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# Doctoral College
# Metabolic & Cardiovascular Disease

Medical University of Graz

UNI GRAZ TU Graz Graz University of Technology

# CYTOPLASMIC NANODOMAINS AND CALCIUM SIGNALLING

**GUEST LECTURE by**

## Prof. Dr. A. Mark Evans

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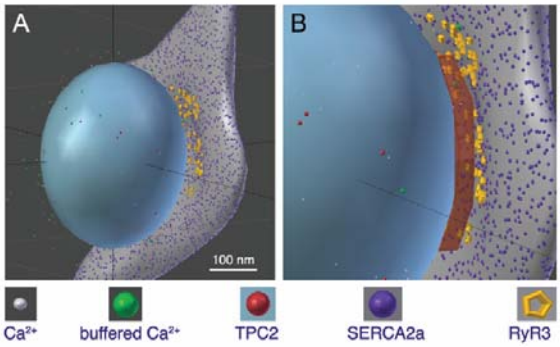
## Monday, 27.10.2014
## 17:00

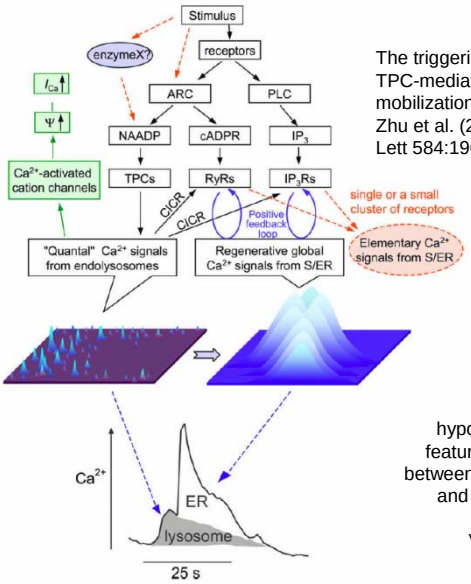
**SR 07.13, Preclinics**
**Harrachgasse 21, MUG**

**Site- and function-specific targeting of calcium pumps and release channels within nano junctions of the SR provides for effective signal demarcation**

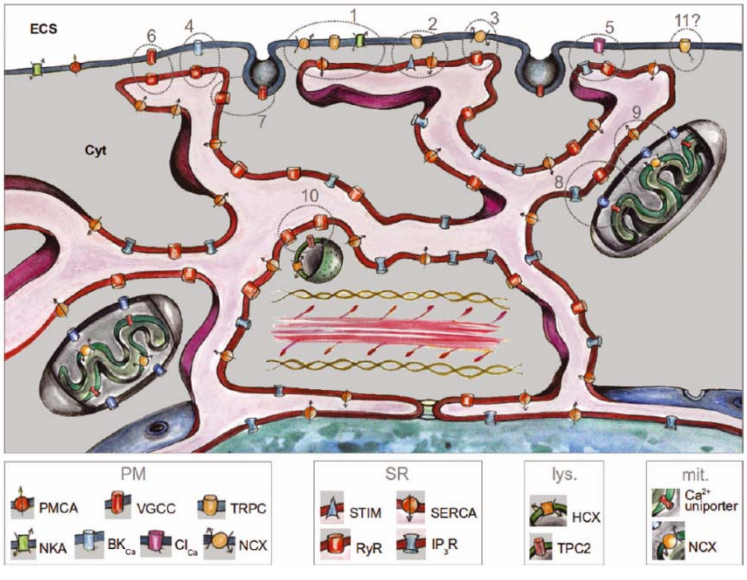
The mechanisms by which calcium signals underpin both contraction and relaxation control smooth muscle cell function, while at the same time coordinating stimulus-transcription coupling remain obscure. The general consensus is that cytoplasmic calcium transients may propagate across the cell in a manner that provides for comparable registration at the plasma membrane and even the nucleus, albeit with the support of direct calcium release into the nucleus from the nucleoplasmic reticulum. However, when considering the array of processes modulated by cytoplasmic calcium signals this model seems wholly inadequate. An attractive alternative would be to consider the possibility that cells segregate calcium signals on the nanoscale.




3D software reproduction of a lysosome closely apposed to a oortion of SR, thereby forming an ~20nm wide L-SR nanojunction. Fameli et al. (2014) F1000Research3:93




The triggering role of TPC-mediated $Ca^{2+}$ mobilization.
Zhu et al. (2010) FEBS Lett 584:1966-74




Graphic illustration of the hypothetical pan-junctional SR featuring multiple nanojunctions between the SR, on the one hand, and the PM, mítochondria and lysosomes, on the other.
Van Breemen et al. (2013) J Physiol 591(8):2043-54