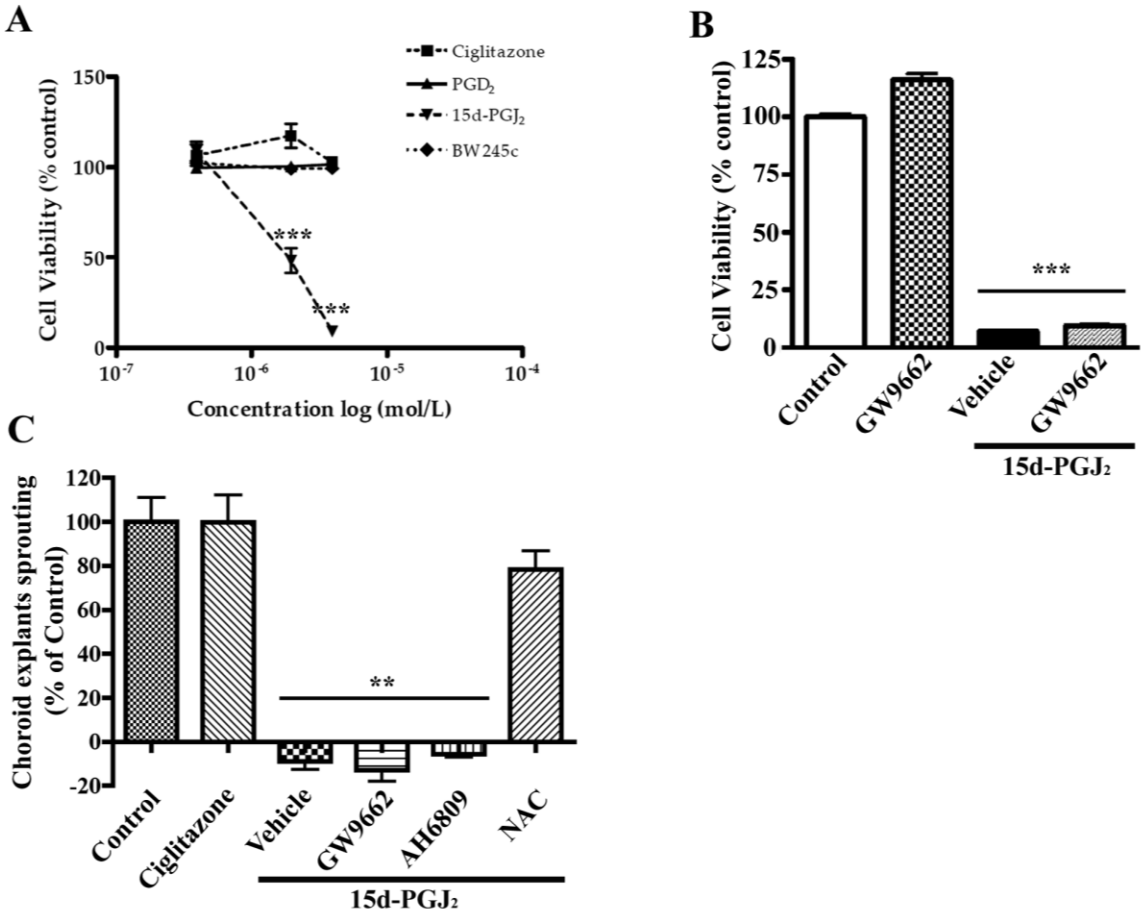
Supplemental Figure 3



Supplemental Figure 3. 15d-PGJ2 induces apoptosis in endothelial cells independent from PPARγ or DP1 receptors but dependent on oxidation status. (A) Treatment of ocular endothelial cells with varying concentrations of PGD2, BW245C (DP receptor agonist), 15d-PGJ2, or Ciglitazone (PPARγ agonist); only 15d-PGJ2 (LD50=1.4 µmol/L) induced endothelial cell death. Values are mean ± SEM of 3-4 experiments; ***p<0.001 compared to all other corresponding values without asterisks. (B) Cultured ocular endothelial cell viability in response to 15d-PGJ2 (5 µmol/L) in the presence or absence of the irreversible PPARγ antagonist GW9662 (330 nmol/L); effects of 15d-PGJ2 are independent of PPARγ. Values are mean ± SEM of 3 separate experiments each performed in triplicate; ***p<0.001 compared to other values without asterisks. (C) Quantification of the sprouting area from choroidal explants in Matrigel after 36 h exposure to Ciglitazone (2.8 µmol/L) or 15d-PGJ2 (5 µmol/L) in presence or absence of GW9662 (330 nmol/L), AH6809 (EP1/DP1 receptor antagonist) (30 µmol/L) or N-acetyl-cysteine (NAC; glutathione precursor) (3 mmol/L); one notes that the vascular sprouting inhibitory effects of 15d-PGJ2 are only prevented by the anti-oxidant NAC. Values are mean ± SEM of 4 separate experiments each performed in triplicate; **p<0.01 compared to other values without asterisks.