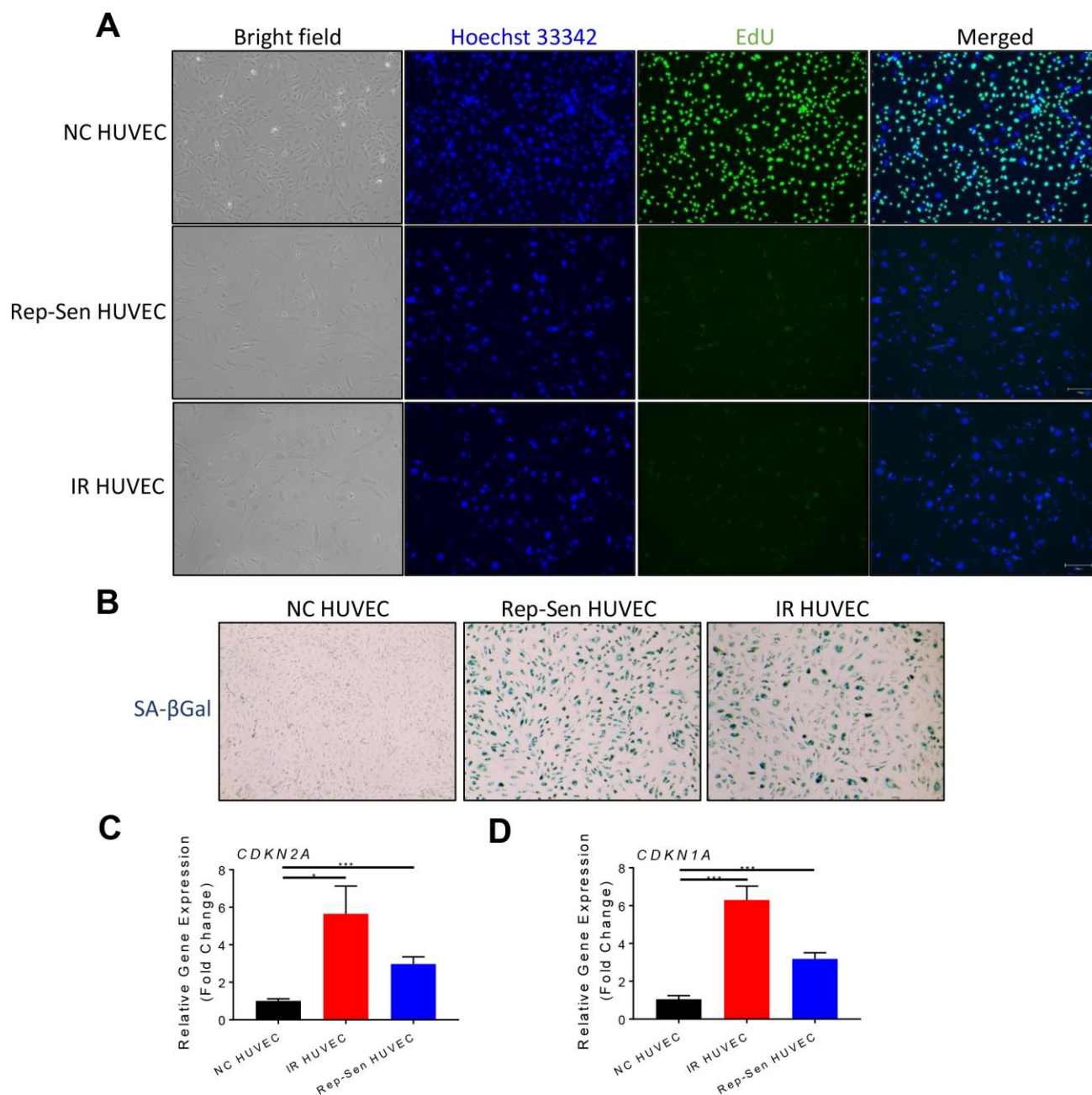
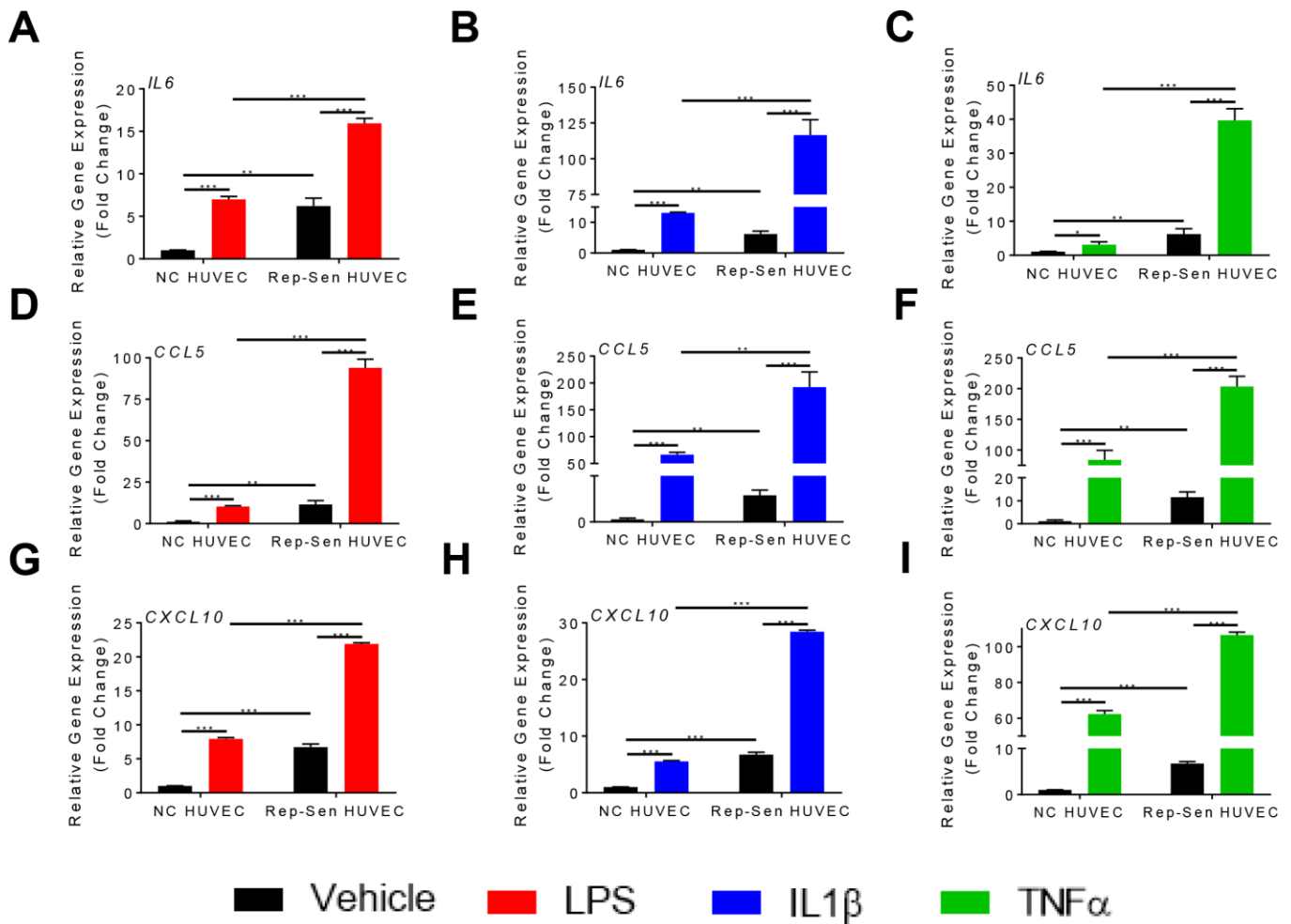


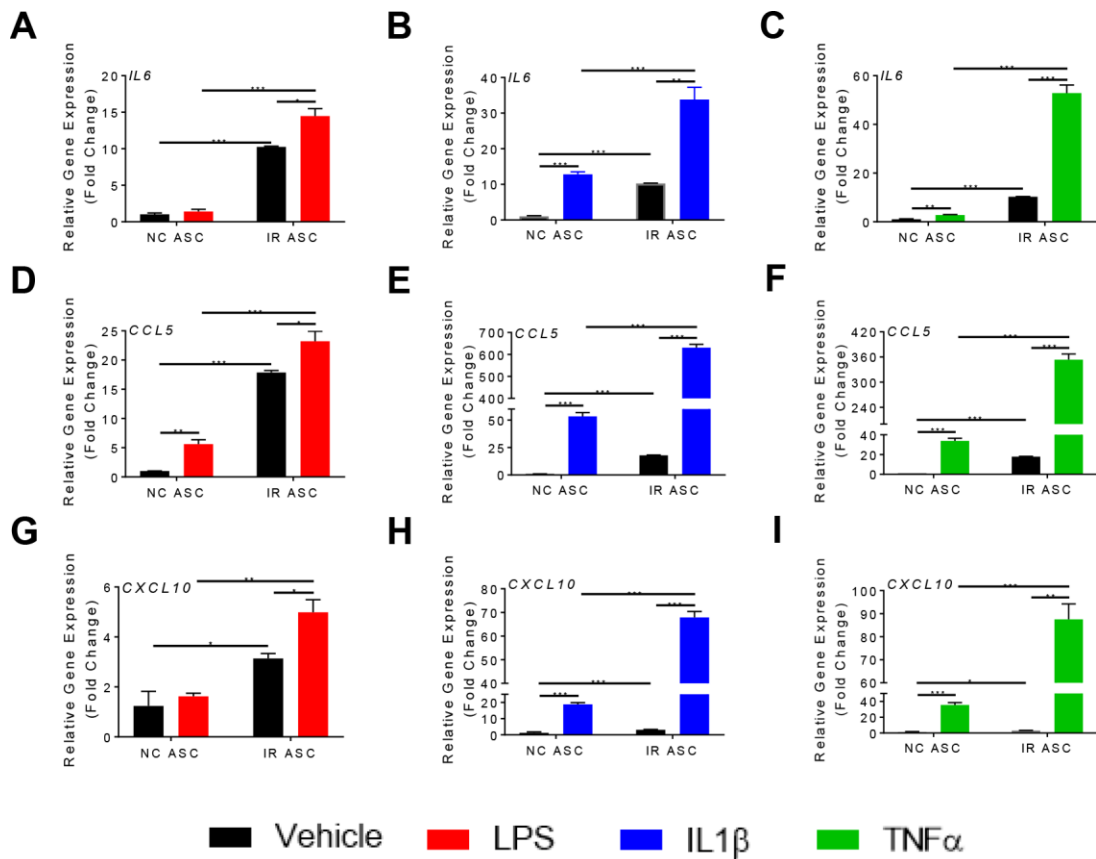
SUPPLEMENTARY FIGURES



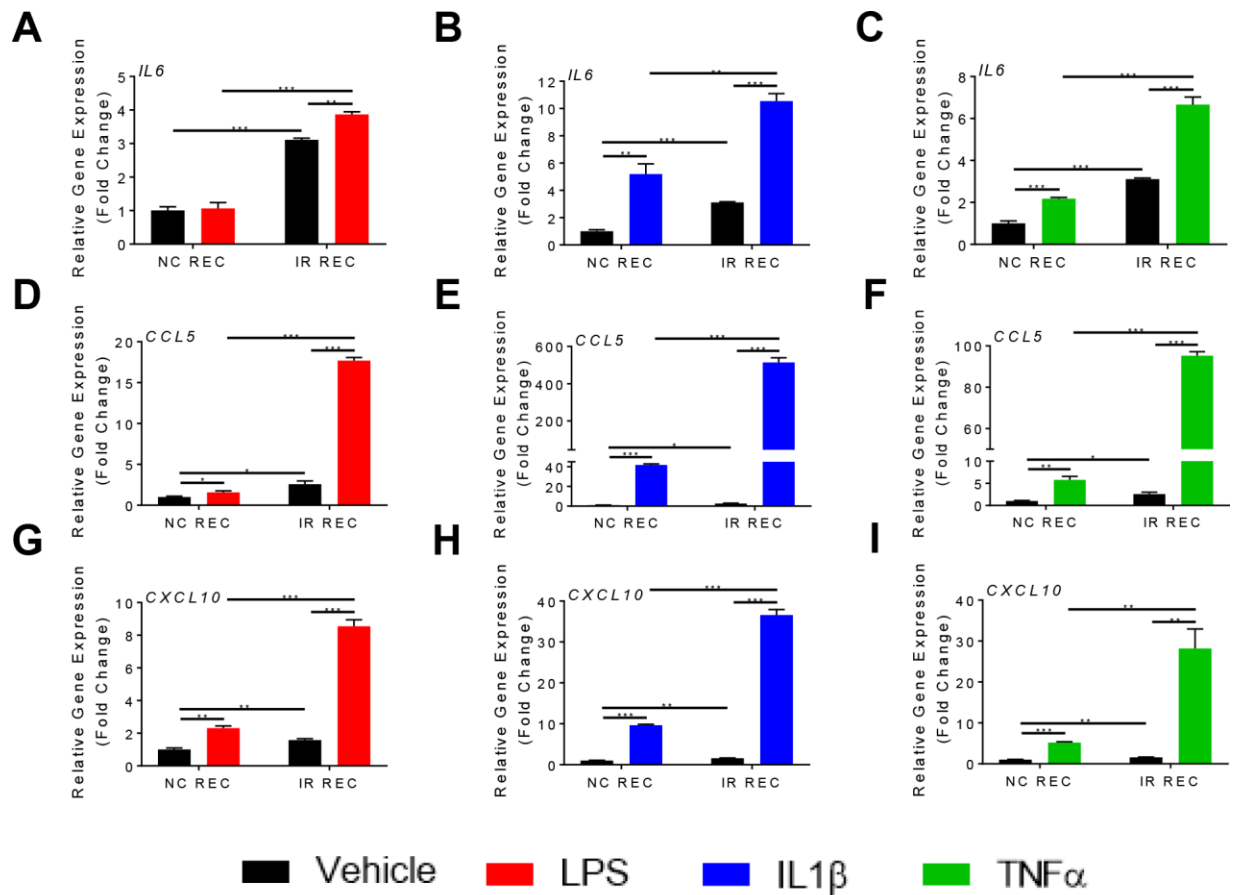
Supplementary Figure 1. Validation of HUVEC senescence. (A) Representative images of non-senescent (NC HUVEC), IR-induced senescent HUVECs (IR HUVEC) and replicative senescent HUVECs (Rep-Sen HUVEC) stained for EdU to analyze cell cycle arrest. Nuclei were revealed with Hoechst 33342 staining. (B) Representative images of NC HUVEC, IR HUVEC and Rep-Sen HUVEC stained for senescence associated β -galactosidase (SA- β -Gal; blue