



Dual action of a dinoflagellate-derived precursor of Pacific ciguatoxins (P-CTX-4B) on voltage-dependent K(+) and Na(+) channels of single myelinated axons

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Résumé en anglais	<p>The effects of Pacific ciguatoxin-4B (P-CTX-4B, also named gambiertoxin), extracted from toxic Gambierdiscus dinoflagellates, were assessed on nodal K(+) and Na(+) currents of frog myelinated axons, using a conventional voltage-clamp technique. P-CTX-4B decreased, within a few minutes, both K(+) and Na(+) currents in a dose-dependent manner, without inducing any marked change in current kinetics. The toxin was more effective in blocking K(+) than Na(+) channels. P-CTX-4B shifted the voltage-dependence of Na(+) conductance by about 14 mV towards more negative membrane potentials. This effect was reversed by increasing Ca(2+) in the external solution. A negative shift of about 16 mV in the steady-state Na(+) inactivation-voltage curve was also observed in the presence of the toxin. Unmodified and P-CTX-4B-modified Na(+) currents were similarly affected by the local anaesthetic lidocaine. The decrease of the two currents by lidocaine was dependent on both the concentration and the membrane potential during pre-pulses. In conclusion, P-CTX-4B appears about four times more effective than P-CTX-1B to affect K(+) channels, whereas it is about 50 times less efficient to affect Na(+) channels of axonal membranes. These actions may be related to subtle differences between the two chemical structures of molecules.</p>
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