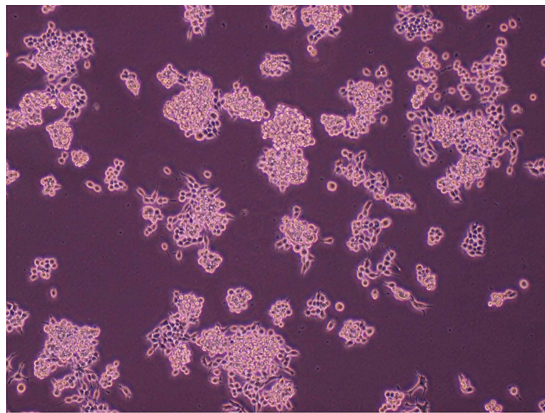
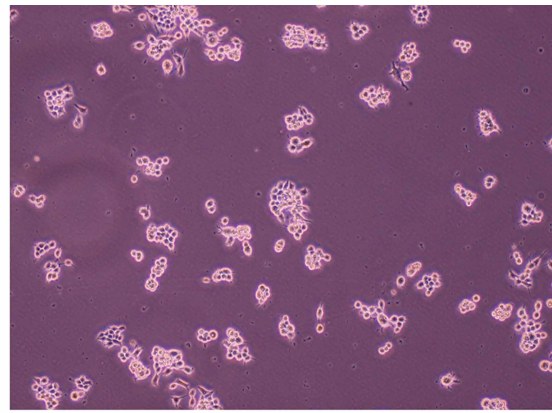


