

**Ca²⁺-dependent Permeabilization of the Inner Mitochondrial Membrane by
Cratylia mollis Seed Lectin Differs between Rat Liver and *T. cruzi*
Mitochondria**

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Lectins are proteins or glycoproteins that serve as tools in glycobiology research and can be employed for the detection of cell surface glycoconjugates. Strong evidences suggest that mitochondrial permeability transition (MPT) is the consequence of oxidative damage to mitochondrial membrane proteins resulting in the formation of a proteinaceous pore. This work was aimed at evaluating the effect of *Cratylia mollis* seed lectin (Cramoll 1,4) on both rat liver and *T. cruzi* mitochondria. Cramoll 1,4 (50 µg/ml) induced mitochondrial swelling, disruption of membrane potential (Ψ_m), Ca²⁺ release and ROS production in rat liver mitochondria (RLM). The use of cyclosporin A (MPT inhibitor) or EGTA (calcium chelator) prevented all these mitochondrial alterations. In contrast, the mitochondrial membrane permeabilization in *T. cruzi* was not prevented by cyclosporin A, but was inhibited by EGTA. Glucose totally prevented Ψ_m disruption in RLM by Cramoll 1,4 while in *T. cruzi* mitochondria the inhibition was only partial. These results indicate that Cramoll 1,4 leads to inner mitochondrial membrane permeabilization through mechanisms Ca²⁺ dependent in both mitochondria. The sensitivity to cyclosporin A in liver mitochondria characterizes lectin as a MPT inducer and the lack of cyclosporin A effect identifies a cyclosporin A-insensitive MPT in *T. cruzi* mitochondria.

Keywords: Lectin, mitochondrial permeability transition, rat liver mitochondria, *T. cruzi* mitochondria.

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