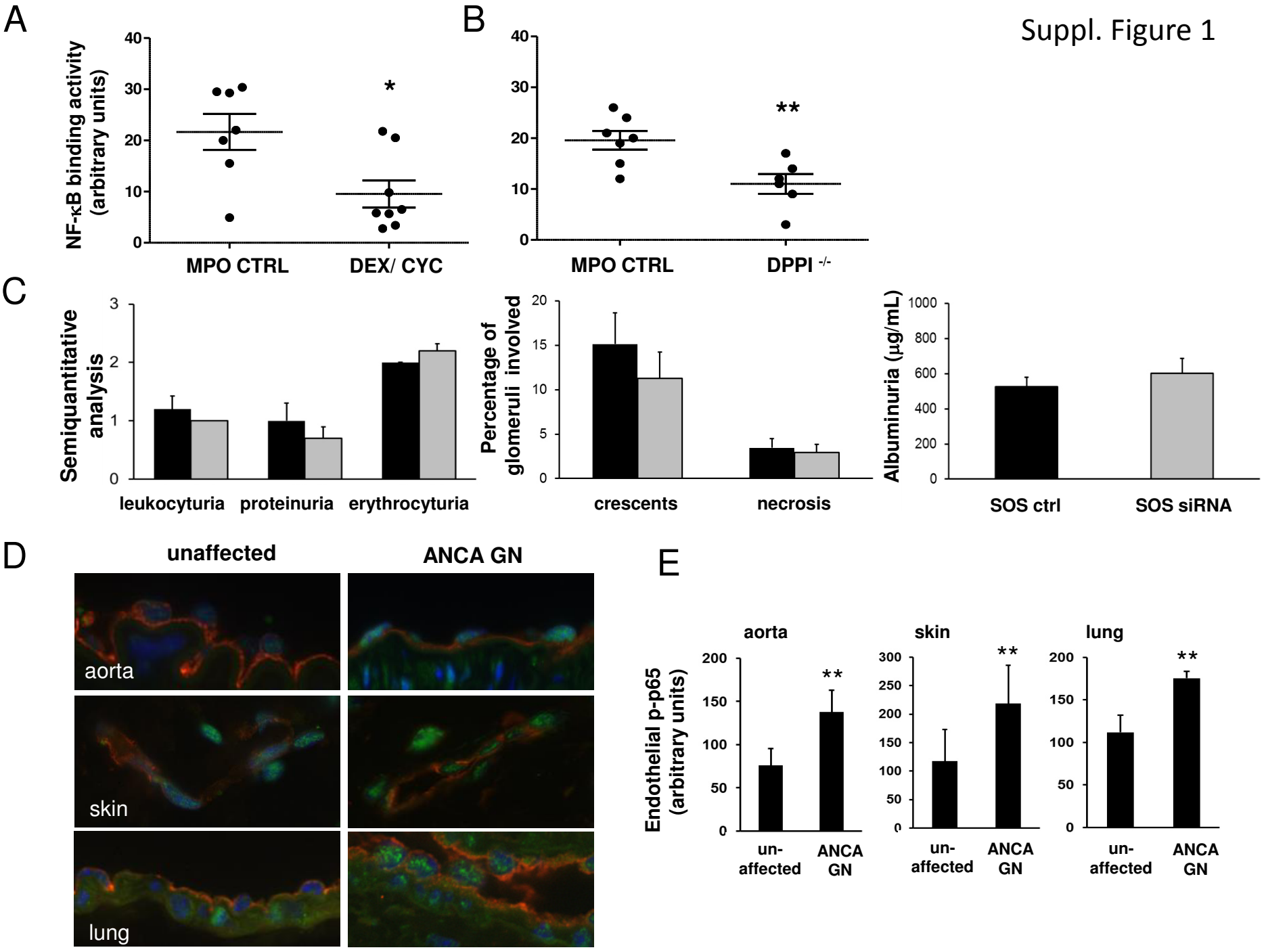
Suppl. Figure 1



Supplementary Figure 1. (A and B) NF-κB activity is diminished in renal tissue from mice with anti-MPO antibody-mediated NCGN that were either left untreated (CTRL, n=7) or treated with (A) dexamethasone/cyclophosphamide (DEX/CYC, n=8)[27], or were (B) deficient in myeloid dipeptidyl peptidase 1 ($DPPI^{-/-}$, n=6)[28], respectively. Optical density of the p50/p65 heterodimer was assessed in nuclear extracts prepared from renal tissue, subjected to EMSA, and expressed as arbitrary units. (C) Application of SOS p65 siRNA without prior TNFα and after anti-MPO antibody and LPS administration did not provide protection from NCGN. Semiquantitative dipstick urine analysis, albumin ELISA, and histology with necrosis and crescents quantification from anti-MPO antibody-treated mice that received SOS control liposomes (black bars, n=5) or SOS p65 siRNA is provided (gray bars, n=5). (D, E) Endothelial NF-κB is activated in vascular beds of aorta, skin, and lung in mice with anti-MPO antibody-induced NCGN from our current study. Co-staining using fluorescence-labeled antibodies to the EC marker CD31 (red), phospho-p65 (green), and DAPI staining for nuclei (blue) was performed (n=3). Typical microscopy examples (high power field) together with the corresponding quantitative assessment of nuclear phospho-p65 staining within CD31-positive glomerular EC using Image J is depicted. $^{**}p<0.01$, $^{*}p<0.05$.