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Axonal Translocation and Anti-proliferative Action of Itm2b in Differentiating N2a Cells[1]

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Itm2b is a type 2 integral transmembrane protein, and its mutation and pro-apoptotic activity were closely coupled in the genetic neurodegenerative disorders, such as familial British (FBD) and Danish dementia (FDD). Although the cellular location and trafficking of Itm2b has been demonstrated in several studies, it is unknown whether the mutant form of Itm2b has a similar cellular effect. The current study was designed to determine the subcellular distribution of wild type Itm2b variants and to investigate its physiological function in neurons. To construct the fusion vectors carrying reporter gene, wild type Itm2b mRNA full sequences were fused with EGFP or myc sequences driven by CMV promoter. Their subcellular locations were obtained from the merged images between EGFP flurescence and subcellular organelle marker. To examine the effects of overexpressed Itm2b on cell proliferation, the incorporation of BrdU was analyzed in the fusion construct-transfected N2a neuroblastoma cells. Fluorescent microscopy showed that the Golgi marker signals were matched completely with GFP or myc signals, indicating Golgi location of Itm2b. The overexpression of Itm2b also resulted in delay of cell proliferation. These results suggest that Golgi distribution of Itm2b may be involved in diverse cellular function such as proliferation and differentiation.

Keywords : Itm2b, Golgi, Proliferation, Differentiation, N2a cell, Brain cells.

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