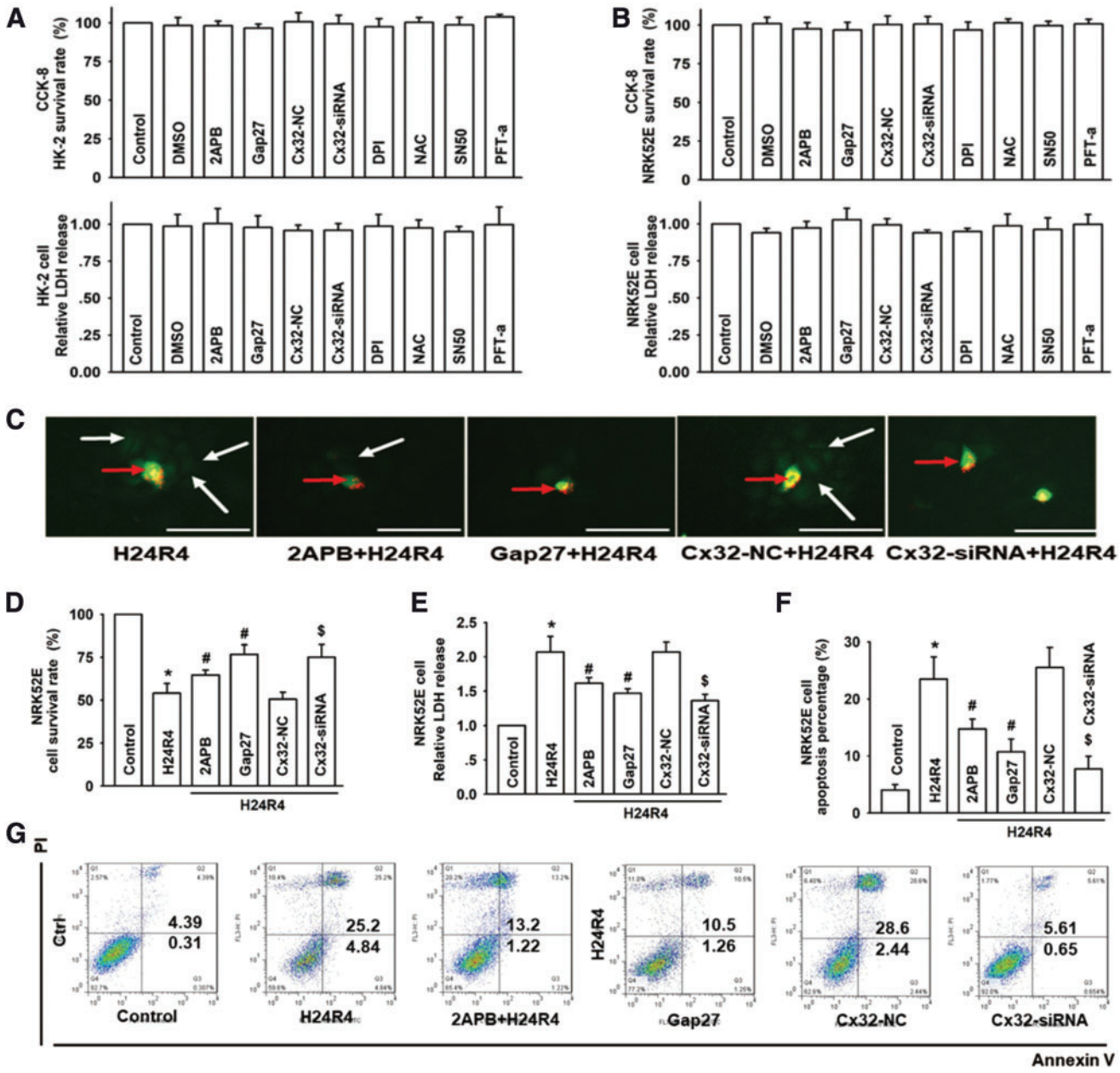

SUPPLEMENTARY FIG. S6. Cx32 inhibition attenuates H24R4-induced cell mitochondrial injuries and apoptosis of NRK52E cells. Before H24R4 exposure, cells were incubated with 2APB (gap junction inhibitor, 25 μM) or DPI (an inhibitor of NADPH oxidase, 1 μM) or NAC (a ROS scavenger, 10 mM) for 1 h, SN50 (a selective inhibitor of NF-κB, 20 μM) or Gap27 (Cx32 peptide, 100 μM) for 24 h, PFT-α (a selective inhibitor of p53, 10 μM) or Cx32-siRNA (50 nM) or vehicle (same volume of DMSO) for 48 h. **(A, B)** Effects of several reagents on HK-2 and NRK-52E cell growth, relative LDH release. **(C)** ''Parachute'' dye-coupling assay was used to determine effects of 2-APB, Gap27, and Cx32-siRNA on decreasing GJ function (scale bar 50 μm). Function of GJ is demonstrated by the spread of GJ-permeable calcein-AM (*white arrow*), and in contrast CM-Dil is impermeable (*red arrow*). **(D, E)** Effects of 2APB, Gap27, and Cx32-siRNA on HK-2 cell growth and relative LDH release. **(F, G)** NRK52E cell apoptotic rates were detected by flow cytometry as described in the Methods and Materials section. Data are presented as mean ± SE ($n = 4$). $^{*}p < 0.05$ compared with control group; $^{\#}p < 0.05$ *versus* H24R4 group. $^{\$}p < 0.05$ compared with Cx32-NC+H24R4 group in **(B–E)**. 2-APB, 2-aminoethoxydiphenyl borate; DPI, diphenyleneiodonium chloride; NAC, N-acetyl cysteine; NRK52E, rat kidney tubular epithelial cell; PFT-α, pifithrin-α.