Quinuclidine compounds differently act as agonists of Kenyon cell nicotinic acetylcholine receptors and induced distinct effect on insect ganglionic depolarizations

Titre	Quinuclidine compounds differently act as agonists of Kenyon cell nicotinic acetylcholine receptors and induced distinct effect on insect ganglionic depolarizations
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Résumé en anglais	We have recently demonstrated that a new quinuclidine benzamide compound named LMA10203 acted as an agonist of insect nicotinic acetylcholine receptors. Its specific pharmacological profile on cockroach dorsal unpaired median neurons (DUM) helped to identify alpha-bungarotoxin-insensitive nAChR2 receptors. In the present study, we tested its effect on cockroach Kenyon cells. We found that it induced an inward current demonstrating that it bounds to nicotinic acetylcholine receptors expressed on Kenyon cells. Interestingly, LMA10203-induced currents were completely blocked by the nicotinic antagonist alpha-bungarotoxin. We suggested that LMA10203 effect occurred through the activation of alpha-bungarotoxin-sensitive receptors and did not involve alpha-bungarotoxin-insensitive nAChR2, previously identified in DUM neurons. In addition, we have synthesized two new compounds, LMA10210 and LMA10211, and compared their effects on Kenyon cells. These compounds were members of the 3-quinuclidinyl benzamide or benzoate families. Interestingly, 1 mM LMA10210 was not able to induce an inward current on Kenyon cells compared to LMA10211. Similarly, we did not find any significant effect of LMA10210 on cockroach ganglionic depolarization, whereas these three compounds were able to induce an effect on the central nervous system of the third instar M. domestica larvae. Our data suggested that these three compounds could bind to distinct cockroach nicotinic acetylcholine receptors.

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