

## Memory impairment in the rats induced by endogenous serotonin-modulating anticonsolidation protein

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### Abstract

Serotonin-modulating anticonsolidation protein (SMAP) has been earlier identified in the rat brain cortex and further purified from the whole rat brains (Mekhtiev, 2000). The conducted biochemical (on the rats) and electrophysiological studies (on the mollusks) have shown that this protein is being in linear relations with serotonin level. During formation of long-term memory on the conditioned shuttle-box model with electric shock reinforcement and on the alternative running model with food reinforcement downregulation of this protein in the parietal and occipital cortex of the rats, achieved 80% of correct trials, was noted ( $p < 0.001$ ). Intra-cerebral administration of SMAP 24 h prior to the learning session significantly impaired formation of memory trace in the animals on the model of passive avoidance ( $p < 0.001$ ), shuttle box model ( $p < 0.001$ ) and alternative running model ( $p < 0.001$ ). In the passive avoidance model SMAP administration 48 h after formation of the memory trace worsened retrieval of the memory 24 h later. On the other hand, blocking activity of SMAP with intra-cerebral administration of anti-SMAP antibodies to the rats prior to the learning session significantly accelerated and strengthened memory formation on the model of alternative running ( $p < 0.001$ ) and 2-lever operant discrimination with food reinforcement ( $p < 0.001$ ). Intra-cerebral administration of the anti-SMAP antibodies to the rats 24 h later brought to downregulation of nerve growth factor in the hippocampus ( $p < 0.001$ ) and left parietal cortex ( $p < 0.001$ ). The results of the undertaken studies give grounds to make proposal concerning the possible role for engagement of SMAP in the neurodegenerative pathologies accompanied with memory disturbances.

### Biography

Arif.A.Mekhtiev is a professor at the Academician Abdulla Garayev Institute of Physiology, Baku. His expertise includes dementia and memory cognition.

[12<sup>th</sup> Annual Congress on Dementia and Dementia Care](#) | June 15-16, 2020

**Citation:** Arif.A.Mekhtiev, Memory impairment in the rats induced by endogenous serotonin-modulating anticonsolidation protein, 15-16 June 2020, pp. 09