Targeting Topics: Recent Scientific References

Reviewed by Matthew Kohls

A putative flip-flop switch for control of REM sleep.
Lu J, Sherman D, Devor M, Saper CB
Nature 441(1):589-594, 2006.

The authors propose a REM sleep regulatory system that involves GABA-ergic and glutaminergic neurons in the mesopontine tegmentum. Two μl of 0.1% orexin-SAP (Cat. #IT-20) was injected into the medial medullary reticular formation of rats. The results suggest the sharp transitions into and out of REM sleep are controlled by recipro-cal interactions between GABAergic REM-off and REM-on neuronal populations.

Antisocial and seizure susceptibility phenotypes in an animal model of epilepsy are normalized by impairment of brain corticotropin-releasing factor.
Turner LH, Lim CE, Heinrichs SC
Epilepsy Behav [Epub Oct 3], 2006.

There appears to be an inverse relationship between seizure susceptibility and social interaction. This work examines the role that CRF may play in this system. 2.5 μg of CRF-SAP (Cat. #IT-13) was administered to the lateral ventricle of rats, and the lesioned animals were assessed in terms of social investigation times as well as handling-induced seizures. The results support the involvement of CRF systems in facilitating evoked seizures and suppression of social activity.

Effects of hypocretin-1 in 192-IgG-saporin-lesioned rats.
Blanco-Centurion CA, Shiromani A, Winston E, Shiromani PJ
Eur J Neurosci 24(7):2084-2088., 2006.

The basal forebrain is a major arousal center. Using 6 μg of 192-IgG-SAP (Cat. #IT-01) injected into the lateral ventricle of rats, the role of non-cholinergic neurons in the basal forebrain was investigated. Administration of orexin, also known as hypocretin, to lesioned animals produced sleep/wake patterns identical to non-lesioned animals. The results indicate that non-cholinergic neurons in the basal forebrain are sufficient to promote arousal in response to orexin.

Ablation of NK(1) receptor bearing neurons in the nucleus of the solitary tract blunts cardiovascular reflexes in awake rats.
Abdala AP, Schoorlemmer GH, Colombari E
Brain Res 1119(1):165-173, 2006.

Cardiovascular function is largely controlled by the nucleus of the tractus solitarius (NTS). This work focuses on the baroreflex, cardiopulmonary chemoreflex, and arterial chemoreflex. Rats were injected with either 20 nl of 2 μM SP-SAP (Cat. #IT-07) into the subpostremal NTS, or 200 nl into the subpostremal and commissural NTS. Saporin (Cat. #PR-01) was used as a control. It was established that NK-1 receptor-expressing neurons in the NTS are critical for these reflexes.

Selective depletion of cortical noradrenalin by anti-dopamine beta-hydroxylase-saporin impairs attentional function and enhances the effects of guanfacine in the rat.
Milstein JA, Lehmann O, Theobald DE, Dalley JW, Robbins TW
Psychopharmacology (Berl) 190(1):51-63, 2007.

The authors examined the effect of cortical noradrenalin depletion on a reaction time task. Rats received 0.2 μg-intracortical infusions of anti-DBH-SAP (Cat. #IT-03), then were trained in a reaction time task. The effect of guanfacine, a selective α-2 adrenergic agonist was also tested in these animals. Lesioned rats were not impaired on the baseline task, but were slower and less accurate during high rate conditions. Guanfacine only affected the lesioned animals.

Spinal NK-1 receptor expressing neurons mediate opioid-induced hyperalgesia and antinociceptive tolerance via activation of descending pathways.
Vera-Portocarrero LP, Zhang ET, King T, Ossipov MH, Vanderah TW, Lai J, Porreca F
Pain [Epub Nov 21], 2006.

Administration of opioids can induce hyperalgesia in humans and other mammals. In this work the authors examined the role of NK-1 receptor-expressing neurons in the spinal dorsal horn during a hyperalgesic condition not induced by tissue injury. Five μl of 10 μM SP-SAP (Cat. #IT-07) was injected into the intrathecal space of rats. Saporin (Cat. #PR-01) was used as a control. Data from the lesioned animals indicate that NK-1 receptor-expressing neurons play a critical role in this hyperalgesic circuit.

Neuronal nitric oxide synthase is upregulated in a subset of primary sensory afferents after nerve injury which are necessary for analgesia from alpha2-adrenoceptor stimulation.
Ma W, Eisenach JC
Brain Res [Epub Nov 16], 2006.

Peripheral nerve injury resulting in neuropathic pain often responds poorly to opioid treatment. α2-adrenoreceptor (AR) agonists, however, perform better after this type of injury. After a spinal nerve ligation, rats were treated with a 0.6 μg-intrathecal injection of 192-IgG-SAP (Cat. #IT-01). The increase of neuronal nitric oxide synthase (nNOS) caused by spinal ligation was abolished

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