

1 Supplementary Information for

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3 ***Erythronium Japonicum* Alleviates Inflammatory Pain by Inhibiting MAPK Activation and by**
4 **Suppressing NF- κ B Activation via the ERK/Nrf2/HO-1 Signaling Pathway**

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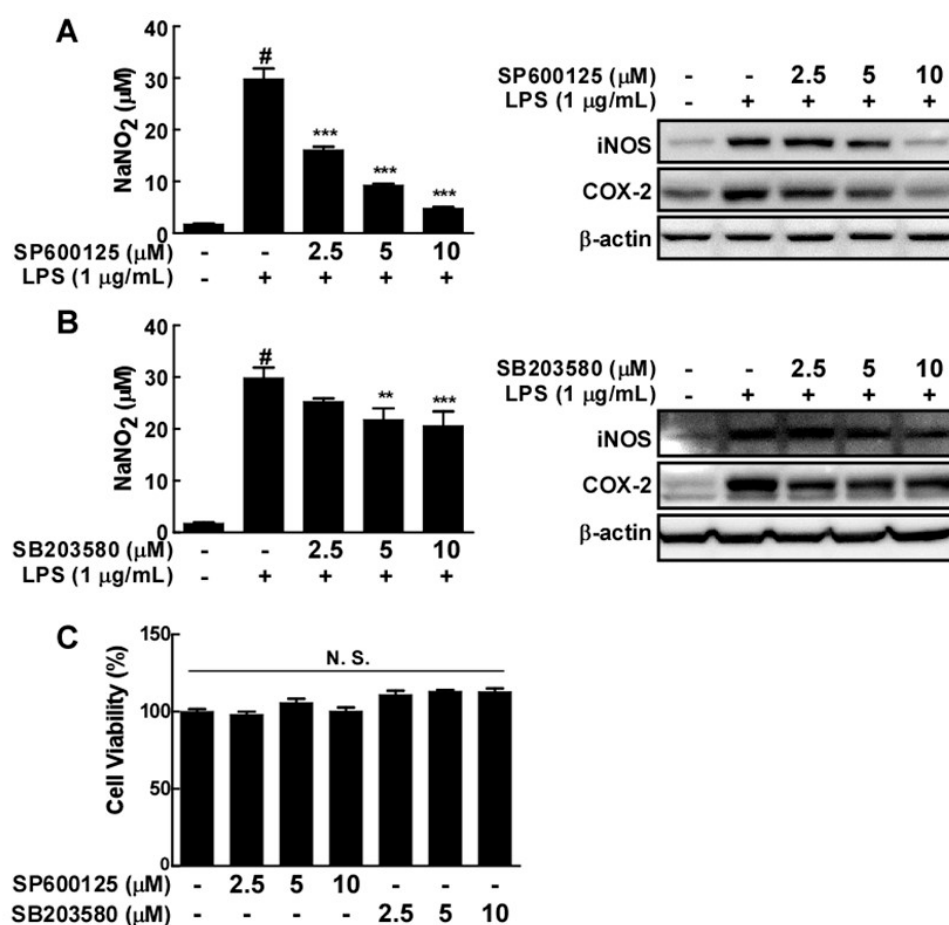
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11 Fig. S1. Effect of MAPK inhibitors on LPS-induced microglial activation in BV2 cells

12 Fig. S2. Effect of MAPK inhibitors, EJE on LPS-induced NF- κ B signaling in BV2 cells

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14 Supplementary Figure Legend



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16 **Fig. S1.** Effect of MAPK inhibitors on LPS-induced microglial activation in BV2 cells (A) JNK
 17 inhibition reduced LPS-induced NO production and iNOS, COX2 expression in dose-dependent
 18 manner. (B) p38 inhibition inhibited LPS-induced NO production and iNOS, COX2 expression in
 19 dose-dependent manner. (C) The inhibitors didn't affect cell viability. Cell viability were
 20 investigated with MTS assay, as indicated in the Materials and Methods. Hash symbols (#) indicate a
 21 significant difference (P < 0.001) between the control group and the group exposed to LPS alone;
 22 Asterisks (** and ***) indicate significant differences (P < 0.01 and < 0.001, respectively) between
 23 groups co-treated LPS and inhibitor and the group exposed to LPS alone. In this and all the following
 24 Figures, data are presented as the mean ± S.E.M. of three independent experiments.

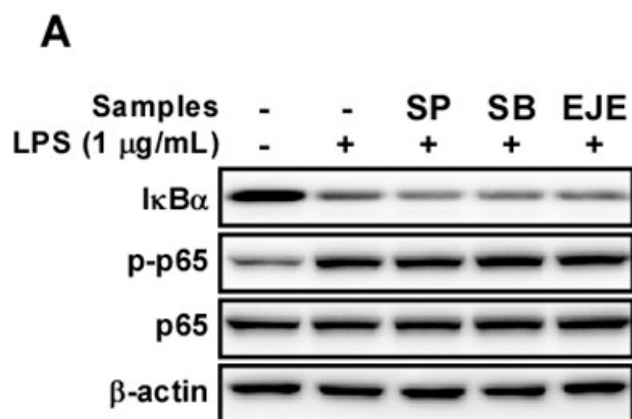
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31 **Fig. S2.** Effect of MAPK inhibitors, EJE on LPS-induced NF-κB signaling in BV2 cells (A) The treatment of
 32 JNK, p38 inhibitors and EJE didn't inhibit LPS-induced NF-κB signaling in BV2 cells at 30 mins after LPS
 33 treatment. SP (SP600125) and SB (SB203580) were treated at concentration of 10 µM. EJE was treated in
 34 BV2 cells at a concentration of 100 µg/mL. Expression and phosphorylation were detected with each specific
 35 antibody.