Anti-inflammatory Effect of *Heracleum moellendorffii* Roots through the Inhibition of NF-κB and MAPK Signaling, and Activation of ROS/Nrf2/HO-1 Signaling in LPS-stimulated RAW264.7 Cells

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Heracleum moellendorffii roots (HM-R) have been long treated for inflammatory diseases such as arthritis, backache and fever. However, an anti-inflammatory effect and the specific mechanism of HM-R were not yet clear. In this study, we for the first time explored the anti-inflammatory of HM-R. Results: HM-R dose-dependently blocked LPS-induced NO and PGE2 production. In addition, HM-R inhibited LPS-induced overexpression of iNOS, COX-2, IL-1β and IL-6 in RAW264.7 cells. HM-R inhibited LPS-induced NF-κB signaling activation through blocking IκB-α degradation and p65 nuclear accumulation. Furthermore, HM-R inhibited MAPK signaling activation by attenuating the phosphorylation of ERK1/2, p38 and JNK. HM-R increased nuclear accumulation of Nrf2 and HO-1 expression. However, NAC reduced the increased nuclear accumulation of Nrf2 and HO-1 expression by HM-R. In HPLC analysis, falcarinol was detected from HM-R as an anti-inflammatory compound. These results indicate that HM-R may exert anti-inflammatory activity by inhibiting NF-κB and MAPK signaling, and activating ROS/Nrf2/HO-1 signaling. From these findings, HM-R may have potential to be a candidate for the development of anti-inflammatory drugs.

Key words: Anti-inflammation, Heracleum moellendorffii, Inflammatory diseases, Inflammatory response

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