TLR2

Toll-like receptor (TLR) 2/4 serves an important regulatory role in nerve tissue injury. However, the downstream and potential mechanisms remain to be elucidated. The present study was designed to investigate the roles of the TLR2/4-major myeloid differentiation response gene 88 (MyD88)-NF-κB signaling pathway in the development of an intracranial aneurysm. The expression of TLR2, TLR4, and MyD88 in the blood of normal controls and patients with intracranial aneurysms were detected by quantitative PCR and ELISA. Human brain vascular smooth muscle cells were treated by Angiotensin II (Ang II) to evaluate the involvement of the TLR2/4-MyD88-NF-κB signaling pathway in the process. The in vitro experiment was divided into four groups: The control group, an Ang Ⅱ group, an Ang Ⅱ + small interfering (si)RNA control group, and an Ang Ⅱ + TLR2-group. Cell viability, migration, apoptosis, and expression of TLR2, TLR4, MyD88, NF-κB, and phosphorylated (p-)p65 expression was detected. The results demonstrated that the expression of TLR2, TLR4, MyD88, and NF-κB at mRNA and protein levels in patients with an intracranial aneurysm was significantly higher compared with corresponding protein in normal controls (P<0.05). In vitro experiments demonstrated that Ang Ⅱ treatment increased the cell proliferation and migration rate but reduced the apoptotic rate compared with the control (P<0.05). The expression of TLR2, TLR4, MyD88, NF-κB, and p-p65 was significantly increased in the Ang II group (vs. control; P<0.05). By contrast, TLR2-short interfering RNA reduced the cell proliferation and migration rate and reduced the expression of TLR2, TLR4, MyD88, NF-κB, and p-p65 (vs. Ang Ⅱ + short interfering RNA control; P<0.05). In conclusion, the data of the present study indicated that the TLR2/4-MyD88-NF-κB signaling pathway is involved in the intracranial aneurysm pathogenesis [1].

1)
Zhang X, Wan Y, Feng J, Li M, Jiang Z. Involvement of TLR2/4-MyD88-NF-κB signaling pathway in the pathogenesis of intracranial aneurysm. Mol Med Rep. 2021 Jan 26. doi: 10.3892/mmr.2021.11869. Epub ahead of print. PMID: 33655339.