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There are some working links on some forums, you can find them there: [-00173j,@B27-antioxidants-09-00173j,@B28-antioxidants-09-00173j,@B29-antioxidants-09-00173j](#)). Recently, we found that RvD1 is able to preserve the endothelium from oxidative damage in vivo. In the present study, the treatment of PA-treated HUVECs with RvD1 protected them from the deleterious effect of PA on intracellular glutathione status and mitochondrial activity. The protective effect of RvD1 was abolished when a putative CysLT-1~ receptor antagonist was added in culture. We have also observed that RvD1 prevents PA-induced intracellular ROS production and reduces the level of 3-nitrotyrosine, which is a biomarker of oxidative damage in PA-treated HUVECs. Our data are consistent with the fact that, in human umbilical vein endothelial cells (HUVECs), PA induced ROS production, oxidative damage, and morphological changes. It has been shown that 3-nitrotyrosine level is elevated in PA-treated HUVECs [\\[@B40-antioxidants-09-00173j\\]](#), which is in line with our results. On the other hand, we found that treatment of PA-treated HUVECs with RvD1 prevented all the changes induced by PA. Furthermore, the protective effect of RvD1 was abolished by a putative CysLT-1~ receptor antagonist. We also observed that RvD1 is able to protect HUVECs from the deleterious effect of PA on mitochondrial membrane potential. Previous studies have shown that PA reduced mitochondrial membrane potential (mΔψm) [\\[@B41-antioxidants-09-00173j\\]](#). In HUVECs, PA induced an increase in the ROS level and in the mΔψm. However, the treatment of PA-treated HUVECs with RvD1 prevented the changes in mΔψm. It is known that the integrity of the endothelial layer is tightly related to the production of cytoprotective factors that regulate the redox status of cells. In endothelial cells, the activation of the MAPK signaling pathway is a [82157476af](#)

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