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Hydrogen Sulfide, A Novel Gaseous Signal Molecule, Elongates Primary Cilia in the Madin-Darby Canine Kidney Cell

Sang Jun Han[1], Jee In Kim[2], Kwon Moo Park[1]

BK21 Plus Biomedical Convergence Program and Department of Anatomy[1],
Kyungpook National University School of Medicine
Department of Molecular Medicine[2], Keimyung Univerity School of Medicine

Primary cilium acts as a mechano- and chemo-sensor in kidney tubular cells. In recent, we have demonstrated that ischemia/reperfusion (I/R) injury alters the length of primary cilia in the kidney tubular epithelial cells and the alteration of lenghthlength is associated with oxidative stress. Hydrogen sulfide (H2S), a novel biological gas, plays a signaling molecule as well as antioxidant in the kidney. Here, we investigated the role of H2S in primary ciliogenesis and its underlying mechanisms in the Madin Darby Canine Kidney (MDCK) cell, an established kidney tubular epithelial cell line. Primary cilia gradually lengthened after confluent growth in the culture dish. Expressions of H2S-producing enzymes, cystathionine beta synthase (CBS) and cystathionine gamma lyase (CSE), gradually increased after confluent growth in the culture dish. Treatment of NaHS, a donor of H2S, lengthened primary cilia in the MDCK cells, whereas treatments of propargylglycine (PAG, an inhibitor of CSE) and hydroxylamine (HA, an inhibitor of CBS) shortened primary cilia. NaHS activated ERK in the MDCK cells. Treatment of U0126, an inhibitor of ERK, prevented the elongation of primary cilia induced by NaHS. Taken together, our results demonstrated that H2S elongated the length of primary cilia through ERK activation, suggesting that H2S plays an important role in the regulation of primary cilia length in kidney tubule epithelial cells.

Key Words: H2S, Primary cilia, ERK