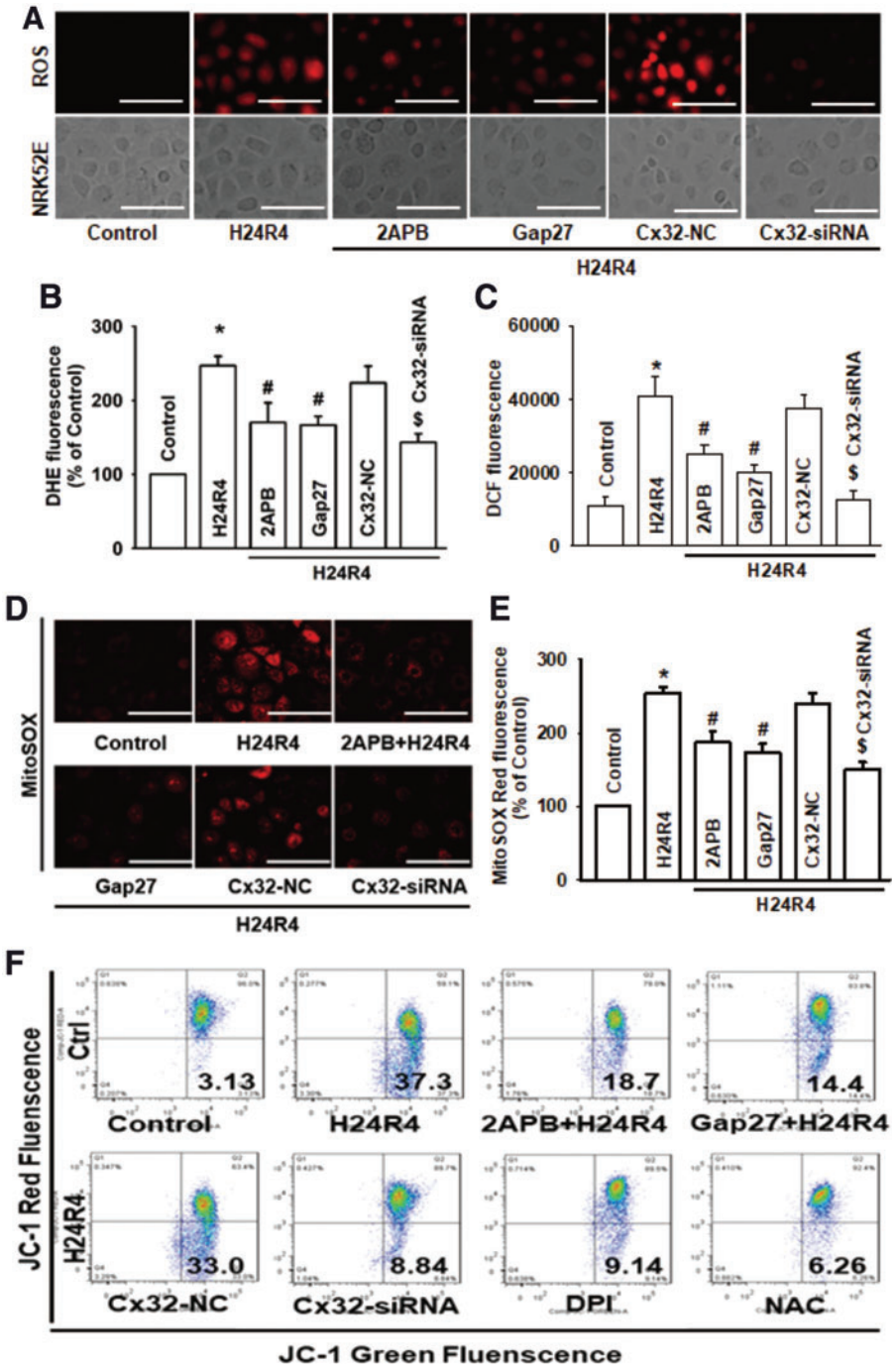


**SUPPLEMENTARY FIG. S7. Cx32 inhibition decreases H24R4-induced ROS generation and apoptotic injury.** Before H24R4 exposure, different methods were used to inhibit GJ function composed of Cx32, including 2APB (gap junction inhibitor, 25 $\mu M$, 1 h pretreatment), Gap27 (Cx32 peptide, 100 $\mu M$, 24 h pretreatment), and specific Cx32-siRNA, to observe effects of Cx32 GJ function on NRK52E cellular ROS production. **(A–C)** Effects of 2APB, Gap27, and Cx32-siRNA on cellular ROS production, detected with DHE staining (**A, B,** stained in *red*, scale bar 50 $\mu$m) and DCFH-DA staining **(C)**. **(D, E)** Effects of 2APB, Gap27, and Cx32-siRNA on mitochondrial superoxide formation, detected by MitoSOX Red dye staining. **(F).** Effects of 2APB, Gap27, Cx32-siRNA, DPI, and NAC on mitochondrial membrane potential. 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.