F-P-3

Rodgersia podophylla Leaves Suppress Inflammatory mediators through activation of Nrf2/HO-1 signaling, and inhibition of LPS-induced NF-κ B and MAPKs signaling in RAW264.7 cells

Ha Na Kim1, Jeong Dong Kim1, Su Bin Park1 and Jin Boo Jeong1,2*

1Department of Medicinal Plant Resources, Andong National University, Andong 36729, Republic of Korea

2Agricultural Science and Technology Research Institute, Andong National University, Andong, 36729, Republic of Korea

In this study, we elucidated the anti-inflammatory mechanisms of leaves extracts from Rodgersia podophylla (RPL) in RAW264.7 cells. RP-L significantly inhibited the production of the pro-inflammatory mediators such as NO, iNOS, IL-1β and IL-6 in LPS-stimulated RAW264.7 cells. RPL increased HO-1 expression in RAW264.7 cells, and the inhibition of HO-1 by ZnPP reduced the inhibitory effect of RPL against LPS-induced NO production in RAW264.7 cells. Inhibition of p38, ROS and GSK3β attenuated RPL-mediated HO-1 expression. Inhibition of ROS inhibited p38 phosphorylation and GSK3β expression induced by RPL. In addition, inhibition of GSK3β blocked RPL-mediated p38 phosphorylation. RPL induced nuclear accumulation of Nrf2, and Inhibition of p38, ROS and GSK3β abolished RPL-mediated nuclear accumulation of Nrf2. Furthermore, RPL blocked LPS-induced degradation of Iκ B-α and nuclear accumulation of p65. RP-L also attenuated LPS-induced phosphorylation of ERK1/2 and p38. Our results suggest that RPL exerts potential anti-inflammatory activity by activating ROS/GSK3β /p38/Nrf2/HO-1 signaling and inhibiting NF-κ B and MAPK signaling in RAW264.7 cells. These findings suggest that RPL may have great potential for the development of anti-inflammatory drug.

Keywords: Anti-inflammation; HO-1; MAPKs; NF-κ B; Rodgersia podophylla

[This work was supported by Basic Science Research Program through the National Research Foundation of Korea (NRF) funded by the Ministry of Education (NRF-2016R1D1A3B03931713 and NRF-2018R1A6A1A03024862).]