Supplementary Figure 6. Effect of stress hormones and various antagonists on endothelial cell migration and proliferation. Mouse mesenteric endothelial cells were used for (a) migration and (b) tube-formation assays. Cell-migration (a) was assayed using the membrane invasion culture system (MICS), as previously described. For tube formation, the endothelial cells were cultured in Matrigel (1 mg/mL) on 48-well plates and visualized 8 h later. Tubule branch points from endothelial cells were counted, and the sum of 4 different fields for each condition was averaged. For both experiments, the endothelial cells were treated according to the following groups: control (media alone); norepinephrine 1 µM (NE); epinephrine 1 µM (Epi), VEGF 5 ng/mL; VEGF + PTK787 100 nM; 24 h conditioned media from SKOV3ip1 cells (SKOV3ip1 CM); 24 h conditioned media from SKOV3ip1 cells treated with norepinephrine 1 µM (SKOV3ip1 NE CM); SKOV3ip1 CM + PTK787; SKOV3ip1 NE CM + PTK787; propranolol 1 µM; SKOV3ip1 CM + propranolol; and SKOV3ip1 NE CM + propranolol. NE and Epi had modest effects on endothelial cell migration and tube-formation, but VEGF induced migration by 468% ($P < 0.01$) and tube formation by 705% ($P < 0.001$) over control. These effects were blocked by the VEGF-R2 inhibitor PTK787. Similarly, conditioned media from SKOV3ip1 cells had some effect on endothelial cells ($P < 0.005$), but the greatest effects were observed when endothelial
cells were exposed to conditioned media from NE treated SKOV3ip1 cells \((P < 0.001)\). These effects were blocked when the SKOV3ip1 cells were pre-treated the \(\beta\)-blocker propranolol prior to NE treatment for collection of conditioned media or by treatment with PTK787. *\(P < 0.01\); **\(P < 0.001\).