Role of Y-box binding protein-1 (YB-1) in epithelial-mesenchymal-driven beast cancer metastasis

For PhD Qualifying Examination (PQE) "Oral Component" – Open Seminar

Epithelial-mesenchymal transition (EMT), is the process whereby epithelial cells transform into mesenchymal cells, leading to metastatic spread. The Y-box binding protein-1 (YB-1) is a transcription and translation regulating protein that is elevated in various human malignancies. The hypothesis of this study is that YB-1 is a key player in EMT, which in turn drives breast cancer metastasis. Breast cancer cell lines examined were found to express both the YB-1 gene and protein. Silencing of YB-1 by siRNA in the mesenchymal cell lines MDA-MB-231 and Hs578T induced a decrease in cell migration but no changes in gene expression of EMT-related markers. Coronin-1C was later identified from the microarray data in *si-YB-1* silenced MDA-MB-231 cells to be a potential downstream target of YB-1 protein, which could mediate cell migration. Similarly, stable knockdown of YB-1 by shRNA in MDA-MB-231 cells did not alter the expression of EMT-related genes. In contrast, overexpression of YB-1 in epithelial-like MCF7 cells led to an increase in cell migration, gene expression of EMT- related markers and cytoskeletal changes. Subsequent transcriptomic analysis of YB-1 overexpressing breast cancer cells, identified the TGF-β signalling pathway (an important EMT- related pathway) to be highly enriched. This study provides evidence that YB-1 could be important in mediating partial EMT-related changes in epithelial-like breast cancer cells but not in mesenchymal cells, thus providing a further understanding of the EMT process.

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