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Emerging Trends in Breast Cancer Therapeutics: Insights into Novel Drug Discovery and Development

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ABSTRACT

Breast cancer is the most-commonly diagnosed malignant tumor in women in the world, as well as the first cause of death from malignant tumours. The incidence of breast cancer is constantly increasing in all regions of the world. Since last two decades, researches related to the breast cancer has led to extraordinary progress in our understanding of the disease, resulting in more efficient and less toxic treatments. Increased public awareness and improved screening have led to earlier diagnosis at stages amenable to complete surgical resection and curative therapies. Consequently, survival rates for breast cancer have improved significantly, particularly in younger women. This article addresses the types, causes, clinical symptoms, diagnosis, treatment (including chemotherapy, gene therapy etc.) and history of drugs used in breast cancer.

INTRODUCTION

When cells in the breast start to proliferate out CONTROL (1)1The breast lobules, tubes, or connective tissue can develop breast cancer, which is the most common cancer in women. The most common forms of breast cancer can be separated into two categories based on site: invasive breast cancer and non-invasive/in situ breast cancer [2], though there are other possible criteria and classifications. Lung Cance redevelops and happens as a result of various external and internal factors. Environmental elements, poor lifestyle decisions, and societal psychological factors are connected to its occurrence. It's actually shown that between 5% and 10% of breast cancer cases can be linked Tod Genetic mutations, family history, and potentially modifiable factors account for 20% to 30% of breast cancer cases. [3]

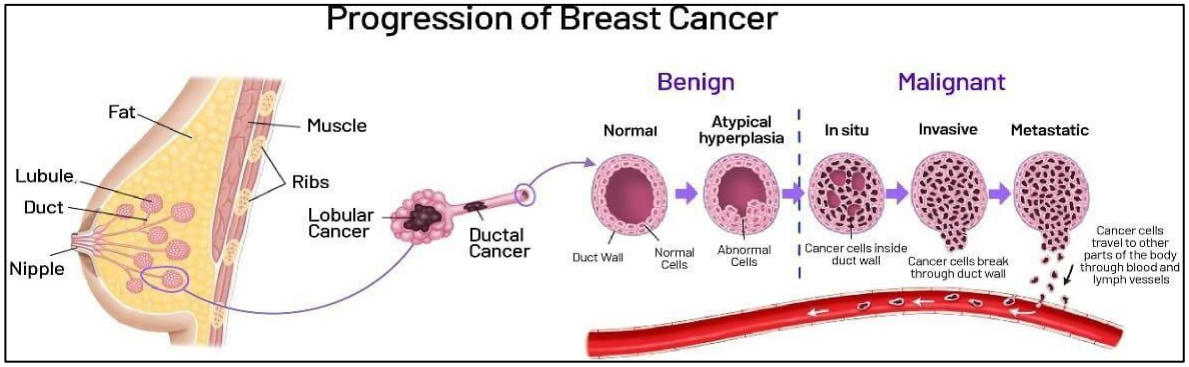

Fig no: 1

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Epidemiology

The most prevalent type of cancer among American women is breast cancer. Approximately 32% of new cases of cancer in women occur each year. Nare cases of breast cancer.r.It. It is anticipated that approximately 316,950 American women will be b59,080 more will be affected by invasive breast cancer. Found to have ductal carcinoma in situ (DCIS), a non-invasive, Sa precursor to breast cancer. Approximately 16% of cases of breast cancer affect women under 50. ancient. Sit is projected that 42,170 women will lose their lives to breast cancer in 2025 [4].2According to GLOBOCAN 2022 data, there are 2.3 million new cases of breast cancer in India. With an estimated 666,103 deaths in 2022, it remains the leading cause of cancer-related deaths among women [5].

Pathophysiology

When a genetically susceptible host comes into contact with an environmental (external) factor, breast cancer develops. Before stopping, normal cells divide as many times as necessary. They stick to other cells and stay in tissues for a very long time. Cells become malignant when they are unable to stop proliferating, adhere to other cells, and die at the right time. Different protein clusters and pathways shield cells from programmed cell death. The RAS/MEK/ERK pathway and the PI3K/AKT pathway are two of the protective pathways. The PI3K/AKT pathway is typically shut down by the PTEN protein when a cell is ready for programmed cell death. The PTEN protein gene is metamorphosed in certain breast cancers, which traps the PI3K/AKT pathway in the "on" position and prevents the cancer cell from self-destructing [6].

