Targeting Topics: Recent Scientific References

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investigate the role of cholinergic MSDB neuron projections to the hippocampus, since many of these neurons express estrogen receptors. The data suggest that septo-hippocampal cholinergic neurons are involved in mediating estrogen effects on the hippocampus.

Immunotoxin lesion of hypothalamically projecting norepinephrine and epinephrine neurons differentially affects circadian and stressor-stimulated corticosterone secretion.

Ritter S, Watts AG, Dinh TT, Sanchez-Watts G, Pedrow C
Endocrinology 144(4):1357-1367, 2003.

Hindbrain norepinephrine (NE) and epinephrine (E) neurons are important in the distribution of internal sensory signals. Injecting 42 ng of anti-DBH-SAP (Cat. #IT-12) into the paraventricular nucleus of rat hypothalamus, the authors were able to specifically destroy NE and E neurons. This study revealed the contribution of NE/E afferents to hypothalamo-pituitary-adrenal activation during stress and confirmed that NE and E neurons are required for specific stress responses.

A role for the basal forebrain cholinergic system in estrogen-induced disinhibition of hippocampal pyramidal cells.

Rudick CN, Gibbs RB, Woolley CS
J Neurosci 23(11):4479-4490, 2003.

Estrogen plays a strong regulatory role in control of synaptic input to the hippocampus of female rats. Injection of 0.22 µg of 192-Saporin (Cat. #IT-01) directly into the medial septum eliminated NGFr-positive cholinergic neurons of the basal forebrain, producing evidence that estrogen-induced disinhibition is partially dependent on these neurons. GABAergic synapses were also found to be involved in this system.

Breathing: Rhythmicity, plasticity, chemosensitivity.

Feldman JL, Mitchell GS, Nattie EE
Annu Rev Neurosci 26:239-66, 2003.

Recent research has indicated that specific areas of the brain exert control over several aspects of breathing, such as rhythm generation, reaction to hypoxia, and regulation of carbon dioxide levels and pH. This review covers many of the latest advances, some of which utilize SP-SAP (Cat. #IT-07) and anti-SERT-SAP (Cat. #IT-23). The use of these targeted toxins allows altered breathing behavior through elimination of very specific cell populations.

192 IgG-saporin lesions to the nucleus basalis magnocellularis (nBM) disrupt acquisition of learning set formation.

Bailey AM, Rudisill ML, Hoof EJ, Loving ML
Brain Res 969:147-159, 2003.

Previous studies by Bailey and others have used quisqualic acid to lesion the nucleus basalis (nBM) in order to understand Alzheimer's disease. Injections of 75 ng of 192-Saporin (Cat. #IT-01) were made into each of four sites in the rat nBM. Behavioral tests showed initial learning set deficits followed by recovery, whereas with quisqualic acid lesions, the deficits were profound. The authors conclude noncholinergic neurons are involved in learning set formation.

Distinct roles of P2X receptors in modulating glutamate release at different primary sensory synapses in rat spinal cord.

Nakatsuka T, Tsuzuki K, Ling JX, Sonobe H, Gu JG
J Neurophysiol 89:3243-3252, 2003.

P2X receptors are important modulating neurons in the spinal cord. These authors used IB4-SAP (Cat. #IT-10) to target a neuronal subset, those neurons expressing $P2X_3$ receptors. 2 µg of IB4-SAP were injected directly into the sciatic nerve on one side. Histological examination showed efficient removal of IB4 and $P2X_3$-staining ipsilaterally in the dorsal horn outer laminae. Behavioral experiments showed intact modulation of glutamate release in the absence of $P2X_3$-positive neurons, indicating involvement by other P2X neurons.

Effects of septal grafts on acetylcholine release from rat hippocampus after 192 IgG-saporin lesion.

Hilgert M, Hartmann J, Loffelholz K, Jeltsch H, Cassel JC, Klein J
Neurochem Res 28:467-472, 2003.

A model for transplantation efficacy was created using injections of 400 ng each into the vertical limb of the rat diagonal band of Broca and the medial septum for the specific removal of cholinergic neurons. Thirteen months after lesioning, sham-operated animals had measured acetylcholine release at 20% of control. 192-Saporin (Cat. #IT-01)-lesioned animals were transplanted with fetal septal cells 15 days after lesioning. Thirteen months later, their septal level of acetylcholine release was near normal (71%) of controls. A serotonin uptake inhibitor briefly stimulated acetylcholine release similar to sham control animals.