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Late phase cell cycle proteins in proteotoxic control in Alzheimer's disease: A possible target for therapy?

Vladan Bajic[1], Biljana Spremo-Potparevic[2], Lada Zivkovic[2], Martina Bruckner[3], Thomas Arendt[3]
[1]International Rice Research Institute, Philippines
[1]Vinca Institute Nuclear Science, Serbia
[2]University of Belgrade, Serbia
[3]University of Leipzig, German

Alzheimer's disease is represented by neuronal loss and this loss is correlated to a constant state of neuronal instability induced by intrinsic and extrinsic factors. Aneuploidy has been seen as a culprit of process leading to Alzheimer's disease (AD). It affects, primarily gene dosage of proteins (proteotoxicity) that are regulators of the APP metabolism but also may affect the protease and autophagy machinery in the AD cell. Late phase cell cycle proteins such as cohesin, centromere associated cycline-CDK11, Bub R1, Mad 2 and Mad 2B are proteins that regulate the anaphase promoting complex and the anaphase cell cycle checkpoint which therefore control proper chromosome segregation and separation. These Late phase cell cycle proteins are essential for maintaining genomic balance and are found when overexpressed lead to reduction in chromosome mis-segregation and hence aneuploidy. Also, knowledge that a number of these proteins that regulate cell division have secondary roles, i.e. coordinate a number of complex processes in neurogenesis (axonal pruning, dendric and spine morphogenesis and etc), neuronal survival and the maintenance of the post-mitotic state of neurons has led to question of how are these protein/s affected in Alzheimer's Disease. Here we wish to present new and previous data on the possible roles of Late phase cell cycle proteins in normal and affected neurons in relation to the alteration of proteotoxic control that has been affected by aneuploidy. Therefore, understanding the mechanisms of proteotoxicity regulated by these proteins may represent a novel strategy for AD treatment.

vladanbajic@yahoo.com