BIOCHEMISTRY 640

(Biomembranes Discussion Group)

Wednesday, February 28, 2018
Room 4-70 Medical Sciences Building
4:00 PM

Qiaolin Hu

Visiting Scholar, Chen Lab Dept. of Physiology

"Maitotoxin Is a Potential Selective Activator of the Endogenous Transient Receptor Potential Canonical Type 1 (TRPC1) Channel in *Xenopus laevis* Oocytes"

Maitotoxin (MTX) is the most potent marine toxin known to date. It is responsible for a particular human intoxication syndrome called ciguatera fish poisoning (CFP). Several reports indicate that MTX is an activator of non-selective cation channels (NSCC) in different cell types. The molecular identity of these channels is still an unresolved topic, and it has been proposed that the transient receptor potential (TRP) channels are involved in this effect. In Xenopus Igevis oocytes, MTX at picomolar (pM) concentrations induces the activation of NSCC with functional and pharmacological properties that resemble the activity of TRP channels. The purpose of this study was to characterize the molecular identity of the TRP channel involved in the MTX response, using the small interference RNA (siRNA) approach and the two-electrode voltage-clamp technique (TEVC). The injection of a specifically designed siRNA to silence the transient receptor potential canonical type 1 (TRPC1) protein expression abolished the MTX response. MTX had no effect on oocytes, even at doses 20-fold higher compared to cells without injection. Total mRNA and protein levels of TRPC1 were notably diminished. The TRPC4 siRNA did not change the MTX effect, even though it was important to note that the protein level was reduced by the silencing of TRPC4. Our results suggest that MTX could be a selective activator of TRPC1 channels in X. laevis oocytes and a useful pharmacological tool for further studies on these TRP channels.

Article:

Flores PL., et al. Maitotoxin Is a Potential Selective Activator of the Endogenous Transient Receptor Potential Canonical Type 1 Channel in Xenopus Iaevis Oocytes. Mar Drugs. 2017 Jun 25;15(7). pii: E198. doi: 10.3390/md15070198