Breast cancer is the most common malignant disease presently ailing western women. Complications from metastases rather than the primary tumor are the cause of death in the majority of cancer patients. Tumor blood flow and cell motility play an essential role in tumor growth and metastasis. Yet, the molecular mechanisms and signaling controlling tumor hemodynamics are poorly understood, as are the mechanisms by which tumor cells navigate through the tumor into neighboring tissues and blood vessels. HGF/SF-Met are master regulators of cell motility. To better understand the molecular mechanisms of tumor cell motility we compared actin organization in cells treated with HGF/SF and untreated cells. Tumor cells expressing YFP-Actin were imaged via STED and Vutara super-resolution microscopy. To visualize and examine tumor cell motility in vivo and ex vivo, we used a xenograft mouse model where mammary adenocarcinoma cells are tagged by the fluorescent protein mCherry (DA3-mCherry). To induce tumor formation, these cells were injected orthotropically into the mammary pad of female mice. Extravagated tumors were cut in half, one half placed in medium containing HGF/SF and the other in control medium. Tumor cells were then imaged overnight using time lapse microscopy. Using the Imaris analysis software we detected the volume of the tumor cells and followed their progression through the tissue over time. Our results demonstrate that Met activation by HGF/SF increases cell velocity and acceleration and induces collective cell motility. To image blood flow, Dextran-FITC was injected i.v. as a blood flow marker. Using confocal microscopy we have demonstrated HGF/SF induced changes in blood flow at single vessel resolution. We hypothesize that Met induced-increase in cell motility and blood flow are key factors in metastasis formation, and that better understanding of these processes and the connection between them may indicate new targets for anti-Met and anti-metastases targeted therapy.