Targeting Topics: Recent Scientific References

(continued from page 3)

(Cat. #IT-01) into the nucleus basalis magnocellularis to examine this connection. Lesioned animals were more affected by propofol and phenobarbital than control animals. Some effects of halothane were also increased. The data indicate a role for acetylcholine in the brain in the response to general anesthesia.

PreBötzinger complex neurokinin-1 receptor-expressing neurons mediate opioid-induced respiratory depression.
Montandon G, Qin W, Liu H, Ren J, Greer JJ, Horner RL.

In order to identify the neurons involved with respiratory depression due to administration of opioids, some neurotransmission networks in the preBötzinger complex were locally manipulated. Among various techniques used to analyze the results was immunohistochemistry with anti-NK1r (Cat. #AB-N04, discontinued). Results show the preBötzinger complex is responsible for suppression of respiratory rate due to opioids.

Gene regulation in the rat prefrontal cortex after learning with or without cholinergic insult.
Paban V, Chambon C, Farioli F, Alescio-Lautier B.

Microarray technology was used to screen gene expression in a model of attention and memory deficit. Rats received bilateral injections of 192-IgG-SAP (Cat. #IT-01) into the medial septum and nucleus basalis magnocellularis (37.5 ng per side and 75 ng per side respectively). Gene expression in memory loss following the lesion was defined by one cluster related to cytoskeleton organization and proliferation, and glial and vascular remodeling. These are processes associated with brain repair after injury.

Human monoclonal antibodies to sialyl-Lewisα (CA19.9), as a target for cancer therapeutics. Human monoclonal antibodies were generated against CA19.9 and characterized using ELISA and flow cytometry. To assess internalization one antibody, 5B1, was combined with Hum-ZAP (Cat. #IT-22) and applied to CA19.9-expressing BxPC3 cells. The cytotoxicity of the 5B1-Hum-ZAP complex indicates that CA19.9 may be a target for cancer therapy.

In this work the authors investigated the use of a carbohydrate antigen, sialyl-Lewisα (CA19.9), as a target for cancer therapeutics. Human monoclonal antibodies were generated against CA19.9 and characterized using ELISA and flow cytometry. To assess internalization one antibody, 5B1, was combined with Hum-ZAP (Cat. #IT-22) and applied to CA19.9-expressing BxPC3 cells. The cytotoxicity of the 5B1-Hum-ZAP complex indicates that CA19.9 may be a target for cancer therapy.

Corticotropin-releasing factor critical for zebrafish camouflage behavior is regulated by light and sensitive to ethanol.
Wagle M, Mathur P, Guo S.

The authors used the hardwired camouflage response of zebrafish to explore neural circuit assembly and function. Corticotropin-releasing factor is a critical component of this pathway. Immunostaining, done with a CRF antibody (Cat. #AB-02) was part of the data that showed how both light exposure and ethanol affect the camouflage response. Understanding this system could provide a tool to further investigate the effect of alcohol on neural circuits.

Sézary syndrome cells overexpress syndecan-4 bearing distinct heparan sulfate moieties that suppress T-cell activation by binding DC-HIL and trapping TGF-β on the cell surface.
Chung JS, Shiue LH, Duvic M, Pandya A, Cruz PDJ, Ariizumi K.

Syndecan-4 (SD-4) is a transmembrane heparan sulfate proteoglycan. The Sézary syndrome (SS) subset of cutaneous T-cell lymphoma overexpresses distinct heparan sulfate moieties, giving the authors a specific target for these cells. Biotinylated DC-HIL-Fc (the extracellular domain of dendritic cell-associated heparan sulfate proteoglycan-integrin ligand fused to Fc of mouse IgG) was combined at a 1:1 molar ratio with streptavidin-ZAP (Cat. #IT-27). In vitro, this targeted toxin eliminated SS cells, preventing their proliferation and suggesting a method for SS treatment.

Selective depletion of Mac-1-expressing microglia in rat subventricular zone does not alter neurogenic response early after stroke.
Heldmann U, Mine Y, Kokaia Z, Ekdahl CT, Lindvall O.
Exp Neurol Epub Mar, 2011.

One result of ischemic stroke is migration of newly formed neuroblasts into the injured area from the subventricular zone (SVZ). The authors investigated the role of microglia, which also accumulate in the SVZ after stroke, in this process. Rats received 5-µg or 10-µg intracerebroventricular injections of Mac-1-SAP (Cat. #IT-33) with varying schedules as to injection and sacrifice. The data indicate that the presence of microglia after stroke does not affect the number or migration of neuroblasts from the SVZ.

Brain stem catecholamines circuitry: Activation by alcohol and role in the hypothalamic-pituitary-adrenal response to this drug.
Lee S, Craddock Z, Rivier C.
J Neuroendocrinol Epub Mar, 2011.

In this work the authors investigated mechanisms underlying the stimulatory effect of alcohol on the hypothalamic-pituitary-adrenal axis (HPA). One method used was 33-ng injections of anti-DBH-SAP (Cat. #IT-03) into the A2/C2/C3 and A1/C1 regions. The data generated show that catecholamines, especially in the brainstem, regulate the HPA response to alcohol. This regulation utilizes α1-adrenergic receptors. Administration of anti-DBH-SAP to the A1-A2/C1-C3 regions disrupted the catecholaminergic input to the paraventricular nucleus.

(continued on page 5)