

Figure S1. Optimization of LPS concentration. Mouse macrophages in 24-wells were preincubated with 5 ng/ $\mu$ l human anti-TLR4 IgG2 for 2 h and then stimulated with different concentrations of LPS. mRNA expression levels of (A) *TNF- $\alpha$* , (B) *IFN- $\beta$*  and (C) *IL-6* were determined by reverse transcription-quantitative PCR and normalized to the internal control,  *$\beta$ -actin*. Data are presented as the mean  $\pm$  SD. N=3. \*P<0.05, \*\*P<0.01 vs. LPS control. LPS, lipopolysaccharide; TLR4, Toll-like receptor 4; L, LPS; A, human anti-TLR4 IgG2; IL, interleukin; IFN- $\beta$ , interferon- $\beta$ ; TNF- $\alpha$ , tumor necrosis factor- $\alpha$ .

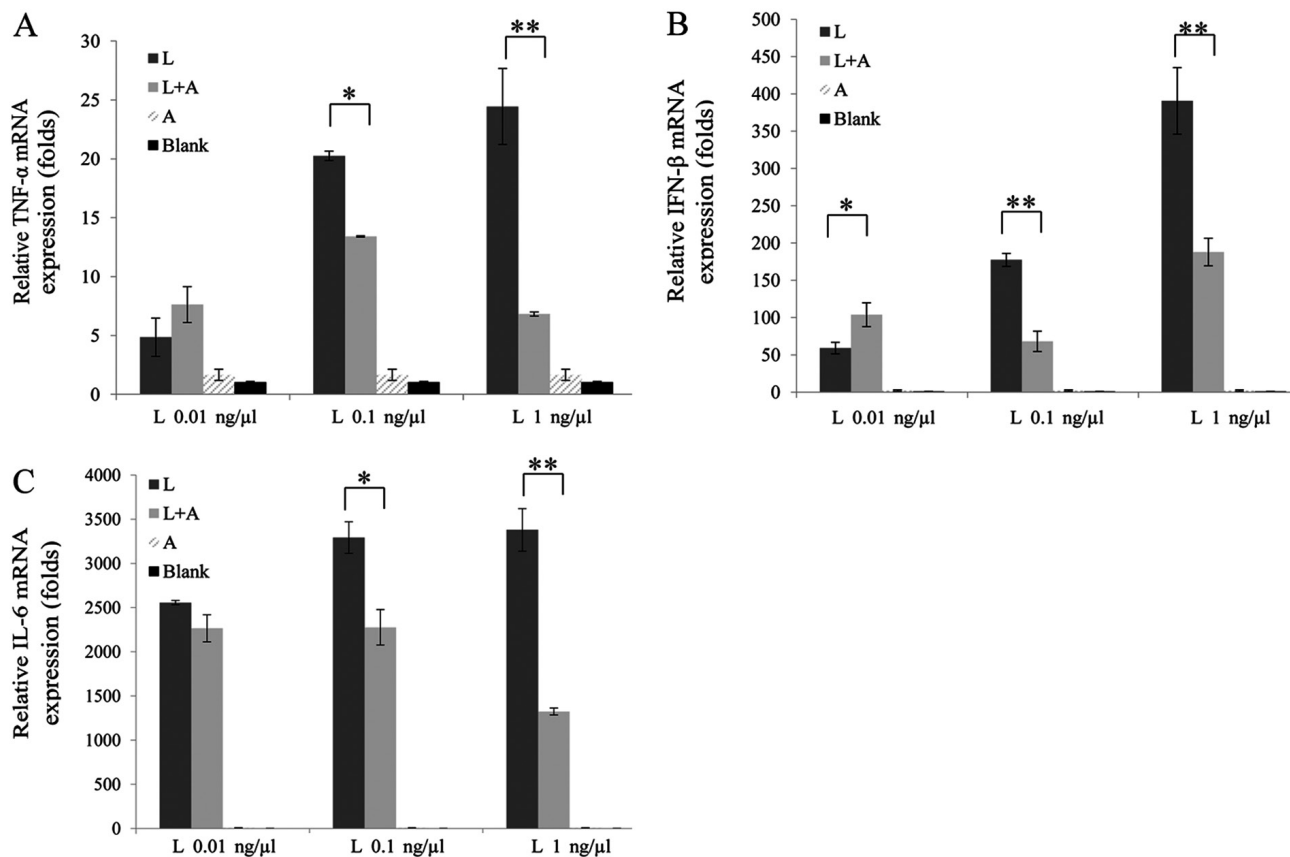


Figure S2. Human anti-TLR4 IgG2 inhibits LPS-induced sepsis in mice by decreasing serum proinflammatory cytokine levels. Mice were intravenously injected with human anti-TLR4 IgG2 (15  $\mu$ g/g) or an equal dose of PBS at 2 h before exposure to LPS (15  $\mu$ g/g). At 2 or 4 h after the LPS challenge, serum concentrations of (A) TNF- $\alpha$ , (B) IFN- $\beta$  and (C) IL-6 were determined by ELISA. N=3. \*\*\*P<0.001 vs. LPS control. LPS, lipopolysaccharide; TLR4, Toll-like receptor 4; L, LPS; A, human anti-TLR4 IgG2; IL, interleukin; IFN- $\beta$ , interferon- $\beta$ ; TNF- $\alpha$ , tumor necrosis factor- $\alpha$ .

