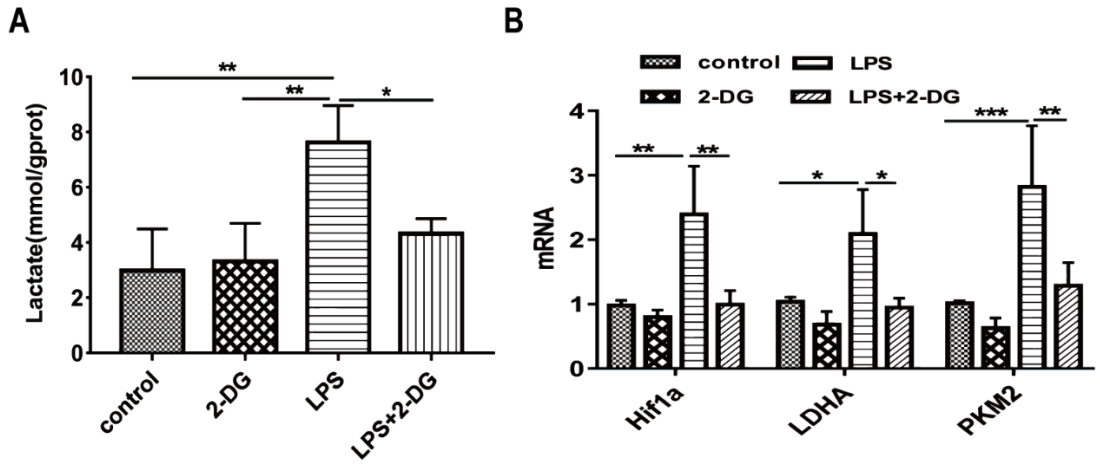
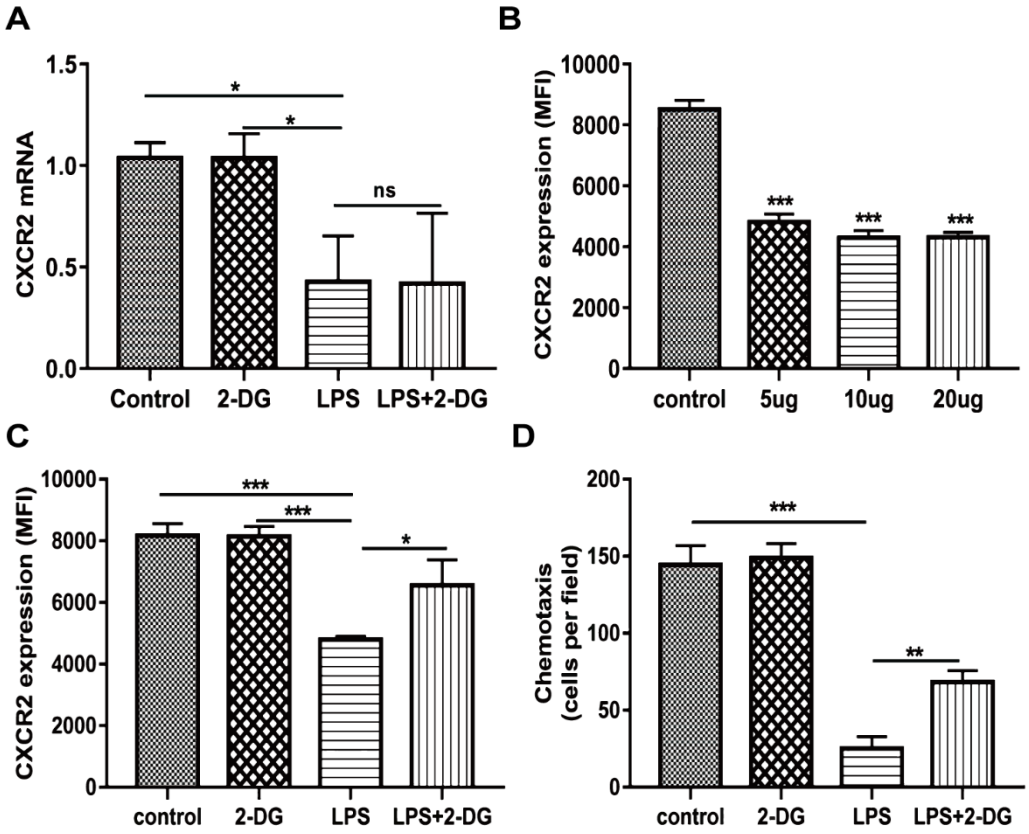
Supplementary Table1. The primers used for QPCR in this study.

