Cell extrinsic and intrinsic factors contribute to breast cancer progression. Outside the cell, an increase in the cell-intrinsic levels. At the extrinsic level, breast cancer cells migrate along the tracks that form in mature collagen fibers when collagen fibrils increase in collagen fibers provide tracks along which breast cancer cells migrate. Meanwhile, cell-intrinsic epithelial-to-intrinsic level, metastasis and general cancer progression mesenchymal transition (EMT) is associated with cancer compound when epithelial cells lose cell-cell adhesion and progression and metastasis [cit]. During EMT, epithelial cells adopt a migration phenotype in a process known as cells undergo changes in gene expression resulting in loss epithelial-to-mesenchymal transition (EMT) [cit]. The induction of EMT during cancer development is currently

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1 Luo CY, Natividad RJ, Lalli ML, Asthagiri AR (2020) Multivariate relationships among nucleus and Golgi properties during fibrillar migration are robust to and unchanged by epithelial-to-mesenchymal transition. PLoS ONE 15(9): e0239188. https://doi.org/10.1371/journal.pone.0239188. Here is the link to the Creative Commons license https://creativecommons.org/licenses/by/4.0/ I do of course make changes to the text.
phenotype. An array of mechanisms, including upregulation attributable to such mechanisms as upregulation of soluble of soluble TGFβ, is implicated in inducing EMT during TGFβ, but a clear mechanistic delineation of EMT and cancer development [cit]. Understanding how EMT affects also a detailed explanation for EMT influence on fibrillar fibrillar migration will provide insights into the etiology migration will help identify the etiology of breast cancer, of breast cancer and help identify potential therapeutic select potential therapeutic targets, and ultimately limit targets. breast cancer progression.