Mocetinostat de-represses miR-203 to reverse EMT-generated stemness in breast cancer cells
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Background

Triple negative breast cancer (TNBC) patients, unlike those with ER, PR, or HER2-positive breast cancers, face higher rates of relapse and mortality, primarily due to the lack of targeted therapies. While cytotoxic neo-adjuvant chemotherapies prove effective in 70% of patients, the remaining often develop recurrence and/or distant metastases. Cancers of epithelial origin grow as tightly compacted tumors, bounded by a basement membrane. In order for metastasis to occur, tumor cells must lose contact with their neighbors, migrate through the extracellular matrix, enter the vasculature, and survive in circulation in a process called epithelial-mesenchymal transition (EMT). In the reverse process, mesenchymal-epithelial transition (MET) cells exit the vasculature and re-initiate proliferation at a new site to form metastases.

Epiogenetic inhibitors upregulate miR-203 expression

We will continue to pursue other small molecules’ effects on miR-203 re-expression and we aim to show mocetinostat’s ability to reverse EMT. Furthermore, we will pursue in vitro studies, evaluating methods to increase endogenous miR-203 expression, as well as these small molecules’ suppression of metastasis.

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About the author: Keighley is beginning the second year of her PhD at Baylor University and is evaluating the role of miR-203 in metastatic triple negative breast cancer. Originally from Wisconsin, the only thing hotter than Texas weather is her new plunge into the cancer field.