



MITOCHONDRIAL Ca²⁺ SIGNALING IN COLORECTAL CANCER INVASION

GUEST LECTURE by

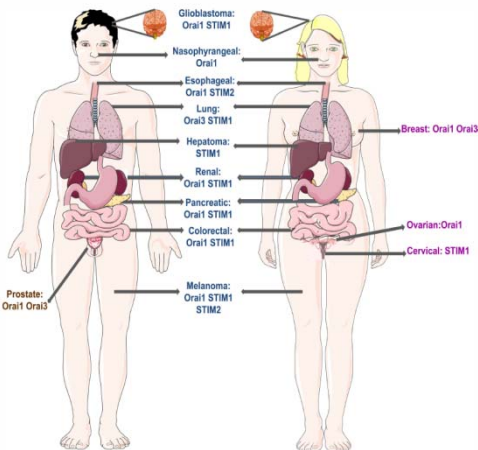


Prof. Mohamed Trebak, PhD

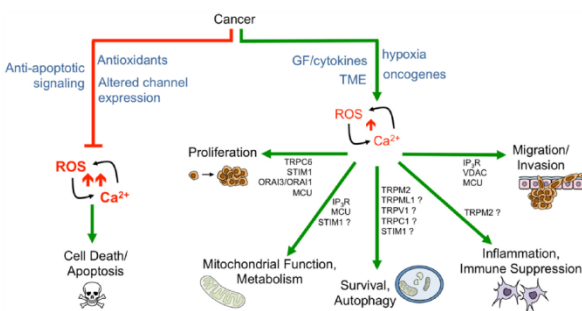
Department of Cellular and Molecular
Physiology & Penn State Cancer Institute,
Pennsylvania State University College of
Medicine, Hershey, USA

Monday, 11.09.2017
17:00

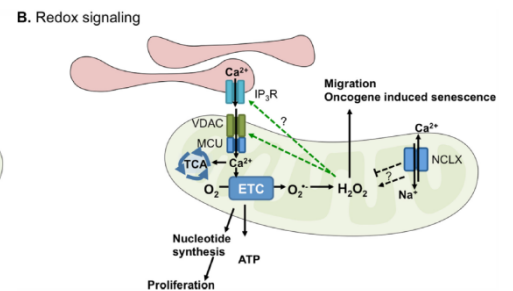
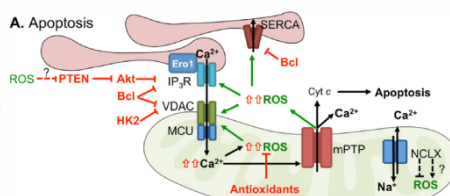
HS E1, Lecture Hall Center, MUG
(Auenbruggerplatz 15)



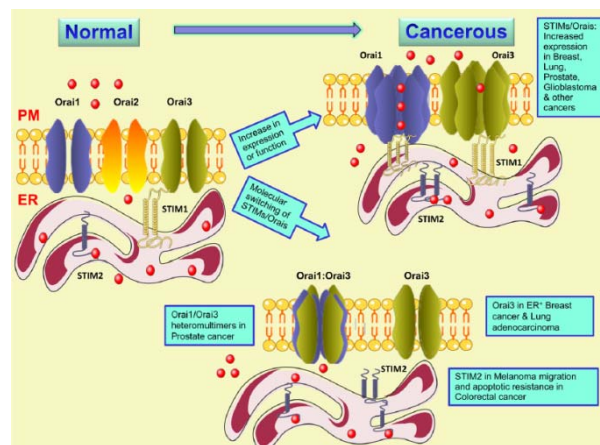
Contribution of STIM and Orai proteins to different types of cancers. Vashint et al. (2015) Am J Physiol Cell Physiol 309:C457-C469



Cancer cells take advantage and manipulate the ROS-Ca²⁺ interplay by inhibiting large ROS-Ca²⁺ surges that mediate apoptosis and by promoting pro-tumorigenic signaling pathways in response to sublethal changes in ROS/Ca²⁺ levels. Hempel & Trebak (2017) Cell Calcium 63:70-96



Cancer cells have developed several mechanisms to evade the pro-apoptotic ROS-Ca²⁺ cross talk at the ER-mitochondrial interface (A), while maintaining regulated mitochondrial Ca²⁺ influx that can regulate mitochondrial redox signaling to support tumor survival, growth and metastasis (B). Hempel & Trebak (2017) Cell Calcium 63:70-96



Mechanisms driving STIM and Orai proteins mediated tumorigenesis. Vashint et al. (2015) Am J Physiol Cell Physiol 309:C457-C469