Adropin

Adropin is a 4.9 kDa peptide that is important for maintenance of metabolic and non-metabolic homeostasis. It regulates glucose and fatty acid metabolism and is involved in endothelial cell function and endothelial nitric oxide (NO) synthase bioactivity as well as physical activity and motor coordination. Adropin is expressed in many tissues and organs including central nervous system (CNS). This peptide plays a crucial role in the development of various CNS disorders such as stroke, schizophrenia, bipolar disorder as well as Alzheimer's, Parkinson's, and Huntington's diseases [1].

In a study of Yu et al. from the Zhejiang University School of Medicine, Hangzhou, Zhejiang, Loma Linda University, California, USA and Departments of Neurosurgery & Brain and Nerve Research Laboratory, The First Affiliated Hospital, Soochow University, Suzhou, China, the role of adropin in collagenase-induced ICH was investigated in mice. At 1-h post-ICH, mice were administered with recombinant human adropin by intranasal. Brain water +content, BBB permeability, and neurological function were measured at different time intervals. Proteins were quantified using western blot analysis, and the localizations of adropin and Notch1 were visualized via immunofluorescence staining. It is shown that adropin reduced brain water content and improved neurological functions. Adropin preserved the functionality of BBB by increasing N-cadherin expression and reducing extravasation of albumin. Moreover, in vivo knockdown of Notch1 and Hes1 both abolished the protective effects of adropin. Taken together, our data demonstrate that adropin constitutes a potential treatment value for ICH by preserving BBB and improving functional outcomes through the Notch1 signaling pathway [2].

1)
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2)
Yu L, Lu Z, Burchell S, Nowrangi D, Manaenko A, Li X, Xu Y, Xu N, Tang J, Dai H, Zhang JH. Adropin preserves the blood-brain barrier through a Notch1/Hes1 pathway after intracerebral hemorrhage in mice. J Neurochem. 2017 Dec;143(6):750-760. doi: 10.1111/jnc.14238. Epub 2017 Nov 17. PubMed PMID: 29030969; PubMed Central PMCID: PMC5729103.