Review Article

Breast Cancer: Innovations in Detection and Treatment

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ABSTRACT

Numerous treatments for breast cancer have surfaced in recent years. Genetic and environmental variables have a role in the heterogeneous disease known as breast cancer. Breast cancer stem cells are the primary cause of various tumours' aggressiveness and provide the biggest obstacle to cancer treatment. According to this assessment, the selection of a promising approach has led to significant progress in the treatment of BC. Brain-metastases, which have a dismal prognosis, are still mostly caused by breast cancer. BMs in patients with breast cancer are being managed with a mix of systemic medications and local therapies (such as radiation or surgery). A revolution in metastatic breast cancer has been spurred by the development of novel antibody-drug conjugates cancer treatment. Technology is being used to enhance patient outcomes and streamline procedures in the rapidly evolving field of modern healthcare. Since breast cancer is the most frequent cancer in women worldwide, successful management calls for creative techniques. This review covers both healthy and unhealthy diets as well as preventive includes the radiation and chemotherapy as well.

Keywords: Breast Cancer, Chemotherapy, Radiation Therapy, Immunotherapy.

INTRODUCTION

One kind of cancer that manifests differently in women is breast cancer. There were 268,670 new BC cases reported in the US in 2018. Worldwide, BC is a prevalent malignancy that primarily affects women. BC might be divided into three types based on molecular and histological evidence: BC expressing human epidermal receptor 2 (HER2+), BC expressing hormone receptors (either progesterone receptor (PR+) or oestrogen receptor (ER+), and triple negative breast cancer (TNBC) (ER−, PR−, HER2−) [1]. There have been notable advancements in the creation of efficient treatment options for people with metastatic breast cancer (MBC) in recent years [2]. In addition to being the fourth most common cause of cancer-related deaths globally, breast cancer is the main cause of death for women. Breast cancer is a major hazard to women's health and has become a global concern due to its high incidence and mortality rate [3]. The disease has a mortality rate of about 1% and an estimated 138 cases per 100,000 people between 2011 and 2020. Now, bulk cell sequencing which is unable to precisely analyse the differences between individual cells is the main focus of breast cancer research [4]. For breast cancer's development, survival, differentiation, regeneration, immunological response, respiration, metabolism and other essential cellular processes, STAT3 is a crucial protein. basic biological processes involved in breast cancer. Upstream signalling molecular proteins including Janus kinase and epidermal growth factor receptor (EGFR) control STAT3 expression and subcellular localisation [5]. Current systemic treatments for breast cancer include surgical resection, local radiotherapy, systemic chemotherapy, endocrine therapy and molecular targeted therapy. Despite relatively advanced treatment strategies, some patients experience progression towards more aggressive disease, and some develop resistance to first-line therapeutic drugs [6].

Mitophagy, another name for mitochondrial autophagy, is a type of selective autophagy that keeps cells and mitochondria in a state of equilibrium. The word "mitophagy" was first used in 2005 and since then, important discoveries have been made about the traditional process of mitophagy, especially about our knowledge of its molecular underpinnings and consequences for both health and sickness. Through lysosomes, damaged or malfunctioning mitochondria are selectively degraded and removed by mitophagy. A complex web of chemicals and signalling channels carefully controls this process. On the other hand, unchecked mitophagy causes excessive mitochondrial breakdown by interfering with the basal mitochondrial turnover [7]. Although certain breast cancer risk variables are out of an individual's control, they are important in determining total risk. These include being a woman, getting older, and having a family history of ovarian or breast cancer all of which are known risk factors. Susceptibility is also influenced by race and ethnicity with certain groups exhibiting greater prevalence rates. Risk is also increased by reproductive history, including early menarche or late menopause, nulliparity, or an earlier first pregnancy. Furthermore, variables that increase the risk of breast cancer include thick breast tissue, a history of non-cancerous breast disorders, a prior diagnosis of breast cancer and previous radiation therapy [8]. Various growth factors, such as transforming growth factor (TGF)-α, TGF-β, insulin-like growth factor (IGF)-I, IGF-II and platelet-derived growth factor (PDGF), have been identified that are secreted by cancer cells and can stimulate stromal cells [9].

The prognosis of BC patients is influenced by several parameters including as age, therapy, histological type, stage, and pathological differentiation. Furthermore, youthful age is a separate risk factor for BC patient survival with prognoses often poorer for younger patients than for older ones [10]. The fact that breast cancer is a diverse disease presents one of the biggest therapeutic problems [11]. In nations with little resources, breast cancer is identified in advanced stages despite medical advancements since early detection, diagnosis, and treatment cannot be effectively promoted. To assess how complicated the medical system is in connection with breast cancer, the Breast In regard to breast health, the Health Global Initiative (BHGI) has attempted to classify the organisational levels of nations cancer [12]. The most prevalent malignancy in women globally is breast cancer. According to recent data, there was an approximately 1% yearly rise in the incidence rate of breast cancer between 2015 and 2019. Over 1750 new instances of breast cancer were detected nationwide in 2022, according to the Jordan Cancer Registry. Consequently, it accounts for one-fifth of all cancer cases and over 40% of all malignancies in women [13]. The study presented the idea of a "4:1 ratio," showing that averting four local recurrences by year five might potentially avoid one breast cancer mortality by year fifteen [14]. Immunotherapy has emerged as a key component of cancer treatment and has been effectively used to a wide variety of solid and liquid tumour forms. They can include gene or cell therapies that involve customising chimeric antigen receptor (CAR) T cells or ex-vivo expansion of desired immune cell products as well as technologies like vaccines and oncolytic viruses that are important in antigen presentation and cancer immune activation, as well as monoclonal and bispecific antibodies that modulate inhibitory and stimulatory immune check points. (adoptive T cells) or substances with anticancer properties (such cytokines) [15].