color). (C, D) Relative mRNA expression of *CDKN2A* (C) and *CDKN1A* (D) in NC HUVEC, IR HUVEC and Rep-Sen HUVEC. Gene expression in unstimulated NC HUVEC was used as baseline and *GAPDH* was used as endogenous control (n = 3; mean \pm SEM; * p<0.05, *** p<0.001).



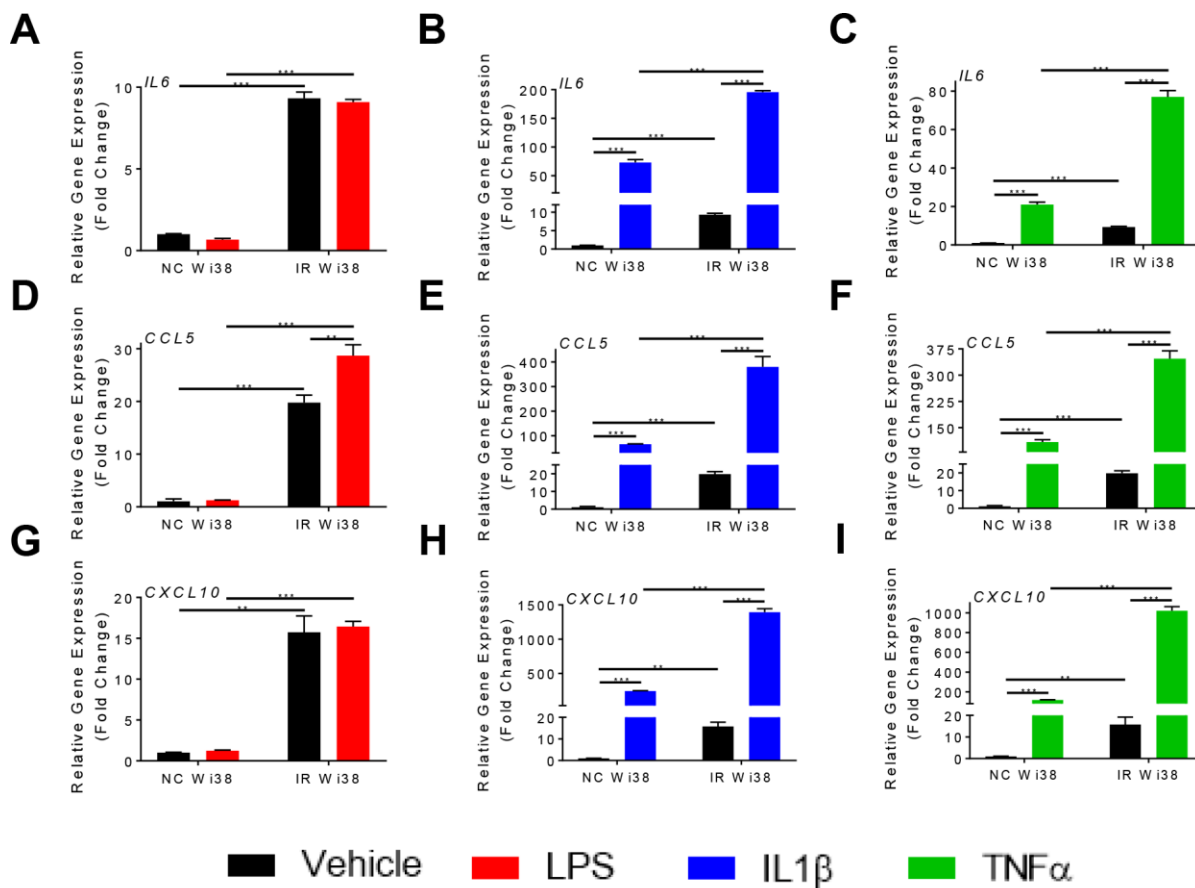
Supplementary Figure 2. Transcriptional response of NC HUVEC and Rep-Sen HUVEC to LPS, IL1 β and TNF α stimulation. Relative change in mRNA expression of *IL6* (A–C), *CCL5* (D–F), and *CXCL10* (G–I) in NC HUVEC and Rep-Sen HUVEC stimulated with 30 ng/mL LPS (A, D, F), 3 ng/ml IL1 β (B, E, H), and 3 ng/ml TNF α (C, F, I) for 3 hours. Gene expression in unstimulated NC HUVEC was used as baseline and *GAPDH* was used as endogenous control (n = 3; mean \pm SEM; * p<0.05, ** p<0.01, *** p<0.001).



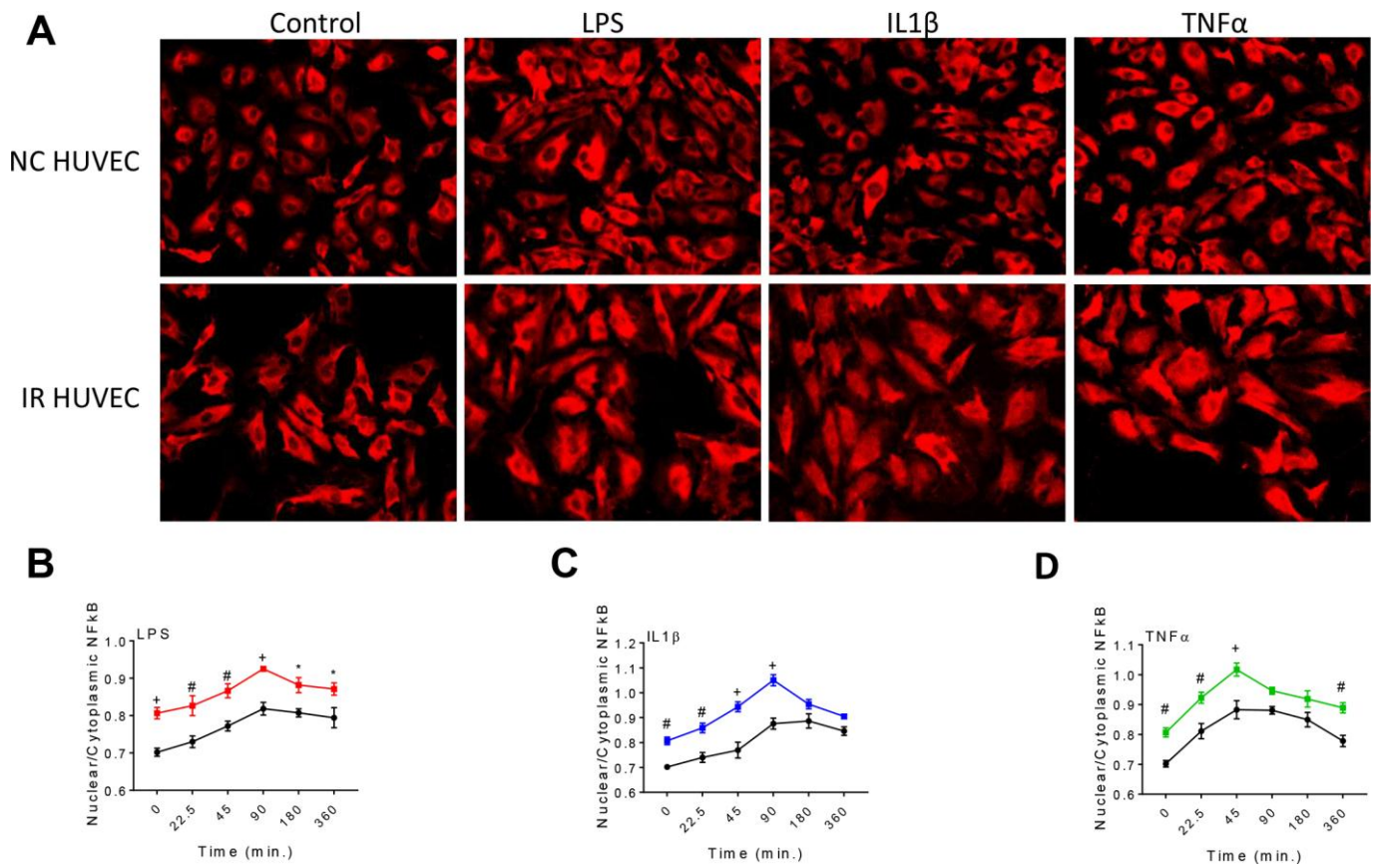
Supplementary Figure 3. Transcriptional response of non-senescent adipose stem/stromal cells (NC ASC) and IR-induced senescent ASC (IR ASC) to LPS, IL1 β and TNF α stimulation. Relative change in mRNA expression of *IL6* (A–C), *CCL5* (D–F), and *CXCL10* (G–I) in NC ASC and IR ASC stimulated with 30 ng/mL LPS (A, D, F), 3 ng/ml IL1 β (B, E, H), and 3 ng/ml TNF α (C, F, I) for 3 hours. Gene