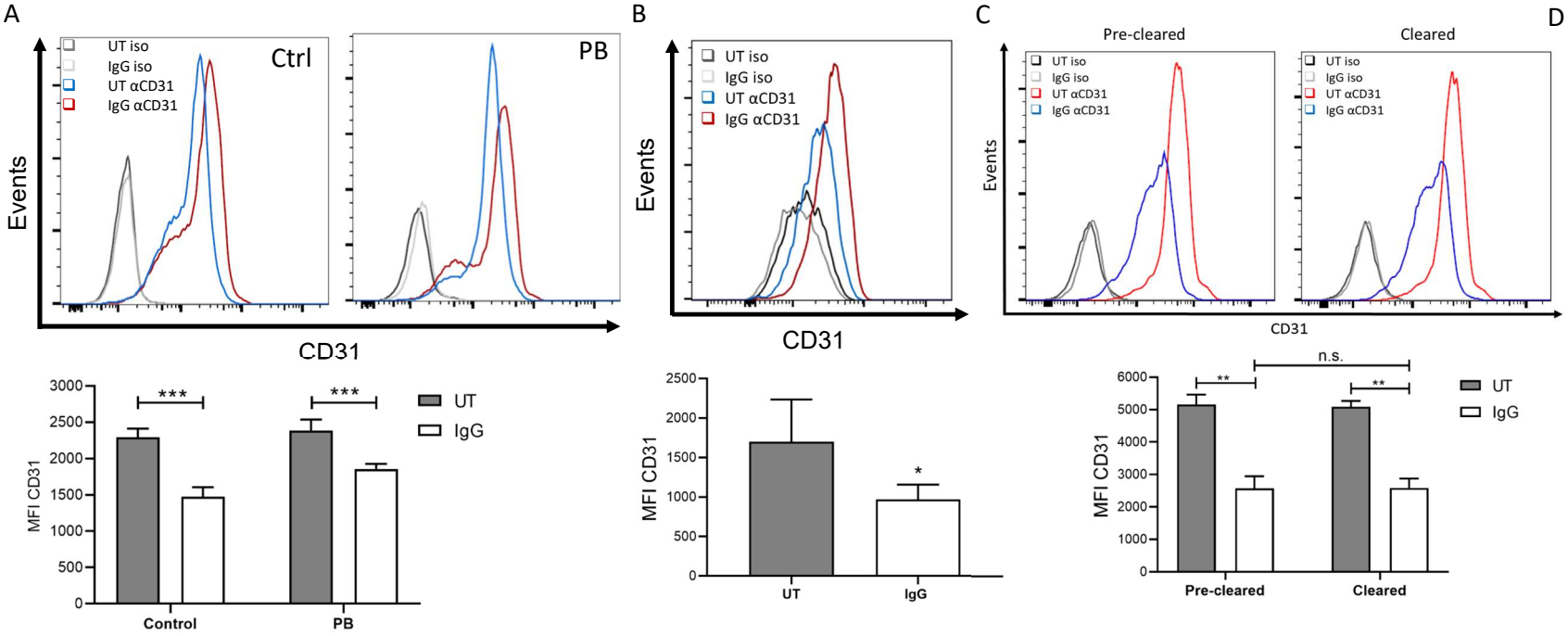
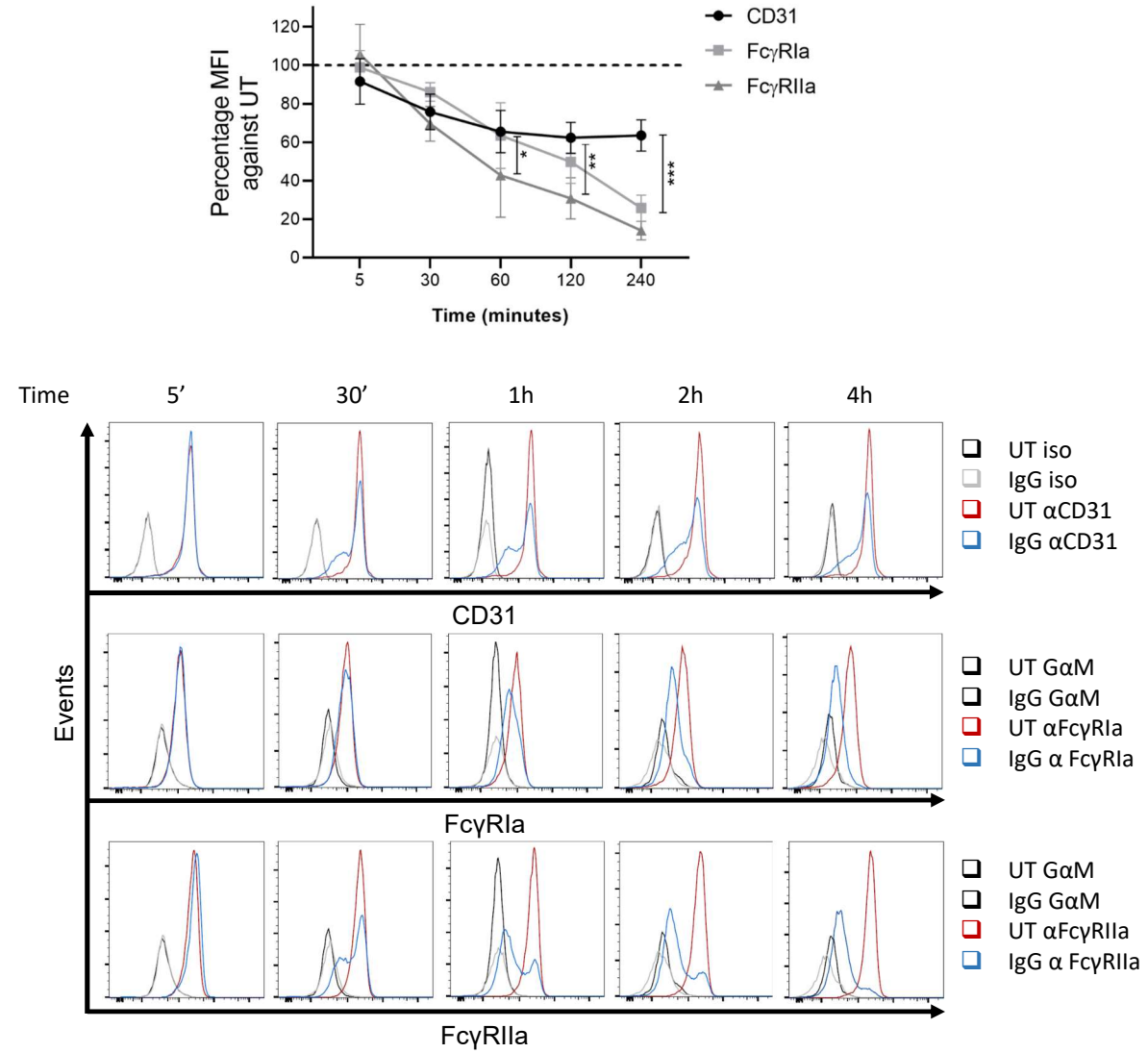


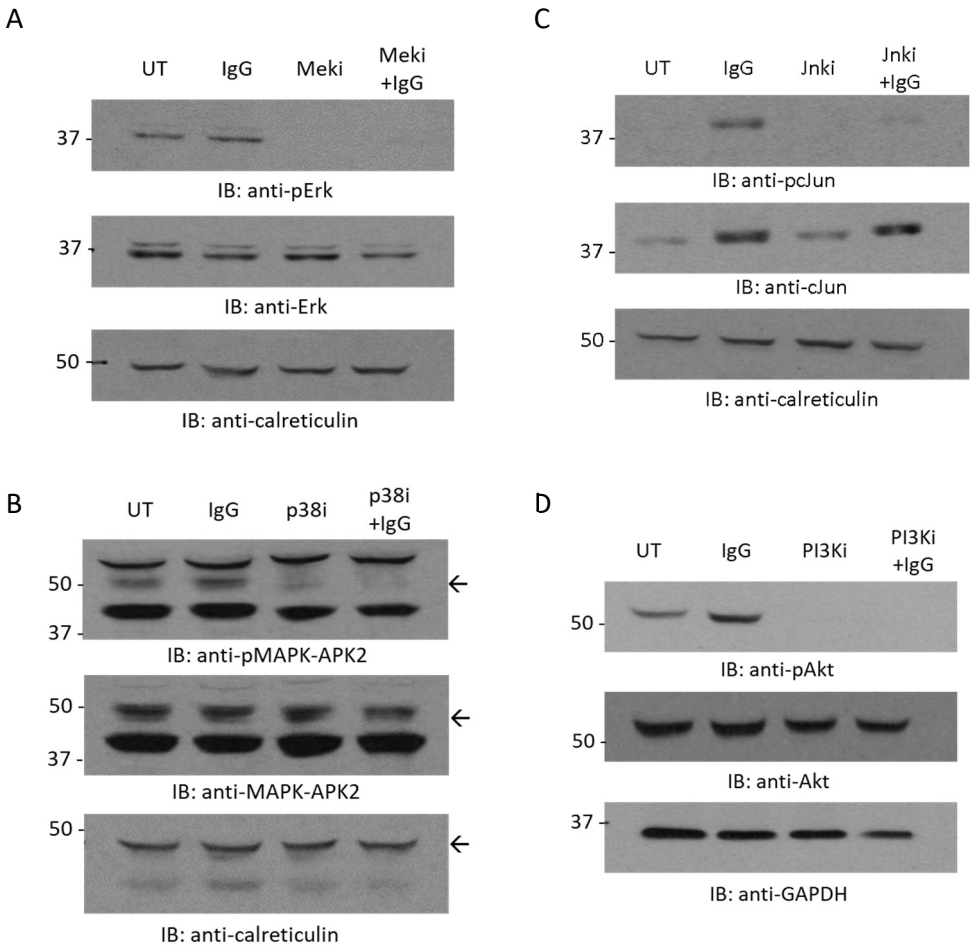
Endotoxin (EU/ml)	*Pre-cleared*	*Cleared*
PBS	<0.005	<0.005
IgG	0.638	0.152

Endotoxin (EU/ml)	*PBS, Pre-cleared*	*PBS, Cleared*
Donor 1	0.0304	0.0341
Donor 2	0.0360	0.0378
Donor 3	0.0300	0.0375
Mean	0.0321	0.0365
S.D.	0.0034	0.0021
	IgG, Pre-cleared	*IgG, Cleared*
Donor 1	0.0457	0.0352
Donor 2	0.0476	0.0307
Donor 3	0.0397	0.0461
Mean	0.0443	0.0373
S.D.	0.0041	0.0079

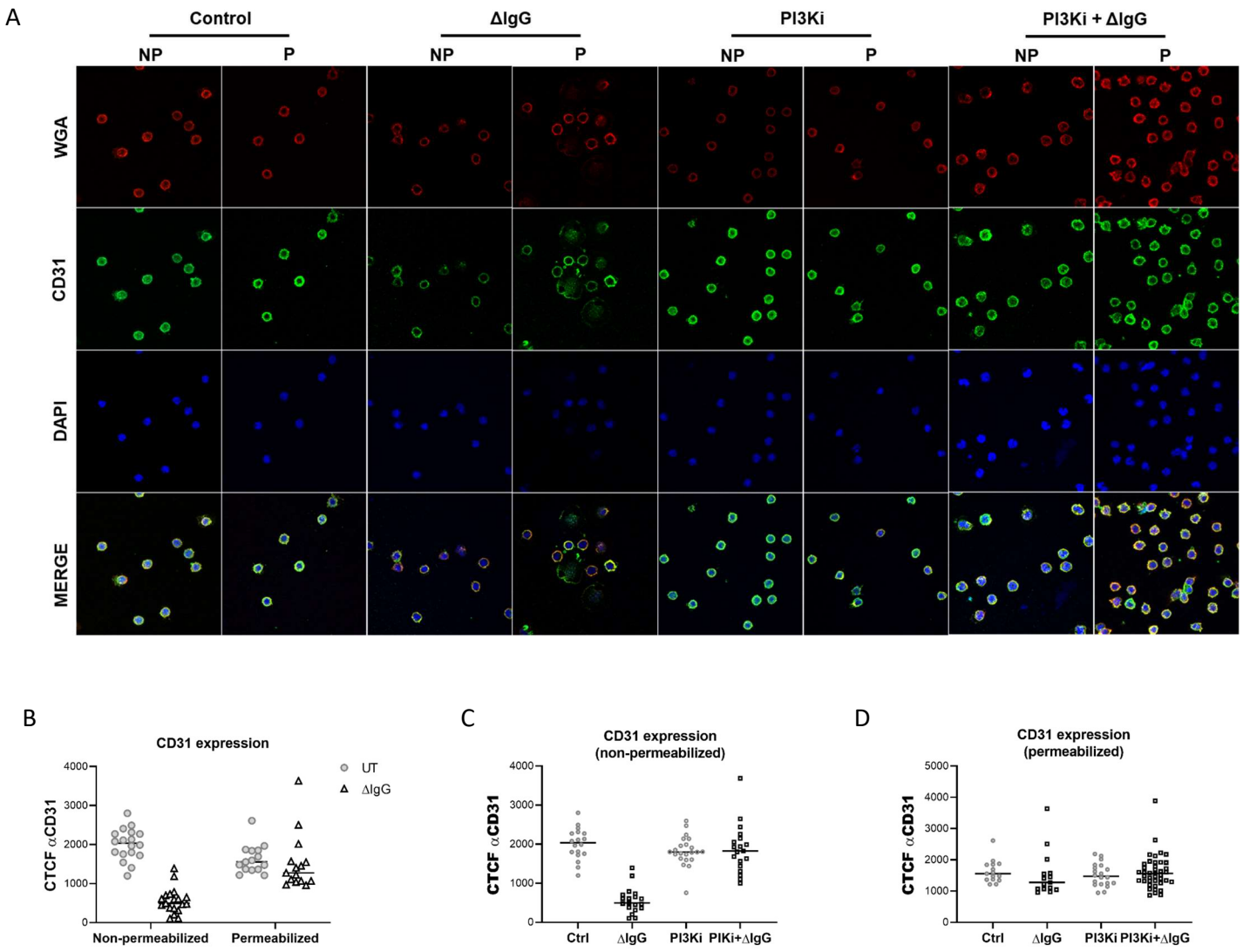
Supplementary Figure 1. CD31 modulation by FcγR is not influenced by LPS. (A-B) PBM were stimulated with plate-bound IgG for 24 hours with or without (Ctrl) polymyxin B (PB) at 10 µg/mL. (**A**) Surface expression of CD31 after stimulation was assessed by flow cytometry (representative histograms shown in the upper panel) and mean fluorescence intensities were quantified in the bar graph (bottom