Ginsenoside Rg3 Mitigates LPS-Induced Injury in Human Bronchial Epithelial Cells by Restoring Autophagic Flux and Inhibiting the TLR4/NF-κB-Mediated Inflammatory Response

Supplementary Figures

Figure S1: G-Rg3 Regulates AMPK/mTOR Signaling Pathway Activation in LPS-Induced Inflammatory Models

(A) AMPK, (B) p-AMPK, (C) mTOR, and (D) p-mTOR. Cells were assigned to five treatment groups: control (Ctrl), ginsenoside Rg3 (Rg3), lipopolysaccharide (LPS), LPS + Rg3, and LPS + Rg3 + compound C (LPS+Rg3+CC); protein expression was detected via Western blot, with GAPDH (~37 kDa) as the loading control.

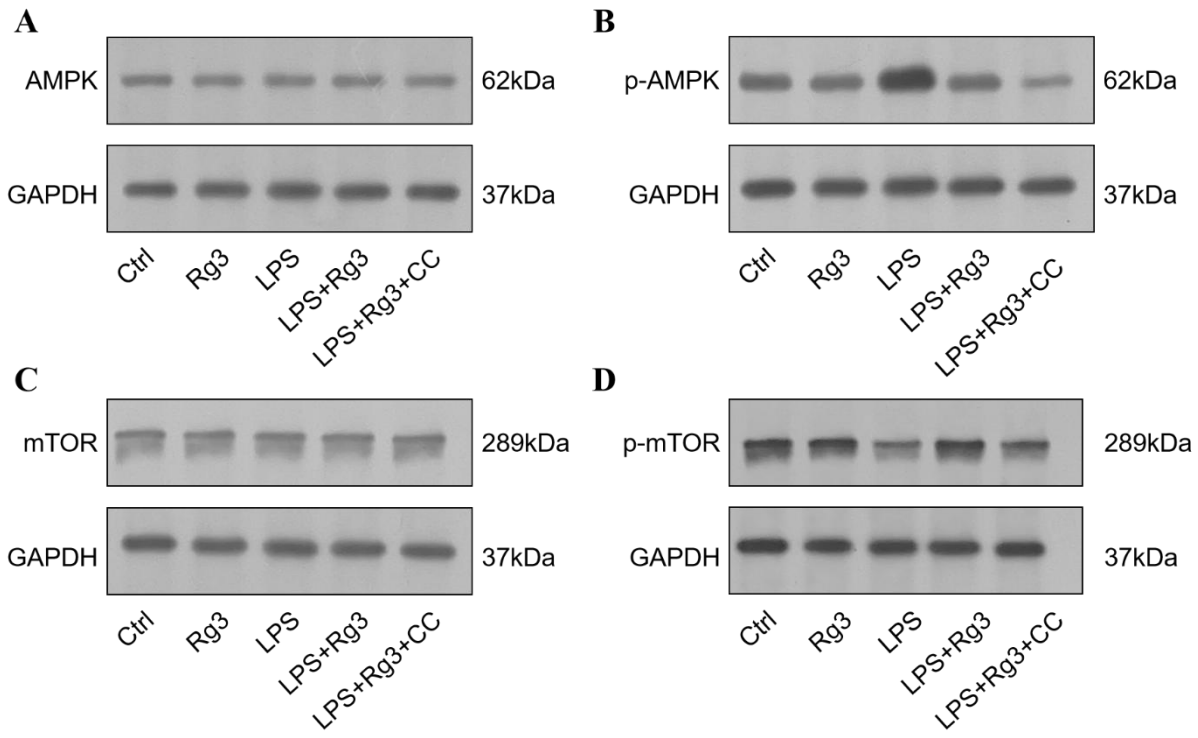

Figure S2: G-Rg3 Regulates NF-κB Signaling Pathway-Related Protein Expression in LPS-Induced Inflammatory Models

(A) p-p65, (B) p65, and (C) IκBα.Cells were assigned to five treatment groups: control (Ctrl), ginsenoside Rg3 (Rg3), lipopolysaccharide (LPS), LPS + Rg3, and LPS + Rg3 + compound C (LPS+Rg3+CC); protein expression was detected via Western blot with corresponding loading controls.

