

Targeting Topics: Recent Scientific References

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3 µg/ml of Saporin (Cat. #PR-01) and subsequent analysis of protein expression. The suppression of ribosome function partially reversed the resistance to ER stress-induced cell death.

Extrinsic regulation of injury/growth-related gene expression in the inferior olive of the adult rat.

Buffo A, Carulli D, Rossi F, Strata P
Eur J Neurosci 18(8):2146-2158, 2003

Inferior olive (IO) cells of the CNS have the ability to regenerate axons after injury, even when the injury is close to the terminal field. After administration of 2.2 µg of 192-Saporin (Cat. #IT-01) and a control immunotoxin (mouse IgG-SAP, Cat #IT-18) to each ventricle in rats, two subsets of IO cells were discovered. Each subset responded differently to injury indicating that multiple mechanisms are responsible for their intrinsic regenerative potential.

Transfer effects and conditional learning in rats with selective lesions of medial septal/diagonal band cholinergic neurons.

Janisiewicz AM, Baxter MG
Behav Neurosci 117(6):1342-1352, 2003

Conditional learning appears to require cholinergic input to the hippocampus and cingulate cortex. Using a total of 0.5 µl of 0.12 µg/µl 192-Saporin (Cat. #IT-01) injected into the medial septal area of rats, the authors investigated the role of cholinergic input in conditional learning. The results suggest that cholinergic neurons of the medial septum/vertical limb of the diagonal band play a role in the transfer of behavioral experience rather than in conditional learning itself.

Environment-spatial conditional learning in rats with selective lesions of medial septal cholinergic neurons.

Janisiewicz AM, Jackson O, Firoz EF, Baxter MG
Hippocampus 14:265-273, 2004

192-Saporin (Cat. #IT-01) has produced varied results when used to determine the role of cholinergic neurons of the medial septum/vertical limb of the diagonal band (MS/VDB) in spatial working memory. The authors used a total of 0.5 µl of 0.12 µg/µl 192-Saporin injected into the MS/VDB to examine "environment-spatial" conditional learning. The findings suggest that cholinergic neurons of the MS/VDB are involved in some aspects of conditional associative learning.



Septohippocampal acetylcholine: involved in but not necessary for learning and memory?

Parent MB, Baxter MG
Learn Mem 11:9-20, 2004

In this review the authors describe some of the methods and rationale behind the investigation of hippocampal acetylcholine and its role in the support

of learning and memory processes. Results produced by the use of 192-Saporin (Cat. #IT-01) are discussed, as well as the differences that have been found between the effects of 192-Saporin and those of less specific lesioning agents.

Extensive lesions of cholinergic basal forebrain neurons do not impair spatial working memory.

Vuckovich JA, Semel ME, Baxter MG
Learn Mem 11:87-94, 2004

The authors wished to examine whether cerebellar Purkinje cells damaged during a cholinergic basal forebrain lesion might be the cause of impaired working memory. Four injections of 0.2-0.3 µl (0.12-0.15 µg/ml, 192-Saporin, Cat. #IT-01) into the medial septum/vertical limb of the diagonal band, two injections into the horizontal limb of the diagonal band of Broca, and four injections into the nucleus basalis magnocellularis/substantia innominata of rats were used to produce a very specific lesion. The results indicate that the cholinergic basal forebrain does not play a substantial role in spatial working memory.

Effects of complete immunotoxin lesions of the cholinergic basal forebrain on fear conditioning and spatial learning.

Frick KM, Jeansok JK, Baxter MG
Hippocampus 14:244-254, 2004

The authors examined the hypothesis that basal forebrain cholinergic neurons are critical for acquisition and consolidation of fear conditioning by lesioning the medial septum/vertical limb of the diagonal band, the horizontal limb of the diagonal band of Broca, and the nucleus basalis magnocellularis of rats with 192-Saporin (Cat. #IT-01). The lesions did not impair contextual fear conditioning, implying that impairments induced by scopolamine may not be mediated by cholinergic input to the hippocampus and neocortex.

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