Metastasizing breast carcinoma cells are believed to mobilize proteolytic systems similar, if not identical, to those employed by normal mammary epithelial cells undergoing branching morphogenesis. However, the key molecular effectors underlying mammary gland morphogenesis have not been identified, nor has their role in breast cancer invasion in vivo been established. In recent studies, we find that the membrane-anchored matrix metalloproteinase, MT1-MMP/MMP14, but not the closely related metalloproteinase, MT2-MMP/MMP15, serves as the dominant proteolytic effector of both mammary gland branching morphogenesis and breast carcinoma cell invasion in vivo. Unexpectedly, while epithelial cell-specific targeting of MT1-MMP in normal epithelial cells fails to impair branching, deleting the proteinase in carcinoma cells abrogates invasion and metastasis. By contrast, deleting MT1-MMP expression from the periductal stroma ablates mammary gland morphogenesis without affecting cancer cell invasion. Together, these data uncover overlapping, but divergent matrix remodeling strategies that underlie mammary gland morphogenesis and breast cancer progression.