Analysis of Caribbean ciguatoxin-1 effects on frog myelinated axons and the neuromuscular junction

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Résumé en anglais	Caribbean ciguatoxin-1 (C-CTX-1) induced, after about 1h exposure, muscle membrane depolarisation and repetitive post-synaptic action potentials (APs) in frog neuromuscular preparations. This depolarising effect was also observed in a Ca(2+)-free medium with a strong enhancement of spontaneous quantal transmitter release, compared with control conditions. The ciguatoxin-induced increase in release could be accelerated when Ca(2+) was present in the extracellular medium. C-CTX-1 also enhanced nerve-evoked quantal acetylcholine (ACh) release. At normal neuromuscular junctions loaded with the fluorescent dye FM1-43, C-CTX-1 induced swelling of nerve terminals, an effect that was reversed by hyperosmotic d-mannitol. In myelinated axons, C-CTX-1 increased nodal membrane excitability, inducing spontaneous and repetitive APs. Also, the toxin enlarged the repolarising phase of APs in control and tetraethylammonium-treated axons. Overall, our data suggest that C-CTX-1 affects nerve excitability and neurotransmitter release at nerve terminals. We conclude that C-CTX-1-induced up-regulation of Na(+) channels and the inhibition of K(+) channels, at low nanomolar concentrations, produce a variety of functional dysfunctions that are in part responsible for the human muscle skeletal symptoms observed in ciguatera. All these dysfunctions seem to result from the subtle balance between ionic currents, intracellular Na(+) and Ca(2+) concentrations, and engaged second messengers.
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