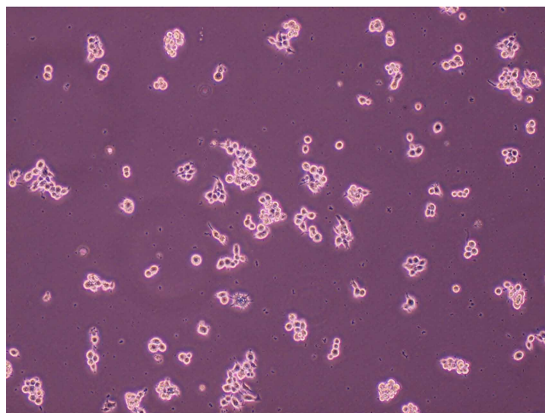
SUPPLEMENTARY FIGURES



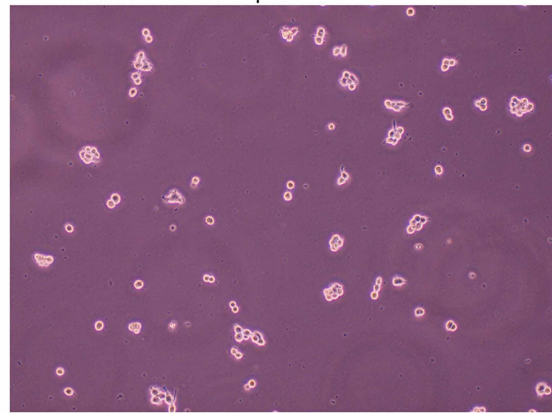
control



10 μ M CAPE

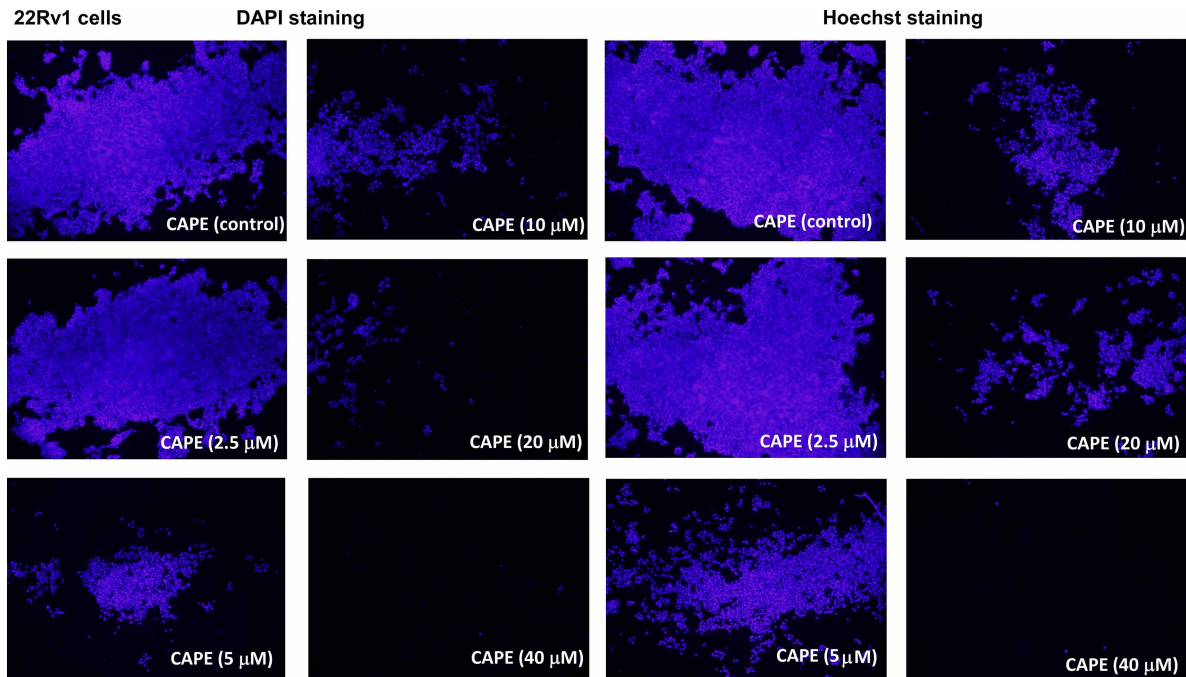


20 μ M CAPE

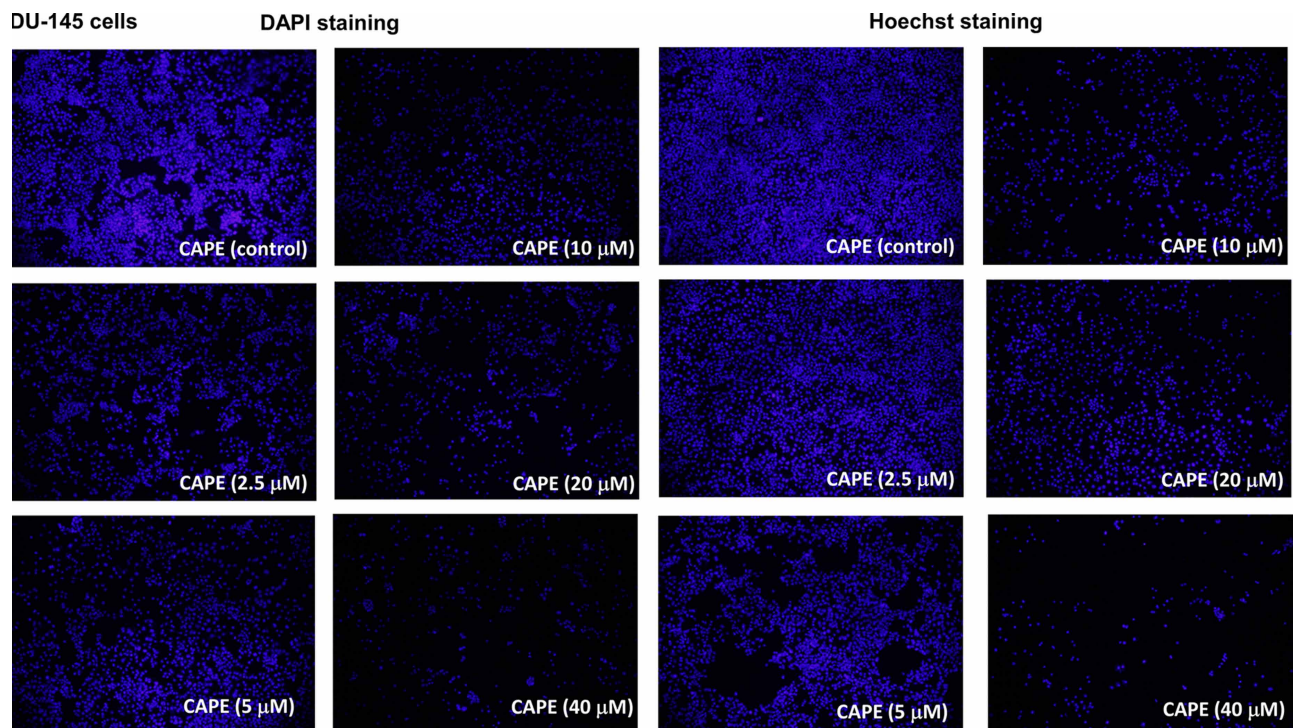


40 μ M CAPE

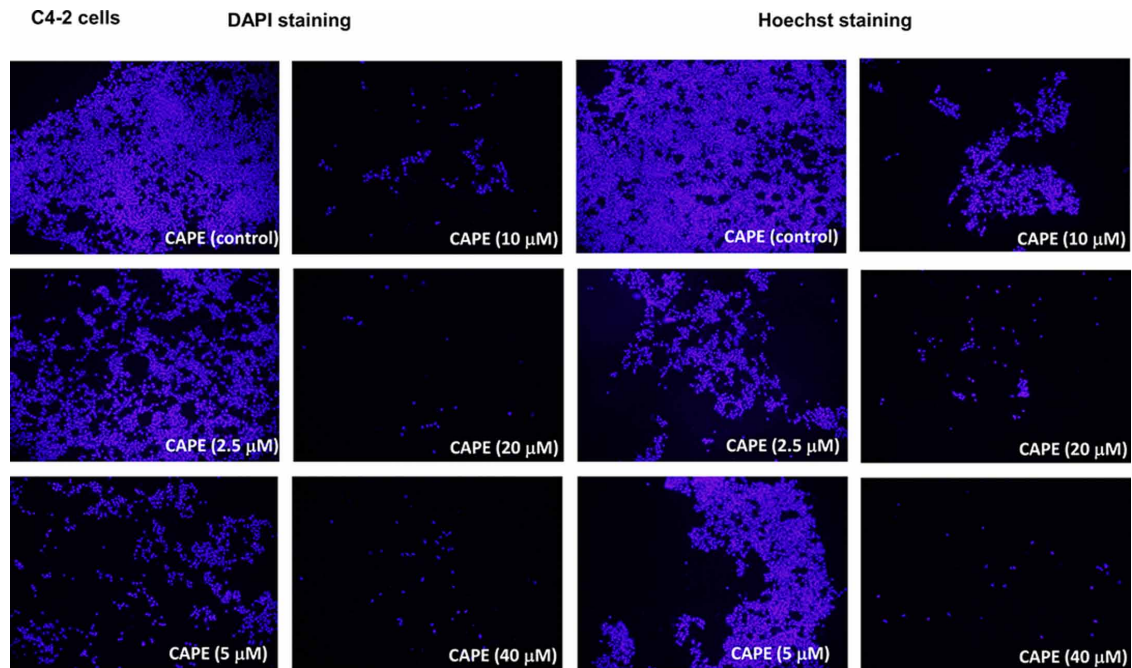
Supplementary Figure 1: CAPE treatment for 96 h reduced cell number of LNCaP 104-R1 cells. Images of LNCaP 104-R1 cells being treated with increasing concentrations of CAPE for 96 h were observed using light microscope with magnification of 100X.



