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The role of transient receptor potential ankyrin 1 (TRPA1) receptors in the H2S-evoked calcitonin gene-related peptide (CGRP) release from isolated rat trachea

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TRPA1 ion channels are expressed by capsaicin-sensitive peptidergic primary sensory neurons. TRPA1 activators, such as allylisothiocyanate (mustard oil), airway irritants, cold temperature stimulate these receptors, thus causing inflammatory neuropeptide release (SP, CGRP) and subsequent airway neurogenic inflammation. It has been established that hydrogen sulfide (H2S) like NO exerts neuromediator function and stimulates the capsaicin-sensitive sensory neurons. The aim of the present study was to investigate the effect of H2S on CGRP release from isolated tracheae comparing the action of the TRPA1 agonist mustard oil. Isolated rat tracheae were perfused in organ bath (37 oC, pH 7.2) containing oxygenized Krebs solution. H2S donor NaHS and mustard oil were applied in different doses to elicit neurotransmitter release. CGRP concentrations of the perfusion fluid were determined by radioimmunoassay. Selective TRPA1 antagonist HC030031 was added into the incubation medium to test the involvement of TRPA1 receptors in the CGRP release. Both NaHS and mustard oil induced dose dependent peptide release from the tracheal tissue which was inhibited by the TRPA1 antagonist. On the basis of these data we conclude that H2S may induce neurogenic airway inflammation by activation of TRPA1 ion channels.

OTKA CNK77793, NNF78917