



Effect of calcium on nicotine-induced current expressed by an atypical alpha-bungarotoxin-insensitive nAChR2

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Résumé en anglais	<p>Two distinct native alpha-bungarotoxin (alpha-Bgt)-insensitive nicotinic acetylcholine receptors (nAChRs), named nAChR1 and nAChR2, were identified in the cockroach <i>Periplaneta americana</i> dorsal unpaired median (DUM) neurons. They differed in their electrophysiological, pharmacological properties and intracellular regulation pathways. nAChR2 being an atypical nicotinic receptor closed upon agonist application and its current-voltage relationship resulted from a reduction in potassium conductance. In this study, using whole-cell patch-clamp technique, we demonstrated that calcium modulated nAChR2-mediated nicotine response. Under 0.5 microM alpha-Bgt and 20 mM d-tubocurarine, the nicotine-induced inward current amplitude was strongly reduced in the presence of intracellularly applied BAPTA or bath application of calcium-free solution. In addition, using cadmium chloride, we showed that nicotine response was modulated by extracellular calcium through plasma membrane calcium channels. Moreover, extracellular application of caffeine and thapsigargin reduced nAChR2-mediated response. Together these experiments revealed a complex calcium-dependent regulation of nAChR2.</p>

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