Thiamethoxam, a poor agonist of nicotinic acetylcholine receptors expressed on isolated cell bodies, acts as a full agonist at cockroach cercal afferent/giant interneuron synapses

Titre	Thiamethoxam, a poor agonist of nicotinic acetylcholine receptors expressed on isolated cell bodies, acts as a full agonist at cockroach cercal afferent/giant interneuron synapses
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Résumé en anglais	Thiamethoxam (TMX) is a second-generation neonicotinoid which is known to induce toxic effects on insects and mammalians. Recently, it has been proposed that TMX is a poor agonist of insect nicotinic acetylcholine receptors (nAChRs) on isolated cell bodies. Here, we have studied its effect on synaptic transmission. Our results demonstrate that TMX acts as an agonist of nAChRs expressed on cockroach cercal afferent giant/interneuron synapses as bath applications of TMX induce a strong reversible depolarization of the sixth abdominal ganglion. This response was reduced by the nicotinic antagonists mecamylamine and methyllicaconitine, but was insensitive to d-tubocurarine. Interestingly, TMX-induced depolarization was partially reduced by the muscarinic antagonist atropine, suggesting that TMX could bind to a 'mixed nicotinic/muscarinic' receptor. Compared to previous studies, we proposed that TMX is able to act as agonist of insect nAChRs expressed at cercal afferent/giant interneuron synapses. Moreover, our results suggest that nAChRs expressed on synaptic ganglion are distinct to nAChRs expressed on isolated cell bodies and that synaptic receptors have higher affinity to TMX resulting to a depolarization of postsynaptic nicotinic receptors.
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