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Micheliolide inhibits LPS-induced production of Proinflammatory cytokines and Type I IFN in macrophages through PI3K/Akt/p70S6K pathway

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Sepsis is still a major cause of fatality in the intensive care unit worldwide. Sepsis involves an uncontrolled inflammatory response by the host immune cells that may result in multi organ failure, DIC and even death. Micheliolide (MCL), a compound of sesquiterpene lactone was reported to inhibit DSS-induced inflammatory intestinal inflammation, colitis-associated cancer and rheumatic arthritis. But, there was no report about whether MCL plays an important role in microbial infection. In this study, we demonstrated that MCL decreased lipopolysaccharide (LPS)-mediated production of interleukin 6 (IL-6), tumor necrotic factor-α (TNF-α), MCP-1 and IFN-β in Raw264.7 macrophage cell line. Also, the same anti-inflammatory role of MCL was detected in primary peritoneal macrophages. Through screen of the signaling transduction, MCL negatively regulated the PI3K/Akt/p70S6K pathway especially dephosphorylating the site of Ser473 on Akt and the site of Thr389 on p70S6K. Meanwhile, MCL has negligible impact on the activation of ERK, JNK, p38 MAPKs and NF-κB pathways. Collectively, our data illustrated that MCL can help to maintain immune equilibrium by down-regulating the production of proinflammatory cytokines, chemokine and type I interferon in TLR4 signaling and thus diminish host damage. MCL may be a new potential immunosuppressive and anti-inflammatory agent in treatment of gram-negative bacterial infection.

Biography

Yuejuan Zheng has completed her PhD from Zhejiang University and Postdoctoral studies from National Key Laboratory of Medical Immunology & Institute of Immunology, Second Military Medical University. She has engaged in the research on the anti-inflammatory roles and mechanisms of natural products for more than six years. She has published more than 8 papers in reputed journals.

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