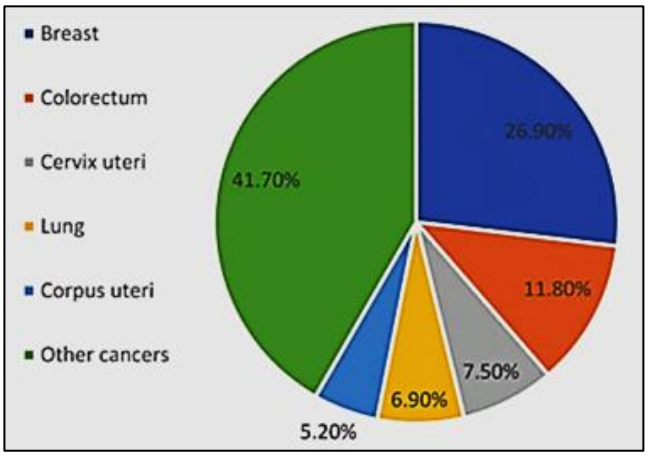


Figure 1: Number of breast cancer new cases in 2020, females, all ages- adapted after Globo can 2020 [8].

PATHOLOGY OF BREAST CANCER

Carcinomas or those originating from breast epithelial components, account for 95% of all breast cancer cases. In situ carcinomas and invasive carcinomas are the two main categories of breast cancer. The ductal or lobular epithelium may give birth to in situ carcinomas, but they stay contained there without invading the basement membrane underneath. membrane that would represent an expansion beyond the borders of the epithelium. Negligible possible metastases are envisaged with such a localised and limited cancer. A ductal or lobular carcinoma is said to be invasive (or infiltrating) when it extends past the basement membrane that makes up the epithelial border [16]

TECHNOLOGY FOR SINGLE-CELL SEQUENCING (SCS)

Single-cell sequencing, which encompasses proteomics, metabolomics, epigenomics, transcriptionomics, and genomics. A new era of single-cell sequencing technology was ushered in 2009 when Tang Fuchou became the first to create highly sensitive next-generation single-cell transcriptome sequencing technology. This technology can perform large-scale libraries of thousands of cells at a comparatively low cost and provide accurate and efficient analyses of the captured cells [4].

BREAST STRUCTURE

The parenchyma that makes up the female breast is subdivided into many lobes by connective tissue and adipose tissue. The mammary fat pad, which is made up of adipocytes, supports the network of glandular ducts formed by the epithelial cells in the parenchyma that are resting on the basement membrane. Adipose tissue also provides the gland with immunological cells and nutrition. The connective tissue, which is characterised by fibroblasts and forms Cooper's ligaments, is further tasked with maintaining the integrity of the breast. This structure also houses the nerves and blood vessels from the chest wall [17].

TYPES OF BREAST CANCER

Breast cancer divided into invasive and non- invasive breast cancers. Depending on the cell types, histological profiles, invasiveness, presence (or absence) of oestrogen and progesterone receptors (ER and PR), human epidermal growth factor receptor 2 (HER2), and some other factors, BC can be classified in many ways. A glimpse of some common types of BC is given in the following [18]. When breast cancer is discovered during pregnancy or within a year after birth, it is referred to as pregnancy-associated breast cancer (PABC) [19].

Invasive

It is a kind of cancer that has not spread outside of the ducts or lobule where it is located. Ductal carcinoma in situ is one kind of non-invasive breast cancer. When atypical cells form inside the milk ducts but do not spread to the exterior or near to tissue, ductal carcinoma in situ manifests. "In situ" refers to being "in place." The atypical cells can develop into invasive breast cancer even when they have not spread to tissues outside of the lobules or ducts [20]. The invasion-metastasis cascade in breast cancer is a series of well controlled events. First, cancer cells enter the circulatory system by invading adjacent lymphatic or blood arteries.

They then spread from the circulation to distant metastatic locations, such as the brain, liver, lungs, or bones. The dynamic interactions between cancer cells and the endothelial cells lining blood arteries are crucial to this metastatic dispersion because they allow the cancer cells to move and infiltrate into new tissues [21]. For patients with metastatic breast cancer, systemic pharmacological therapy is the primary approach. The pharmacological treatment of breast cancer includes chemotherapy, targeted therapies, immunotherapeutic drugs, and endocrine therapy agents [22].

Non-Invasive

It arises when aberrant cells from the milk ducts or lobules divide and spread out into the breast tissue. Through the immune system or the systemic circulation, cancer cells can travel from the breast to other areas of the body. They could move when the tumour is just a minute in size or later when it is enormous. The most common type of general cancer in women is invasive breast cancer. The wealthy populations of Australia and Europe, where 6% of women develop invasive breast cancer before the age of 75 are the areas of greatest peril [20].

BREAST CANCER WITH LAMINATION

About 70% of all cases of breast cancer in Western nations are luminal breast cancers, which are ER-positive tumours. Although they can infrequently progress to invasive micropapillary, invasive lobular, invasive cribriform, mucinous, and tubular carcinomas, luminal-like malignancies usually present as invasive breast cancer (IBC) without a distinct subtype. The two main biological processes that distinguish luminal-like tumours into luminal A and luminal B variations, each with a unique clinical presentation, are proliferation-related pathways and luminal-regulated pathways [18].

Luminal- A

The presence of oestrogen receptors (ER) and/or progesterone receptors (PR) and the lack of HER2 (HER2 negative) are characteristics of luminal A tumours. This subtype is characterized by the activation of genes by ER transcription factors that are specific to the luminal epithelium lining the mammary ducts. Additionally, it exhibits decreased gene expression, which is involved in cell division. They are easy to diagnose, inferior, and grow slowly [18].

Luminal-B

Luminal B tumours have a worse prognosis than luminal A tumours. They may also be HER2 positive and PR-negative in addition to being ER positive. Moreover, proliferative genes including MKI67 and AURKA show increased expression levels in luminal B tumours. Compared to the ER, this subtype expresses the PR and FOXA1 genes and proteins, which are characteristic of the luminal epithelium, less often. Luminal and non-luminal disorders are distinguished by oestrogen receptors, which show up equally in A and B subtypes [18].

