Targeting Topics: Recent Scientific References

(continued from page 3)

Leptin-sensitive neurons in the arcuate nucleus integrate activity and temperature circadian rhythms and anticipatory responses to food restriction.

Wiater MF, Li AJ, Dinh TT, Jansen HT, Ritter S.

Am J Physiol Regul Integr Comp Physiol Epub2013.

The arcuate nucleus (Arc) of the hypothalamus is known to participate in the regulation of feeding, adiposity, and leptin-dependent metabolism. The authors examined the role of leptin-receptive neurons in locomotor and temperature rhythms. Rats received four bilateral injections of Leptin-SAP (Cat. #IT-47) into the Arc; Blank-SAP (Cat. #IT-21) was used as a control. The lesion affected learning connected to light cycles, but not learning connected to food schedules, suggesting a mechanism for internal desynchrony that might play a role in obesity and other metabolic disorders.

C1 neurons: the body's EMTs.

Guyenet PG, Stornetta RL, Bochorishvili G, Depuy SD, Burke PG, Abbott SB.

Am J Physiol Regul Integr Comp Physiol 305(3):R187-204, 2013.

Although mainly known for their involvement in the control of arterial pressure, C1 neurons are also suspected to participate in numerous other physiological processes such as neuroendocrine response, glucose homeostasis, food consumption, and others. This review discusses the role of these neurons as 'emergency medical technicians' – cells that produce and modulate physiological survival responses to acute physical stress. The use of Anti-DBH-SAP (Cat. #IT-03) to delineate C1 neurons in the rostral ventrolateral aspect of the medulla oblongata is discussed.

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Loss of neurons in rostral ventromedial medulla that express neurokinin-1 receptors decreases the development of hyperalgesia.

Khasabov SG, Simone DA.

Neuroscience 250C:151-165, 2013.

Previous data has indicated that neurokinin-1 receptors are located on ON cells in the rostral ventromedial medulla (RVM). ON cells are considered pronociceptive because noxious stimulation is stimulatory. In this work the authors eliminated ON cells using 0.3-µl injections of 1 µM SSP-SAP (Cat. #IT-11) into the left and right side of the RVM. Blank-SAP (Cat. #IT-21) was used as a control. SSP-SAP treatment did not change mechanical or heat withdrawal responses, or change morphine-induced analgesia. A significant reduction in the duration of nocifensive behaviors induced by various hyperalgesic stimulators indicated that these neurons are involved in pain facilitation rather than modulation.

Selective Immunotoxic Lesions of Basal Forebrain Cholinergic Neurons: Twenty Years of Research and New Directions.

Baxter MG, Bucci DJ.

Behav Neurosci Epub2013.

This review covers twenty years of basal forebrain cholinergic lesioning. The initial use of 192-IgG-SAP (Cat. #IT-01) is discussed, as well as other immunotoxins such as GAT-1-SAP (Cat. #IT-32) and OX7-SAP (Cat. #IT-02). The findings generated by the use of 192-IgG-SAP and how those data have helped forward the understanding of how the cholinergic system functions in the basal forebrain are detailed. The authors also discuss new directions in the field.

Noggin and Sonic hedgehog are involved in compensatory changes within the motoneuron-depleted mouse spinal cord.

Gulino R, Gulisano M.

J Neurol Sci 332(1-2):102-109, 2013.

Noggin (NOG) and Sonic hedgehog (Shh) are both involved in the generation and organization of neural tissues. In order to clarify the role of these two proteins in the regulation of neurogenesis and/or neuroplasticity the authors used a motoneuron depletion model in the mouse spinal cord. 3 µg of CTB-SAP (Cat. #IT-14) was injected into each of the medial and lateral gastrocnemius muscles and the expression of NOG and Shh were monitored. Motor performance also correlated with NOG and Shh levels, indicating that these proteins could play roles in regeneration and functional restoration.

Cortical Metabolic Deficits in a Rat Model of Cholinergic Basal Forebrain Degeneration.

Gelfo F, Petrosini L, Graziano A, De Bartolo P, Burello L, Vitale E, Polverino A, Iuliano A, Sorrentino G, Mandolesi L.

Neurochem Res Epub2013.

In this work the authors investigated the connection between cholinergic depletion caused by conditions such as Alzheimer's disease and cerebral energy metabolism deficits. Rats received a 0.4-µg injection of 192-IgG-SAP (Cat. #IT-01) into the nucleus basalis magnocellularis. Neuronal metabolic activity was measured by assaying cytochrome oxidase (CO) activity. The unilateral injection produced a bilateral deficit in CO activity throughout the cortex, and the front and parietal cortices showed CO deficits before the lesion was complete. The data suggest a link between cholinergic hypofunctionality and metabolic deficit.