Figure S1. Cd-induced cytotoxicity in RBE4 cells. Morphologic alterations in RBE4 cells treated with CdCl₂ 10 µM and 30 µM for 8 and 24 hours. Images were taken with an Optika XDS-2 inverted phase contrast microscope equipped with a TrueChrome HDII camera. Total magnification 100X. Scale bar: 50 µm.
Figure S2. αTocopheryl acetate counteracts CdCl₂ effects on ZO-1, F-actin and vimentin localization. The changes induced by CdCl₂ 10 µM, both at 8 and 16 h, in the distribution of ZO-1 (A), F-actin (B), and vimentin (C) in RBE4 cells, was clearly counteracted by the presence of αTA 10 µM. In panel A it is important to note the holes (asterisks) formed between endothelial cells, and the morphological alterations in intercellular junctions (arrows), indicating the loss of junctional function. These alterations are counteracted by the presence of αTA during CdCl₂ treatment. Panel B show the absence of stress fibers during the αTA treatment in presence of CdCl₂. In panel C, αTA presence, both at 8 and 16 h, clearly show a decrease in vimentin aggregates and clumps (arrowheads). Total magnification 400X, n=135; bar: 50 µm.
Figure S3. αTocopheryl acetate counteracts the Cd-induced ER stress. Western blotting analysis reveal that αTA 10 µM counteract the Cd-induced upregulation of GRP78 expression both at 8 (panel A) and 16 h (panel B). Control condition was arbitrarily set as 100 % and results are expressed as mean ± S.E.M., n=9; *p<0.05 vs control (untreated cells); #p<0.05 vs Cd.

Figure S4. αTocopheryl acetate counteracts the Cd-induced caspase-3 activation. Western blotting analysis show that αTA 10 µM counteract the Cd-induced caspase-3 activation, both at 8 (panel A) and 16 h (panel B). Control condition was arbitrarily set as 100 % and results are expressed as mean ± S.E.M., n=9; *p<0.05 vs control (untreated cells); #p<0.05 vs Cd.