Tumor p38MAPK signaling enhances breast carcinoma vascularization and growth by promoting expression and deposition of pro-tumorigenic factors

**SUPPLEMENTARY FIGURES**

Supplementary Figure 1: Immunoblots of phospho-HSP27, phospho-ERK and beta-actin in lysates from control-EGFP and dn-p38 MDA-MB-231 cells treated with 2 ng/mL TGF-β1 for 2 hours.
Supplementary Figure 2: Growth of control-EGFP and dn-p38 MDA-MB-231 cells in media containing 5% serum for indicated time.
Supplementary Figure 3: Assessment of the effect of p38 inhibitor on TGF-β1 signaling. Immunoblots of phospho-SMAD2, phospho-HSP27 and GAPDH in lysates from MDA-MB-231 cells treated for 2 hours with 2 ng/mL TGF-β1 ± 1-2μM SB202190, a p38MAPK inhibitor.
Supplementary Figure 4: Relapse-free status and mRNA levels of fibronectin in the breast cancer stroma. The data are obtained using Oncomine tools (www.oncomine.org) and the Finak Breast dataset (Nature Medicine, 2008). The Finak study assesses gene expression in the stroma adjacent to invasive breast carcinomas and normal tissues. The data show that a 5-year disease-free status inversely correlates with elevated mRNA levels of Fibronectin.