

Targeting Topics: Recent Scientific References

(continued from page 3)

in the lesioned animals. The data indicate that AR agonists may reduce sensitization by activating nNOS fibers in the superficial dorsal horn.

Noradrenergic inputs to the bed nucleus of the stria terminalis and paraventricular nucleus of the hypothalamus underlie hypothalamic-pituitary-adrenal axis but not hypophagic or conditioned avoidance responses to systemic yohimbine.

Banihashemi L, Rinaman L
J Neurosci 26(44):11442-11453, 2006.

Yohimbine (YO) is an α_2 adrenoceptor antagonist that increases transmitter release from adrenergic/noradrenergic (NA) neurons. The authors investigated whether NA inputs to the bed nucleus of the stria terminalis (BNST) were required for YO effects. After receiving 11 ng of anti-DBH-SAP (Cat. #IT-03) in the left and right BNST, rats displayed a marked decrease in the hypothalamic-pituitary-adrenal axis in response to YO administration.

Lack of localization of 5-HT₆ receptors on cholinergic neurons: implication of multiple neurotransmitter systems in 5-HT₆ receptor-mediated acetylcholine release.

Marcos B, Gil-Bea FJ, Hirst WD, Garcia-Alloza M, Ramirez MJ
Eur J Neurosci 24(5):1299-1306, 2006.

The authors investigated a potential link between 5-HT₆ receptors, cholinergic activity, and learning. After 0.067 μ g of 192-IgG-SAP (Cat. #IT-01) was injected into each hemisphere of the nucleus basalis magnocellularis in the basal forebrain of rats, 5-HT₆ receptor mRNA and protein expression were measured. Results demonstrate the involvement of multiple neurotransmitter systems in neurochemical actions following 5-HT₆ receptor blockade.

Please visit www.ATSBio.com to see a complete list of references.

Long-term effects of immunotoxic cholinergic lesions in the septum on acquisition of the cone-field task and noncognitive measures in rats.

van der Staay FJ, Bouger P, Lehmann O, Lazarus C, Cosquer B, Koenig J, Stump V, Cassel JC
Hippocampus 16(12):1061-1079, 2006.

192-IgG-SAP (Cat. #IT-01) has been used to make extremely specific lesions in the septohippocampal cholinergic system of the brain. The specificity of these lesions is allowing researchers to more accurately map the involvement of the septohippocampal cholinergic system in spatial learning and memory. Here, rats received 0.8 μ g of 192-IgG-SAP in the medial septum and the vertical limb of diagonal band of Broca. Lesioned animals only exhibited deficits in attentional learning.



Inhibition within the nucleus tractus solitarius (NTS) ameliorates environmental exploration deficits due to cerebellum lesions in an animal model for autism.

Walker BR, Diefenbach KS, Parikh TN
Behav Brain Res [Epub Sep 11], 2006.

In this work the authors use environmental exploration deficits in rats as a model for autism. Animals received 2 μ g of either OX7-SAP (Cat. #IT-02) or 192-IgG-SAP (Cat. #IT-01) into each ventricle. Only the OX7-SAP-treated rats displayed a reduction in exploration behavior, and the anticonvulsant

muscimol restored exploration behavior to control levels. This system may have use in controlling behavior deficits seen in autism.

Up-regulation of cation-independent mannose 6-phosphate receptor and endosomal-lysosomal markers in surviving neurons after 192-IgG-saporin administrations into the adult rat brain.

Hawkes C, Kabogo D, Amritraj A, Kar S
Am J Pathol 169(4):1140-1154, 2006.

The cation-independent mannose 6-phosphate receptor (CI-MPR) plays a major role in the endosomal-lysosomal (EL) system. One of the tasks carried out by the EL system is clearance of abnormal proteins after injury. By administering 2.0 μ g bilateral injections of 192-IgG-SAP (Cat. #IT-01) to rats, the researchers were able to increase CI-MPR expression levels, as well as other EL markers in response to the lesion. The up-regulation of EL components suggests that the EL system may be able to repair neuronal abnormalities induced by injury.

Basal forebrain cholinergic lesions reduce heat shock protein 72 response but not pathology induced by the NMDA antagonist MK-801 in the rat cingulate cortex.

Willis CL, Ray DE, Marshall H, Elliot G, Evans JG, Kind CN
Neurosci Lett 407(2):112-117, 2006.

The NMDA receptor antagonist MK-801 may have use in establishing a model for schizophrenia. The mechanism by which cortical neurons are damaged by these antagonists is unknown. The authors tested the theory that cholinergic hyperstimulation of cingulate neurons is involved by administering 80 ng of 192-IgG-SAP (Cat. #IT-01) unilaterally to rats. The results indicate that although cholinergic neurons are involved in the heat shock response to MK-801, the pathological effects follow a different pathway.

(continued on page 6)