Salvianolic Acid B (Sal B) is considered as one of the most active anti-oxidant and the major pharmacological component of the herb, Salvia miltiorrhiza. Its beneficial effects including hepatoprotection, elicitation of endothelium-dependent vasodilation, lowering of blood pressure in hypertension, inhibition of HIV-1 replication and suppressing inflammatory cytokine- stimulated endothelial adhesiveness to human monocyctic cells by its strong antioxidant activities.

Our study demonstrated the protective effects of Sal B on hydrogen peroxide (H2O2)-induced injury in human umbilical vein endothelial cells (HUVECs). Treatment with H2O2 significantly decreased the cell viability and increased the lactate dehydrogenase (LDH) leakage that is an apoptotic feature. Pretreatment with Sal B prevented significantly from H2O2-induced cell apoptosis and other damages in a concentration-dependent manner. The mechanisms of Sal B protection was studied with two-dimensional gel electrophoresis (2-DE) coupled to hybrid quadrupole time-of-flight mass spectrometry (Q-TOF) mass spectrometer. Data base searching implicated glucose-regulated protein 78 (GRP78), a central regulator for ER stress, was up-regulated in Sal B-exposed HUVECs. After exposure to Sal B, the level of activating transcription factor 4 (ATF4) was raised, with a transient phosphorylation of the α subunit of eukaryotic translation initiation factor (eIF2α). Knock-down of GRP78 by siRNA significantly reduced protective effects of Sal B. These results suggest that Sal B-induced GRP78 upregulation via phosphorylation of eIF2α and resultant translation of ATF4. And up-regulation of ER chaperones induced by Sal B may play an important role in protecting human endothelial cells from oxidative stress-induced cellular damage.