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BACE1 expression in post-natal rat brain regions: Where goes wrong?

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Science through decades has reported that BACE1 activity towards APP promotes toxic Aβ-generation during conditions like Alzheimer's disease (AD). In spite of several other proteins that are involved in AD pathology it is not clear why BACE1 alone serves as target? Age and sex are considered to be the major risk factors for AD, while their influence on BACE1 expression yet poorly investigated. In other hand, brain has parcellation of neuronal-circuits containing 180-regions and therefore the origin of pathogenesis become mysterious; again, why hippocampus alone delineated for cognitive decline and AD is unclear. Therefore, present study aims to understand the levels of BACE1 expression in Post-natal-developmental (PND) stages of rat at various brain regions. Based on human-age Vs rat-age calculation, 10-groups of Wistar rats in their PND-stages viz., P1, P8, P24, P30, 3M, 3M-UMF, 6-M, 9M-MF, 12-M and 24-M were used to determine the sensitive period of pathogenesis in the present study. Vulnerability at 9-different brain regions (viz., olfactory-bulb, frontal-cortex, parietal-cortex, temporal-cortex, occipital-cortex, hippocampus, cerebellum, hypothalamus-thalamus and pons--medulla-oblongata) were analyzed for BACE1. Immunoblotting results show an unprecedented higher expression of BACE1 even during the first post-natal week that contradicts literature, which states that BACE1 increases in expression during aging. Interestingly, BACE1 is found to be significantly similar in all the PND-stages and in all the regions of brain. If BACE1 is observed to be expressed throughout the period of life, then how does the young get spared from SAD pathogenesis is astonishing. Further, to contradict, the levels of BACE1 is expressed with higher significant in young rather than aged. Although the level of BACE1 was down regulated during aging, the pyramidal cell rich hippocampus had a greater expression than the neocortical regions. To corroborate, it is inconclusive to claim BACE1 as a potent-target against AD-like pathology. Further, APP during aging has to be elucidated given that much more substrates available for BACE1 cleavage, and speculations on region specific action of BACE1 has to be intensively investigated to consider BACE1 as potent-target against SAD pathogenesis.

Biography

Sathya M is currently working in the Department of Biochemistry at Bharathidasan University in Trichy, India. She has published several original research papers in the reputed journals and participated in the several scientific meetings.

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