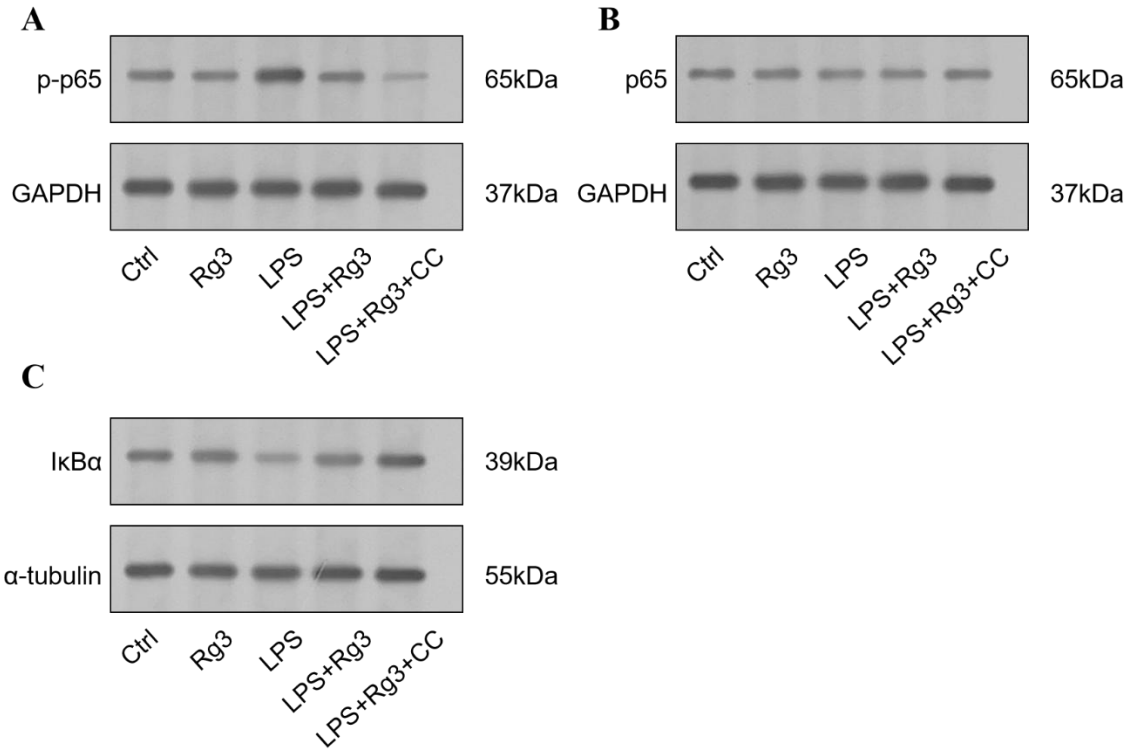

Figure S3: G-Rg3 Regulates Autophagy-Related Protein Expression in LPS-Induced Models (Including ATG5 Knockdown Intervention)

(A) ATG5, (B) LC3-II/I, (C) LC3-II/I, (D) SQSTM1/p62, (E) SQSTM1/p62.Cells were assigned to different treatment groups: For panels A, B, D: control (Ctrl), negative control siRNA (si-NC), ATG5-targeting siRNA (si-ATG5), lipopolysaccharide (LPS), LPS + ginsenoside Rg3 (LPS+Rg3), LPS+Rg3+si-ATG5; For panels C, E: Ctrl, Rg3, LPS, LPS+Rg3, LPS+Rg3+compound C (LPS+Rg3+CC). Protein expression was detected via Western blot with corresponding loading controls