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Neurovascular Pathology in ApoB-100 Transgenic Mice

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Apolipoprotein B-100 (ApoB-100) is the major protein component of the low density and very-low-density lipoproteins that are responsible for cholesterol and triglyceride transport from the liver to the peripheral tissues. Therefore, the ApoB-100 overexpressing mouse strain is a frequently used model of atherosclerosis, as they are more susceptible to the cholesterol-enriched diet induced myocardial dysfunction. Moreover, previously we detected widespread neurodegeneration, neuronal apoptosis, synaptic dysfunction and tau-hyperphosphorylation in the brain of ApoB-100 transgenic mice finally leading to cognitive impairment. Since the neurovascular origin of various neurodegenerative diseases is becoming more and more accepted, our aim was to study the structural and functional impairments of the blood-brain barrier in ApoB-100 transgenic mice. In vivo permeability studies showed increased blood-brain barrier permeability in the hippocampus, while the disruption of the brain capillary endothelial tight junction structure and edematous swelling of astrocyte end-feet were demonstrated using transmission electron microscopy in transgenic brains. We found decreased P-glycoprotein (Abcb1) and vimentin immunostainings and altered Gfap immunostaining pattern related to the neurovascular unit by confocal microscopy. Real-time PCR showed increased *Lox-1, Aqp4*, and decreased *Meox-2, Mfsd2a, Abcb1a, Lrp2, Glut-1, Nos2, Nos3* gene expression level in isolated microcapillary fraction of transgenic mice. These results underline the neurovascular origin of neurodegeneration in hypertriglyceridemicApoB-100 transgenic mice.

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Biography:

Melinda E. Tóth is currently a research associate scientist in the Institute of Biochemistry of the Biological Research Centre (Szeged, Hungary). She graduated from the University of Szeged with a master degree in biology in 2006. After graduation she continued her work in the lab of MiklósSántha, the Laboratory of Animal Genetics and Molecular Neurobiology as a PhD student and got her PhD from the University of Szeged in 2013. Her research interest is investigating the molecular basis of the protective role of heat shock proteins in diseases like Alzheimer's disease and hyperlipidemia induced neurodegeneration in transgenic mouse models.