panel) (n=7). (**B**) **S**pontaneously-differentiated monocyte-derived macrophages were stimulated for 24 hours with plate-bound IgG and PB, and surface levels of CD31 were quantified by flow cytometry (representative histogram shown in the upper panel, with bar graph of all donors (n=5) in the below panel). **(C-D)** Next, to verify the polymyxin B results, PBM were left untreated (UT) or stimulated with plate-bound IgG either before (Pre-cleared) or after endotoxin removal (Cleared) for 24 hours. **(C)** Surface expression of CD31 after stimulation was assessed by flow cytometry (representative histograms shown in the top panel) and mean fluorescence intensities were quantified (bottom panel) (n=3). **(D)** Endotoxin levels were quantified in IgG stock before and after clearing (top table). Cleared media was collected from the samples after the 24-hour IgG incubation and also tested (bottom table). ** $p \leq 0.01$, *** $p \leq 0.001$, between stimulated and unstimulated (UT) cells.



Supplementary Figure 2. Internalization of CD31 and FcγR are different. PBM were stimulated with plate-bound IgG in presence of PB for the indicated time points. Percentage of surface levels against UT control of CD31, FcγRIa and FcγRIIa are shown (n=4). Representative histograms are shown below. * $p \leq 0.05$; ** $p \leq 0.01$; *** $p \leq 0.001$ against unstimulated (UT) control.



Supplementary Figure 3. Inhibition of FcγR-related signaling pathways. For all experiments shown in Figures 4 and 5, whole cell lysates were prepared to confirm inhibition with (**A**) U0126 (Meki; phospho-Erk) (**B**) SB203580 (p38i; phospho-MAPK-APK2); (**C**) SP60025 (Jnki; phospho-c-Jun) or (**D**) LY294002 (PI3Ki; phospho-Akt). Each blot was re-probed for levels of total signaling protein, or with either GAPDH or calreticulin as loading controls. Representative blots are shown. **UT** = unstimulated cells.



Supplementary Figure 4. PI3K is required for cytoplasmic localization of CD31. PBM were preincubated with the PI3K inhibitor LY294002 and PB for 45 minutes and then stimulated with ΔIgG for 4 hours. Cells were allowed to adhere to poly-L-lysine coverslips, then fixed and stained in non-permeabilizing (NP) or permeabilizing (P) conditions for CD31. DAPI and wheat germ agglutinin (WGA) were used to label the nuclei and the membrane, respectively. (**A**) Representative image from one of two donors. Corrected total cell fluorescence (CTCFs) quantification was done for (**B**) UT (unstimulated) and IgG controls in non-permeabilized and permeabilized conditions, as well as with the PI3K inhibitor in (**C**) non-permeabilized and (**D**) permeabilized conditions for one field in each of the two donors tested. Control UT and ΔIgG are from the same experiment as that in Fig. 4B.