

Researchers find novel role for calcium channels in pacemaker cell function

March 9 2011

Pacemaker cells in the sinoatrial node control heart rate, but what controls the ticking of these pacemaker cells? New research by Angelo Torrente and his colleagues of the M.E. Mangoni group's, reveals, for the first time, a critical functional interaction between Cav1.3 calcium ion (Ca^{2+}) channels and ryanodine-receptor (RyR) mediated Ca^{2+} signaling.

The study also sheds light on a long-standing debate regarding the relative contributions of the 'funny current' generated by [ion channels](#) and the RyR dependent spontaneous diastolic Ca^{2+} release theory in determining [heart rate](#).

The investigation by the research team compared pacemaker cells in normal mice with mutants that lacked the L-type Cav1.3 channels to contrast how they handled calcium. They found that the absence of Cav1.3 channels in sinoatrial node (SAN) cells reduced the frequency of Ca^{2+} transients, which determine the rate of cardiac muscle contraction. The Cav1.3 channels were also found to be important regulators of ryanodine-receptor dependent local calcium release in the diastolic pacemaker phase. Overall, their results show that local calcium release in SAN cells is tightly controlled by the Cav1.3 channels.

Defects in [calcium channels](#) controlling heart [muscle function](#) are known to cause [heart failure](#), and this study reveals that Cav1.3 [mutant mice](#) also suffer from bradycardia and other cardiac arrhythmias.

"Our results clarify the role of Cav1.3 channels in pacemaker generation, and are a step towards using it as a target for drug therapy to treat heart dysfunction related to the sinoatrial node", says A. Torrente of CNRS in Montpellier, France, who was the lead author on the study.

Not only Cav1.3 channels are critical to the heart pacemaker cell function, they appear to be important to several other cellular mechanisms as well. In both humans and mice, Cav1.3 mutations have been linked to sinoatrial node dysfunction and deafness (or SANDD) syndrome. Cav1.3 channels are believed to play a role in pancreatic β -cell stimulation, and they may also serve as pacemaker channels in the central nervous system, playing a pathophysiological role in Parkinson's disease.

"A better understanding of these channels in SAN could help us to comprehend the mechanism of calcium release in many other tissues and disease conditions as well", says Torrente.

More information: The presentation, "CAV1.3 L-TYPE CALCIUM CHANNELS-MEDIATED RYANODINE RECEPTOR DEPENDENT CALCIUM RELEASE CONTROLS HEART RATE" is at 10:30 a.m. on Wednesday, March 9, 2011 in Hall C of the Baltimore Convention Center. ABSTRACT: tinyurl.com/4pkjxgk

Provided by American Institute of Physics

Citation: Researchers find novel role for calcium channels in pacemaker cell function (2011, March 9) retrieved 24 April 2024 from <https://phys.org/news/2011-03-role-calcium-channels-pacemaker-cell.html>

This document is subject to copyright. Apart from any fair dealing for the purpose of private

study or research, no part may be reproduced without the written permission. The content is provided for information purposes only.