Targeting Topics: Recent Scientific References

Reviewed by Matthew Kohls

Targeted ablation of cholinergic interneurons in the dorsolateral striatum produces behavioral manifestations of Tourette syndrome.

Xu M, Kobets A, Du JC, Lennington J, Li L, Banasr M, Duman RS, Vaccarino FM, DiLeone RJ, Pittenger C.

Proc Natl Acad Sci U S A 112(3):893-898, 2015.

Postmortem studies of Tourette syndrome patients has revealed a reduction in the number of specific striatal interneurons. The authors explored the hypothesis that this neuronal deficit is enough to produce the symptoms of Tourette syndrome in mice. Animals received 90-ng injections of Anti-ChAT-SAP (Cat. #IT-42) into the striatum. Rabbit IgG-SAP (Cat. #IT-35) was used as a control. The data suggest that loss of the striatal interneurons is enough to produce some, but not all, of the symptoms caused by Tourette syndrome.

Role of striatal cholinergic interneurons in set-shifting in the rat.

Aoki S, Liu AW, Zucca A, Zucca S, Wickens JR.

J Neurosci 35(25):9424-9431, 2015.

The authors examined the role that cholinergic interneurons in the striatum play in a process called strategy set-shifting, where an attentional shift is required. Rats received bilateral injections of Anti-ChAT-SAP (Cat. #IT-42) into either the dorsomedial striatum or ventral striatum (500 ng total). Initial task learning was unaffected by either lesion. Lesioned animals displayed set-shifting deficits, and the deficit characteristics depended on the location of the lesion.

A central role for spinal dorsal horn neurons that express neurokinin-1 receptors in chronic itch.

Akiyama T, Nguyen T, Curtis E, Nishida K, Devireddy J, Delahanty J, Carstens MI, Carstens E.

Pain 156(7):1240-1246, 2015.

Chronic itch is caused by increased sensitivity of itch-signaling pathways. It can be generated by normally itchy stimuli (hyperknesis) and by normally non-itchy light touch (alloknesis). The authors used an ovalbumin-induced atopic dermatitis model to study chronic itch in mice. The mice received 400-ng intrathecal injections of Bombesin-SAP (Cat. #IT-40), SSP-SAP (Cat. #IT-11), or the control Blank-SAP (Cat. #IT-21). While Bombesin-SAP significantly attenuated hyperknesis, it had no effect on spontaneous scratching or alloknesis. SP-SAP reduced all behavioral signs of chronic itch.

Neurokinin 3 Receptor-Expressing Neurons in the Median Preoptic Nucleus Modulate Heat-Dissipation Effectors in the Female Rat.

Mittelman-Smith MA, Krajewski-Hall SJ, McMullen NT, Rance NE.

Endocrinology 156(7):2552-2562, 2015.

Kisspeptin and Neurokinin B (NKB) expression in the infundibular, or arcuate, nucleus is increased after menopause. Here the authors investigate whether KNDy (kisspeptin, NKB, and dynorphin expressing) neurons are able to influence cutaneous vasodilation through Neurokinin 3 (NK3)-expressing projections from the median preoptic nucleus (MnPO). Rats received two 10-ng injections of NK3-SAP (Cat. #IT-63) into the MnPO. Blank-SAP (Cat. #IT-21) was used as a control. The data indicate that NK3-expressing neurons in the MnPO facilitate vasodilation.

Hindbrain catecholamine neurons activate orexin neurons during systemic glucoprivation in male rats.

Li AJ, Wang Q, Elsarelli MM, Brown RL, Ritter S.

Endocrinology Epub2015.

Norepinephrine and epinephrine-secreting catecholamine neurons are strong stimulators of food intake. The authors investigated the interaction between these catecholamine neurons and orexin neurons in the perifornical lateral hypothalamus (PeFLH), which are known to be involved with the stimulation of food intake, increased arousal, and behavioral activation. Rats received 82-ng injections of Anti-DBH-SAP (Cat. #IT-03) into the PeFLH terminal field in order to lesion catecholamine neurons. Saporin (Cat. #PR-01) was used as a control. Assessment of food intake in response to 2-deoxy-D-glucose, as well as selective catecholamine activation, indicated that orexin neuron activation may be involved in glucoprivic appetite responses.

Orexin-A Enhances Feeding in Male Rats by Activating Hindbrain Catecholamine Neurons.

Li AJ, Wang Q, Davis H, Wang R, Ritter S.

Am J Physiol Regul Integr Comp Physiol Epub2015.

Although administration of orexin, norepinephrine, and epinephrine all induce significantly increased food intake, the potential interaction between the networks affected by these molecules has not been studied. In this work, the authors investigate the hypothesis that orexin neurons may stimulate feeding through the activation of catecholamine neurons. Rats received 82-ng injections of Anti-DBH-SAP (Cat. #IT-03) into the hypothalamus in order to lesion hypothalamically-projecting catecholamine neurons. Saporin (Cat. #PR-01) was used as a control. While the normal response to orexin A is increased food intake, lesioned animals did not display this response, indicating that catecholamine neurons are necessary for orexin modulation of food intake.

Selective optogenetic stimulation of the retrotrapezoid nucleus in sleeping rats activates breathing without changing blood pressure or causing arousal or sighs.

Burke PG, Kanbar R, Viar KE, Stornetta RL, Guyenet PG.

J Appl Physiol (1985) 118(12):1491-1501, 2015.

Hypoxia and hypercapnia both play roles in the activation of normal breathing. If either one is severe enough, arousal will also occur. The authors looked to better define the CNS pathways utilized by hypoxia and hypercapnia, as well as the pathways responsible for activation of arousal due to these conditions. The authors used optogenetic activation of the retrotrapezoid nucleus and C1 and A5 catecholaminergic

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