

Supplementary Material

CD47 Potentiates Inflammatory Response in Systemic Lupus Erythematosus

Jin Kyun Park ^{1,2,†}, Ye Ji Lee ^{2,†}, Ji Soo Park ¹, Eun Bong Lee ^{1,2}, and Yeong Wook Song ^{1,2,*}

¹ Department of Molecular Medicine and Biopharmaceutical Sciences, Graduate School of Convergence Science and Technology and College of Medicine, Medical Research Center, Seoul National University, Seoul, Korea; jinkyunpark@snu.ac.kr (J.K.P.); vjisuev@gmail.com (J.S.P.); leb7616@snu.ac.kr (E.B.L.)

² Division of Rheumatology, Department of Internal Medicine, Seoul National University College of Medicine, Seoul, Korea; lyj901120@naver.com

* Correspondence: ysong@snu.ac.kr; Tel: + 82-2-2072-4765

† Both authors contributed equally to work

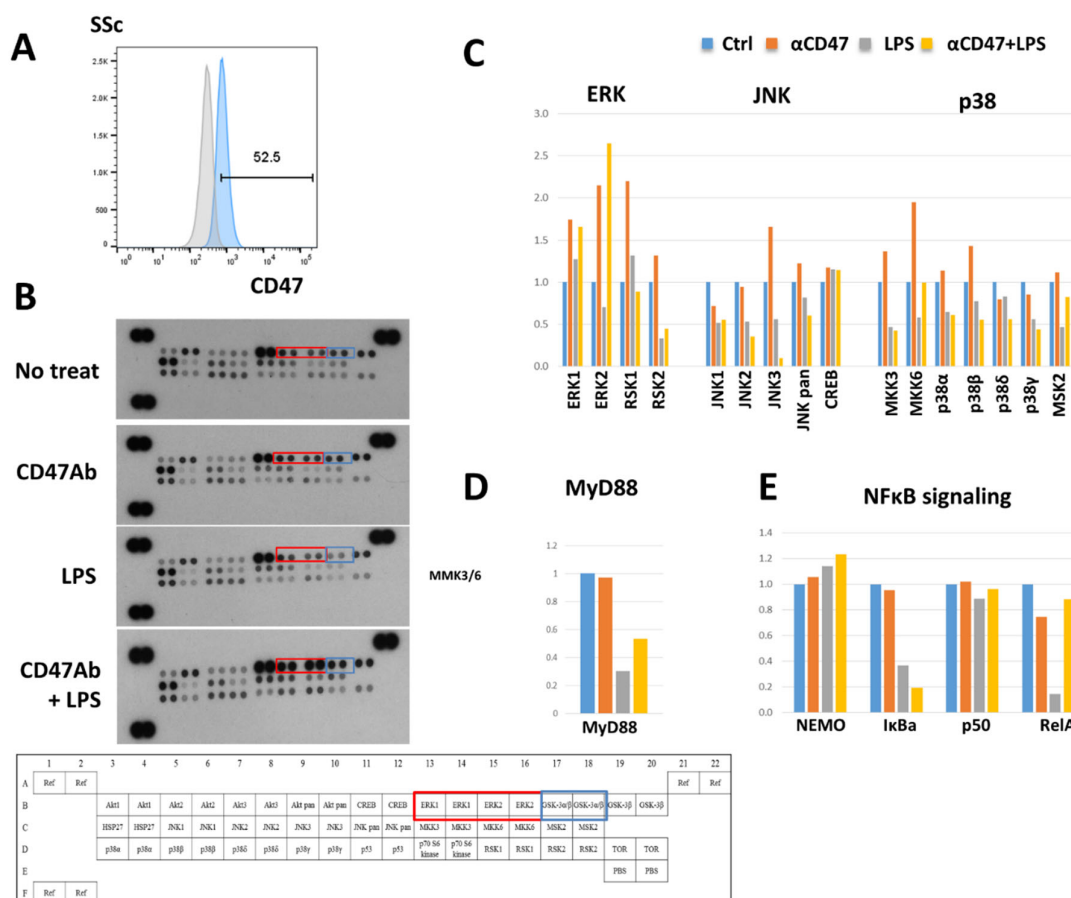


Figure S1. CD47 activation activates MAPK signaling. (A) Expression of CD47 on THP1 cells was examined by flow cytometry. (B,C) THP1 cells were treated with 1 µg/mL anti-CD47 antibody and/or 3 ng/mL LPS for 30 min and phospho-MAKP array was performed. (B) Image of phospho-MAKP-array blot with the array map (bottom panel) is shown. Phosphorylation of ERK (red rectangle) and GSK-3 (blue rectangle) is marked. (C) Densitometry analysis of the phospho-MAKP-array according to the 3 major arms of MAPK pathways (i.e., ERK, JNK, p38). (D,E) Densitometry analysis of the phospho-MyD88 and phospho-NFκB array after treatment with CD47 and/or LPS is shown. Phosphorylation is shown as fold change compared to no treatment. Ctrl, control; LPS, lipopolysaccharide; SSc, Side scatter.