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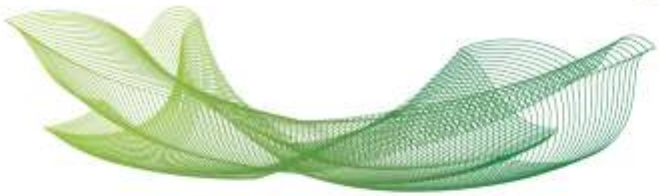
Resumo	Systemic scorpion envenomation is characterized by massive neurotransmitter release from peripheral nerves mediated primarily by scorpion venoms neurotoxins. Tityus bahiensis is one of the medically most important species in Brazil, but its venom pharmacology, especially regarding to peripheral nervous system, is poorly understood. Here, we evaluated the T. bahiensis venom activity on autonomic (sympathetic) neurotransmission by using a variety of approaches, including vas deferens twitch-tension recordings, electrophysiological measurements (resting membrane potentials, spontaneous excitatory junctional potentials and whole-cell patch-clamp), calcium imaging and histomorphological analysis. Low concentrations of venom (≤ 3 µg/mL) facilitated the electrically stimulated vas deferens contractions without affecting postsynaptic receptors or damaging the smooth muscle cells. Transient TTX-sensitive sustained contractions and resting membrane depolarization were mediated mainly by massive spontaneous ATP release. High venom concentrations (≥ 10 µg/mL) blocked the muscle contractions and induced membrane depolarization. In neuronal cells (ND7-23wt), the venom increased the peak sodium current, modified the current-voltage relationship by left-shifting the Na_v-channel activation curve, thereby facilitating the opening of these channels. The venom also caused a time-dependent increase in neuronal calcium influx. These results indicate that the sympathetic hyperstimulation observed in systemic envenomation is presynaptically driven, probably through the interaction of α- and β-toxins with neuronal sodium channels.
Fomento	



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