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## The regulating affects of receptor interacting protein 3 on retina ganglion cell-5 necroptosis following elevated hydrostatic pressure

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Necroptosis is an important neuronal death mode in retinal ischemia, but the mechanism still needs clarify. *RIP3* is characterized as an N-terminal Serine/Threonine kinase, which participates in cell death signaling. Previous studies indicated *RIP3* may participate in neuronal necroptosis, and the activation of caspase-8 could cleave *RIP3* to inactive form. In the present study, we explored the effects of *RIP3* in retinal necroptosis following elevated hydrostatic pressure (EHP) and discussed the possible role of caspase-8 on regulation of *RIP3* activity. Necrosis levels detection were repeated with pretreatment of Nec-1of 24 h to confirm the existence of necroptosis. The expression of *RIP3*, downstream molecules in the pathway of *RIP3*-induced necroptosis and necrosis levels of RGC-5 cells were detected by immunoblotting, immunofluorescence and flow cytometry at 6 h, 12 h or 24 h after EHP. Then, RNAi to *rip3* was used for further confirming *RIP3*'s effects on retinal necroptosis. Finally, caspase-8 inhibitor and activity peptide were applied to try to unveil the regulated mechanism of *RIP3* activity. The results showed that, *RIP3* expression was up-regulated and *RIP3* enhance-labeled cells were coexisted with PI-positive cells after injury. PI-positive cells were reduced and ratios of necrosis were decreased after *rip3* knockdown. Caspase-8 inhibitor and activity peptide usage affected ratio of necrosis and levels of ROS or PYGL. Our results indicated *RIP3* participated in RGC-5 necroptosis following EHP and caspase-8 may interference *RIP3*-induced necroptosis.

## **Biography**

Lei Shang has completed his PhD from Central South University. He is the research worker of Hunan Cancer Hospital in the department of Translational Medicine Research, a premier Cancer Research Institute in Central South Region of China. He has published more than 10 papers in reputed journals.

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