Thioredoxin interacting protein

Liang et al., found that thioredoxin-interacting protein (TXNIP) links endoplasmic reticulum stress (ER stress) to neuronal apoptosis and aggravates EBI. However, the other underlying mechanisms remain unknown. Mitochondria are considered to be the central points in integrating apoptotic cell death. However, whether crosstalk between TXNIP and the mitochondria-mediated intrinsic apoptotic pathway is effective on EBI has not been previously reported. Therefore, we created an endovascular perforation SAH model in Sprague-Dawley rats to determine the possible mechanism. We found that TXNIP expression in apoptotic neurons significantly increased in the SAH group compared with the sham group. In addition, increased TXNIP expression was accompanied by remarkable changes in mitochondrial-related antiapoptotic and proapoptotic factors. Furthermore, resveratrol (RES, a TXNIP inhibitor) administration significantly downregulated the expression of TXNIP and mitochondria-related proapoptotic factors. Additionally, it attenuated SAH prognostic indicators, such as brain edema, blood-brain barrier permeability, and neurological deficits. Therefore, our study further confirms that TXNIP may participate in neuronal apoptosis through the mitochondrial signaling pathway and that TXNIP may be a target for SAH treatment [1].

In a study, a possible association of thioredoxin-interacting protein (TXNIP) with malignant glioma was evaluated. Initially, semi-quantitative and quantitative analysis of the expression levels of TXNIP in clinical specimens of primary glioma was performed via immunohistochemistry (IHC) and reverse transcription-quantitative polymerase chain reaction (RT-qPCR), respectively, and expression levels were further correlated to the overall survival time of the patients. The proliferative, migratory and invasive properties of the glioblastoma U251 cell line, engineered to downregulate TXNIP by lentiviral transfection of a specific short hairpin RNA, were evaluated by means of in vitro assays. Consequently, IHC and RT-qPCR analysis revealed a negative association between the expression level of TXNIP and the histopathological grade of the tumor. Higher TXNIP expression level was associated with extended patient survival time. In vitro analysis revealed increased growth, migration and invasion in U251 cells with downregulated TXNIP expression compared with their non-transfected counterparts. These findings strongly indicate that TXNIP functions as a tumor suppressor in malignant glioma cells and underscore its potential as a novel therapeutic target and prognostic indicator of the condition [2].

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2)
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