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Effect of inflammatory inhibition in rheumatoid fibroblast-like synoviocytes through two polypeptides, extracts from *Rubia philippinensis***Hee Sun Byun and Gang Min Hur**

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Rheumatoid arthritis (RA) is a chronic inflammatory disease by autoimmune disorder that primarily affects joints. We studied the isolated two polypeptides (Rp53, Rp54) from *Rubia philippinensis*, traditional medicine plant, about anti-inflammation effects in fibroblast-like synoviocytes (FLS) derived from patients with RA. Here we found that the Rp53 strongly inhibited the nuclear factor κ B (NF- κ B) signaling pathway induced by TNF α , but not that induced by IL-1 β . Analysis of the upstream signaling events affected by Rp53 revealed that it strikingly inhibited the TNF-induced recruitment of TNFR1-associated death domain protein (TRADD) and receptor-interacting protein (RIP) to TNFR1. Interestingly, Rp53 reduced the interaction with TNFR1 to TNF cytokines and enhanced the activation of the mitogen-activated protein (MAP) kinase. According to these findings, Rp53, the polypeptides induced the proteolytic cleavage of TNF-R1 and its release into the culture medium. That was the ectodomain shedding of TNF receptor 1 by TNF- α -converting enzyme (TACE). Along with the TACE inhibitor TAPI-2, the p38 kinase inhibitor SB203580 suppressed the ectodomain shedding of TNF receptor 1 induced by Rp53. Thus, our results indicate that Rp53 induces the TACE-dependent ectodomain shedding of TNF receptor 1 through the activation of p38 MAP kinase, and thereby inhibits the TNF α induced NF- κ B signaling pathway in RA-FLS cells.

Biography

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