

## **P2.29.**

### **Intracellular redistribution of nitric oxide synthase in snail neurons: role of hypothermia**

Rőszer, T.<sup>1,4\*</sup>; Kiss-Tóth, É.<sup>1</sup>; Rózsa, D.<sup>1</sup>; Józsa, T.<sup>2</sup>; Szentmiklósi, A. J.<sup>3</sup>; Bánfalvi, G.<sup>1</sup>

*1: Department of Microbial Biotechnology and Cell Biology (formerly Animal Anatomy and Physiology), Faculty of Science and Technology*

*2: Department of Pediatrics, Medical Health Science Center, University of Debrecen, Debrecen, Hungary*

*3: Department of Pharmacology and Pharmacotherapy, Medical and Health Science Center, University of Debrecen, Debrecen, Hungary*

*4: Department of Regenerative Cardiology, Spanish National Cardiovascular Research Center, Madrid, E-mail: troszer@cnic.es*

Neuronal nitric oxide (NO) levels are modulated through the control of NO synthase (NOS) catalytic activity. Although signals limiting excess NO synthesis are being extensively studied in the vertebrate nervous system, our knowledge is rather limited on the modulation of NOS in neurons of invertebrates. We have previously reported a transient inactivation of NOS in hibernating snails. In the present study, we investigated further the mechanism leading to blocked NO production during hypothermic periods of *Helix pomatia*. Our studies with intact ganglia and cultured neurons revealed that hypothermic challenge translocated NOS from the cytosol to the perinuclear endoplasmic reticulum, and that this cytosol to membrane trafficking was essential for inhibition of NO synthesis. Cold stress also downregulated NOS mRNA levels in snail neurons, although the amount of NOS protein remained unaffected in response to hypothermia. We provide evidence that hypothermia keeps NO synthesis "hibernated" through subcellular redistribution of NOS.