SUPPLEMENTARY MATERIALS

miR--30c--1 inhibitor induces the senescence of hCECs

The number of SA- β -gal positive cells increased in treated with miR-30c-1-3p inhibitor or miR-30c-1-5p inhibitor (p < 0.001 for all; Supplementary Figure 1A–1B). Intracellular oxidative stress levels were elevated in treated with miR-30c-1-3p inhibitor or miR-30c-1-5p inhibitor (p < 0.001 for all; Supplementary Figure 1C–1D). Cell viability decreased in treated with miR-30c-1-3p inhibitor or miR-30c-1-5p inhibitor (p = 0.004 and <0.001; Supplementary Figure 1E). BrdU proliferation rate decreased in treated with miR-30c-1-3p inhibitor or miR-30c-1-5p inhibitor (p = 0.001 and <0.001; Supplementary Figure 1F)

TGF-\(\beta\)1 induces the senescence of hCECs

BrdU proliferation rate decreased at 24 h, 48 h, and 72 h compared with 0 h after TGF- β 1 treatment (p = 0.026, p = 0.047, and p = 0.023, respectively; Supplementary Figure 2A). Representative images of cell cycle analysis are shown in Supplementary Figure 2B. Cell cycle analysis showed that the number of cells in G0/G1

phase increased at 48 h and 72 h compared with 0 h (p < 0.001 for both; Supplementary Figure 2C), while the number of cells in S phase decreased at 48 h and 72 h compared with 0 h (p = 0.005 and p = 0.003, respectively; Supplementary Figure 2D), and the number of cells in G2/M phase decreased at 48 h and 72 h compared with 0 h (p = 0.006 and p = 0.010, respectively; Supplementary Figure 2E). The number of SA-β-gal positive cells increased at 24 h, 48 h, and 72 h compared with 0 h after TGF-β1 treatment (p < 0.001, p < 0.001, and p < 0.001, respectively; Supplementary Figure 2F–2G). Cell size increased at 48 h and 72 h compared with 0 h (p = 0.005 and p < 0.001, respectively; Supplementary Figure 2H–2I).

Intracellular oxidative stress levels were elevated by TGF- β 1 at 48 h and 72 h compared with 0 h (p=0.034 and p=0.001, respectively; Supplementary Figure 3A–3B). The pERK1/2 level increased over time after TGF- β 1 treatment (Supplementary Figure 3C). The percentage of cells with depolarized mitochondrial membrane potential was elevated at 48 h and 72 h compared with 0 h (p=0.002 and p=0.001, respectively; Supplementary Figure 3D–3E).