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Participation of cardiac Ryanodine Receptors in cardiovascular complications associated to Metabolic Syndrome

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Abstract

The prevalence of Metabolic Syndrome (MS) in México has increased in the last 10 years. MS is a serious health problem due to its related cardiovascular disorders, such as hypertension and heart failure. The latter is among the major causes of death in México; however, the molecular mechanisms responsible for cardiac dysfunction in MS patients are unclear and could be related to anomalies in the cardiac excitation-contraction coupling (E-C coupling). The cardiac Ryanodine Receptor (RyR2) is a macromolecular complex that participates in releasing Ca^{2+} from internal stores and is involved in E-C coupling. Our aim has been to examine alterations at the expression level, phosphorylation status, Ca^{2+} sensitivity and *in situ* function (Ca^{2+} sparks and Ca^{2+} transients) of RyR2 that could explain the cardiac dysfunction associated with MS.

In an experimental model of MS we found that cardiomyocytes displayed diminished intracellular Ca^{2+} transients with impaired cell contractility and decrease in Ca^{2+} spark frequency. [^{3}H]Ryanodine binding showed that functional RyR2 are decreased in MS hearts with no changes of its Ca^{2+} sensitivity. However MS did not alter the phosphorylation of RyR2 at Serine-2809 linked to the development of heart failure due to leaky RyR2. Then, the impaired RyR2 functionality could be attributable to additional stress-induced RyR2 modifications but not to changes at RyR2 phosphorylation status. RyR2 alterations may account for the poor overall cardiac outcome found in MS patients and could be targeting for future therapies. Funding: Instituto de Ciencia y Tecnología of México City (ICyTDF, Project 331/2010).

Biography

Angélica Rueda is Professor at the Department of Biochemistry, Cinvestav-IPN, México, since 2008. She was a fellow of the AHA during her postdoctoral stay at the Department of Physiology, Medical School, University of Wisconsin-Madison. In 2006 she moved to Ana Maria Gómez Laboratory (Montpellier France) to study calcium handling alterations in vascular smooth muscle cells from the diabetic db/db mice. She has 17 publications, one book chapter, and is part of the National System of Researchers in México. In 2010, she was awarded with Dr. Jorge Rosenkranz prize from Roche of Mexico, in the area of diabetes.