

## CODEN [USA]: IAJPBB

ISSN: 2349-7750

# INDO AMERICAN JOURNAL OF PHARMACEUTICAL SCIENCES

http://doi.org/10.5281/zenodo.997173

Available online at: <u>http://www.iajps.com</u>

**Review Article** 

## MOLECULAR GENETICS AND BREAST CANCER TREATMENT-A REVIEW

Fateme parooei, Sara Zamanpour and Morteza Salarzaei \*

Medical Student, Student Research Committee, Zabol University of Medical Sciences,

Zabol, Iran

## Abstract:

In this review article, the databases Medline, Cochrane, Science Direct, and Google Scholar were thoroughly searched to identify the Molecular genetics and breast cancer treatment. In this review, the papers published until early January 2017, that was conducted to study the relationship between the Molecular genetics and breast cancer treatment were selected.

Key words: cancer, breast cancer, and colorectal cancer

**Corresponding author:** 

Morteza Salarzaei, Medical student, Student Research Committee, Zabol University of Medical Sciences, Zabol, Iran Email: <u>mr.mortezasalar@gmail.com</u> Tell : +989120644917



Please cite this article in press as Morteza Salarzaei et al, **Molecular Genetics and Breast Cancer Treatment-A Review**, Indo Am. J. P. Sci, 2017; 4(09).

## **INTRODUCTION:**

According to published statistics by the World Health Organization in 2011, cancer is the second leading cause of death after cardiovascular diseases throughout the world. The American Cancer Society announced in its latest report that out of every eight women, one is diagnosed with breast cancer [1]. The rate of cancer in developed countries is increasing from 1 to 0.2% and in developing countries about 0.5% annually. According to a report by the World Health Organization in 2011, cancer in Iran was reported to be 12% widespread and was recognized as the third most common cause of death [2]. Gastric cancer, breast cancer, and colorectal cancer are the three common cancers in Iran respectively. Breast cancer is the first place cancer widespread among women [3]. The average age of breast cancer diagnosis in the Western countries is 56 years and in Iran 45 years. New developments in the patients care with breast cancer have increased the overall survival rate of the patients in recent years. This increase in survival has doubled the importance of predictive factors of local recurrence and distant metastases of the disease [4]. In addition, it should be noted that the progression or regression of some diseases are not constant over time, as in the stages of recovery or worsening of the disease, the occurrence of some consequences changes the course of the disease, and the disease progress declines and this risk begins to decrease in the 2-5 years after treatment, which make the recovery process speed [5].

### **METHODS:**

In this review article, the databases Medline, Cochrane, Science Direct, and Google Scholar were thoroughly searched to identify the Molecular genetics and breast cancer treatment. In this review, the papers published until early January 2017 that were conducted to study the relationship between the Molecular genetics and breast cancer treatment were selected.

### **FINDINGS:**

Breast cancer is a heterogeneous disease. It has been believed for many years that tumors with different biological characteristics have different clinical results and therapeutic outcomes [6]. At present, the prognosis as well as the treatment selection for breast cancer is based on determining the status of growth hormone receptors [ER, PR, HER2] in the tumor. Four functional groups of tumor can be determined by using these indicators:

- 1. Positive hormone receptor and negative HER2
- 2. Negative hormone receptor and negative HER2 [triple negative tumor]
- 3. Tumors with too much HER2 or those lacking hormone receptor

Cell proliferation is an important feature of cancer and Ki67 Non-histone nuclear proteins are proper indicators for this process [7]. Ki67 staining can be used both as a durable indicator of proliferation and as an appropriate treatment indicator through various measurements on continuous tissue samples during the treatment process [8]. Some researchers have managed to classify different kinds of breast cancer to subcategories with different prognosis by using cDNA microarrays. These studies have applied hierarchical clustering analysis to identify subcategories having different gene expression patterns [9]. The differences existing in gene expression patterns of these subcategories indicate the fundamental differences in the cellular biology of these tumors that show itself through the clinical findings of these tumors. Clinical specialists consider the process method of these cellular subcategories as distinct diseases [10].

#### DISCUSSION AND CONCLUSION

Breast cancer tumors include heterogeneous groups of cells a small part of which is formed by the stem cells. Given their capabilities of proliferation and self-renewal, these cells effective in tumor creation process. The loss of self-renewal process regulation leads to the increase of stem cells and this is likely to affect the early stages of cancer formation [11]. The capability of miRNAs in the simultaneous regulation of several target genes have made them a good candidate for regulating the renewal process of stem cells and making decision about the fate of the cells [12]. There are numerous evidences that indicate special miRNAs are expressed differently in the stem cells. Destroying dicer, an important enzyme in the mRNA production process, is fatal for the embryos of mice and results in the destruction of stem cells population [13]. Moreover, in stem cells lacking dicer proliferation and distinction are disturbed. Furthermore, miRNAs are necessary in order to have the capability to dominate G1-S control point and conduct self-renewal. The identification and manipulation of these miRNAs can facilitate the population regulation of the stem cells affecting the formation of cancer. Discovering the role of miRNAs in the creation and development of human malignancies has made it possible to improve the current strategies of diagnosing and treating patients suffering from cancer [14]. The identification of new miRNAs, determining their target mRNA, and defining their functional effect will improve our awareness about the role of these indicators in the creation of different kinds of cancer including breast cancer and will provide new possibilities for the medical interventions needed.

