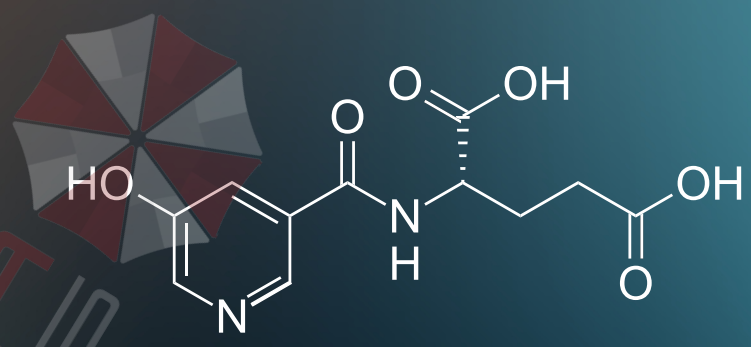


Nooglutyl - Assessing its Role in Neuroprotection and Neuronal Activity

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History

Nooglutyl was first appeared in the scientific literature in the middle of the 1990`s. It was discovered by a group at the Russian Academy of Sciences, focusing on the treatment of amnesia.

Effects in Animals Studies

Adult rats with a dose of 25 mg/kg/day administered with an intracutaneous injection normalized their behavior after a 2 hour oxygen deprivation, which leads to cognitive decrease in untreated rats. (PMID: 8704601) This was attributed to a change in local blood flow. A similar neuroprotective effect was also found in a stroke scenario, where Nooglutyl was found to reduce the amount of damaged tissue by 7%.[1]

Another study on rats and cats showed Nooglutyl was able to reduce motion sickness, shown by an increase in food intake after the provocation of nausea.[2]

Trauma in rat brains lead to negative changes in brain cell metabolism. These changes were prevented by Nooglutyl in a dose of 50 mg/kg.[3]

In a mechanistic study it was shown that AP7, a specific antagonist of the NMDA-glutamate receptor, blocks the effects of Nooglutyl and Emoxypine in rat hippocampus, which suggests Nooglutyl does affect glutamineric transmission using this receptor.[4] This is consistent with its molecular structure, which contains a glutamate unit. Nooglutyl and Emoxypine, but not piracetam inhibited the neurotransmission in this area. Later it was also found that Nooglutyl also affects the AMPA receptor, which represents another subtype of glutamate receptor.

Further experiments showed Nooglutyl (injected in doses and 50 mg/kg and 100 mg/kg) did decrease anxiety in animals during benzodiazepine withdrawal, but not in healthy rats , while no binding affinity towards the D2 receptor was reported.[5]

A comparative study found that the addition of the surfactant Tween-80 to Nooglutyl increased oral bioavailability by around 5%.[6]

Conclusion

So far, there is some evidence that Nooglutyl has neuroprotective effects under conditions of reduced blood flow in the brain and some other unspecific effects. It seems clear that it negatively affects neurotransmission for glutamate, which is the most important excitatory neurotransmitter in the brain. The protective and anti-nausea effect

can potentially be explained by a lower activity of neurons in certain brain areas, slowing down the need for energy and oxygen. There are no toxicological or studies on humans reported, which means the safety profile is not defined. Overdoses will have unforeseeable effects, but will likely involve confusion, loss of consciousness and later coma. From the published data, it cannot be concluded that this substance has potential use for increasing cognitive or physiological fitness in healthy individuals.

No studies were published after the year 2005.

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