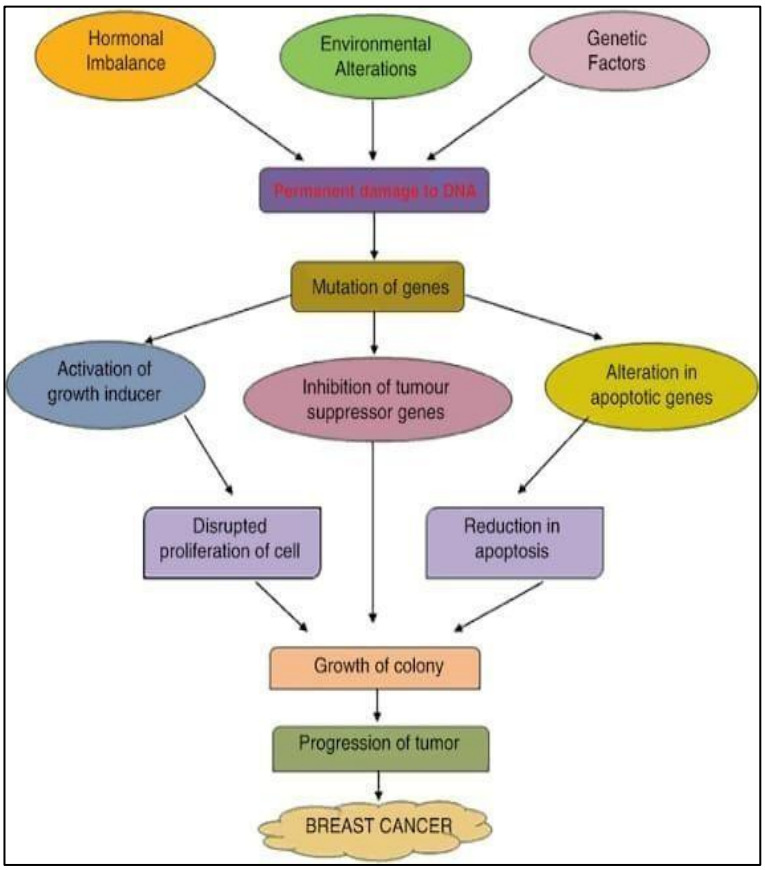

Figure 2.

Types

[A]**Invasive Breast Cancer:** cells that pierce the duct and lobular wall and infiltrate the breast's surrounding fatty and connective tissues [7].

1. **Invasive breast cancer** that begins in the milk ducts—the tubes that transport milk to the nipple—is known as invasive ductal carcinoma (IDC)...Invasive ductal carcinomas account for approximately 80% of all cases of breast cancer, making it the most prevalent type.

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2. **Invasive lobular carcinoma (ILC):** This type of breast cancer begins in the breast's milk- producing glands, or lobules. About 10% of all invasive breast cancers are invasive carcinomas, making it the second most prevalent type of the disease. (8).

[B] **Breast cancer that is not invasive:** Cells that are limited to the educts and do not penetrate the breast's surrounding fatty and connective tissues [7].

1. **Ductal carcinoma in situ (DCIS):** When a tumor is discovered in the milk ducts but has not spread outside of them, treatment is recommended because the tumor may eventually become invasive (9)9.

2. **Lobular neoplasia, also known as lobular carcinoma in situ:** -non-invasive breast cancer that has not progressed beyond the original lobules. Din spite of its name, LCIS is not actually breast cancer; rather, it is a benign breast condition. [8]

