Figure S1 | LECs increase VEGFR3-dependent invasion of human breast cancer cells after treatment with docetaxel in human 3D *in vitro* model of breast tumor microenvironment.



Figure S1 | LECs increase invasion of human breast cancer cells after treatment with docetaxel in human 3D *in vitro* co-culture system. Representative images of cancer cell invasion of pre-labeled MDA-MB-231 cells in our 3D microenvironment system following treatment with and without docetaxel (0.1 μ M) with and without MAZ51 (1 μ M) in the presence or absence of LECs, as described in Fig. 1.

Figure S2 | Blockade of VEGFR3 in combination with docetaxel reduces primary tumor growth and lung metastasis in 4T1.



Figure S2 | Blockade of VEGFR3 in combination with docetaxel reduces primary tumor growth and lung metastasis in 4T1. (A) Representative H&E images of lung metastases from 4T1 mice treated with docetaxel and/or anti-VEGFR3. Metastatic lesions are noted by black arrowheads. (n=5/cohort) (B) Quantification of lung metastasis. Columns represent number of metastatic foci per mouse. Numbers above columns represent number of mice in each cohort that developed any lung metastasis *p< 0.05. (n=5). (C) Representative bioluminescence imaging of 4T1-luciferase tumors in mammary fat pads of mice treated with docetaxel (or vehicle) and anti-VEGFR3 antibody (or control IgG) as previously described in Figure 2.

 Table S1 | LECs increase EC50 of docetaxel in three human breast cancer cell lines.

Cell Death EC50 of Docetaxel (µM)					
HCC 38		MDA-MB-231		HCC 1806	
- LEC	+ LEC	- LEC	+ LEC	- LEC	+ LEC
0.052	101.2	0.3	81.2	513.7	657.0

Table S1 | LECs increase EC50 of docetaxel in three human breast cancer cell lines. EC50 calculated for cancer cell death as quantified by flow cytometry following docetaxel treatment in 3D *in vitro* system for HCC38, MDAMB231, and HCC1806 human breast cancer cell lines with or without LECs present in the system as described in Fig. 1.

Figure S3 | LEC-mediated reduction in docetaxel-induced cytotoxicity is independent of VEGFR3.



Figure S3 | LEC-mediated reduction in docetaxel-induced cytotoxicity is independent of VEGFR3. (A) Cancer cell death of MDA-MB-231 TNBC cells in 3D microenvironment system with and without docetaxel treatment (10 μ M) in the presence of LECs treated with or without VEGFR3 inhibitor MAZ51. (B) Cancer cell death of HCC 38 TNBC cells in 3D microenvironment system with and without docetaxel treatment (10 μ M) in the presence of LECs treated with or without VEGFR3 inhibitor MAZ51. (B) Cancer cell death of HCC 38 TNBC cells in 3D microenvironment system with and without docetaxel treatment (10 μ M) in the presence of LECs treated with or without VEGFR3 inhibitor MAZ51. (C) Cancer cell death of HCC 1806 TNBC cells in 3D microenvironment system with and without docetaxel treatment (10 μ M) in the presence of LECs treated with or without VEGFR3 inhibitor MAZ51. Results are indicated as % dead cancer cells as assessed by flow cytometry. #p<0.1, **p<0.01