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MEETING ABSTRACT

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**The role of hydrogen sulfide in the autonomic nervous system**

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**Background:** Hydrogen sulfide (H<sub>2</sub>S) is a toxic gas also produced in mammalian tissues where it can exert various functions as gasotransmitter, such as opening of smooth muscle K<sub>ATP</sub> channels and resulting in vasorelaxation. A recent study showed that H<sub>2</sub>S is endogenously generated and released in sympathetic ganglia and potentiates ganglionic transmission [1].

**Methods:** Experiments were performed on primary cultures of rat superior cervical ganglion (SCG) or on transfected tsA cells. Neurotransmitter release was determined by measuring the outflow of radioactivity from cultures labelled with [<sup>3</sup>H]noradrenaline. Electrophysiological recordings were performed by using the perforated patch-clamp technique.

**Results:** In SCG primary cultures, we found that in radiotracer release experiments, basal tritium overflow as well as outflow triggered by either electrical fields or depolarizing K<sup>+</sup> concentrations were enhanced by 0.1 to 1 mM of the H<sub>2</sub>S donor NaHS in a concentration-dependent manner. In electrophysiological experiments, H<sub>2</sub>S hyperpolarized the SCG membrane potential and reduced action potential firing. In SCG neurons, hyperpolarisation of membrane potential can be caused by an enhancement of currents through K<sub>V7</sub> channels [2]. Unexpectedly, NaHS inhibited currents through K<sub>V7</sub> channels in a concentration-dependent manner, whether endogenously expressed in SCG neurons or heterologously expressed in tsA cells.

**Discussion:** These results show that H<sub>2</sub>S regulates various functions of ganglionic neurons. Nevertheless, diazoxide, a well-known K<sub>ATP</sub> channel opener, also hyperpolarized the SCG membrane potential leading to the hypothesis that the membrane hyperpolarization caused by H<sub>2</sub>S could be an effect mediated by K<sub>ATP</sub> channels.

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**References**

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