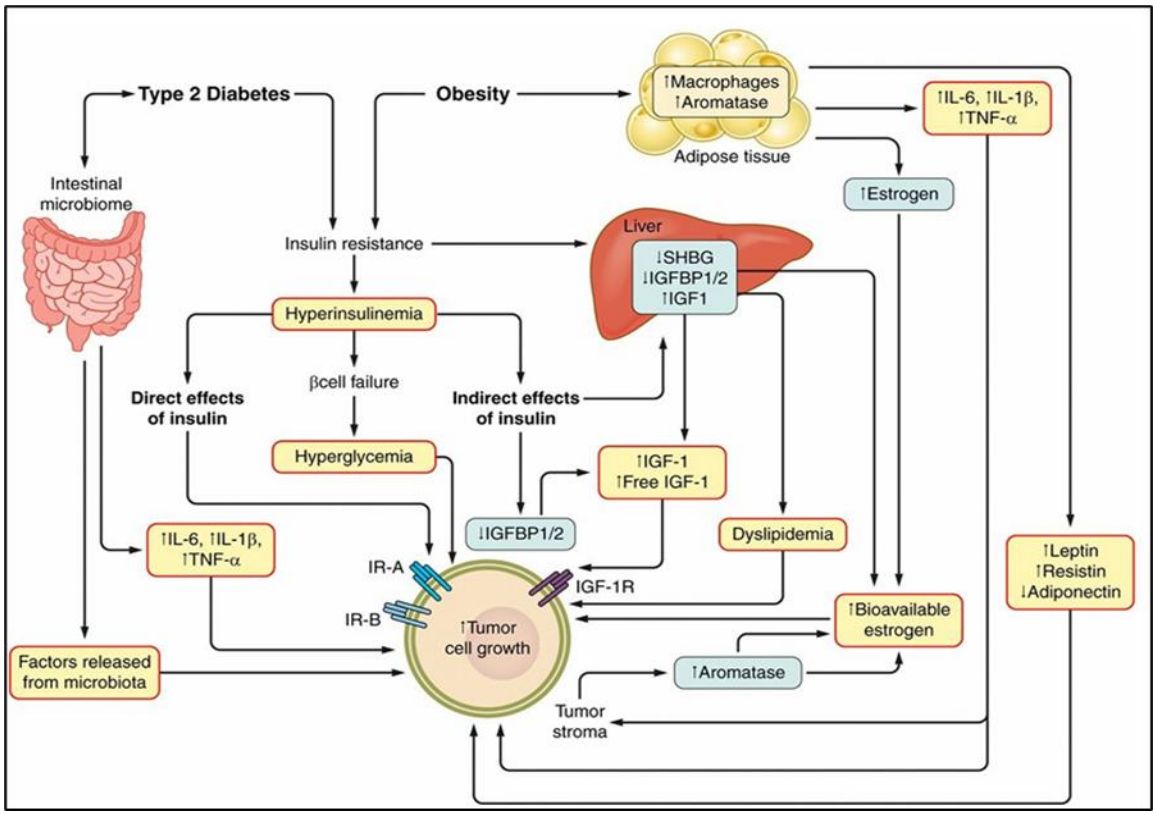


Figure 2. Potential mechanisms linking obesity/diabetes and cancer. IGFBP, IGF-binding protein; IR, insulin receptor; SHBG, sex hormone–binding globulin. Reproduced with permission from Gallagher EJ. Obesity and diabetes: the increased risk of cancer and cancer-related mortality [23].

MECHANISMS OF BREAST CANCER DEVELOPMENT AND METASTASIS

A number of complex cyto-biological processes contribute to the development and metastasis of breast cancer, converting a healthy breast cell into a malignant one that grows, invades, and finally spreads to other locations. Comprehending these mechanisms is essential for creating tailored treatments and enhancing patient results [21]. Approximately 70% of breast cancer metastases occur to the bone, making it the most frequent location. It is commonly linked to metastatic lesions of the osteolytic type because of hyperactive osteoclast-mediated bone resorption. All subtypes are susceptible to bone metastases, although the risk of bone metastasis is much greater for luminal subtype tumours (80.5%) compared to HER2-like tumours (55.6%) and basal-like tumours (41.7%) [24]. Patients with non-metastatic BC may ultimately acquire distant metastases, even if initial therapy was effective, it is important to comprehend the mechanisms behind the metastatic process and the intricate tumour-host interactions leading the disease's evolution [25].

CHEMOTHERAPY

The effect of chemotherapy-induced menopause on cognitive function needs to be better explored as the use of chemotherapy to treat early-stage breast cancer grows. The effects of chemotherapy are far more widespread, the majority of data points to the oestrogen impact being at least partially unique to verbal memory. Since verbal memory functioning as assessed by comparable neuropsychological tests is influenced by both oestrogen level and chemotherapy, there may be a significant interaction between menopausal state and chemotherapy in the verbal memory domain [26]. There is growing evidence that anticancer treatment can alter the microbiome of breast cancer. Neoadjuvant chemotherapy, which typically consists of anthracycline, alkylating agents, and taxes, is sometimes administered to patients with breast cancer to shrink the tumour before surgery. Given the dearth of efficient inhibitors and the progressive up-regulation of these proteins with continued chemotherapy treatment, evaluation of MDR expression may one day be used as an extra stratification for detecting high-risk malignancies, although its use is probably limited [27]. The chemotherapy regimen a patient receives will depend on the grade and severity of their breast cancer. The most common signs and symptoms and examined the connection between adjuvant chemotherapy side effects and outcomes in elderly patients with breast cancer. These most often include mucositis, diarrhoea, nausea, vomiting and infection symptoms brought on by a low white blood cell count. These clinical characteristics may have a negative impact on a patient's quality of life as well as that of their family members and support system. Before starting chemotherapy, patients can feel scared and anxious since they may have had direct or indirect experience with the side effects in the past [28]. The majority of patients with breast cancer who get chemotherapy do it adjuvant or just after surgery. However, as a so-called neo-adjuvant treatment, chemotherapy could be administered prior to surgery. "Primary medical therapy" and "induction chemotherapy" are synonyms. The treatment of locally progressed and incurable breast cancer is where neoadjuvant chemotherapy first appeared. Primary chemotherapy plays a major role in the unique instance of inflammatory breast cancer; many centres choose to treat patients with cytotoxic medicines and radiation therapy instead of surgery [29]. Some chemotherapeutic drugs have a direct effect on natural killer cells. Epirubicin pretreatment dramatically increases Killer cell-mediated cytotoxicity against tumour cells, according to in vitro research. This implies that Knell-based immunotherapy in conjunction with anthracycline-based chemotherapy may be a powerful therapeutic approach for breast cancer. Killer cell responses were first seen to be compromised by cytotoxic chemotherapeutics in patients with breast cancer; nevertheless, Killer cell numbers (CD56) usually return to normal following adjuvant treatment [30]. When primary breast conservation is not feasible because to tumour size or the relationship between the cancer and breast size, neoadjuvant systemic treatment has become the gold standard of care, as long as the patient has a chemotherapeutic rationale [31].

