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D043 - The role of TRPV1 / A1 receptors in the effects of hydrogen sulfide in rat trigeminal system

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Aim. Hydrogen sulfide(H_2S) is involved in a variety of physiological functions, including pro-nociceptive action in the sensory system. Cystathionine-β-synthase endogenously produces H_2S in nervous system and is abundantly expressed in rat trigeminal (TG) neurons. Its expression is upregulated in response to inflammatory pain with subsequent increase of the excitability of TG neurons. The aim of our study was to explore the role of TRP-receptors in the firing of TG nerve fibers induced by NaHS.
Methods. Extracellular recordings of peripheral branches of the TG nerve in isolated rat meninges and patch clamp recordings of TRPV1/A1 currents also Ca2+-imaging of rat TG neurons.
Results. The donor of H_2S—NaHS(100mkM) significantly increases firing in TG nerve. This effect was annulled by capsazepine (the TRPV1 inhibitor) but was partially prevented by HC030031 (the TRPA1 blocker). In a group of isolated TG neurons, NaHS transiently increased amplitude of capsaicin-induced currents. In addition, NaHS by itself induced inward currents in sensory neurons, which were prevented by capsazepine. In contrast, HC030031 did not prevent the NaHS-induced currents. Imaging of a large population of TG neurons revealed that NaHS induced calcium transients in 41% of tested neurons. Interestingly, this effect of NaHS in some neurons was inhibited by the TRPV1 antagonist capsazepine whereas in others it was sensitive to the TRPA1 blocker HC 030031.
Conclusions. Our data suggest that H2S activate both TRPV1 and TRPA1-receptors during neuro-inflammation conditions contribute to the nociceptive firing in primary afferents underlying migraine pain.
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