A Review of the Signal Transduction Pathways involved in Epithelial-Mesenchymal Transition induced in Breast Cancer Metastasis and Their Cross-talks by Kasey Cervantes

Figure 1. Shown above are the signal transduction pathways involved in Epithelial-Mesenchymal Transition (EMT), induced in breast cancer metastasis, by promoting an EMT-associated transcription factor (Slug (SNAI2), SNAI1 (Snail), Zeb1, Zeb2, and Twist) caused by downstream targets. EMT is the biological process of which epithelial cells lose their cell-cell adhesion and polarity, allowing for the cells to transition into a motile mesenchymal state triggering metastasis. A hallmark of EMT is the repression of E-cadherin, which occurs when an EMT-associated transcription factor is activated. EMT is involved in the initiation of metastasis, wound healing, and development. Second, angiogenesis or lymphangiogenesis must occur for metastasis to occur, which is the formation of new blood vessels or lymphatic vessels. Cancer stem cells utilize the bloodstream or lymphatic stream in order to metastasize to different parts in the body. The process of angiogenesis is initiated by vascular endothelial growth factor (VEGF), which binds to VEGFR on the interior of blood vessel walls. This causes endothelial cells to begin to proliferate, to sprout out of the normal blood vessel to form a new blood vessel connected to the tumor. Activated matrix metalloproteinase (MMP-3/9) aids in this process by degrading the proteins that keep the vessel walls solid.