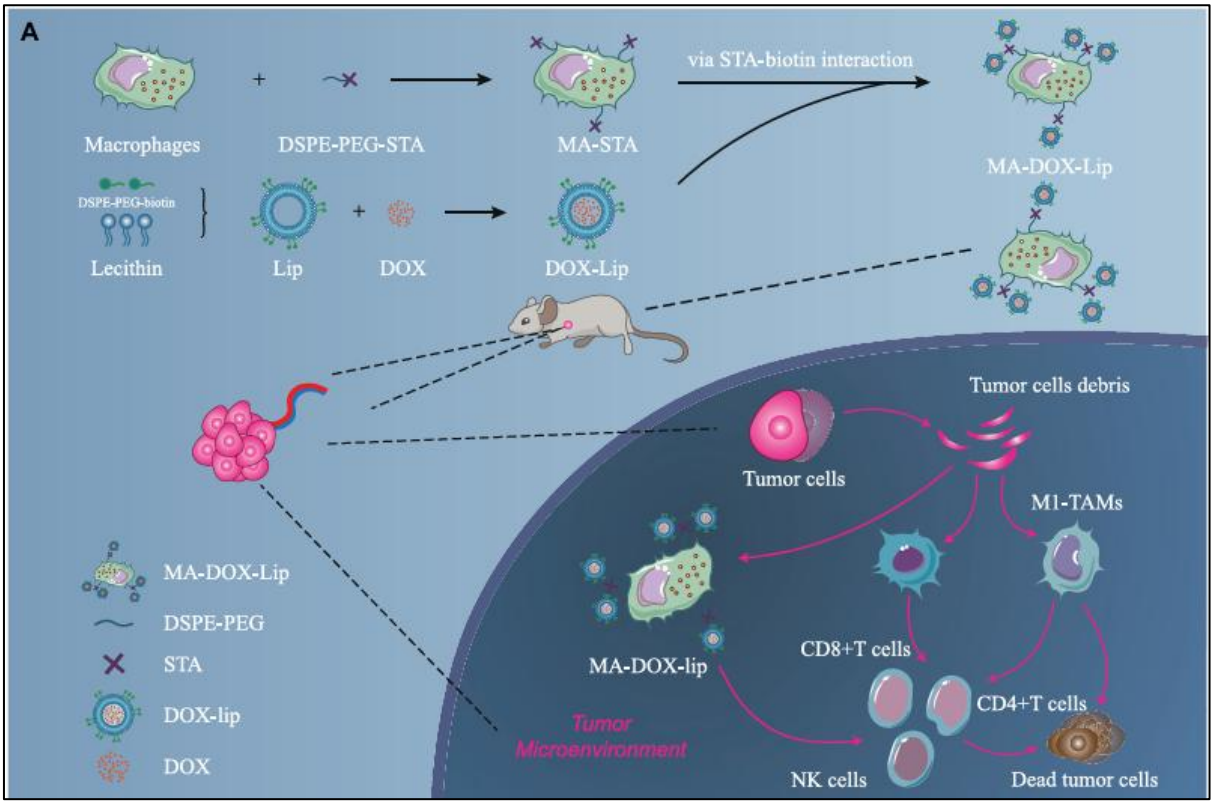
"The use of pharmacologic or natural agents that inhibit the development of invasive breast cancer either by blocking the DNA damage that initiates carcinogenesis, or by arresting or reversing the progression of premalignant cells in which such damage has already occurred" is the traditional definition of chemotherapy given by Sporn. Since over 70% of breast tumours are ER-positive, the oestrogen receptor is a key target for chemotherapy. Two main kinds of anti-oestrogen medications are aromatase inhibitors (AIs) and selective oestrogen receptor modulators (SERMs) [32]. About 70% of all instances of breast cancer are hormone receptor-positive (HR+) cases [33]. The long-term chemotherapy regimens that combine taxeme and tricyclene schedules mirror everyday routines. Standard fractionation is still used to provide radiotherapy [34]. Given the high number of breast cancer survivors, more knowledge on the long-term effects of anticancer therapies is necessary. Every anticancer treatment has unique side effects, some of which overlap [35]. The use of cytotoxic chemotherapy in both advanced and early-stage breast cancer have made significant progress in the last 10 years with several landmark studies identifying clear survival benefits for newer therapies. Despite these developments the optimal approach for any specific patient cannot be determined from a literature review or decision -making algorithm alone [36]. 5-fluorouracil (CMF), methotrexate, and cyclophosphamide. In a prospective clinical study, CMF was the first combined adjuvant chemotherapy treatment to be studied. Regardless of lymph node, menopausal, or hormone receptor status, the majority of women with localised breast cancer should get adjuvant polychemotherapy, according to a National Institutes of Health consensus group [37]. According to a National Institutes of Health consensus committee, adjuvant

polychemotherapy should be given to most women with localised breast cancer, regardless of their lymph node, menopausal, or hormone receptor status. These factors ultimately decide the prognosis of breast cancer [38]. Combination therapies based on taxeme, anthracycline, cyclophosphamide, cisplatin, and fluorouracil are advised by the national comprehensive cancer network recommendations [39].

