Targeting Topics: Recent Scientific References

Reviewed by Matthew Kohls

Endogenous opioid activity in the anterior cingulate cortex is required for relief of pain.

Navratilova E, Xie JY, Meske D, Qu C, Morimura K, Okun A, Arakawa N, Ossipov M, Fields HL, Porreca F

J Neurosci 35(18): 7264-7271, 2015.

There are a number of neuronal circuits involved in the processing of pain, including those that control somatosensory, affective, and cognitive aspects of pain perception. Opioid signaling in the anterior cingulate cortex (ACC) plays a part in pain modulation - this area has also been implicated in the encoding of pain aversiveness. In order to examine the neuronal mechanisms of pain relief and the following reward, the authors of this paper administered 48 ng of Dermorphin-SAP (Cat. #IT-12) into the rostral ACC of rats. Saporin (Cat. #PR-01) was used as a control. The results illuminate the opioid pathway during pain treatment, and the dependence of nucleus accumbens dopaminergic transmission on upstream ACC opioid circuits during pain processing.

proBDNF and p75NTR Control Excitability and Persistent Firing of Cortical Pyramidal Neurons.

Gibon J, Buckley SM, Unsain N, Kaartinen V, Seguela P, Barker PA

J Neurosci 35(26): 9741-9753, 2015.

Principal neurons in the entorhinal cortex (EC) display persistent firing (PF) during working-memory tasks. Much of the communication between the hippocampus and the neocortex passes through the EC, and the EC also receives some cholinergic input from the medial septum and diagonal band of Broca. In this work the authors investigated the role of pro-brain-derived neurotrophic factor (proBDNF) and the p75 receptor in excitability and PF in the EC. The authors propose the proBDNF/p75 system as a regulator for pyramidal neuron excitability and PF in the EC, preventing runaway activity. Some of the western blot and current-clamp data was generated using Anti-p75 (Cat. #AB-N01; no concentration information provided).

New versions of Orexin-SAP are now in development and production. If you are interested in testing Orexin-SAP and/or orexin receptor antibodies, please contact us.

Repeated Mu-Opioid Exposure Induces a Novel Form of the Hyperalgesic Priming Model for Transition to Chronic Pain.

Araldi D, Ferrari LF, Levine JD

J Neurosci 35(36): 12502-12517, 2015.

Repeated administration of mu-opioid receptor agonists can lead to persistent mechanical hyperalgesia. One current hypothesis is that a form of hyperalgesic priming is triggered by the repeated activation of these receptors. Classic hyperalgesic priming is associated with signaling via protein kinase Cε (PKε), which is mediated by isolectin-B4+ (IB4) nociceptors. In this work the authors eliminated the IB4+ nociceptors with a 3.2 µg intrathecal injection of recombinant IB4-SAP (Cat. #IT-10). The authors found that hyperalgesic priming induced through the use of DAMGO was dependent on protein kinase A activation rather than activation of PKε. This work demonstrates a novel model for hyperalgesic priming transitioning to chronic pain.

Roles of isolectin B4-binding afferents in colorectal mechanical nociception.

La JH, Feng B, Kaji K, Schwartz ES, Gebhart GF

Pain. 2015 Oct 5. [Epub ahead of print]

Primary afferent neurons are often classified as peptidergic or non-peptidergic. One characteristic of the non-peptidergic neurons is that they bind isolectin-B4. In the spinal cord these neurons terminate mainly in inner lamina II. Non-peptidergic neurons in the spinal cord have been found to be involved in various aspects of pain response. In this work the authors examined the role of non-peptidergic neurons in the viscerosensory system. Rats received 1.5 µg of intrathecal recombinant IB4-SAP (Cat. #IT-10) between the L5 and L6 vertebrae. Saporin (Cat. #PR-01) was used as a control. While IHC demonstrated that a majority of viscerosensory L6 colon DRG neurons are IB4+, they do not play a significant role in colorectal mechano-nociception.

Phenotypic and functional characterization of Bst+/- mouse retina.

Riazifar H, Sun G, Wang X, Rupp A, Vemaraju S, Ross-Cisneros FN, Lang RA, Sadun AA, Hattar S, Guan MX, Huang T

Dis Model Mech 8(8): 969-976, 2015.

The belly spot and tail mutant mouse strain was first reported on in 1976. Among other phenotypic changes, it carries ocular mutations including retinal colobomas, reduced retinal ganglion cells (RGCs), and axon misrouting. In order to assess the use of this strain as a murine model for stem cell therapies of retinal degenerative diseases the authors performed a number of characterization experiments including electron microscopy, immunohistochemistry, testing of circadian rhythms, and morphological studies. Some of the immunohistochemistry was done using Anti-Melanopsin (Cat. #AB-N38) at a 1:5000 dilution.

Disrupting spinal noradrenergic activation delays recovery of acute incision induced hypersensitivity and increases spinal glial activation in the rat.

Arora V, Morado-Urbina CE, Aschenbrenner CA, Hayashida KI, Wang F, Martin TJ, Eisenach JC, Peters CM

J Pain. 2015 Nov 3. pii: S1526-5900(15)00924-4 [Epub ahead of print]

A significant percentage of patients who undergo surgery experience prolonged clinically impactful pain, reducing the quality of life and physical function. Disruption of the descending noradrenergic input has been hypothesized to be important to the generation of this type of pain state. Using an acute incision model, the authors administered 5 µg ofAnti-DBH-SAP (Cat. #IT-03) to the L5-L6 interspace of rats. Mouse IgG-SAP (Cat. #IT-18) was used as a control. Lesioned animals demonstrated a

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