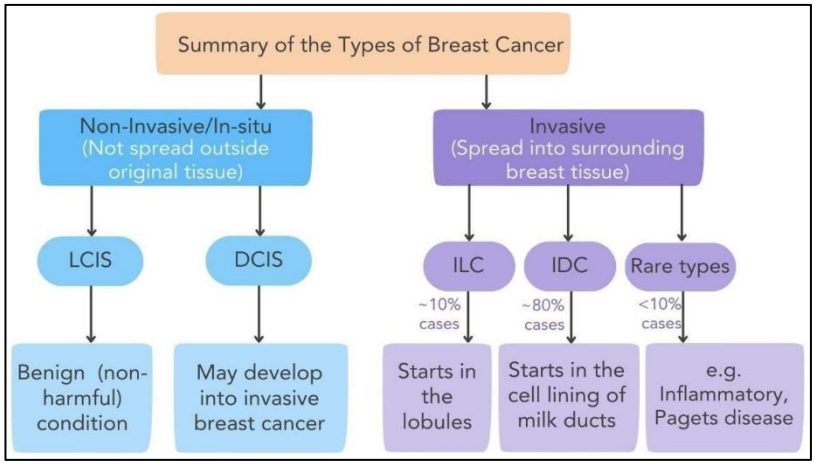

Figure 3.

Symptoms

- A new lump under the armpit or breast.
- Swelling or thickening of a portion of the breast.
- Pain or pulling in the nipple region.
- Blood and other non-breast milk-related discharge from the breast.
- Any alteration to the breast's dimensions or form. Pain in any area of the breast. (9), (10).

Risk Factor:

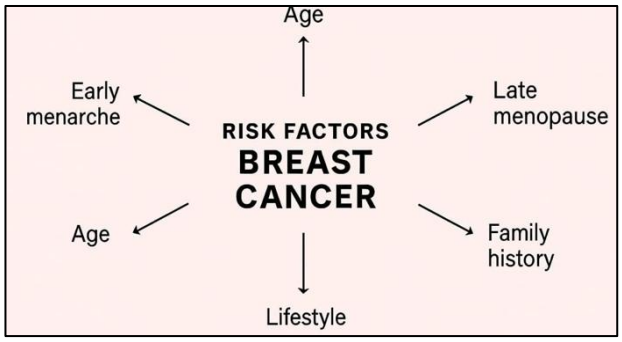

Figure 4.

Diagnosis

Mammography

The most popular screening method for early breast cancer detection is mammography, which also significantly lowers the death rate from breast cancer [11]. In contrast to diagnostic mammography, which is used to assess a patient with a positive clinical finding, such as a breast lump or an abnormal screening mammogram, screening mammography consists of two standard radiographic images of each breast. Additional views, such as spot compression or magnification views, are included in a diagnostic mammogram to look into the specific finding.

Ultrasound

When a palpable mass is not clearly visible on a mammogram, ultrasound screening can help distinguish between solid and cystic breast masses. When a palpable mass cannot be seen on a mammogram, ultrasound is particularly useful for young women with dense breast tissue. Because microcalcifications cannot be seen and the yield of carcinomas is very low, ultrasound should not be used for routine screening. [12].

biopsy

Biopsies are necessary to provide an accurate diagnosis following the identification of an anomaly in breast tissue using imaging techniques. A biopsy is an invasive procedure that involves removing abnormal breast tissue or fluid for molecular, histological, and cytological analysis. According to radiologists' use of the BI-RADS lexicon scale, the test is only advised in suspected cancer cases (Elmore et al., 2015). The gold standard method for determining whether a tumor is benign is still tumor biopsy [13].

MRI

By using RF signals and a strong magnetic field, MRI produces images at various cross- sections. Contrast agents can be used to improve the image's resolution. Due to its high false- positive rate, high cost, time commitment, insufficient number of units, requirement for skilled radiologists, and lack of clinical utility, breast MRI has been recommended for individuals with a high risk of breast cancer but not for the general population. The American College of Radiology (ACS) has published guidelines for MRI as an adjunctive tool to mammography, and annual MRI tests have been recommended for certain population groups, such as individuals with high risk of breast cancer and carriers of BRCA mutations [33]. Compared to mammography and ultrasound, MRI is less specific but more sensitive to detect small tumours in subjects with high breast cancer risk [14].