expression in unstimulated NC ASC was used as baseline and *GAPDH* was used as endogenous control (n = 3; mean \pm SEM; * p<0.05, ** p<0.01, *** p<0.001).



Supplementary Figure 4. Transcriptional response of non-senescent human renal epithelial cells (NC REC) and IR-induced senescent RECs (IR REC) to LPS, IL1β and TNFα stimulation. Relative change in mRNA expression of *IL6* (A–C), *CCL5* (D–F), and *CXCL10* (G–I) in NC REC and IR REC stimulated with 30 ng/mL LPS (A, D, F), 3 ng/ml IL1β (B, E, H), and 3 ng/ml TNFα (C, F, I) for 3 hours. Gene expression in unstimulated NC REC was used as baseline and *GAPDH* was used as endogenous control (n = 3; mean ± SEM; * p<0.05, ** p<0.01, *** p<0.001).



Supplementary Figure 5. Transcriptional response of non-senescent human WI38 lung fibroblast cells (NC WI38) and IR-induced senescent WI38 cells (IR WI38) to LPS, IL1 β and TNF α stimulation. Relative change in mRNA expression of *IL6* (A–C), *CCL5* (D–F), and *CXCL10* (G–I) in NC WI38 and IR WI38 stimulated with 30 ng/mL LPS (A, D, F) 3 ng/ml IL1 β (B, E, H), and 3 ng/ml TNF α (C, F, I) for 3 hours. Gene expression in unstimulated NC WI38 was used as baseline and *GAPDH* was used as endogenous control (n = 3; mean \pm SEM; * p<0.05, ** p<0.01, *** p<0.001).



Supplementary Figure 6. Assessment of NF- κ B nuclear translocation in HUVEC in response to LPS, IL1 β and TNF α stimulation. (A) Representative images of NF- κ B p65 immunostaining in NC HUVEC and IR HUVEC cultured with vehicle (Control) or stimulated with LPS (30 ng/ml), IL1 β (3 ng/ml) or TNF α (3 ng/ml) for 90 min. (B–D) Quantitative analysis of the nuclear/cytoplasmic ratio of NF- κ B p65 immunostaining in NC HUVEC and IR HUVEC stimulated with 30 ng/ml LPS (B), 3 ng/ml IL1 β (C) or 3 ng/ml TNF α (D) at various time points (n = 4; mean \pm SEM; * p<0.05, ** p<0.01, *** p<0.001).