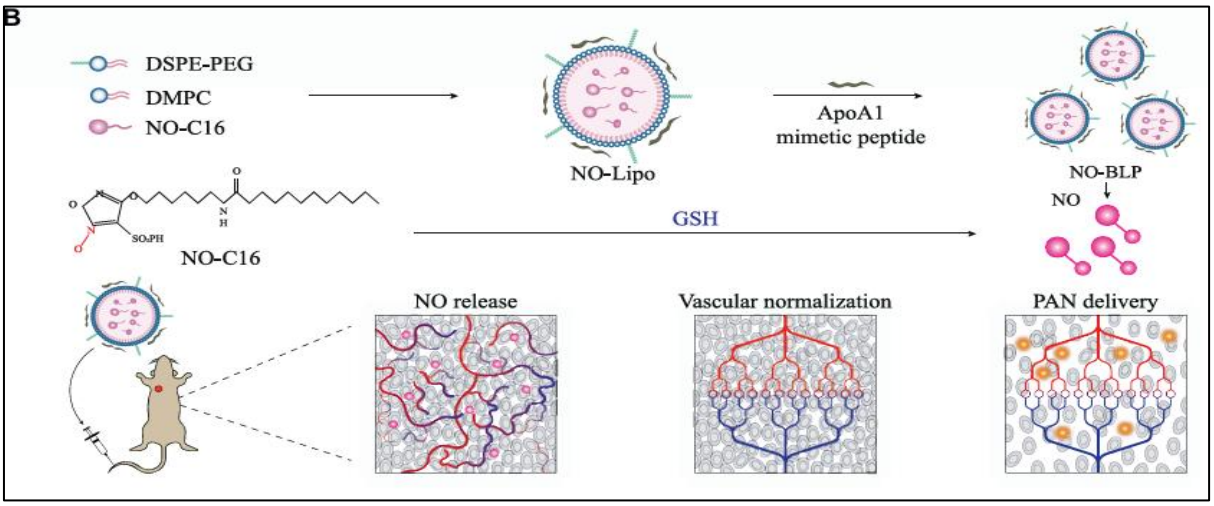
RADIATION THERAPY

Because the breasts are positioned on the chest wall while the patient is supine, patients with big breasts may get higher doses of radiation to vital organs such the heart or lungs. Using prone whole-breast radiation, which has been shown to provide favourable toxicity profiles, is one method to reduce the toxicities related to radiotherapy in patients with larger breasts and/or greater BMI [40].

Figure 3: Prolonging the retention times of chemotherapeutic drugs in tumour tissues and activating strong immune responses [41].



(A)Through DSPE-PEG-STA interactions, DOX-Lip links interacts with macrophages; the mechanism of in vivo action of self-assembled lipid-based nanoparticles (Yangtal.,2022) ⓒ2022, American Chemical Society.



(B) Diagrammatic representation of self-assembled lipid-based nanoparticles acting on breast cancer tissue (Wuetal.,2023) ⓒ2023, American Chemical Society. PAN stands for paclitaxel nanoparticles, NO-BLP for donor-loaded bioinspired lipoprotein system, DSPE-PEG for 1,2-distearoyl-sn-glycero-3-phosphoethanolamine poly (ethylene glycol), and DMPC for 1,2-dimyristoyl-sn-glycero-3-phosphatidylcholine.

VACCINES

Bivalent vaccines containing the immunologic adjuvant OPT-821 and the neuroblastoma-associated antigens GD2 and GD3 are now undergoing phase II trials. In NB patients, these vaccinations have resulted in anti-GD2 and anti-GD3 IgG antibody responses [42].

BREAST CANCER STEM CELLS

Although they make up a small portion of the tumour, breast cancer stem cells (CSC) are believed to play a crucial role in tumours because of their capacity for unrestricted multiplication [27]. High levels of progesterone in the blood can induce pluripotent stem cells of immature breast tissue to undergo terminal differentiation, and the first full-term pregnancy before the age of 20 is protective against breast cancer. Malignant stem cells are resistant to traditional treatment and either quiescent or cycle slowly. Their capacity for self-renewal offers the chance for both clinical recurrence of cancer and regeneration. Selective targeting of this significant subgroup of tumour cells will be possible if biochemical mechanisms specific to cancer stem cells are identified. Anticipate the cellular response to treatments and co-target escape mechanisms [43].

MAMMARY GLAND INFLAMMATION

Increased inflammation in breast tissue can result from both obesity and sudden involution. By itself, obesity is defined as a persistent condition of systemic inflammation that raises local inflammation and raises the chance of developing cancer. The mammary gland has acute inflammation because of abrupt involution, which persists over time [44]. The majority indicates that rather than eliciting potent antitumor responses, the inflammatory cells and cytokines present in tumours are more likely to aid in immunosuppression. Additionally, women with weakened immune systems had a lower relative chance of developing common epithelial malignancies, such as breast adenocarcinoma. According to one earlier study, women who took NSAIDs at least twice a week for at least five years had a 21% lower chance of developing breast cancer [45]. When cancer cells obstruct lymph veins or channels in the skin covering the breast, the result is swollen, heated, and red breasts with dimples and/or wide ridges. This condition is known as inflammatory breast cancer. Despite being uncommon, inflammatory breast cancer grows quite quickly. All multidisciplinary approaches, including as radiation therapy, surgery, chemotherapy, and imaging, must be carefully coordinated during treatment [46].

HEALTHY DIETS

BC has been linked to good circadian dietary practices, which, when disturbed, contribute to BCR. Additionally, the biomarkers of inflammation and BCR can be downregulated by eating more often, consuming less calories in the evening, and fasting for extended periods of time. Breakfast is the first meal of the day and is frequently referred to as "the most important meal of the day". Research indicates that eating a healthy breakfast and eating foods high in nutrients are associated with a lower body mass index (BMI), greater life satisfaction, and improved cognitive function [47]. Through the nuclear factor kappa-light-chain-enhancer of activated B cells (NF-κB) and mitogen-activated protein kinase (MAPK) signalling pathways, it has been demonstrated that the anti-inflammatory and anticancer mechanisms of several compounds in pomegranate extract, such as ellagitannins, anthocyanins, and phenols, reduce the expression of cyclooxygenase-2 (COX-2). This decrease results in less cell proliferation and less pro-inflammatory prostaglandin synthesis. reduced cell proliferation and the production of pro-inflammatory prostaglandins. Furthermore, the downregulation of inflammatory gene transcription is further facilitated by the inhibition of phosphatidylinositol 3-kinases (PI3K), protein kinase B (Akt), or NF-κB itself [48].

UNHEALTHY DIETS

While a long-term anti-inflammatory diet can extend the longevity of BC patients, a pro-inflammatory diet that is centred on a high intake of red, processed meat and alcohol was linked to an elevated BCR. Increased BCR is also linked to the unhealthy dietary pattern, which includes sugars, processed juices, soft drinks, potato chips and mayonnaise, desserts, solid oils, red and processed meat, and high salt intake, as well as the Western dietary pattern, which is high in hydrogenated fat, soft drinks, animal fat, fast food, refined cereals, sweets, and processed meat [47].