Treatment

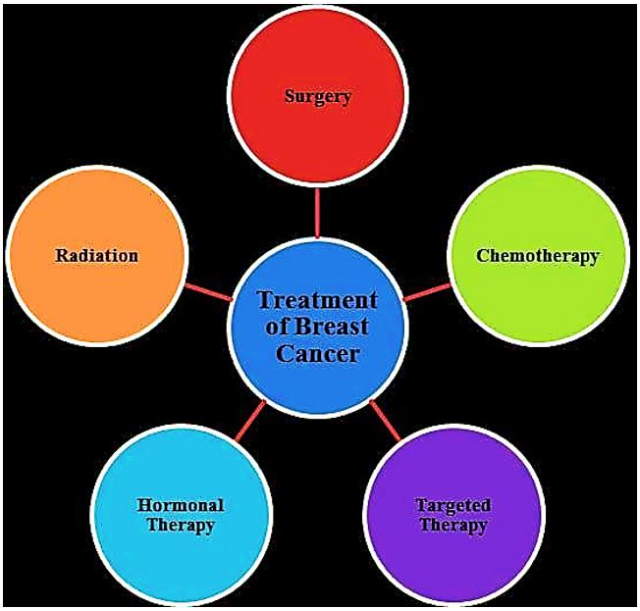

Figure 5.

Chemotherapy:

For women with node-positive cancer or tumours larger than one centimetre, chemotherapy is the recommended course of treatment. Chemotherapy employs medications to eradicate breast cancer cells. These medications are administered as pills or by vein [15].

Neoadjuvant chemotherapy: This type of treatment is administered prior to the main course of treatment.

Adjuvant chemotherapy: Adjuvant therapy is the treatment given in addition to the initial therapy, which can suppress or eliminate the growth of occult cancer cells[16].

Side effects:

* Hair loss
* Fatigue
* Loss of appetite
* Nausea and vomiting
* Constipation or diarrhoea
* Mouth sores
* Skin and nail changes

Drugs used:

* Anthracyclines
* Taxanes.

Hormone therapy

Cancers with progesterone and estragon receptors may be treated with hormone therapy. Either prior to surgery to reduce the tumor size or following surgery to stop recurrence. For those who are unable to receive chemotherapy, radiation, or surgery, hormone-blocking therapy may be an option. It is only effective for cancers that are hormone receptor-positive (HR-positive), not for–negative only [17].

Side effects

* Hot flashes
* Night sweats
* Virginal dryness
* Disturb the menstrual cycle

Drugs used

* Tamoxifen
* Aromatase inhibitors.

Targeted therapy

Targeted therapies are medications that use particular genetic alterations to target cancer cells. Targeted cancer medications target particular proteins found in cancer cells that aid in their development and spread. These medications eliminate cancer cells Oro decelerate their growth. Some, like monoclonal antibodies, also help the immune system fight cancer [18].

Side effects

* Skin problems like rash or dry skin
* Fatigue
* Diarrhoea
* High blood pressure
* And mouth sores.

Drugs used

* Tyrosine kinase inhibitors
* PARP inhibitors.

Radiation therapy

High-energy X-rays, protons, or other particles are used in radiation therapy for breast cancer to destroy cancer cells. Cancer cells and other rapidly proliferating cells are more vulnerable to the effects of radiation therapy than normal cells.

The particles or X-rays don't hurt and are undetectable. You are not radioactive after treatment, so it is safe to be around other people, including children [19].

Side effects

* Irritation and darkening of the skin
* Fatigue
* Lymphoedema
* Breast swelling

Drugs used

* Cisplatin
* Nimorazole.

History Of Drugs Used In Breast Cancer

Since breast cancer is the most common cancer diagnosed in women globally, developing new medications is a top priority. We examined oncology drug approvals by the US Food and Drug Administration (FDA) over a 70-year period from 1953 (the year of the first oncology drug approval) to 2020 in order to gain a better understanding of the development of new drug approvals for breast cancer [20].

