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Enhanced cleavage of amyloid β-peptides in the casein injected inflammatory brain of mice

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The excessive accumulation of amyloid β-Peptides (Aβs) in the brain is the causative factor in all genetic as well as sporadic cases of Alzheimer's disease (AD). Two enzymes namely beta-secretase and gamma-secretase are involved in the defective cleavage of Amyloid Precursor Protein (APP) and the alpha-secretase is involved in its normal processing. The core protein of the amyloid senile plaques within the brains of afflicted individuals contains peptide of 39-43 residues, but mostly terminating with residues 40 and 42. The longer Aβ42 is more abundant in the amyloid of the neuritic plaques while the shorter peptides ($A\beta_{40}$) found more in the vascular deposits. Factors that lead to the over expression of Aβs are yet to be identified. In the present study, we have shown that the prolonged subcutaneous injection of casein, which is known to develop organomegaly of the liver, spleen and kidneys in mice (the Congo red stain of the liver confirmed the presence of a fibrillary protein deposits with amyloid characteristics in chronic inflammation and associated systemic amyloidosis) triggers about 20 times more Abeta accumulation (in sixty six days period) in the mice brain than that of the control mice which was confirmed as well as quantified by RP-HPLC. From the Mass Spectroscopic analysis, we have shown the occurrence of a new type of proteolytic zeta cleavage fragment, a 1-54 residue Abeta in the mice brain along with the Abeta peptides.

Biography

Asokan C has completed his PhD from University of Madras and Postdoctoral studies from Columbia University, NY, USA. He is an Associate Professor at the Department of Biochemistry, Sokoto State University, Sokoto, Nigeria. He has published more than 36 papers in reputed journals and has been serving as an Editorial Board Member of repute.

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