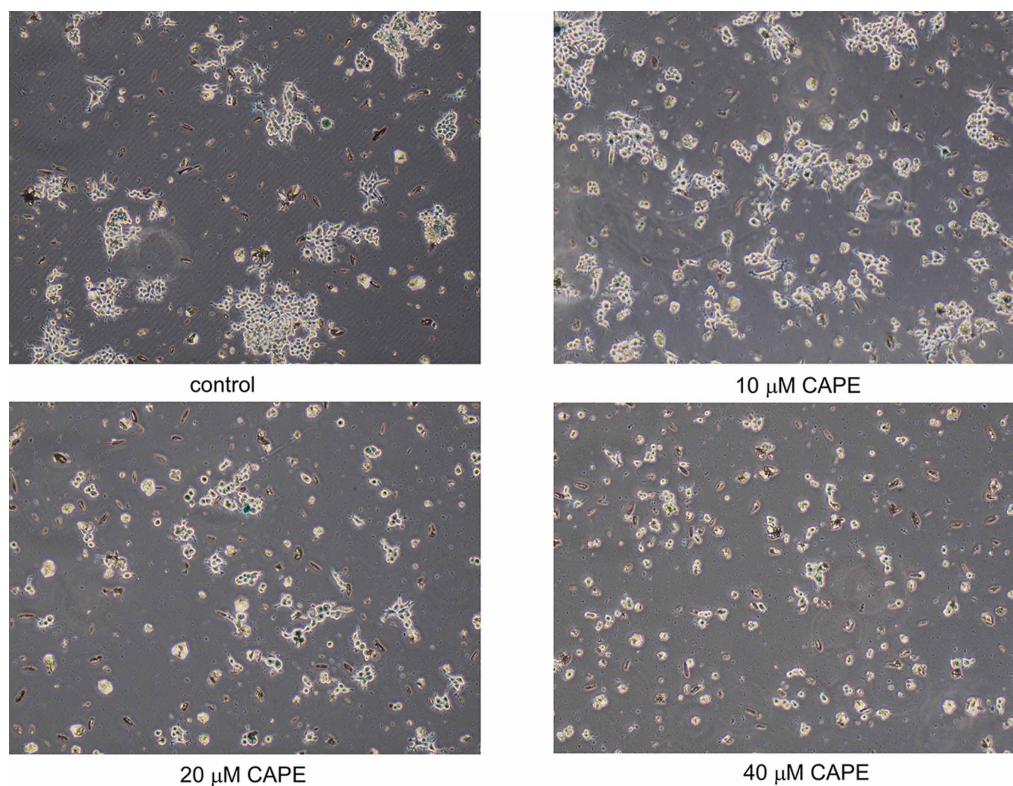
Supplementary Figure 2: CAPE treatment for 96 h reduced cell proliferation of 22Rv1 cells. DAPI staining and Hoechst dye-staining of 22Rv1 cells being treated with increasing concentrations of CAPE for 96 h was used to monitor cell proliferation of 22Rv1 cells using fluorescent microscope with magnification of 100X.



Supplementary Figure 3: CAPE treatment for 96 h reduced cell proliferation of DU-145 cells. DAPI staining and Hoechst dye-staining of DU-145 cells being treated with increasing concentrations of CAPE for 96 h was used to monitor cell proliferation of DU-145 cells using fluorescent microscope with magnification of 100X.



Supplementary Figure 4: CAPE treatment for 96 h reduced cell proliferation of C4-2 cells. DAPI staining and Hoechst dye-staining of C4-2 cells being treated with increasing concentrations of CAPE for 96 h was used to monitor cell proliferation of C4-2 cells using fluorescent microscope with magnification of 100X.



Supplementary Figure 5: CAPE treatment induced moderate β -galactosidase staining in LNCaP 104-R1 cells as determined by β -galactosidase staining. LNCaP 104-R1 cells were treated with 0, 10, 20, 40 μ M CAPE for 96 h. Expression of β -galactosidase was stained to determine if CAPE treatment causes cellular senescence and observed using light microscope with magnification of 100X.