

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)-1 β , 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⁾

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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.

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