#### **REFERENCES:**

1.Xing Y, Foy M, Cox DD, Kuerer HM, Hunt KK, Cormier JN. Meta-analysis of sentinel lymph node biopsy after preoperative chemotherapy in patients with breast cancer. British journal of surgery. 2006 May 1;93[5]:539-46.

2.Berry DA, Cirrincione C, Henderson IC, Citron ML, Budman DR, Goldstein LJ, Martino S, Perez EA, Muss HB, Norton L, Hudis C. Estrogenreceptor status and outcomes of modern chemotherapy for patients with node-positive breast cancer. Jama. 2006 Apr 12;295[14]:1658-67.

3.Jiralerspong S, Palla SL, Giordano SH, Meric-Bernstam F, Liedtke C, Barnett CM, Hsu L, Hung MC, Hortobagyi GN, Gonzalez-Angulo AM. Metformin and pathologic complete responses to neoadjuvant chemotherapy in diabetic patients with breast cancer. Journal of clinical oncology. 2009 Jun 1;27[20]:3297-302.

4.Paik S, Tang G, Shak S, Kim C, Baker J, Kim W, Cronin M, Baehner FL, Watson D, Bryant J, Costantino JP. Gene expression and benefit of chemotherapy in women with node-negative, estrogen receptor–positive breast cancer. Journal of clinical oncology. 2006 Aug 10;24[23]:3726-34.

5.Borges S, Desta Z, Li L, Skaar TC, Ward BA, Nguyen A, Jin Y, Storniolo AM, Nikoloff DM, Wu L, Hillman G. Quantitative effect of CYP2D6 genotype and inhibitors on tamoxifen metabolism: implication for optimization of breast cancer treatment. Clinical Pharmacology & Therapeutics. 2006 Jul 1;80[1]:61-74.

6.Berns K, Horlings HM, Hennessy BT, Madiredjo M, Hijmans EM, Beelen K, Linn SC, Gonzalez-Angulo AM, Stemke-Hale K, Hauptmann M, Beijersbergen RL. A functional genetic approach identifies the PI3K pathway as a major determinant of trastuzumab resistance in breast cancer. Cancer cell. 2007 Oct 16;12[4]:395-402.

7.Li C, Heidt DG, Dalerba P, Burant CF, Zhang L, Adsay V, Wicha M, Clarke MF, Simeone DM. Identification of pancreatic cancer stem cells. Cancer research. 2007 Feb 1;67[3]:1030-7.

8.Miller KD, Chap LI, Holmes FA, Cobleigh MA, Marcom PK, Fehrenbacher L, Dickler M, Overmoyer BA, Reimann JD, Sing AP, Langmuir V. Randomized phase III trial of capecitabine compared with bevacizumab plus capecitabine in patients with previously treated metastatic breast cancer. Journal of clinical oncology. 2005 Feb 1;23[4]:792-9.

9.Wacholder S, Hartge P, Prentice R, Garcia-Closas M, Feigelson HS, Diver WR, Thun MJ, Cox DG, Hankinson SE, Kraft P, Rosner B. Performance of common genetic variants in breastcancer risk models. New England Journal of Medicine. 2010 Mar 18;362[11]:986-93.

10.Simpson PT, Reis-Filho JS, Gale T, Lakhani SR. Molecular evolution of breast cancer. The Journal of pathology. 2005 Jan 1;205[2]:248-54.

11.Carey LA, Perou CM, Livasy CA, Dressler LG, Cowan D, Conway K, Karaca G, Troester MA, Tse CK, Edmiston S, Deming SL. Race, breast cancer subtypes, and survival in the Carolina Breast Cancer Study. Jama. 2006 Jun 7;295[21]:2492-502. 12.Weigelt B, Horlings HM, Kreike B, Hayes MM, Hauptmann M, Wessels LF, De Jong D, Van de Vijver MJ, Van't Veer LJ, Peterse JL. Refinement of breast cancer classification by molecular characterization of histological special types. The Journal of pathology. 2008 Oct 1;216[2]:141-50.

13.Loi S, Haibe-Kains B, Desmedt C, Wirapati P, Lallemand F, Tutt AM, Gillet C, Ellis P, Ryder K, Reid JF, Daidone MG. Predicting prognosis using molecular profiling in estrogen receptor-positive breast cancer treated with tamoxifen. BMC genomics. 2008 May 22;9[1]:239.

14.Lujambio A, Ropero S, Ballestar E, Fraga MF, Cerrato C, Setién F, Casado S, Suarez-Gauthier A, Sanchez-Cespedes M, Gitt A, Spiteri I. Genetic unmasking of an epigenetically silenced microRNA in human cancer cells. Cancer research. 2007 Feb 15;67[4]:1424-9.