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# Mechanisms of action of insecticides on ligand-gated ion channels

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**Abstract:**
Ligand-gated ion channels (LGICs) mediate fast synaptic neurotransmission and are important targets for insecticides. Thus, the actions of several insecticides have been explored in electrophysiological studies on recombinant and native insect neuronal LGICs. I have shown that non-competitive antagonists of γ-aminobutyric acid gated $Cl^-$ channels also act on glutamate-gated $Cl^-$ channels, albeit at higher concentrations. Neonicotinoids are more potent agonists on recombinant hybrid nicotinic acetylcholine receptors (nAChRs) consisting of *Drosophila* Dα2 and vertebrate β2 subunits than those consisting of only vertebrate nAChR subunits (α4β2). Using this hybrid nAChR, clothianidin and related compounds containing a acyclic guanidine moiety were found to be super-agonists. Similar super-agonist actions of neonicotinoids were also observed on cultured *Drosophila* cholinergic neurons. Single channel nAChR recordings show that a clothianidin analogue induces a high conductance state in channel opening more frequently than acetylcholine, thereby offering a possible explanation for its super-agonist action. Unlike the case for clothianidin, imidacloprid attenuates the acetylcholine-induced re-sponse of native neuronal nAChRs when co-applied with ACh. These new discoveries add to our understanding of both the selectivity and the diverse actions of insecticides targeting LGICs.

**Keywords:**
ligand-gated ion channels (LGIC), electrophysiology, neurotoxic insecticides

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