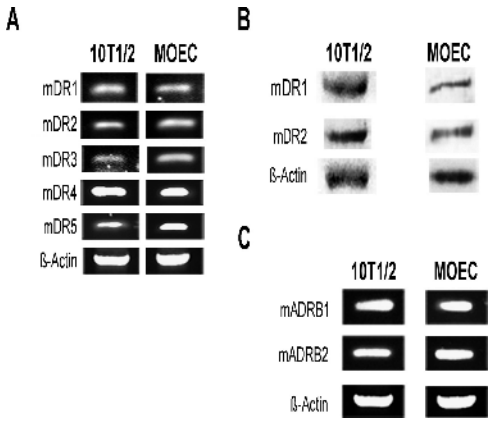


**Figure W1.** Expression of DA and β-adrenergic receptors in murine pericyte 10T1/2 cells and MOEC. (A) RT-PCR analysis of mDR1 to mDR5. (B) Protein expression of DR1, DR2 in pericytes, and MOEC. (C) RNA expression of β-adrenergic receptors in pericytes and MOEC.

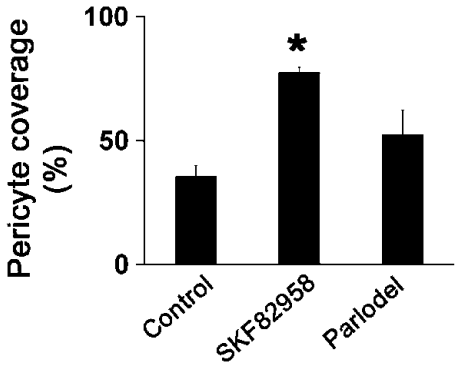


**Figure W2.** Pericyte coverage of SKOV3ip1 tumor endothelial cells. Tumor tissues from mice treated with DA and SKF 82958 showed significantly higher percentage of pericyte coverage compared to the control group (*$P < .0001$). Tumor tissues from mice treated with parlodel showed an increase in pericyte coverage; however, this increase was not statistical significant compared to controls.

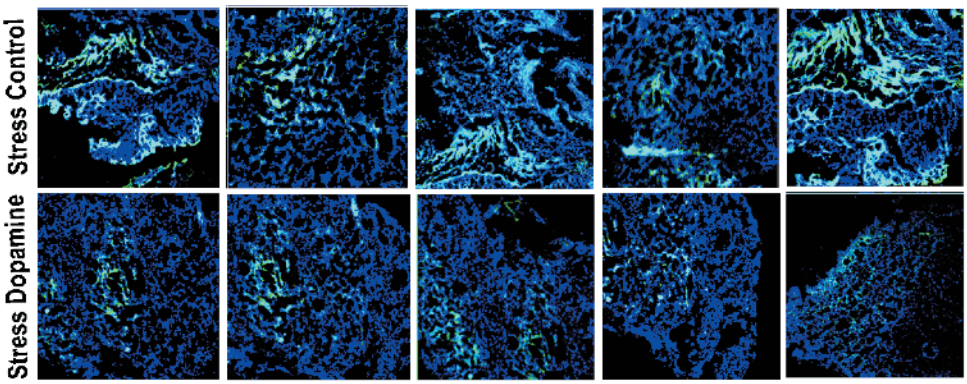


**Figure W3.** Hypoxic areas in ovarian cancer tumors from stressed mice. Representative confocal images (100×) of SKOV3ip1 tumor tissues from chronically stressed mice stained for CA9 (green fluorescence). Tumors from stressed mice treated with DA (lower panel) showed decreased regions of hypoxia (green fluorescence) compared with those from control (untreated) stressed mice (upper panel).

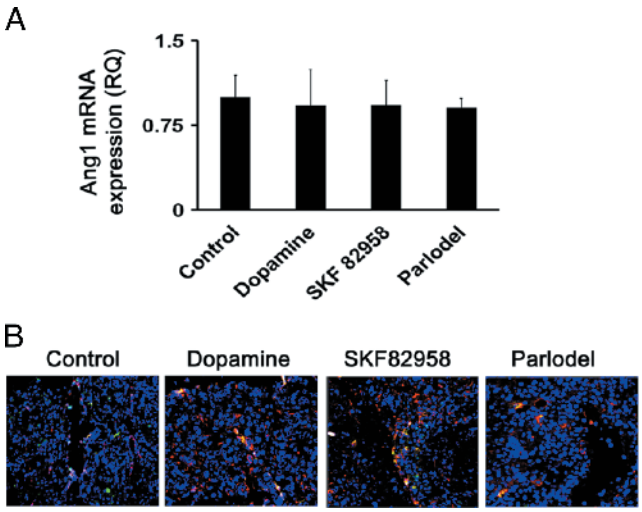


**Figure W4.** Ang1 expression in SKOV3ip1 tumor tissues from stressed mice. Treatments of DA, DR1 agonist SKF 82958, and DR2 agonist parlodel did not affect mRNA (A) and protein (B) expression of Ang1. Ang1 mRNA was determined by quantitative RT-PCR. Ang1 protein was detected in tumor pericytes by a double immunofluorescence staining for Ang1 (green fluorescence) and RGS5 (as pericyte marker; red fluorescence).

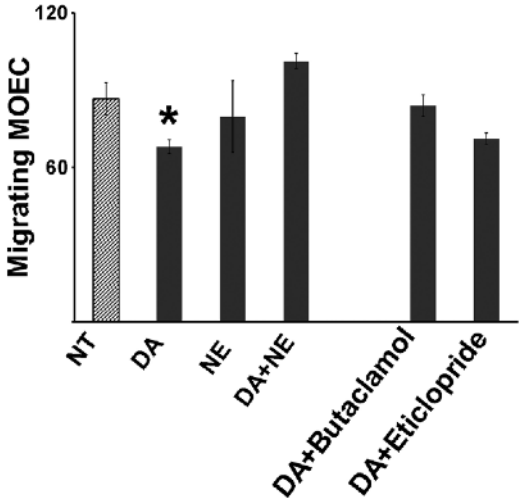


**Figure W5.** Migration of MOECs in the presence of DA, NE, and DA antagonist. DA exposure resulted in a 22% ($P < .05$) decrease in MOEC migration. This effect, however, was not significantly abolished with either combined treatment of DA and DR1 antagonist butaclamol (DA/butaclamol) or combined treatment of DA and DR2 antagonist eticlopride (DA/eticlopride). MOEC migration was not significantly affected by treatment with NE or DA + NE.