

Supplementary Materials

The Protective Role of Bioactive Quinones in Stress-induced Senescence Phenotype of Endothelial Cells Exposed to Cigarette Smoke Extract

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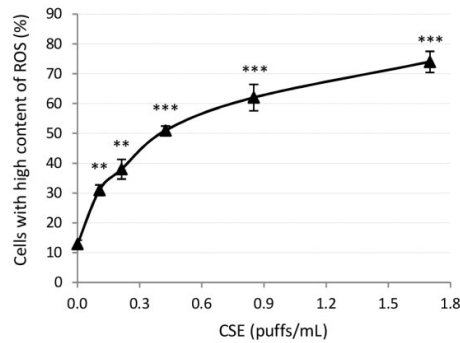


Figure S1. Cells with high content of ROS after 24 h treatment of young HUVECs with increasing doses of CSE. Significance was calculated with respect to young untreated cells (0 puffs/mL CSE). * $p \leq 0.05$, ** $p \leq 0.01$, *** $p \leq 0.001$.

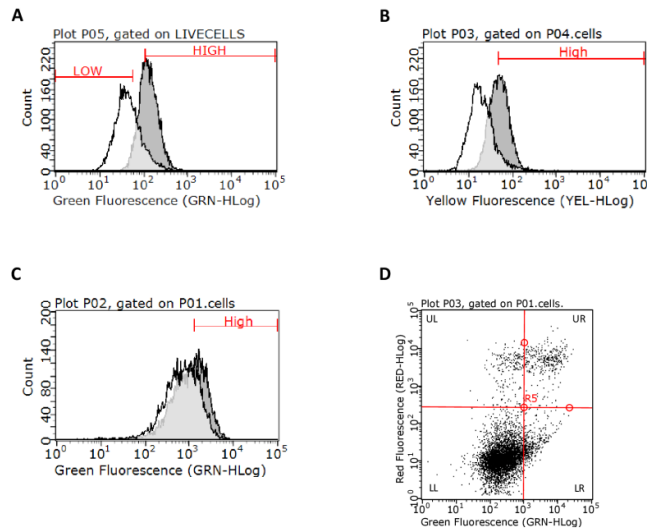


Figure S2. Representative flow cytometry graphs. Fluorescence distribution of (A) total ROS, (B) mitochondrial ROS, (C) mPTP opening, (D) caspase-1: LL: live cells; LR: live cells with activated caspase-1; UR: apoptotic or death cells with activated caspase-1; UL: apoptotic or death cells without activated caspase-1.

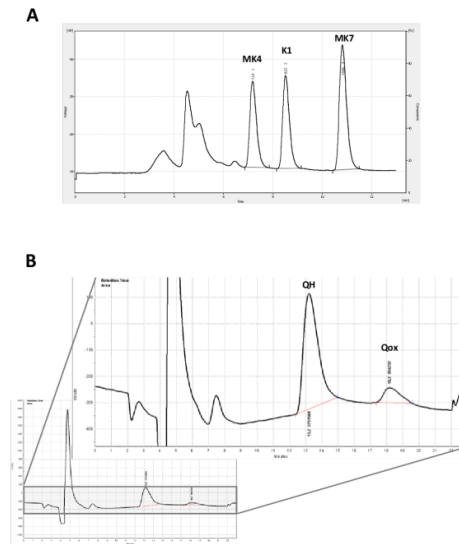


Figure S3. Representative chromatogram obtained by HPLC system for (A) K vitamins and (B) CoQ₁₀.

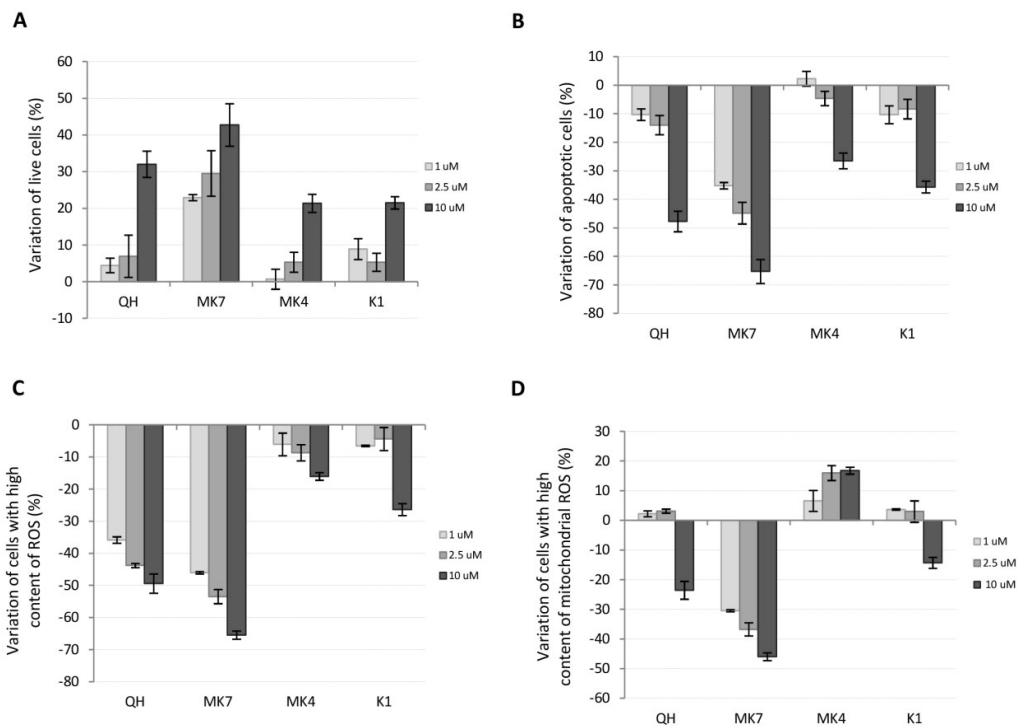


Figure S4. Effect of quinones supplementation on viability and oxidative stress in young HUVECs exposed to CSE (0.425 puffs/mL for 24 h). Cells were supplemented with different concentrations of quinones for 24 h followed by replacement with CSE medium containing the same quinones and incubated for a further 24 h. Variation of the percentage of: (A) live cells, (B) apoptotic cells, (C) cells with high ROS content, (D) cells with high mitochondrial ROS content.