S.No.	Drug name	Year of approval	Recommended dose	Drug delivery type
1.	**Tucatinib**	2020	300 mg	Combination
2.	Margetuximab	2020	15 mg/kg over 120 min	Single and combination both
3.	Pembrolizumab	2020	100 mg	Single and combination both
4.	Alpelisib	2019	90mg per day	Single and combination both
5.	Atezolizumab	2019	840 mg/mL	B Single and combination both
6.	Tolazoparib	2018	1 mg	Single
7.	Olaparib	2018	150 mg	Single and combination both
8.	Neratinib Maleate	2017	240 mg	Single
9.	Ribociclib	2017	200 mg	Combination
10.	Abemaciclib	2017	150 mg	Combination
11.	Palbociclib	2015	125 mg	combination
12.	Pertuzumab	2012	220 mg	Single and combination both
13.	Eribulin mesylate	2010	1.5 mg/m 2	Single
14.	Everolimus	2009	5mg once daily	Single and combination both
15.	Lapatinib	2007	250mg	250mg
16.	Ixabepilone	2007	15 mg	Single and combination both
17.	Fulvestrant	2002	5 ml	single
18.	Epirubicin	1999	2–10 mg/m^2 daily	Single
19.	Exemestane	1999	25 mg tablet once daily	Single
20.	Capecitabine	1998	500 m tablet twice daily	Single and combination both
21.	Transtuzumab	1998	440 mg	single
22.	Megestral Acetate	1998	40 mg	Single and combination both
23.	Toremifene	1997	60 mg once daily	Single
24.	Letrozole	1997	2.5 mg once daily	single
25.	Docetaxel	1996	20 mgh	Single and combination both
26.	Gemcitabine	1996	200 mg	Single and combination both
27.	Pamidronate	1996	Pamidronate	Single
28.	Anastrozole	1995	1 mg capsule once daily	single
29.	Paclitaxel	1992	20 ml	Single and combination both
30.	Goserelin Acetate	1989	3.6 mg	Single and combination both
31.	Tamoxifen	1977	10mg per day	single
32.	Doxorubicin	1974	10mg/5 mL	single
33.	Methyl testosterone	1973	50 mg	Single and combination both
34.	5-Fulurouracil	1970	10 mg	single
35.	Vinblastin Sulfate	1965	10 mg/mL	combination
36.	Cyclophosphamide	1959	200 mg	Single and combination both
37.	Tepadina	1957	0.3-0.4 mg/kg	combination
38.	Fluoxymesterone	1956	10–40 mg daily	single
39.	Methotrexate	1953	15 mg/mL	Combination [21]

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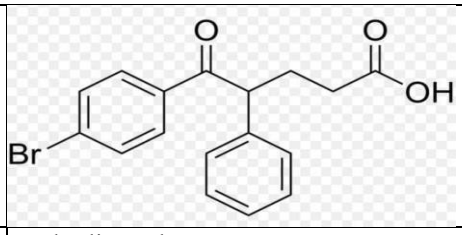
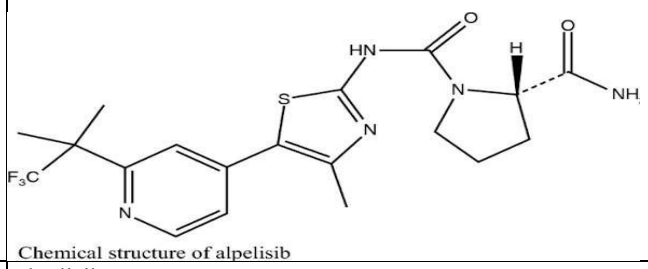
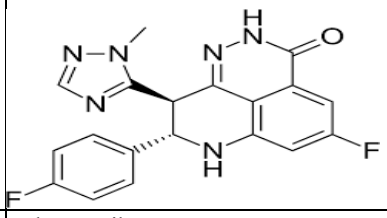
1.Pembrolizumab

Structure	
Generic name	pembrolizumab
Brand name	Keytruda
Chemical Formula	C6534H10004N1716O2036S46
Molecular weight	146648.64 g·mol−1
Mechanism of action	Pembrolizumab targets and blocks a protein called PD-1 on the surface of certain immune cells called T cells Open a glossary item. Blocking PD-1 triggers the T cells to find and kill cancer cells.
Route of administration	Intravenous [22], [23]