Primers name	Forward (5'-3')	Reverse (5'-3')
Human-PKM2	ATTATTTGAGGAACTCCGCCGCCT	ATTCCGGGTCACAGCAATGATGG
Human-LDHA	ATCTTGACCTACGTGGCTTGGA	CCATACAGGCACACTGGAATCTC
Human-HIF-1α	TTGGCAGCAACGACACAGAAACTG	TTGAGTGCAGGGTCAGCACTACTT
Human-CXCR2	AAGGTGAATGGCTGGATTTTTG	CCCAGATGCTGAGACATATGAA
Human-18S	ATCCTCAGTGAGTTCTCCCG	CTTTGCCATCACTGCCATTA
Mouse- PKM2	TGTCTGGAGAAACAGCCAAG	TCCTCGAATAGCTGCAAGTG
Mouse-LDHA	AGAGCGGGAGGGCAGCTTTCT	GGGCAAGCTCATCCGCCAAGT
Mouse-HIF-1α	AGCTTCTGTTATGAGGCTCACC	TGACTTGATGTTCATCGTCCTC
Mouse-CXCR2	TCACAAACAGCGTCGTAGA	GACAGCATCTGGCAGAATAG
Mouse-actin	AGAAAATCTGGCACCACACC	CAGAGGCGTACAGGGATAGC



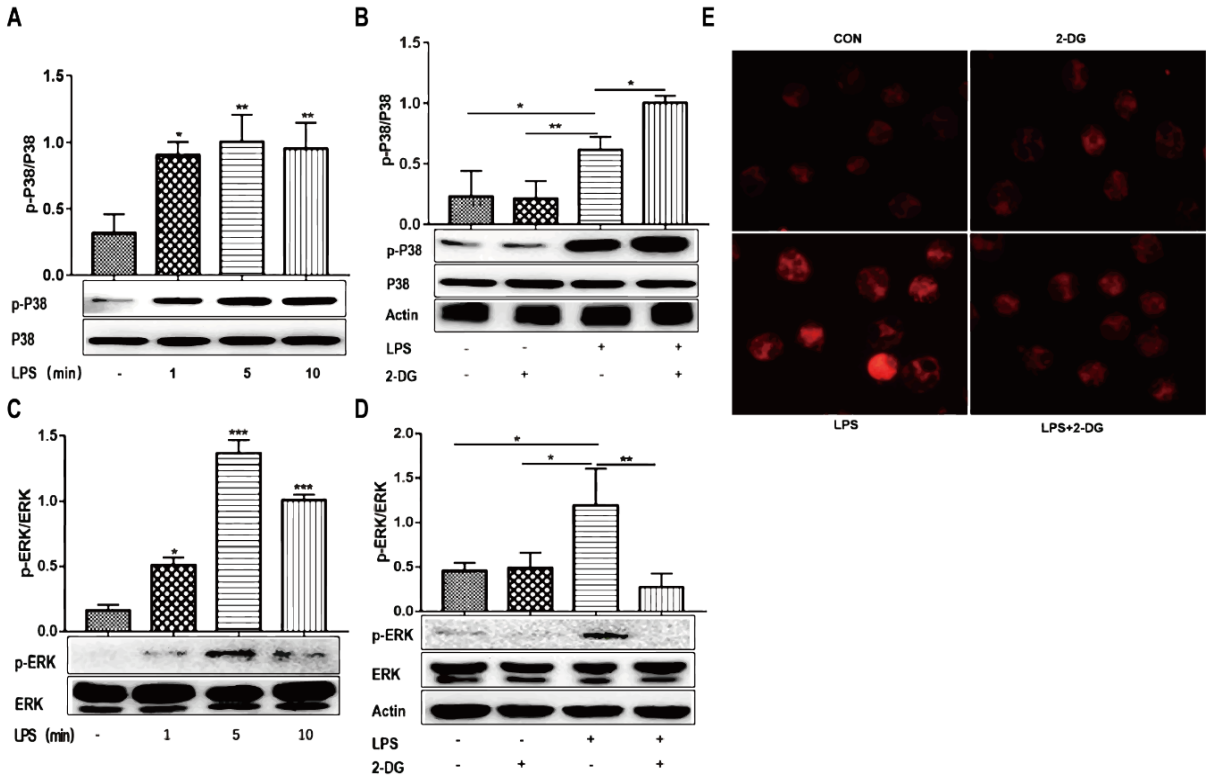
Supplementary Figure.1 Inhibition of aerobic glycolysis attenuates LPS-induced up-regulation of aerobic glycolysis and production of lactate in human neutrophils

