The tumor microenvironment is a complex tissue containing cancer cells and various stromal cells embedded in an extracellular matrix network. Due to the role of stroma in cancer initiation and progression, targeting the stroma and its interactions with cancer cells is being studied as a potential treatment strategy. To address the need for novel tumor models to study tumor-stromal interactions, we developed an organotypic model of human triple negative breast cancer (TNBC) that mimics the architecture of solid TNBC tumors and contains a mass of cancer cells surrounded by carcinoma-associated fibroblasts (CAFs) embedded in a collagen I matrix of stiffness similar to advanced TNBC tumors. Our results showed that CAFs significantly and cell density-dependently contracted the collagen matrix via activation of the Rho-ROCK pathway. The largest matrix contraction over two weeks of culture was over 7 folds. Additionally, signaling of TNBC cells and CAFs promoted matrix invasion of cancer cells by activating the oncogenic MAPK/ERK signaling pathway. In contrast, when we included normal human mammary fibroblasts (HMFs) in the tumor model, TNBC cells did not show the ability to invade the matrix, indicating the importance of CAFs in TNBC cell invasiveness. To further validate this finding, we demonstrated that blocking the signaling between a CAFs-produced CXCL12 chemokine and its cognate receptor CXCR4 on TNBC cells inhibits invasiveness of cancer cells, suggesting blocking of CAFs-cancer cells signaling as a therapeutic strategy. Our convenient-to-use and scalable tumor model is a promising tool for drug discovery against tumor-stromal interactions.