

Figure S1. LPS induces the mRNA expression levels of *Irg-1* in BV2 cells. Cells were treated with LPS (1.5 $\mu\text{g/ml}$) for 5 h and ATP (3 mM) for an additional 1 h. The mRNA expression levels of *Irg-1* were measured by reverse-transcription quantitative PCR. Data are presented as the mean \pm SD of three independent experiments. *** $P < 0.001$. *Irg-1*, immune responsive gene 1; LPS, lipopolysaccharide.

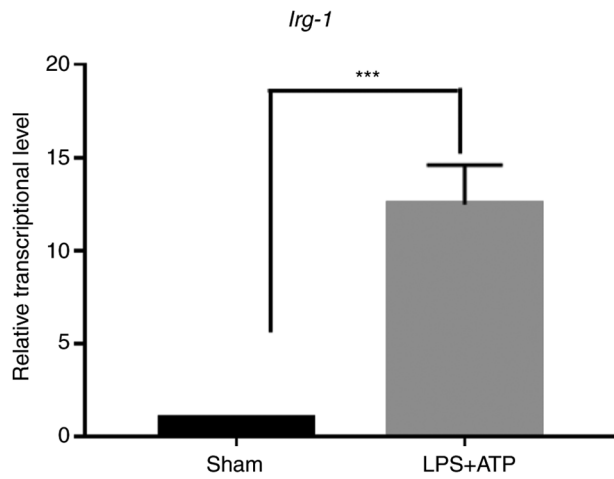


Figure S2. DI inhibits *nlrp3* expression in LPS-stimulated BV2 microglia. BV2 cells were treated with LPS (1.5 $\mu\text{g/ml}$) and ATP (3 mM) together with DI (200 μM) for 6 h. The mRNA expression levels of *nlrp3*, *nlrp1* and *aim2* were measured by reverse-transcription quantitative PCR. Data are presented as the mean + SD of three independent experiments. *** $P < 0.001$ vs. sham group; ### $P < 0.001$ vs. LPS + ATP group. *aim2*, absent in melanoma 2; DI, dimethyl itaconate; LPS, lipopolysaccharide; *nlrp*, NLR family pyrin domain-containing 3.

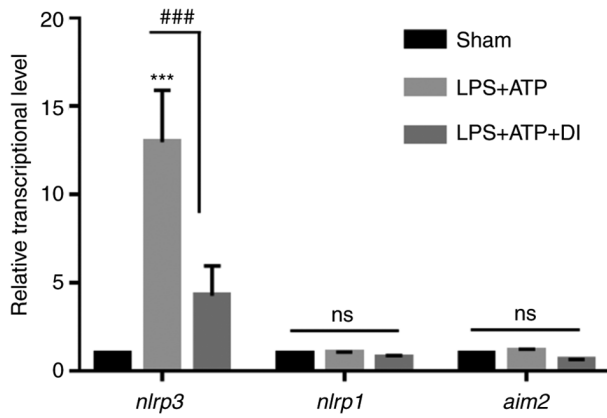


Figure S3. Blocking NLRP3 prevents cleavage of IL-1 β and GSDMD. (A) Effect of siRNA-NLRP3 on *nlrp3* expression levels were detected by reverse-transcription quantitative PCR. (B) Western blot analysis was performed. Protein expression levels of (C) NLRP3, (D) pro-IL1 β , (E) cleaved IL-1 β and (F) cleaved GSDMD were semi-quantified after siRNA-NLRP3 transfection. **P<0.01, ***P<0.001 vs. sham group or as indicated; #P<0.05, ##P<0.01, ###P<0.001 vs. LPS + ATP group or as indicated. GSDMD, gasdermin D; LPS, lipopolysaccharide; NLRP3, NLR family pyrin domain-containing 3; siRNA, small interfering RNA.

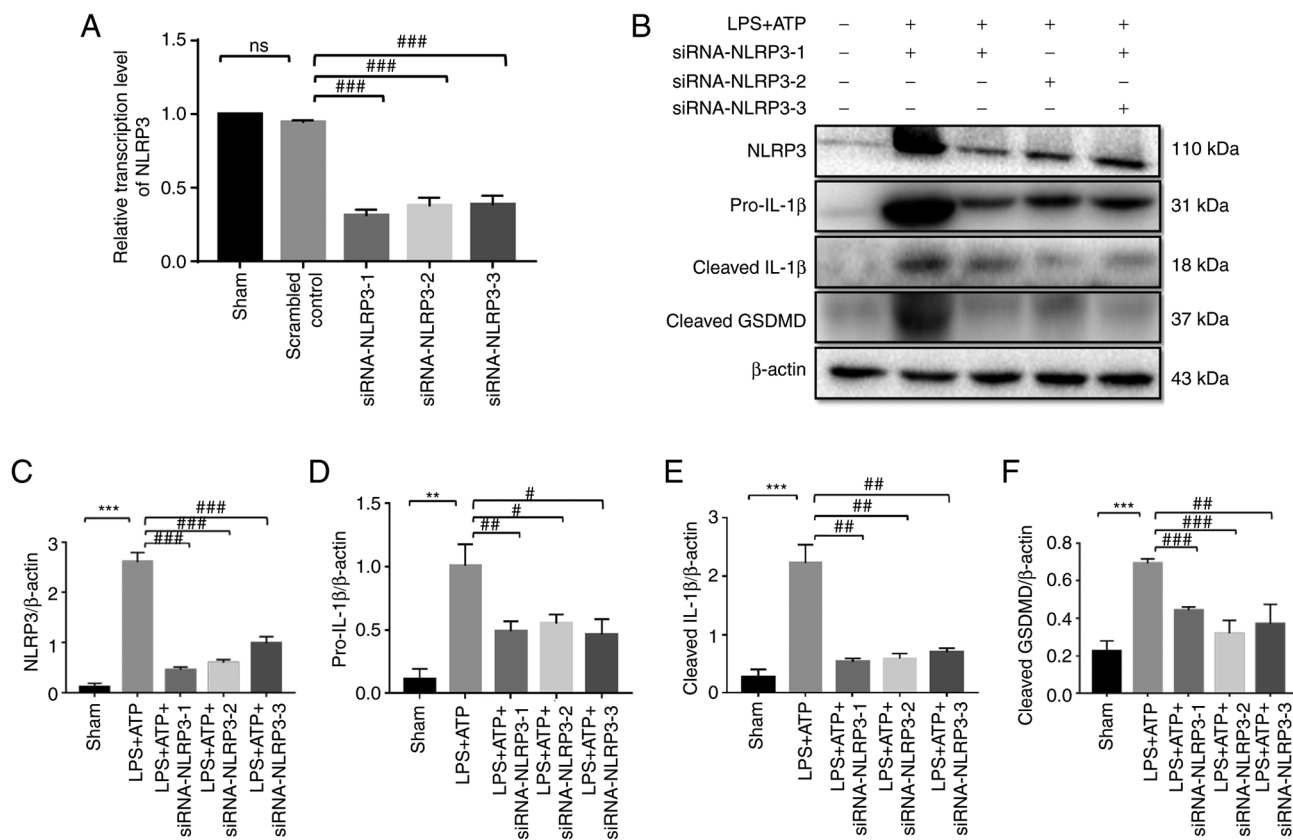


Figure S4. Cell morphology detection of microglia polarization. Cell morphology of microglia in (A) sham group, (B) LPS + ATP group and (C) LPS + ATP + DI group. Images were captured using a DMI8 microscope. DI, dimethyl itaconate; LPS, lipopolysaccharide.