ROLE OF P53 IN BREAST CANCER

One important tumour suppressor protein that keeps the genome stable is p53. Replication stress, oxidative stress, hypoxia, DNA damage, food deprivation, and telomere shortening are all examples of stress responses that are known to activate p53, the primary regulatory transcription factor. A loss of tumour suppressive activity or an increase in carcinogenic qualities are linked to p53 gene mutations, which are common in breast cancer. These mutations are linked to metastasis, tumour growth, and treatment resistance [49].

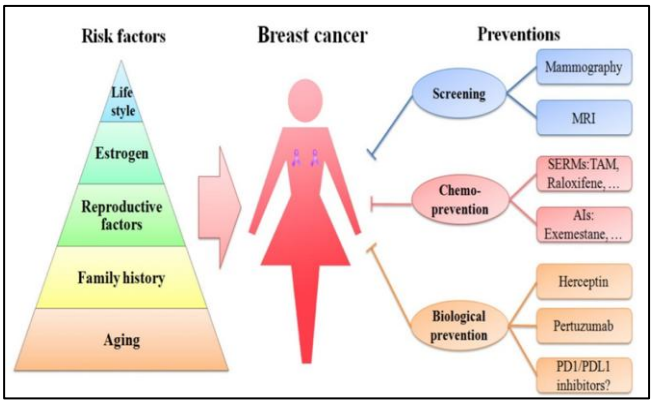


Figure 4: shows a schematic representation of breast cancer risk factors and preventative measures. The pyramid graphic illustrates the five key risk factors for breast cancer: age, family history, reproductive factors, oestrogen, and lifestyle. Breast cancer is presently prevented via screening

(mammography and MRI), chemoprevention (using SERMs and AIs), and biological prevention (using Herceptin and epratuzumab). Immunotherapy medications known as PD1/PDL1 inhibitors may be effective treatments for TNBC[32].

AVOIDANCES

Clinical and theoretical research on breast cancer has advanced significantly thus far (Figure 4). Compared to earlier preventative strategies, the present ones such as screening, chemoprevention, and biological prevention are more straightforward and successful (Figure 4). The death rate from breast cancer has dropped. However, among women aged 20 to 59, breast cancer continues to be the primary cause of cancer-related deaths [32].

CONCLUSION

According to this review, the selection of a promising approach has led to significant progress in the treatment of BC. Hormone treatments and other conventional methods were insufficiently effective. In conclusion, ongoing research and learning are necessary for the diagnosis and management of phyllodes tumours. One of the most prevalent cancers affecting women globally, breast cancer is characterised by a variety of genetic and epigenetic changes. One of the biggest threats to world health is breast cancer. Knowing its categorisation, molecular causes, and available treatments is vital. Patients with breast cancer who are obese make up a special patient group. They are more susceptible to the development of breast cancer and can encounter additional issues with radiation and surgery. Obese women are more likely than normal-weight women to experience a local recurrence even with proper local illness therapy.

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REFERENCES

1. Barzaman K, Karami J, Zarei Z, Hosseinzadeh A, Kazemi MH, Moradi-Kalbolandi S, Safari E, Farahmand L. Breast cancer: Biology, biomarkers, and treatments. Int Immunopharmacol. 2020 Jul;84:106535. doi: 10.1016/j.intimp.2020.106535. Epub 2020 Apr 29. PMID: 32361569.
2. Pan S, Gadrey JY, Sammons S, Lin NU, Tolaney SM, Tarantino P, Schlam I. Role of antibody drug conjugates in the treatment of patients with breast cancer brain metastases. Ther Adv Med Oncol. 2024 Nov 10; 16: 17588359241292266. doi: 10.1177/17588359241292266. PMID: 39529890; PMCID: PMC11552056.
3. Zhang H, Hussin H, Hoh CC, Cheong SH, Lee WK, Yahaya BH. Big data in breast cancer: Towards precision treatment. Digit Health. 2024 Nov 3;10:20552076241293695. doi: 10.1177/20552076241293695. PMID: 39502482; PMCID: PMC11536614.
4. Chen M, Feng M, Lei H, Dan Mo, Ren S, Yang D. The development of the occurrence and metastasis of breast cancer by single-cell sequencing. Cancer Cell Int. 2024 Oct 26;24(1):349. doi: 10.1186/s12935-024-03531-x. PMID: 39462368; PMCID: PMC11515250.
5. Jiang RY, Zhu JY, Zhang HP, Yu Y, Dong ZX, Zhou HH, Wang X. STAT3: Key targets of growth-promoting receptor positive breast cancer. Cancer Cell Int. 2024 Oct 28;24(1):356. doi: 10.1186/s12935-024-03541-9. PMID: 39468521; PMCID: PMC11520424.
6. Zhang G, Cheng C, Wang X, Wang S. N6-Methyladenosine methylation modification in breast cancer: current insights. J Transl Med. 2024 Oct 28;22(1):971. doi: 10.1186/s12967-024-05771-x. PMID: 39468547; PMCID: PMC11514918.
7. Chen C, Xiang A, Lin X, Guo J, Liu J, Hu S, Rui T, Ye Q. Mitophagy: insights into its signaling molecules, biological functions, and therapeutic potential in breast cancer. Cell Death Discov. 2024 Oct 29;10(1):457. doi: 10.1038/s41420-024-02226-6. PMID: 39472438; PMCID: PMC11522701.
8. Mihai AM, Ianculescu LM, Creţoiu D, Suciu N. In vitro fertilization impact on the risk of breast cancer. Arch Clin Cases. 2024 Oct 9;11(3):73-82. doi: 10.22551/2024.44.1103.10292. PMID: 39479256; PMCID: PMC11520175.
9. Walker RA. The complexities of breast cancer desmoplasia. Breast Cancer Res. 2001;3(3):143-5. doi: 10.1186/bcr287. Epub 2001 Feb 1. PMID: 11305947; PMCID: PMC138677.
10. Li Y, Tao X, Ye Y, Tang Y, Xu Z, Tian Y, Liu Z, Zhao J. Prognostic nomograms for young breast cancer: A retrospective study based on the SEER and METABRIC databases. Cancer Innov. 2024 Oct 25;3(6):e152. doi: 10.1002/cai2.152. PMID: 39464427; PMCID: PMC11503687.
11. Arribas J, Baselga J, Pedersen K, Parra-Palau JL. p95HER2 and breast cancer. Cancer Res. 2011 Mar 1;71(5):1515-9. doi: 10.1158/0008-5472.CAN-10-3795. Epub 2011 Feb 22. PMID: 21343397.
12. da Costa Vieira RA, Biller G, Uemura G, Ruiz CA, Curado MP. Breast cancer screening in developing countries. Clinics (Sao Paulo). 2017 Apr;72(4):244-253. doi: 10.6061/clinics/2017(04)09. PMID: 28492725; PMCID: PMC5401614.
13. Abdel-Razeq H, Sharaf B, Tamimi F, Hani HB, Alsmadi O, Khalil H, Abunasser M, Edaily S, Mansour A. Establishment of a clinical cancer genetics program for breast cancer in a resource-limited country; challenges and opportunities. Front Oncol. 2024 Oct 23;14:1431985. doi: 10.3389/fonc.2024.1431985. PMID: 39507757; PMCID: PMC11537866.
14. Alzibdeh A, Abuhijlih R, Abuhijla F. Breast cancer radiobiology: The renaissance of whole breast radiation fractionation (Review). Mol Clin Oncol. 2024 Oct 22;21(6):97. doi: 10.3892/mco.2024.2795. PMID: 39484288; PMCID: PMC11526245.
15. Hrubesz G, Leigh J, Ng TL. Understanding the relationship between breast cancer, immune checkpoint inhibitors, and gut microbiota: a narrative review. Transl Breast Cancer Res. 2024 Oct 21;5:31. doi: 10.21037/tbcr-24-14. PMID: 39534584; PMCID: PMC11557166.

