Targeting Topics: Recent Scientific References

(continued from page 3)

Increased phencyclidine-induced hyperactivity following cortical cholinergic denervation.

Mattsson A, Lindqvist E, Ogren SO, Olson L

Neuroreport 16(16):1815-1819, 2005

A potential contribution to schizophrenia is altered cholinergic function. The authors investigated how lesioning cholinergic corticopetal projections might affect glutaminergic activity. Rats were injected with 0.134 μg of 192-IgG-SAP (Cat. #IT-01) into the nucleus basalis magnocellularis. The authors found that cholinergic lesioning of the neocortex led to enhanced sensitivity to phencyclidine, which has been shown to induce clinical symptoms similar to those of schizophrenia. These data suggest that glutaminergic dysfunction may be relevant to schizophrenia pathophysiology.

Selective loss of basal forebrain cholinergic neurons by 192 IgG-saporin is associated with decreased phosphorylation of Ser glycogen synthase kinase-3beta.

Hawkes C, Jhamandas JH, Kar S

J Neurochem 95(1):263-272, 2005

Glycogen synthase kinase-3ß (GSK-3ß) is an enzyme involved in a variety of biological events. In this study the authors examined the potential role of GSK-3ß in degeneration of basal forebrain cholinergic neurons. Rats were treated with 2.0 μg per ventricle injections of 192-IgG-SAP (Cat. #IT-01), then GSK-3ß and other cholinergic marker levels were assayed. The results indicate that increased GSK-3ß activity can provide some protection from 192-IgG-SAP-induced degeneration of forebrain cholinergic neurons.

Insomnia following hypocretin2-saporin lesions of the substantia nigra.

Gerashchenko D, Blanco-Centurion CA, Miller JD, Shiromani PJ

Neuroscience [epub Nov 10], 2005

It is known that orexin (also known as hypocretin) is involved in waking. Here the authors investigate which regions of major arousal areas might be responsible for the changes in sleep-wake architecture upon treatment with orexin-SAP (Cat. #IT-20). Bilateral injection of orexin-SAP into the ventral tegmental (VT) area and the substantia nigra (SN; 92 and 184 ng/µl, 0.25 µl in the VT area and 0.5 µl in the SN) of rats induced insomnia, as well as hyperactivity and stereotypic movements. The results suggest that motor activity is under inhibitory control of the SN.

The septohippocampal cholinergic system and spatial working memory in the Morris water maze.

Frielingsdorf H, Thal LJ, Pizzo DP

Behav Brain Res [epub Dec 2], 2005

The authors examined whether an optimized Morris water maze test could reveal the role of the septohippocampal cholinergic system in spatial working memory. Rats were treated with bilateral 75-ng injections of 192-IgG-SAP (Cat. #IT-01) followed by acquisition of the water maze task, and two independent phases of working memory testing. Test optimization was followed by icv infusion of nerve growth factor in unlesioned animals. The data demonstrate that working memory impairments cannot be revealed by the Morris water maze test.

Cholinergic septo-hippocampal innervation is required for trace eyeblink classical conditioning.

Fontan-Lozano A, Troncoso J, Munera A, Carrion AM, Delgado-Garcia JM

Learn Mem 12(6):557-563, 2005

Classical conditioning of eyeblink responses can be used to evaluate cognitive deficits. The authors lesioned the medial septum/diagonal band of rats with 200 ng of 192-IgG-SAP (Cat. #IT-01), then examined classical and instrumental conditioning paradigms. Lesioned animals displayed a deficit in the acquisition, but not retrieval of eyeblink classical conditioning. The deficit was reversed by carbachol, a cholinergic muscarinic agonist, suggesting a role for the muscarinic system in the acquisition of new motor abilities.

Upcoming Events

Spring Brain Conference
March 15-18, 2006
Sedona, Arizona

Experimental Biology
April 2-4, 2006
San Francisco, CA
Booth #837