

Inherited Mutations in *BRCA1* Increases the Risk of Female Breast and Ovarian Cancers

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EDITORIAL NOTE

Journal of Cell Science & Therapy commemorates its decade long service to the scientific community by consistently publishing peer-reviewed articles and tracking the progress and significant advancements in the field of cell science. Ever since its inception in the year 2010, in addition to regular issue releases on a quarterly basis, this transdisciplinary journal is also releasing special issues and conference proceedings from time to time, thus comprehensively covering a wide range of topics and emerging challenges in cell science. The journal focuses on application oriented research on cell therapy, hematology, and cell science. In this issue some of the recent and impactful research articles that were published by the journal will be discussed.

The susceptible to develop cancer due to synergism of *BRCA1* mutation and impaired estrogen signaling in oxidative stress modulation has been done in this study. Nazmeen et al. [1] reported their research work wherein, a specific inherited mutation in *BRCA1* increases the risk of female breast and ovarian cancers. *BRCA1* critically maintains genome stability and cell cycle progression. *BRCA1* is a well-known tumor suppressor gene; germline mutations in this gene confer increased susceptibility to developing breast and ovarian cancer. Though breast cancer associated with *BRCA1* mutations were considered sporadic for mostly being $ER\alpha(-)$. Significant numbers of $ER\alpha(+)$ *BRCA1* mutated breast cancer patients were also discovered.

There are two questions prevailing with *BRCA1* breast cancers. Why *BRCA1* related patients have higher risk for cancer development mainly in estrogen responsive tissues such as breast and ovary. And the second is, the therapeutic approach for $ER\alpha(+)$ *BRCA1* breast cancers may not be same as $ER\alpha(-)$ *BRCA1* cancers. Recently *BRCA1* in context with oxidative stress is been widely studied. The association of *BRCA1* and cancers in estrogen responsive tissues may be explained by *BRCA1*, estrogen and ER cooperation mediated by oxidative stress.

In this study the proteomics profiling of pancreatic cancer and pancreatitis for biomarkers discovery has been done, Sanh et al. [2] have performed a study where the identified proteins that are altered in expression in pancreatic cancer and pancreatitis compared to normal using proteomic technology. Proteins were extracted from laser captured micro-dissected tissues and separated in 2-DPAGE and imaged. The protein profiles of pancreatic cancer and pancreatitis are similar but differed with the protein profile of normal adjacent tissues. Representative proteins, overexpressed in tumor and pancreatitis but not normal tissues, were excised from gels, subjected to in-gel digestion, and analyzed by MALDI-TOF mass spectrometry. Proteins identified included transferrin, ER-60 protein, proapolipoprotein, tropomyosin 1, alpha 1 actin precursor, ACTB protein, and gamma 2 propeptide, aldehyde dehydrogenase 1A1, pancreatic lipase and annexin A1. Several proteins, which were shown in pancreatic cancer, were also observed in pancreatitis samples. Understanding the role of these specific proteins and their mechanistic action will give insights into their involvement in pancreatic cancers.

These research articles published by the journal have immense relevance and significance in susceptible to develop cancer due to synergism of *BRCA1* mutation and impaired estrogen signaling in oxidative stress and proteomics profiling of pancreatic cancer and pancreatitis for biomarkers.

REFERENCES

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