

**2<sup>nd</sup> JOINT MEETING OF SLOVAK AND SERBIAN PHYSIOLOGICAL  
SOCIETIES**

**“PHYSIOLOGY WITHOUT FRONTIERS”**

**May 15-18, 2016**

**Smolenice Castle**

**Congress Center of the Slovak Academy of Sciences, Bratislava,  
Slovakia**



**Book of Abstracts**

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## **Redox regulation: from redox congeners to ATP-mediated contractility**

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Redox reactions regulate cellular functions by linking ATP production with oxygen demand, reactive oxygen species (ROS) production and mitochondrial functioning. When ROS production is controlled and compartmentalized, ROS participate in different subtle redox modifications of regulatory molecules. Redox balance is provided by a set of antioxidant enzymes that utilize NADPH and GSH to prevent an uncontrolled shift towards a more oxidative cellular milieu. The sum of these processes is stable but dynamic redox homeostasis; the small redox congeners form a discrete cellular redox-based and reversible network referred to as redox signaling, which is characterized by reversibility and short-lasting activity. These mediators are ideally suited for local effects by virtue of their ability to diffuse through tissue. This is especially important in physiological regulation and fine-tuning of small vessel vascular tone and smooth muscle contractility. These effects can be mediated by redox regulation of signaling molecules and/or by cellular receptors. Our results show that hydrogen peroxide relaxes the uterus in a dose-dependent manner through changes in voltage-gated potassium channels. The chemical properties of the generated signaling molecules determine the extent and magnitude of their physiological impact. The thiol group (SH) accomplishes its physiological activity rapidly by the SH<sup>-</sup> anion, whereas the CH<sub>3</sub>SH form has a prolonged effect. The link between local oxygen demands, the energetic state and physiological regulation is mediated by complex redox interactions. In erythrocytes the redox state can trigger different output signals ranging from ATP to NO. Stability of NO is achieved partially by SOD activity, which has been proposed to represent an enzymatic stabilizer of the cellular feedback mechanism that controls the cell's redox equilibrium, and the physiological levels of NO.