Targeting Topics: Recent Scientific References

(continued from page 3)

Facilitation of dopamine-mediated locomotor activity in adult rats following cholinergic denervation

Mattsson A, Ögren SO, Olson L
Exp Neurology 174:96-108, 2002.

It has been hypothesized that hyperactivity of dopaminergic systems is involved in schizophrenia. The authors examine the effect of central cholinergic denervation on dopamine-mediated functions. Adult rats received bilateral injections of 2.5 µg of 192-Saporin (Cat. #IT-01) into the lateral ventricles. Neonatal rats received 400 ng 192-Saporin in bilateral i.c.v. injections. The results indicate that severe cholinergic deficiencies in the forebrain can lead to dopaminergic overfunction, one of the possible mechanisms in schizophrenia.

Isoflurane and nociception: Spinal alpha2A adrenoceptors mediate antinociception while supraspinal alpha1 adrenoceptors mediate pronociception.

Kingery WS, Agashe GS, Guo TZ, Sawamura S, Davies MF, Clark JD, Kobilka BK, Maze M
Anesthesiol 96:367-74, 2002.

The authors injected 3 µg/3 µl of anti-DBH-SAP (Cat. #IT-03) into the lateral ventricle of rats to determine whether noradrenergic brainstem nuclei and descending spinal pathways are responsible for the antinociceptive actions of isoflurane. The results indicate that isoflurane modulates nociception by as many as three mechanisms, utilizing various combinations of noradrenergic neurons, adrenoceptors, and descending spinal pathways.

Cytotoxic targeting of isolectin IB4-binding sensory neurons

Vulchanova L, Olson TH, Stone LS, Riedl MS, Elde R, Honda CN.
Neurosci 108(1):143-55, 2001.

Vulchanova *et al.* examine the role of IB4-binding neurons in nociception. IB4-SAP (Cat. #IT-10) was injected into rats (2 µg in left sciatic nerve). The resulting ablation of IB4-binding neurons provides evidence for their role in nociceptive processing and demonstrates a rapid compensatory response to signalling of acute pain.

Lesions of the nucleus basalis magnocellularis induced by 192 IgG-saporin block memory enhancement with posttraining norepinephrine in the basolateral amygdala

Power AE, Thal LJ, McGaugh JL
PNAS 99(4):2315-2319, 2002.

There is evidence that memory consolidation (retention) can be modulated by drugs and stress hormones acting in the basolateral amygdala (BLA). The BLA sends projections to the nucleus basalis magnocellularis (NBM), which in turn sends cholinergic projections to the neocortex. The authors used 100 ng bilateral infusions of 192-Saporin (Cat. #IT-01) in 500 nl 0.1 M PBS to investigate whether lesions of the cholinergic NBM projections affect BLA modulation of memory. 192-Saporin lesions blocked memory enhancement normally induced by norepinephrine infusions into the BLA. This finding suggests NBM-cortex projections may mediate BLA modulation of memory storage or processing in the neocortex.

Cholinergic and noncholinergic septal neurons modulate strategy selection in spatial learning

Cahill JFX, Baxter MG
Eur J Neurosci 14:1856-1864, 2001.

The authors compared ibotenic acid (IA)-treated rats with those injected with 45 ng and 30 ng of 192-Saporin (Cat. #IT-01) into two separate coordinates of the medial septum/vertical limb of the diagonal band (MS/VDB) to investigate the role of basal forebrain projections in modulating strategy selection in spatial learning. While rats with IA lesions in the MS/VDB demonstrated significant disruption of the learning process, the 192-Saporin-lesioned rats did not show this effect.

Please visit our website (www.ATSbio.com) to see a complete list of references.