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Effects of thiazole analogs of vitamin B1 on neuromuscular transmission and α-latrotoxin-induced transmitter release in skeletal muscles

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Abstract

Thiazole analogs of vitamin B1 3-decyloxycarbonylmethyl-4-methyl-5-(2-hydroxyethyl)thiazole chloride (DMHT) and 3-decyloxycarbonylmethyl-4-methylthiazole chloride (DMT) suppress quantum transmitter release from nerve terminals in the frog skeletal muscle. Intraperitoneal administration of these compounds to mice suppresses behavioral motor activity, diminishes motor coordination, and suppresses the corazol-induced seizures. Application of DMHT reduces the α-latrotoxin-induced massive transmitter release from nerve terminals in the frog skeletal muscle and suppresses latrotoxin-induced seizures in mice. In model experiments, DMHT blocks Ca2+ entry through the ion channels formed by α-latrotoxin in a bilayer lipid membrane. It has been suggested that the effectiveness of DMHT and DMT is determined by the presence of a thiazole cycle in their molecules that, among all endogenous biologically active compounds, is possessed only by vitamin B1 and its metabolites.

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* **Nerve Terminal**

