Evidence has come to light eliciting the neuroprotective function of SNHG16 in cerebrovascular diseases. A study sought to analyze the regulatory mechanism of long non-coding RNA small nucleolar RNA host gene16 (SNHG16) in oxidative stress (OS) injury and cell inflammation. Firstly, models of oxygen-glucose deprivation and reoxygenation (OGD/R) were established in SK-N-SH cells. Cell proliferation and apoptosis were appraised using cell counting kit-8 and flow cytometry. Additionally, SNHG16, X-linked inhibitor of apoptosis protein (XIAP), microRNA (miR 421), reactive oxygen species (ROS), lactate dehydrogenase (LDH), malondialdehyde (MDA), superoxide dismutase (SOD), tumor necrosis factor -α, interleukin (IL)-1β, and IL-10 expression patterns were determined. In addition, determined and validated the subcellular localization of SNHG16 and the binding relationships between SNHG16 and miR-421, and miR-421 and XIAP. It was found that SNHG16 was poorly-expressed in OGD/R-treated cells. On the other hand, SNHG16 over-expression enhanced cell proliferation, inhibited apoptosis, and alleviated OS and cell inflammation. Furthermore, SNHG16 is bound to miR-421 to facilitate the expression of XIAP. Up-regulation of miR-421 or down-regulation of XIAP could reverse the suppressive effects of SNHG16 on OS and cell inflammation. Collectively, these findings indicated that SNHG16 bound to miR-421 to facilitate XIAP expression, thus alleviating OS injury and inflammation in OGD/R-induced SK-N-SH cells [1)]

Wang D, Zheng J, Liu X, Xue Y, Liu L, Ma J, He Q, Li Z, Cai H, Liu Y. Knockdown of USF1 Inhibits the Vasculogenic Mimicry of Glioma Cells via Stimulating SNHG16/miR-212-3p and linc00667/miR-429 Axis. Mol Ther Nucleic Acids. 2019 Mar 1;14:465-482. doi: 10.1016/j.omtn.2018.12.017. Epub 2019 Jan 15. PubMed PMID: 30743215; PubMed Central PMCID: PMC6369224.

1)
Cao X, Ma J, Li S. Mechanism of lncRNA SNHG16 in oxidative stress and inflammation in oxygen-glucose deprivation and reoxygenation-induced SK-N-SH cells. Bioengineered. 2022 Mar;13(3):5021-5034. doi: 10.1080/21655979.2022.2026861. PMID: 35170375.