cause apoptosis. The linkages inferred, but not directly tested, are indicated with dashed arrows.

## **Supplemental information**

**Figure S1.** *A.* 15d-PGJ<sub>2</sub> induces ROS generation in HUVECs. Cells were pretreated with 10 mM NAC for 30 min before exposure to 10 μM 15d-PGJ<sub>2</sub>. DCF fluorescence was examined after treatment of 15d-PGJ<sub>2</sub> for 1 h. Nuclei were stained with Hoechst 33342 (*blue*). Cells were then washed and imaged on an inverted fluorescence microscope. Magnification: × 20. Merged images indicate the DCF-fluorescence emitted from 15d-PGJ<sub>2</sub>-induced HUVECs but not in NAC-pretreated HUVECs. *B-C*. NAC prevents phosphorylations of p38 MAPK, JNK and p53 (Ser392) induced by 15d-PGJ<sub>2</sub> in vascular ECs. Alkali burn mouse corneas were treated with either 20 μl of PBS or 10 mM NAC before treatment with 15d-PGJ<sub>2</sub> (20 μl, 20 μM). After treatment for 8 h, corneas were harvested and subjected to immunofluorescent assay for eNOS (*red*; specific for vascular endothelium) and p-JNK, anti-p-p38 MAPK or anti-p-p53 (Ser392) (*green*). Nuclei were stained by Hoechst 33342 (*blue*). Magnification: × 40. Bar: 75 μm. Merged images indicate that 15d-PGJ<sub>2</sub> induces the increase of p-JNK, p-p38 MAPK, and p-p53 that are localized in the nuclei (*pale blue*) and co-localized with eNOS (*yellow*) in the cornea. NAC pretreatment prevents these inductions.