عنوان مقاله:

Knockdown of TRPMγ attenuates apoptosis and inflammation in neonatal necrotizing enterocolitis model cell IEC-۶ via modulating TLR۴/NF-ĸB and MEK/ERK pathways

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خلاصه مقاله:

Objective(s): Neonatal necrotizing enterocolitis (NEC) is the most common gastrointestinal critical illness in neonatal infants. TRPMγ reportedly plays a role in human inflammatory bowel disease (IBD) and colorectal cancer, but the role of TRPMγ in the pathogenesis of NEC remains vague.Materials and Methods: The expression of TRPMγ was determined in intestinal tissues of NEC patients and lipopolysaccharide (LPS)-induced IEC-۶ cells. Subsequently, a loss-of-function assay was performed to assess the effects of TRPMγ on cell apoptosis and inflammatory response in IEC-۶ cells after LPS induction. Furthermore, the modulation of TRPMγ on TLR۴/NF-ĸB and MEK/ERK signaling pathways was validated.Results: The expression of TRPMγ was higher in the intestinal tissues of NEC patients compared with the normal human intestinal tissues. Moreover, the expression level of TRPMγ was elevated in LPS stimulation IEC-۶ cells. Knockdown of TRPMγ enhanced cell viability and suppressed apoptosis, accompanied by the decreased Bax/Bcl-۱ ratio and cleaved-caspase۳ expression in LPS-induced IEC-۶ cells. Additionally, TRPMγ silencing attenuated LPS-induced expressions and secretions of proinflammatory cytokines. Mechanistically, TRPMγ knockdown inhibited the TLR۴/NF-ĸB activation, while enhancing the MEK/ERK activation in LPS-treated IEC-۶ cells. Overexpression of TLR۴ or inhibition of MEK attenuated the inhibitory effects of TRPMγ knockdown on LPS-induced apoptosis and inflammation in IEC-۶ cells.Conclusion: Knockdown of TRPMγ attenuated LPS-induced IEC-۶ cell apoptosis and inflammation by modulating TLR۴/NF-ĸB and MEK/ERK pathways.

کلمات کلیدی:

Apoptosis, Inflammation, Necrotizing Enterocolitis, TRPMγ, Viability

لینک ثابت مقاله در پایگاه سیویلیکا: