
RESEARCHERS IN FRANCE AND AUSTRIA FIND NOVEL ROLE FOR
CALCIUM CHANNELS IN PACEMAKER CELL FUNCTION

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WASHINGTON, D.C. (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 $Ca_v1.3$ 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 $Ca_v1.3$ channels to contrast how they handled calcium. They found that the absence of $Ca_v1.3$ channels in sinoatrial node (SAN) cells reduced the frequency of Ca^{2+} transients, which determine the rate of cardiac muscle contraction. The $Ca_v1.3$ 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 $Ca_v1.3$ channels.

Defects in calcium channels controlling heart muscle function are known to cause heart failure, and this study reveals that $Ca_v1.3$ mutant mice also suffer from bradycardia and other cardiac arrhythmias.

"Our results clarify the role of $Ca_v1.3$ 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 $Ca_v1.3$ 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, $Ca_v1.3$ mutations have been

linked to sinoatrial node dysfunction and deafness (or SANDD) syndrome. $Ca_v1.3$ 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.

NOTE TO EDITORS: An image is available to accompany this story.

IMAGE CAPTION: A mouse pacemaker cell initiates local Ca^{2+} releases in the diastolic phase. Red spots are the regions with maximal $[Ca^{2+}]_i$ released.

This project was supported by funding from the European Union Research Programme (Ca_v Net project) and the French National Agency for Research

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: <http://tinyurl.com/4pkjxgk>

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