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Poster Sessions

The protective effect of a novel radical scavenger, NSP-116 on cerebral ischemia injury

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For patient outcome with stroke, recanalization of occlusion vessels is an effective therapy. However, recanalization induces oxidative stress via oxygen over-supply, which leads to neuronal damage as ischemia reperfusion injury (IRI). NSP-116 is a novel radical scavenger, and our previous study showed that NSP-116 suppressed oxidative stress. Therefore, we speculated that NSP-116 could ameliorate IRI damage. The purpose of this study is to investigate the effect of NSP-116 on IRI.

We established the IRI mouse model by using middle cerebral artery occlusion/ reperfusion (MCAO/R). NSP-116 (30 mg/kg) was orally administrated at 1 day before surgery and immediately after reperfusion. We evaluated the cerebral blood flow (CBF), neurological symptom and infarct volume after MCAO/R. In addition, using *in vitro* neural injury models, we assessed whether NSP-116 had the directly neuroprotective effect. NSP-116 administration improved neurological deficit and reduction of CBF. In infarct volume assay, NSP-116 had protective tendency. Moreover, NSP-116 pre-treatment suppressed the neuronal cell death in *in vitro* experiments.

Collectively, our findings suggested that NSP-116 could be useful for ischemic stroke therapy.