Targeting Topics: Recent Scientific References

(continued from page 3)

Regulation of sympathetic tone and arterial pressure by rostral ventrolateral medulla after depletion of C1 cells in rat

Schreihofer AM, Stornetta RL, Guyenet PG
J Physiol 529(1):221-236, 2000.

The rostral ventrolateral medulla (RVLM) controls and maintains basal sympathetic vasomotor tone, and is also vital to many sympathetic reflexes. Sympathetic nerve activity and arterial pressure correlate with the C1 adrenergic neurons in the RVLM, but there are also non-catecholaminergic neurons present. Schreihofer *et al.* used anti-DBH-SAP (Cat. # IT-03) to eliminate the C1 cells of the RVLM to investigate the non-catecholaminergic neuron contribution to vasomotor tone. Their data indicate C1 cells are necessary for full expression of sympathoexcitatory responses generated by the RVLM.

Neuronal lesioning with axonally transported toxins

Wiley RG, Kline IV RH
J Neurosci Meth 103:73-82, 2000.

Functional neuroanatomy studies have long utilized lesioning. Given the complexity of heterogeneous neuron populations, conventional lesioning methods have proved relatively crude and have provided limited information. Wiley and Kline detail some of the immunotoxins utilizing saporin as well as neuropeptide-saporin conjugates that have found use in recent neurological research. These products include SP-SAP (Cat. #IT-07), which eliminates neurons expressing the neurokinin 1 receptor, 192-Saporin (Cat. #IT-01), which eliminates neurons expressing the p75 receptor in rats, anti-DBH-SAP (Cat #IT-03), which destroys noradrenergic and adrenergic neurons, and OX7-SAP (Cat. #IT-02), which is a suicide transport agent targeting all rat neurons. The authors also discuss some of the protocols and methods utilized with these compounds.

Non-linear cortico-cortical interactions modulated by cholinergic afferences from the rat basal forebrain

Villa AEP, Tetko IV, Dutoit P, Vantini G
BioSystems 58:219-228, 2000.

Elimination of the cholinergic neurons of the basal forebrain (BF) is an excellent model for some aspects of Alzheimer's Disease (AD). 192-Saporin (Cat. # IT-01) is a very effective tool for elimination of cholinergic neurons in the BF. Villa *et al.* investigate whether field potential changes in the brains of lesioned animals mimic changes observed in the brains of human AD patients. The data presented indicate depletion of cholinergic neurons from the BF of both rats and humans produces similar changes in field potential.

Antinociceptive action of nitrous oxide is mediated by stimulation of noradrenergic neurons in the brainstem and activation of α_{2B} adrenoceptors

Sawamura S, Kingery WS, Davies MF, Agashe GS, Clark JD, Kobilka BK, Hashimoto T, Maze M
J Neurosci 20(24):9242-9251, 2000.

Nitrous oxide has been used extensively in surgical anesthesia for more than 150 years, but the molecular mechanism of action has not yet been defined. Sawamura *et al.* investigate whether noradrenergic neurons in the brainstem are involved in the analgesic action of nitrous oxide. The authors injected rats with anti-DBH-SAP (Cat. #IT-03) to destroy pontine noradrenergic neurons. The treated rats demonstrated the usual sedative effects of nitrous oxide, but the analgesic effects were reduced or blocked. Coupled with data from null mice for the α_{2B} adrenoceptor, the data indicates that α_2 adrenoceptor subtypes and ligands are involved in the analgesic but not sedative effects of nitrous oxide.

Cortical cholinergic inputs mediate processing capacity: Effects of 192 IgG-saporin-induced lesions on olfactory span performance

Turchi J, Sarter M
Eur J Neurosci 12:4505-4514, 2000.

Many experiments support the theory that the basal forebrain (BF) is involved in major aspects of attention that influence learning and memory. Elimination of cholinergic neurons in the BF by 192-Saporin (Cat. #IT-01) has been shown to reduce the ability of rats to perform a task while paying attention to more than one thing. The authors tested the treated rat's ability to identify one olfactory stimuli from an increasing number of such stimuli. While the performance of the treated rats returned to control levels within four weeks post-lesion, their performance reflected increased time between tests. These data indicate that cholinergic neurons of the BF play a role in attentional capacities.

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