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BAY 60-7550

A Novel Potent & Selective PDE2 Inhibitor

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ALX-270-421-M005 5 mg
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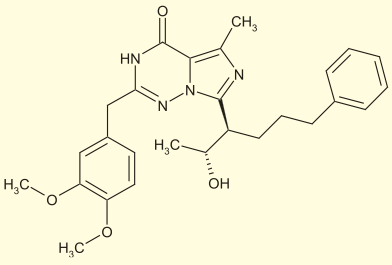
Inhibitor of human phosphodiesterase 2 (PDE2) (IC_{50}=5nM). Selective (>50-fold) versus other human PDEs tested (1C, 3A, 4B, 5A, 6, 7B, 8A, 9A, 10A, 11A). Does not affect adenosine deaminase. Raises cGMP levels in cell culture, particularly in combination with a suitable activator of soluble guanylyl cyclase (sGC). Raises cAMP in combination with forskolin (Prod. No. ALX-350-001). Increases memory performance in rodents *in vivo*.

LIT: Inhibition of phosphodiesterase 2 increases neuronal cGMP, synaptic plasticity and memory performance: F.G. Boess, et al.; Neuropharmacology **47,** 1081 (2004)



Cyclic Nucleotide Phosphodiesterases

The nucleotides cyclic adenosine 3',5'-monophosphate (cAMP) and cyclic guanosine 3',5'-monophosphate (cGMP) are intracellular second messengers that play a key role in mediating cellular responses to various hormones and neurotransmitters. Levels of cyclic nucleotides are tightly regulated at the level of synthesis by adenylate and guanylate cyclase as well as degradation by cyclic nucleotide phosphodiesterases (PDEs). Of the now 11 PDEs characterized, PDE1, -2, -3, -10 and -11 act on both cyclic nucleotides, PDE4, -7 and -8 are specific for cAMP and PDE5, -6 and -9 are specific for cGMP. This isozyme superfamily has become a major focus of drug discovery efforts owing to its diversity, molecular nature, differential regulation and expression in different cell types, and the range of biological functions. Inhibition of these isozymes can lead to an increase in cAMP and cGMP levels, which can affect a variety of physiological responses.

BAY 60-7550

An essential element of the signalling cascade leading to synaptic plasticity is the intracellular second messenger molecule guanosine 3',5'-cyclic monophosphate (cGMP). Using the novel, potent, and selective inhibitor BAY 60-7550, F.G. Boess, et al. showed that PDE2 is responsible for the degradation of newly synthesized cGMP in cultured neurons and hippocampal slices. Inhibition of PDE2 enhanced long-term potentiation of synaptic transmission without altering basal synaptic transmission. Inhibition of PDE2 also improved the performance of rats in social and object recognition memory tasks, and reversed MK-801 (Prod. No. ALX-550-337)-induced deficits in spontaneous alternation in mice in a T-maze. The data provide strong evidence that inhibition of PDE2 can improve memory functions by enhancing neuronal plasticity.

LIT: Inhibition of phosphodiesterase 2 increases neuronal cGMP, synaptic plasticity and memory performance: F.G. Boess, et al.; Neuropharmacology **47,** 1081 (2004)

Selected Latest Review Articles

Dynamic regulation of cAMP signaling by cGMP in the cardiovascular system: roles of phosphodiesterase 2 and phosphodiesterase 3 enzymes: D. H. Maurice; Proc. West. Pharmacol. Soc. **46,** 32 (2003)

Cyclic nucleotide phosphodiesterases and their role in immunomodulatory responses: advances in the development of specific phosphodiesterase inhibitors: A. Castro, et al.; Med. Res. Rev. **25,** 229 (2005)

The next generation of phosphodiesterase inhibitors: structural clues to ligand and substrate selectivity of phosphodiesterases: D. T. Manallack, et al.; J. Med.Chem. **48,** 3449 (2005)

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