

P013 Dysregulation of Caveolin processing in Alzheimer disease mice
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Alzheimer's disease (AD) is a neurodegenerative disorder characterized by progressive dementia and the accumulation of neuritic plaques in limbic and neocortical brain regions. Cholesterol is not only modulates amyloid beta synthesis but also interactions between amyloid beta and neuronal membranes that may initiate a neurotoxic cascade. Caveolin, which binds with cholesterol and plays a prominent role in cellular cholesterol transport. High levels of cholesterol and caveolin in the caveolae-enriched fractions were found in AD patients compared with control. In the present study, we have used transgenic mice overexpressing mutated amyloid precursor protein (APP_{K670N/M671L+V717F}) under the control of PrP gene promoter, and unique is that exhibits amyloid beta deposits by three month of age with dense-cored plaques and neuritic pathology, in concert with cognitive deficits. Western blot analysis of Caveolar markers, such as, Caveolin and Caveolin-1 showed significant increase in brain homogenates from 6 and 9 month old transgenic mice but no increase in young mice at 2 to 4 month of age compared with littermate control. These results, taken together, suggest that caveolin expression is directly correlated with high Amyloid beta levels and amyloid deposits.