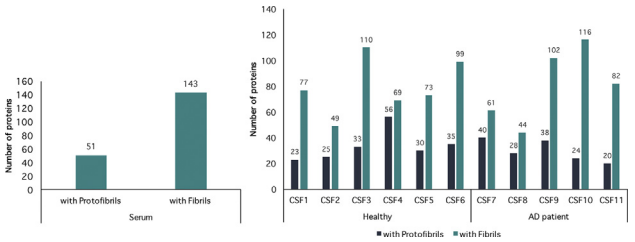

		Healthy						Alzheimer's				
Protein name	Acc#	CSF-1	CSF-2	CSF-3	CSF-4	CSF-5	CSF-6	CSF-8	CSF-9	CSF-10	CSF-11	CSF-12
Neurexin-1	Q9ULB1											
Neurexin-2	Q9P2S2											
Neurexin-2-beta	P58401											
Neurexin-3	Q9Y4C0											
Agrin	O00468											
Testican-3	Q9BQ16											
Complement C4-A	P0C0L4											
Complement C4-B	P0C0L5											
Neuronal pentraxin-1	Q15818											
Decorin	P07585											
Protein AMBP	P02760											
Calreticulin	P27797											
Complement C1r subcomponent	P00736											
Complement factor H-related protein 1	Q03591											
EGF-containing fibulin-like extracellular matrix protein 2	O95967											
Apolipoprotein L1	O14791											
Collagen alpha-1(I) chain	P02452											
Complement factor H	P08603											

(APP) containing vesicles in neuron. Kinesin-1 is a tetrameric protein composed of two heavy chains (KHCs) and two light chains (KLCs). The tetratricopeptide repeat (TPR) domain of KLC1 may be responsible for binding APP either directly or via interaction with C-jun N-terminal kinase-interacting protein 1 (JIP1). However, the binding partners of the TPR domain of KLCs have not yet been fully identified. **Methods:** We were used the yeast two-hybrid system to identify the binding proteins that interact with the TPR domain of KLC1. The binding affinity was quantified by measuring β-galactosidase activity in liquid cultures of yeast transformed cells. Direct interaction between binding proteins and KLC1 in mammalian cells as well as *in vitro* was assayed using the co-immunoprecipitation with the antibodies. The cellular co-localization in cells was used the immunocytochemistry. **Results:** We revealed an interaction between the TPR domain of KLC1 anddynamin-1-like protein (Dnm1L), also known as dynamin-related protein 1. Dnm1L bound to the six TPR domain of KLC1 and did not interact with KIF5s. Dnm1L interacts with KLC1 through itsGTPase effector domain (GED) domain. When co-expressed in HEK-293T cells, co-localized with KLC1 and co-immunoprecipitated with KLC1, but not KIF5B. **Conclusions:** We suggest that, after mitochondrial fission, interaction of Dnm1L with KLC1 may lead to dissociation of kinesin-1 tetramer, allowing KIF5 to interact with milton and transport mitochondria.

P1-231 A CROSS-SECTIONAL STUDY OF SERUM BRAIN DERIVED NEUROTROPIC FACTOR (BDNF) CONCENTRATIONS IN A SAUDI POPULATION AND ALZHEIMER'S DISEASE

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Background: Brain-derived neurotrophic factor(BDNF) is a protein, a member of the neurotrophin family of growth factors. It is found in the brain and peripheral tissues; it mainly helps to support the survival of existing neurons and encourages the growth and differentiation of new neurons and synapses. BDNF concentrations tend to decline with age. Post-mortem studies of AD patients showing decreased mRNA BDNF in brain regions commonly affected by AD have ignited an interest in BDNF as a potential marker. **Methods:** The research variables are the serum BDNF concentrations and cognition changes. The enzyme-linked immunosorbent assay (ELISA) technique was used to assess the BDNF concentrations. While the clinical rating scales were used to assess the cognitive performance. Moreover, there were independent variables such as age, gender, BMI, DM and the use of medications were assessed. **Results:** The total healthy group was 123 participants made up of younger healthy subject's age range 25-35 years (n=34), middle age group range from 36-59 years (n=41) and the elderly healthy volunteer's age was above 60 years (n=48). As the result showed, elderly subjects had a lower mean serum BDNF level than younger age participants (338.9±124.30 vs. 80.3±27.84pg/ml, $P < 0.001$). Additionally, the Alzheimer's patients were above 60 years old (n=27) and all categorised according to the CDR scores into four categories (very mild cognitive impairment n=4(14.8%), Mild dementia n=6(22.2%), Moderate dementia n=13 (48.1%) then severe dementia n=4 (14.8%). **Conclusions:** Our research has evidence that the serum BDNF concentrations of healthy participants decrease with ageing in comparison to younger healthy control group. Within the patient population, serum BDNF concentrations were found to be significantly decreased only in patients with severe AD. Those with mild to moderate did not have serum BDNF concentrations significantly different to those of healthy, elderly controls.

P1-232 HIGH-SPEED ATOMIC FORCE MICROSCOPY REVEALS STRUCTURAL DYNAMICS OF AMYLOID β1-42 AGGREGATES

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Background: Alzheimer's disease (AD) is characterized by the accumulation of amyloid plaques and neurofibrillary tangles. Aggregation of amyloidogenic proteins into insoluble amyloid fibrils is implicated in various neurodegenerative diseases. This process involves protein assembly into oligomeric intermediates and fibrils with highly polymorphic molecular structures. These structural differences may be responsible for different disease presentations. **Methods:** In order to elucidate the structural features and assembly kinetics of amyloid β-protein (Aβ), we used high-speed atomic force microscopy (HS-AFM) studies of fibril formation and elongation by the 42-residue form of Aβ1-42, a key pathogenetic agent of AD. **Results:** 1st, our video-imaging visualized the growth manner of individual filament of Aβ1-42 fibrils: polarized growth and stepwise elongation. 2nd, our data demonstrate two different growth modes of Aβ1-42, one producing straight fibrils and the other