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Modulation of spatial memory and neurotransmitter markers in the prefrontal cortex by cholinergic and GABAergic neurons of the nucleus basalis magnocellularis

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Nearly all hypotheses of psychiatric disorders, including Alzheimer's disease, theorize that abnormal Prefrontal Cortex (PFC) function is a result of the dysregulation of the ascending neuromodulatory systems. The present study was designed to investigate the effects of selective lesions of cholinergic and GABAergic neurons (using immunotxins 192 IgG-saporin and GAT1-SAP, respectively) of the nucleus basalis magnocellularis (NBM), that projects to the PFC, on spatial memory and cholinergic and GABAergic markers in the PFC in rats. Immunohistochemical evaluation revealed that: microinjection of 192-IgG saporin into the NBM significantly reduces number of AChE-sensitive neurons, but do not modulates GABAergic transmission in PFC; microinjection of GAT1-SAP into NBM significantly reduces number of $GABA_A$ receptors immunostaining and do not reduce number of AChE-sensitive neurons in the PFC. In the present study acquisition of a spatial memory and its subsequent recall was assessed in the water maze. The behavioral experiments indicate that rats with NBM GABAergic lesions exhibited learning deficit. In marked contrast from GABAergic neurons, behavioral analysis revealed that NBM cholinergic lesions affect only long-term spatial memory which was assessed 24 h later after training and do not affect learning process and short-term spatial memory assessed 30 min later after training, The results for the first time demonstrate a functional dissociation between basalocortical cholinergic and GABAergic systems in aspects of cognitive function: selective lesions of NBM cholinergic and GABAergic neurons differentially affect neurotransmitters activity in the PFC, learning process, short- and long-term spatial memory.

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