Pflugers Archiv European Journal of Physiology 2010 vol.459 N3, pages 389-397

Hydrogen sulfide increases calcium-activated potassium (BK) channel activity of rat pituitary tumor cells

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Abstract

Hydrogen sulfide (H2S) is the third gasotransmitter found to be produced endogenously in living cells to exert physiological functions. Large conductance (maxi) calcium-activated potassium channels (BK), which play an important role in the regulation of electrical activity in many cells, are targets of gasotransmitters. We examined the modulating action of H2S on BK channels from rat GH3 pituitary tumor cells using patch clamp techniques. Application of sodium hydrogen sulfide as H2S donor to the bath solution in whole cell experiments caused an increase of calcium-activated potassium outward currents. In single channel recordings, H2S increased BK channel activity in a concentration-dependent manner. Hydrogen sulfide induced a reversible increase in channel open probability in a voltagedependent, but calcium independent manner. The reducing agent, dithiothreitol, prevented the increase of open probability by H 2S, whereas, the oxidizing agent thimerosal increased channel open probability in the presence of H2S. Our data show that H2S augments BK channel activity, and this effect can be linked to its reducing action on sulfhydryl groups of the channel protein.

http://dx.doi.org/10.1007/s00424-009-0737-0

Keywords

Gasotransmitter, GH3 cells, H2s, Maxi calcium-activated potassium channel patch clamp