16. Richie RC, Swanson JO. Breast cancer: a review of the literature. J Insur Med. 2003;35(2):85-101. PMID: 14733031.

17. Surdacka LM, Jakubas A, Jagiełło J, Daniłowska K, Picheta N, Gil-Kulik P. Epigenetic and Immune Mechanisms Linking Breastfeeding to Lower Breast Cancer Rates. Med Sci Monit. 2024 Nov 5;30:e945451. doi: 10.12659/MSM.945451. PMID: 39497379; PMCID: PMC11549897.

18. Mukherjee A, Bandyopadhyay D. Targeted Therapy in Breast Cancer: Advantages and Advancements of Antibody-Drug Conjugates, a Type of Chemo-Biologic Hybrid Drugs. Cancers (Basel). 2024 Oct 17;16(20):3517. doi: 10.3390/cancers16203517. Erratum in: Cancers (Basel). 2025 May 12;17(10):1633. doi: 10.3390/cancers17101633. PMID: 39456611; PMCID: PMC11505910.

19. Bao W, Ma X, Xue Y, Zou X, Guo Y. Pregnancy-associated triple-negative breast cancer: A case report and literature review. Medicine (Baltimore). 2024 Oct 11;103(41):e40059. doi: 10.1097/MD.0000000000040059. PMID: 39465823; PMCID: PMC11479494.

20. Akram M, Iqbal M, Daniyal M, Khan AU. Awareness and current knowledge of breast cancer. Biol Res. 2017 Oct 2;50(1):33. doi: 10.1186/s40659-017-0140-9. PMID: 28969709; PMCID: PMC5625777.

21. Akl MM, Ahmed A. Cytobiological Alterations Induced by Celecoxib as an Anticancer Agent for Breast and Metastatic Breast Cancer. Adv Pharm Bull. 2024 Oct;14(3):604-612. doi: 10.34172/apb.2024.055. Epub 2024 Jun 29. PMID: 39494258; PMCID: PMC11530885.

22. Tang Z, Tian X. *Astragalus membranaceus*: A Traditional Chinese Medicine with Multifaceted Impacts on Breast Cancer Treatment. Biomolecules. 2024 Oct 21;14(10):1339. doi: 10.3390/biom14101339. PMID: 39456271; PMCID: PMC11506204.

23. Kang C, LeRoith D, Gallagher EJ. Diabetes, Obesity, and Breast Cancer. Endocrinology. 2018 Nov 1;159(11):3801-3812. doi: 10.1210/en.2018-00574. PMID: 30215698; PMCID: PMC6202853.

24. Chen W, Hoffmann AD, Liu H, Liu X. Organotropism: new insights into molecular mechanisms of breast cancer metastasis. NPJ Precis Oncol. 2018 Feb 16;2(1):4. doi: 10.1038/s41698-018-0047-0. PMID: 29872722; PMCID: PMC5871901.

25. Rusnáková DŠ, Aziri R, Dubovan P, Jurík M, Mego M, Pinďák D. Detection, significance and potential utility of circulating tumor cells in clinical practice in breast cancer (Review). Oncol Lett. 2024 Oct 17;29(1):10. doi: 10.3892/ol.2024.14756. PMID: 39492933; PMCID: PMC11526295.

26. Ahles TA, Saykin AJ. Breast cancer chemotherapy-related cognitive dysfunction. Clin Breast Cancer. 2002 Dec;3 Suppl 3:S84-90. doi: 10.3816/cbc.2002.s.018. PMID: 12533268.

27. Marquette C, Nabell L. Chemotherapy-resistant metastatic breast cancer. Curr Treat Options Oncol. 2012 Jun;13(2):263-75. doi: 10.1007/s11864-012-0184-6. PMID: 22528367.

28. Graham J. Breast Cancer: The Psychological Impact of Diagnosis, Treatment, and Remission. Cureus. 2024 Oct 4;16(10):e70814. doi: 10.7759/cureus.70814. PMID: 39493150; PMCID: PMC11531659.

29. Cleator S, Parton M, Dowsett M. The biology of neoadjuvant chemotherapy for breast cancer. Endocr Relat Cancer. 2002 Sep;9(3):183-95. doi: 10.1677/erc.0.0090183. PMID: 12237246.

30. Naji O, Ghouzlani A, Rafii S, Sadiqi RU, Kone AS, Harmak Z, Choukri K, Kandoussi S, Karkouri M, Badou A. Investigating tumor immunogenicity in breast cancer: deciphering the tumor immune response to enhance therapeutic approaches. Front Immunol. 2024 Oct 23;15:1399754. doi: 10.3389/fimmu.2024.1399754. Erratum in: Front Immunol. 2024 Dec 11;15:1532921. doi: 10.3389/fimmu.2024.1532921. PMID: 39507526; PMCID: PMC11538072.