(A). The levels of lactate in culture supernatant of human peripheral blood neutrophils were measured at 1h after stimulated by LPS in the presence/absence of 2-DG (2mM). (B). mRNA expression of glycolysis-related genes was analyzed by real time-PCR in human peripheral blood neutrophils at 1h after stimulated by LPS in the presence /absence of 2-DG. The data are means ± SEM, n=5 mice per group. *P < 0.05, **P<0.01, ***P<0.001 versus indicated groups.



Supplementary Figure.2 Inhibition of aerobic glycolysis attenuates LPS-induced impairment of neutrophil chemotaxis via reversing the downregulation of CXCR2 in human neutrophils

(A). The mRNA expression of CXCR2 was analyzed by RT-PCR in human peripheral blood neutrophils at 1h after stimulated by LPS (1μg/ml) in the presence/absence of 2-DG. (B). cell surface CXCR2 expression was analyzed by FACS in human peripheral blood neutrophils at 1h after stimulated with different concentration of LPS. *** P<0.001 versus control group. (C). The mean fluorescence intensity (MFI) of CXCR2 in human neutrophils stimulated by LPS (5μg/ml) in the presence/absence of 2-DG were also shown. (D). Human neutrophils were analyzed for chemotaxis at 1h after stimulated by LPS (1μg/ml) in the presence/absence of 2-DG. The data are means ± SEM, n=5 mice per group. *P < 0.05, **P<0.01, ***P<0.001 versus indicated groups.



Supplementary Figure.3 Inhibition of aerobic glycolysis attenuates the induction of GRK2 by LPS via inhibiting ERK phosphorylation and increasing P38 phosphorylation in human neutrophils.

(A, C). Immunoblot analysis (below) and quantification (above) of phosphorylated and total p38 (A) or ERK (C) in human neutrophils stimulated with LPS(1μg/ml) for 1, 5, 10min. *P < 0.05, **P<0.01 versus control group. (B, D). Immunoblot analysis (below) and quantification (above) of phosphorylated and total p38 (B) or ERK (D) in human neutrophils stimulated with LPS (1μg/ml) for 5min in presence/absence of 2-DG (2mM). (E). Immunofluorescence staining of GRK2 expression (red) in fixed human neutrophils and cultured for 1h with LPS (1μg/ml), 2-DG (2mM) or a combination of LPS and 2-DG. The data are means ± SEM, n=3 mice per group. *P < 0.05, **P<0.01, ***P<0.001 versus indicated groups.