Emerging studies have demonstrated the important physiological and pathophysiological roles of hydrogen sulphide (H2S) as a gasotransmitter for NOD-like receptor family pyrin domain-containing 3 (NLRP3) inflammasome-associated neuroinflammation in the central nervous system. However, the effects of H2S on neuroinflammation after intracerebral haemorrhage (ICH), especially on the NLRP3 inflammasome, remain unknown.

Zhao et al. employed a Sprague-Dawley rat of collagenase-induced ICH in the present study. The time course of H2S content and the spatial expression of cystathionine-β-synthase (CBS) after ICH, the effects of endogenous and exogenous H2S after ICH, the effects of endogenous and exogenous H2S on NLRP3 inflammasome activation under P2X7 receptor (P2X7R) overexpression after ICH, and the involvement of the P2X7R in the mechanism by which microglia-derived H2S prevented NLRP3 inflammasome activation were investigated.

They found ICH induced significant downregulation of endogenous H2S production in the brain, which may be the result of decreasing in CBS, the predominant cerebral H2S-generating enzyme. Administration of S-adenosyl-L-methionine (SAM), a CBS-specific agonist, or sodium hydrosulfide (NaHS), a classical exogenous H2S donor, not only restored brain and plasma H2S content but also attenuated brain oedema, microglial accumulation and neurological deficits at 1 day post-ICH by inhibiting the P2X7R/NLRP3 inflammasome cascade. Endogenous H2S production, which was derived mainly by microglia and above treatments, was verified by adenovirus-overexpressed P2X7R and in vitro primary microglia studies.

These results indicated endogenous H2S synthesis was impaired after ICH, which plays a pivotal role in the P2X7R/NLRP3 inflammasome-associated neuroinflammatory response in the pathogenesis of secondary brain injury. Maintaining appropriate H2S concentrations in the central nervous system may represent a potential therapeutic strategy for managing post-ICH secondary brain injury and associated neurological deficits [1)].

1)
Zhao H, Pan P, Yang Y, Ge H, Chen W, Qu J, Shi J, Cui G, Liu X, Feng H, Chen Y. Endogenous hydrogen sulphide attenuates NLRP3 inflammasome-mediated neuroinflammation by suppressing the P2X7 receptor after intracerebral haemorrhage in rats. J Neuroinflammation. 2017 Aug 18;14(1):163. doi: 10.1186/s12974-017-0940-4. PubMed PMID: 28821266.