31. Harbeck N, Gnant M, Thomssen C. Breast Cancer Is Our Global Responsibility. Breast Care (Basel). 2015 Dec;10(6):360. doi: 10.1159/000443159. Epub 2015 Dec 16. PMID: 26989353; PMCID: PMC4789920.

32. Sun YS, Zhao Z, Yang ZN, Xu F, Lu HJ, Zhu ZY, Shi W, Jiang J, Yao PP, Zhu HP. Risk Factors and Preventions of Breast Cancer. Int J Biol Sci. 2017 Nov 1;13(11):1387-1397. doi: 10.7150/ijbs.21635. PMID: 29209143; PMCID: PMC5715522.

33. Lőczi LL, Vleskó G, Éliás M, Turan C, Kajtár P, Tóth R, Sipos M, Nagy R, Hegyi P, Ács N, Várbíró S, Keszthelyi M. Effect of Vaginal Laser and Topical Therapies on Vulvovaginal Atrophy Symptoms in Breast Cancer Patients: A Systematic Review and Meta-Analysis. J Clin Med. 2024 Oct 15;13(20):6131. doi: 10.3390/jcm13206131. PMID: 39458081; PMCID: PMC11508551.

34. Lazzari G, Montagna A, D'Andrea B, Bianculli A, Calice G, Tucciariello R, Castaldo G, Metallo V, De Marco G, Benevento I. Breast Cancer Adjuvant Radiotherapy and Chemotherapy Sequencing: Sequential, Concomitant, or What Else? A Comprehensive Review of the Adjuvant Combinations Journey. J Clin Med. 2024 Oct 19;13(20):6251. doi: 10.3390/jcm13206251. PMID: 39458200; PMCID: PMC11508402.

35. Catalano O, Fusco R, Carriero S, Tamburrini S, Granata V. Ultrasound Findings After Breast Cancer Radiation Therapy: Cutaneous, Pleural, Pulmonary, and Cardiac Changes. Korean J Radiol. 2024 Nov;25(11):982-991. doi: 10.3348/kjr.2024.0672. PMID: 39473089; PMCID: PMC11524688.

36. Hassan MS, Ansari J, Spooner D, Hussain SA. Chemotherapy for breast cancer (Review). Oncol Rep. 2010 Nov;24(5):1121-31. doi: 10.3892/or_00000963. PMID: 20878101.

37. Anampa J, Makower D, Sparano JA. Progress in adjuvant chemotherapy for breast cancer: an overview. BMC Med. 2015 Aug 17;13:195. doi: 10.1186/s12916-015-0439-8. PMID: 26278220; PMCID: PMC4538915.

38. Xu X, Zhang M, Xu F, Jiang S. Wnt signaling in breast cancer: biological mechanisms, challenges and opportunities. Mol Cancer. 2020 Nov 24;19(1):165. doi: 10.1186/s12943-020-01276-5. PMID: 33234169; PMCID: PMC7686704.

39. Yin L, Duan JJ, Bian XW, Yu SC. Triple-negative breast cancer molecular subtyping and treatment progress. Breast Cancer Res. 2020 Jun 9;22(1):61. doi: 10.1186/s13058-020-01296-5. PMID: 32517735; PMCID: PMC7285581.

40. Lee K, Kruper L, Dieli-Conwright CM, Mortimer JE. The Impact of Obesity on Breast Cancer Diagnosis and Treatment. Curr

Oncol Rep. 2019 Mar 27;21(5):41. doi: 10.1007/s11912-019-0787-1. PMID: 30919143; PMCID: PMC6437123.

41. Liu S. Self-assembled lipid-based nanoparticles for chemotherapy against breast cancer. Front Bioeng Biotechnol. 2024 Oct 29;12:1482637. doi: 10.3389/fbioe.2024.1482637. PMID: 39534673; PMCID: PMC11555772.
42. Zhang G, Zeng J, Li C, Wei C. Breast tumor with giant borderline phyllodes: Case report and literature review. Medicine (Baltimore). 2024 Nov 1;103(44):e37260. doi: 10.1097/MD.0000000000037260. PMID: 39496019; PMCID: PMC11537575.
43. Benson JR, Jatoi I, Keisch M, Esteva FJ, Makris A, Jordan VC. Early breast cancer. Lancet. 2009 Apr 25;373(9673):1463-79. doi: 10.1016/S0140-6736(09)60316-0. PMID: 19394537.
44. Ormiston K, Kulkarni A, Sarathy G, Alsammerai S, Shankar E, Majumder S, Stanford KI, Ganju RK, Ramaswamy B. Obesity and lack of breastfeeding: a perfect storm to augment risk of breast cancer? Front Oncol. 2024 Oct 25;14:1432208. doi: 10.3389/fonc.2024.1432208. PMID: 39525621; PMCID: PMC11543574.
45. Bates JP, Derakhshandeh R, Jones L, Webb TJ. Mechanisms of immune evasion in breast cancer. BMC Cancer. 2018 May 11;18(1):556. doi: 10.1186/s12885-018-4441-3. PMID: 29751789; PMCID: PMC5948714.
46. Akram M, Iqbal M, Daniyal M, Khan AU. Awareness and current knowledge of breast cancer. Biol Res. 2017 Oct 2;50(1):33. doi: 10.1186/s40659-017-0140-9. PMID: 28969709; PMCID: PMC5625777.
47. Neagu AN, Josan CL, Jayaweera TM, Weraduwage K, Nuru N, Darie CC. Double-Edged Sword Effect of Diet and Nutrition on Carcinogenic Molecular Pathways in Breast Cancer. Int J Mol Sci. 2024 Oct 15;25(20):11078. doi: 10.3390/ijms252011078. PMID: 39456858; PMCID: PMC11508170.
48. Jang JY, Kim D, Im E, Kim ND. Therapeutic Potential of Pomegranate Extract for Women's Reproductive Health and Breast Cancer. Life (Basel). 2024 Oct 3;14(10):1264. doi: 10.3390/life14101264. PMID: 39459564; PMCID: PMC11509572.
49. Song J, Cui Q, Gao J. Roles of lncRNAs related to the p53 network in breast cancer progression. Front Oncol. 2024 Oct 16;14:1453807. doi: 10.3389/fonc.2024.1453807. PMID: 39479021; PMCID: PMC11521785.