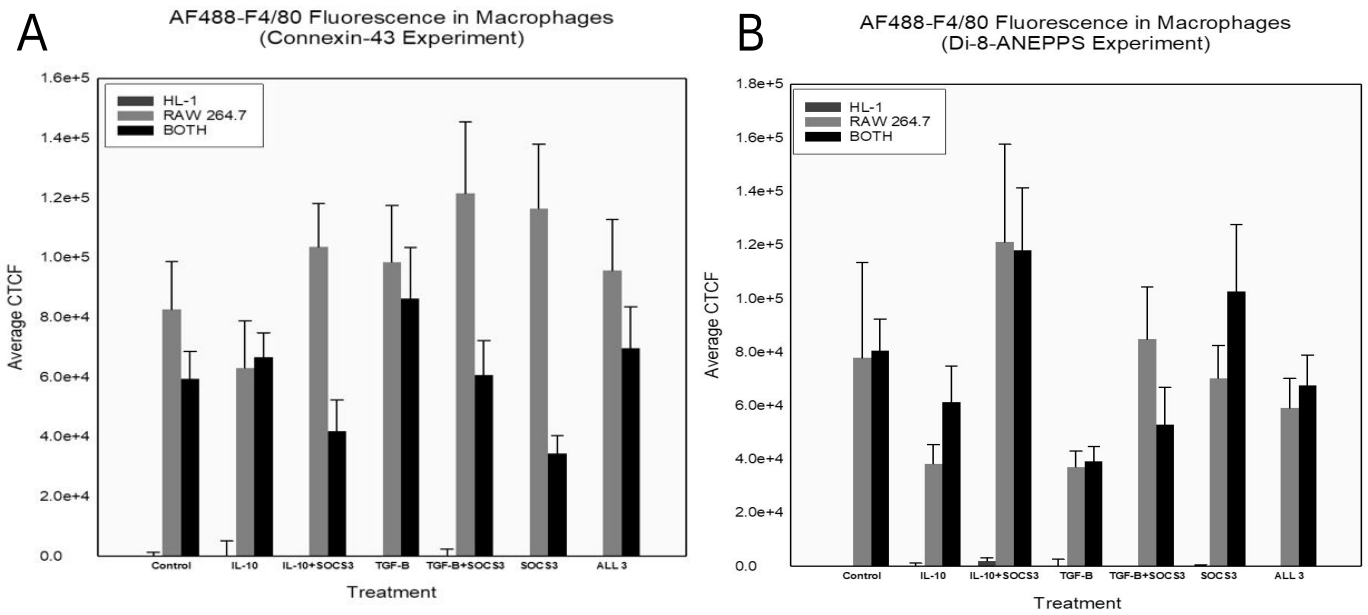
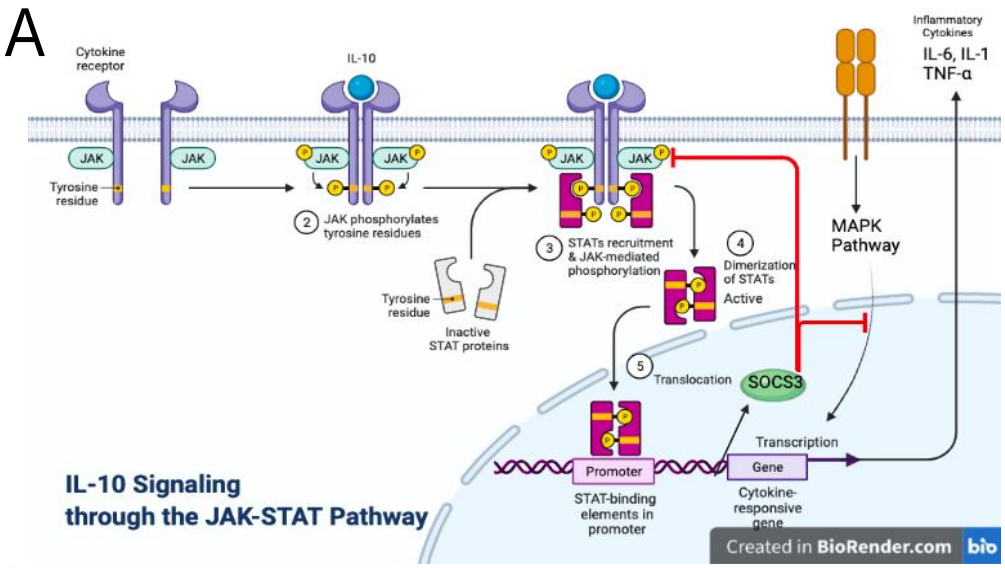
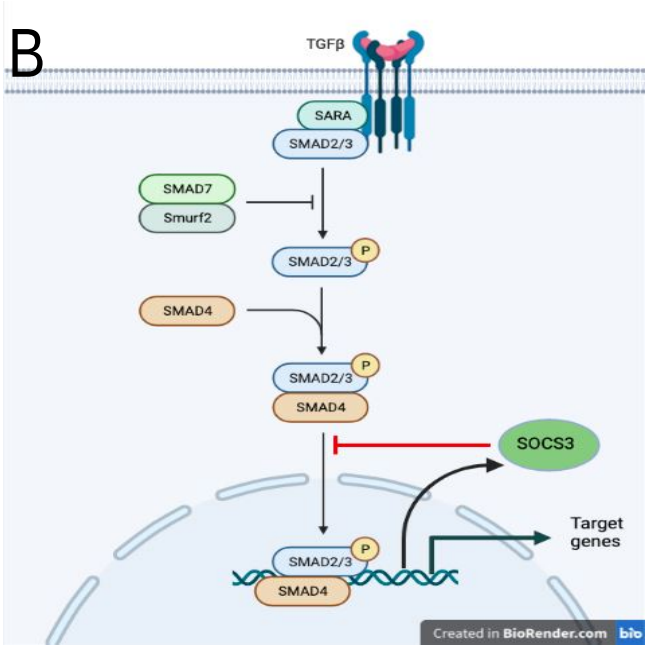
Supplementary Figures



- *S1: Confirmation of AF488-F4/80 Fluorescence in Macrophages for Connexin-43 Experiment and Di-8-ANEPPS Experiment*
  AF488-F4/80 fluorescence shows presence of macrophages. To ensure macrophages are in their respective wells (RAW 264.7 and BOTH), fluorescence was compared between the three cell types (e.g., HL-1, RAW 264.7 and BOTH) .
  A. For the connexin-43 experiments, F4/80 fluorescence can be seen in RAW 264.7 wells and BOTH wells. As indicated by lack of fluorescence, macrophages are not present in HL-1 wells. CTCF=corrected total cell fluorescence. Each bar represents the mean CTCF and error bars represent the standard error for each group.
  B. For the Di-8-ANEPPS experiments, F4/80 fluorescence can be seen in RAW 264.7 wells and BOTH wells. As indicated by lack of fluorescence, macrophages are not present in HL-1 wells. CTCF=corrected total cell fluorescence. Error bars=standard error.





- *S2: The Regulatory Role of SOCS3 in the IL-10 Signaling Pathway and the TGF-beta Signaling Pathway*
  - A. IL-10 signaling through the JAK/STATpathway shows how IL-10 is capable of causingtranscription of various inflammatory cytokines but that it also causes transcription of SOCS3. SOCS3 inhibits production of inflammatory cytokines (by blocking the MAPKpathway) as well as inhibiting IL-10 via blocking the phosphorylation of JAK. Created in BioRender - "IL-10 Signaling through the JAK-STATPathway" template.
  - B. Binding of TGF-beta1 to its receptor causes SMAD3 to phosphorylate and create a complexwith SMAD4. This complex then translocates to the nucleus, binds to the promotor and activates transcription of inflammatory cytokines, regulatory cytokines and SOCS3. SOCS3 inhibits the SMAD3/4 complex from translocating to the nucleus. Created in BioRender - "TGF-beta1 Signaling Pathway" template.