Experimental Results and Discussion

Methods

MCF10A, A1N4 and MDA231 cells are obtained from the American Type Culture Collection (ATCC). SUM159 cell line is from Asterand Bioscience. Cybrids are generated by the transmitochondrial cybrid technology and are generated using the SUM159, and MDA-MB-231 (MDA231) TNBC cells. Label free shotgun Jetstream proteomics data suggested that differential statistics and pathway analysis on the expression profiles resulted from spectrum MIL.

Mitochondrial-Nuclear Crosstalk in Cancer

Mitochondrial retrograde regulation (MRR) is a bidirectional communication between the mitochondria and the nucleus that influences many cellular activities.

Transmitochondrial cybrid technology is an excellent tool to understand mitochondria-nuclear crosstalk in a defined nuclear background.

Any other subgroup of patients with breast cancer, except for triple negative breast cancer (TNBC), there is a current lack of understanding of driver pathways.

The Src kinase activity is frequently over-expressed in TNBC and in association with metastatic disease progression

Mitochondria-nuclear crosstalk affects mitochondria-regulated cancer pathways and performed pathway based approach to understand such as PI3K-AKT, MAPK, JAK-STAT, and Focal adhesion kinase; these pathways were significantly increased in metastatic cybrids and cells. The analyses confirmed that mitochondria-nuclear crosstalk regulates tumor property of cancer cell.

Shotgun Jetstream proteomics data suggested that pathways related to downstream signaling of Src activation such as PI3K-AKT, MAPK, JAK-STAT, and Focal adhesion kinase have altered in metastatic cybrids and cells. The analyses confirmed that mitochondria-nuclear crosstalk regulates tumor property of cancer cell.

Results and Discussion

Conclusions

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