Subchronic exposure to sublethal dose of imidacloprid changes electrophysiological properties and expression pattern of nicotinic acetylcholine receptor subtypes in insect neurosecretory cells

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Résumé en anglais	Neonicotinoids are the most important class of insecticides used in agriculture over the last decade. They act as selective agonists of insect nicotinic acetylcholine receptors (nAChRs). The emergence of insect resistance to these insecticides is one of the major problems, which limit the use of neonicotinoids. The aim of our study is to better understand physiological changes appearing after subchronic exposure to sublethal doses of insecticide using complementary approaches that include toxicology, electrophysiology, molecular biology and calcium imaging. We used cockroach neurosecretory cells identified as dorsal unpaired median (DUM) neurons, known to express two α-bungarotoxin-insensitive (α-bgt-insensitive) nAChR subtypes, nAChR1 and nAChR2, which differ in their sensitivity to imidacloprid. Although nAChR1 is sensitive to imidacloprid, nAChR2 is insensitive to this insecticide. In this study, we demonstrate that subchronic exposure to sublethal dose of imidacloprid differentially changes physiological and molecular properties of nAChR1 and nAChR2. Our findings reported that this treatment decreased the sensitivity of nAChR1 to imidacloprid, reduced current density flowing through this nAChR subtype but did not affect its subunit composition (α3, α8 and β1). Subchronic exposure to sublethal dose of imidacloprid also affected nAChR2 functions. However, these effects were different from those reported on nAChR1. We observed changes in nAChR2 conformational state, which could be related to modification of the subunit composition (α1, α2 and β1). Finally, the subchronic exposure affecting both nAChR1 and nAChR2 seemed to be linked to the elevation of the steady-state resting intracellular calcium level. In conclusion, under subchronic exposure to sublethal dose of imidacloprid, cockroaches are capable of triggering adaptive mechanisms by reducing the participation of imidacloprid-sensitive nAChR1 and by optimizing functional properties of nAChR2, which is insensitive to this insecticide.
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