Additional Table 1: Experimental studies of hydrogen sulfide in ischemic stroke of recent years (until 2021)

Study	Year	Model	Animals/cells	Main results
Jiang et al.[20]	2017	MACO OGD/R	Rats PC cells	Inhibition of overactivated autopagy may contribute to the attenuation of MCAO - induced cerebral ischemia/reperfusion injury in rats and OGD/R - induced cellular injury in PC12 cells by exogenous supplementation of NaHS.
Woo et al.[23]	2017	tMCAO	Rats	The NaHS-1 group (NaHS delivered at 1 min before reperfusion, respectively) had the lowest apoptosis rate compared with other group such as sham and NaHS30 groups (NaHS delivered at 30 min before reperfusion, respectively).
Zhu et al.[21]	2017	OGD/R Cerebral I/R	SH-SY5Y cells Mice	NaHS (intraperitoneal) shows best protection at 2 mg/kg, less protection at 1 or 4 mg/kg, and no protection at 8 or 16 mg/kg against mouse cerebral I/R injury through single injection. The neuroprotectiive effects of exogenous H_2S on ischemia/hypoxia and reperfusion/reoxygenation injury is mediated by enhanced autophagic degradation.
Wen et al.[18]	2018	MCAO	Rats Endothelial cells	1×10^{-5} - 1×10^{-7} mol/kg NaHS supplement: H_2S has the protective effects on brain I/R injury by upregulation of endothelium-dependent vasoconstriction and dilation function of cerebral vessels, which may be associated with activating potassium channel.
Bai et al.[25]	2019	tGCI	Rats	NaHS (24 μmol/kg) postconditioning effectively protected hippocampal CA1 neurons from tGCI-induced injury, at least in part by activating ERK1/2 signaling pathway.
Song et al.[22]	2020	MCAO	Rats	NaHS (28 μmol/kg) could down-regulate the phosphorylation of p38 by reducing the assembly of CaMKII with the ASK1-MKK3-p38 signal module, thus inhibiting brain I/R injury.
Tao et al.[27]	2020	MCAO	mice	Exogenous H_2S treatment suppressed inflammation and reduced behavioral impairment. The anti-inflammatory effect of H_2S was mediated by inhibiting NF-κB.
Wang et al.[16]	2018	MCAO	Rats	8e, a H_2S derivation released by 3-n-butylphthalide, significantly reduced neural apoptosis, focal infarction, brain edema and sensorimotor deficits within 72 h after transient middle cerebral artery occlusion.
Han et al.[14]	2020	MCAO	Rats	H_2S sustained release agent GYY4137 inhibited apoptosis by regulating p38MAPK, ERK1/2 and JNK signaling pathways, improved neural function after brain I/R injury,

				and reduced infarct area.
Pomiernyet al.[13]	2021	MCAO	Rats	AP39 (50 nmol/kg), an H_2S delivery molecule which can release slowly and target at mitochondria. After administration, this compound was found to have the neuroprotective activity and the notably reduced infarct volume and neurological deficit in the experimental groups treated with AP39 and subjected to MCAO.

Note: ASK1: Apoptosis signal-regulating kinase 1; CaMKII: calmodulin-dependent protein kinase II; ERK: extracellular-regulated kinase; H_2S: hydrogen sulfide; JNK: c-Jun N-terminal kinase; MAPK: mitogen-activated protein kinase; MCAO: middle cerebral artery occlusion; MKK3: mitogen-activated protein kinase kinase 3; NaHS: sodium hydrosulfide; NF-κB: nuclear factor kappa B; tGCI: transient global cerebral ischemia.