Supplementary Data

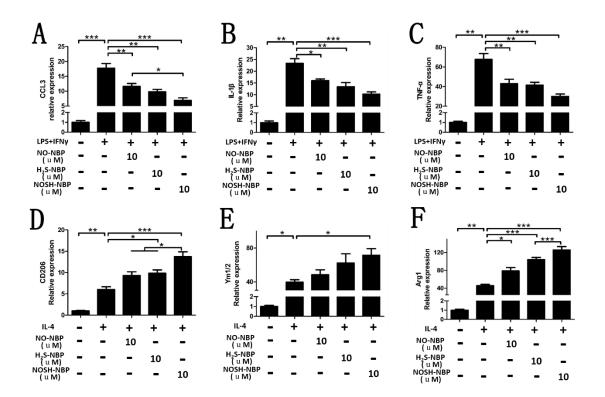


Figure S1. The effect of NO-NBP, H₂S-NBP and NOSH-NBP on macrophage M1/M2 polarization in vitro. BMDM were treated with LPS (100 ng/mL) + IFN- γ (20 ng/mL), IL-4 (20 ng/mL), NO-NBP (10 μ M), H2S-NBP (10 μ M), NOSH-NBP (10 μ M) or a combination of these treatments. Quantitative analysis of mRNA expression of M1 markers (A) CCL3, (B) IL-1 β and (C) TNF- α (n = 6). Quantitative analysis of mRNA expression of M2 markers (D) CD206, (E) Ym1/2 and (F) Arg1 (n = 6). Data were expressed as the mean \pm SD and analyzed by one-way analysis of variance (ANOVA) followed by post hoc Tukey test: ****P* < 0.001, ***P* < 0.01, **P* < 0.05.

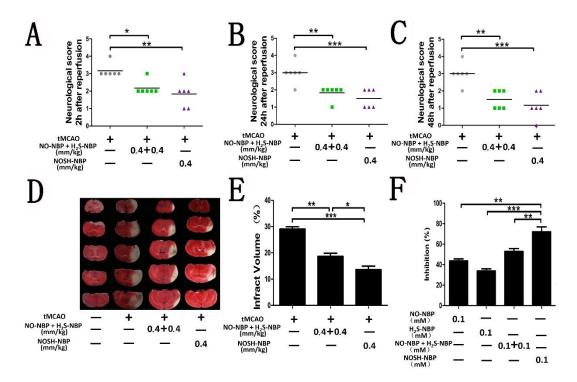


Figure S2. The effect of NOSH-NBP and combination of NO-NBP and H₂S-NBP on neurologlical deficit score, infarct volume in response to tMCAO injury, and on the ADP-induced platelet aggregation in vitro. The model group and test compounds group underwent tMCAO while the sham group underwent the same surgical procedure without the filament being inserted. After MCAO being performed, mice in test compounds group were administrated with NOSH-NBP (0.4 mm/kg) or NO-NBP + H_2 S-NBP (0.4 + 0.4 mm/kg) for 3 days. (A-C) The neurological deficit evaluation were performed at 24 h, 48 h and 72 h after reperfusion according to longa's method with minor modifications (n = 6). (**D**) Representative examples of TTC staining from each treatment group of mice. And infarct volume percentage data indicated in (E) were calculated as infarct area/whole area (n = 6). Platelet suspensions were preincubated with the testing compound (0.1 mM) at 37 °C for 5 min and exposed to 10 μ M of ADP, followed by continually monitoring. Rabbit platelet suspensions that had been treated with the vehicle and exposed to ADP were used as positive controls. (F) Inhibition of platelet aggregation (%). Data were present as mean \pm SD and were analyzed by one-way analysis of variance (ANOVA) followed by post hoc Tukey test: ***P < 0.001, **P < 0.01, *P < 0.05.