation of AD patients from normally aged individuals.

**Method:** We made use of a sandwich enzyme-linked immunosorbent assay (ELISA) to quantify blood serum Aβ<sub>1-40</sub> level. Clinical diagnoses were carried out on the basis of the criteria of the NINCDS-ADRDA (National Institute of Neurological and Communicative Disorders Association Stroke and Alzheimer's Disease and Related Disorders Association). We analyzed 45 samples in three groups, i.e., 15 AD patients (aged 73.5±9.9), aged control (AC) group (10 individuals, aged 65.8±8.1) and young control (YC) group (20 individuals aged 28±3).

**Results:** The serum Aβ<sub>1-40</sub> levels in the AD group were significantly lower than those in the other groups. Considering the relationship between Aβ<sub>1-40</sub> level and age in AD group it becomes clear that Aβ<sub>1-40</sub> level decreases with age in both females and males with AD.

**Conclusions:** These results are consistent with our hypothesis that low serum Aβ<sub>1-40</sub> levels is an indicator of risk for AD and declines with onset and progression of Alzheimer and which might reflect the sequestration of Aβ<sub>1-40</sub> in the senile plaques or the formation of semi-soluble oligomers.

**Keywords:** Protein misfolding, Amyloid plaques, Biomarker, Amyloid beta.

**Presentation:** Poster