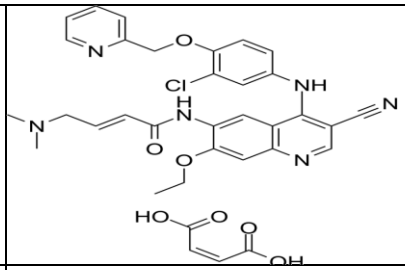
2.Alpelisib

Structure	 Chemical structure of alpelisib
Generic name	Alpelisib
Brand names	Piqray, Vijoice
Chemical Formula	C19H22F3N5O2Se
Molecular weight	441.47 g/mol
Mechanism of action	Phosphatidylinositol 4,5-bisphosphate 3-kinase catalytic subunit alpha isoform Inhibitor Phosphatidylinositol 3-kinaseInhibitor
Route of administration	Oral [24], [25]

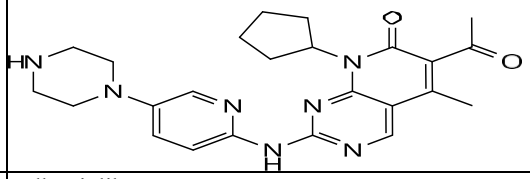
3. Talazoparib

Structure	
Generic name	Talazoparib
Brand names	Talzenna, Talzenna
Molecular Weight	380.4 g/
Chemical Formula	C19H14F2N6On
Mechanism of Action	Poly [ADP-ribose] polymerase 1Inhibitor Poly [ADP-ribose] polymerase 2Inhibitor
Route of administration	Oral [26]

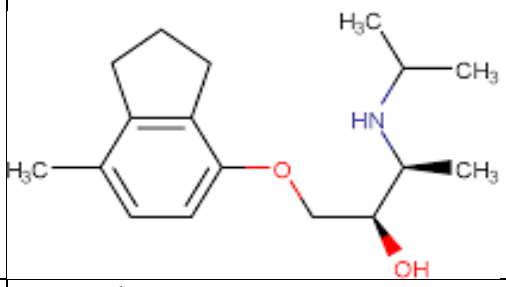
4.Neratinib maleate

Structure	
Generic name	Neratinib
Brand name	Nerlynx
Chemical formula	C34H33ClN6O7
Molecular weight	673.11 g/mla
Mechanism of action	Neratinib irreversibly inhibits EGFR, HER2, and HER4, blocking receptor autophosphorylation and reducing oncogenic signalling via the MAPK and Akt pathways
Route of administration	Oral [27]

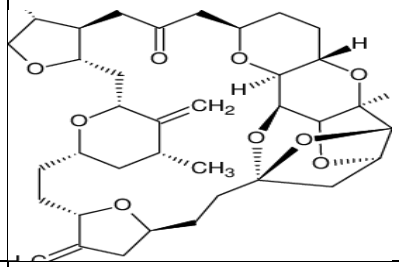
5.Palbociclib

Structure	
Generic name	Palbociclib
Brand name	Ibrance
Chemical formula	C24H29N7O2
Molecular weight	447.533 g/Bol
Mechanism of action	It is a selective inhibitor of the cyclin- dependent kinases CDK4 and CDK6.
Route of administration	Oral [28],[29]

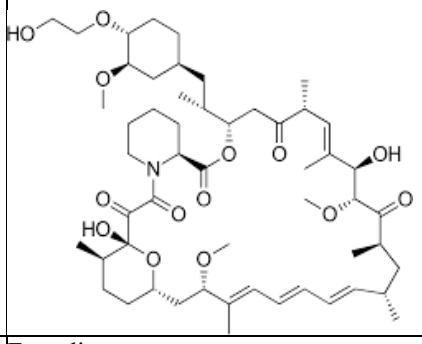
6.Pertuzumab

Structure	
Generic name	pertuzumab
Brand name	Perjeta, Phesgo
Chemical formula	(C_{6476}H_{9974}N_{1710}O_{2016}S_{44}\)
Molecular weight	148,000 Da
Mechanism of action	Receptor tyrosine-protein kinase erbB-2Inhibito
Route of administration	Intravenous [30]

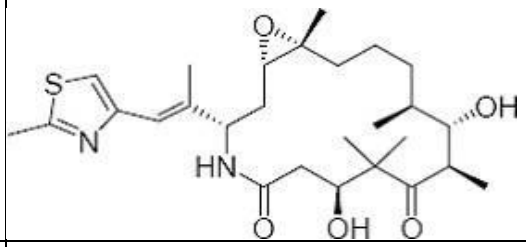
7.Eribulin mesylate

Structure	
Generic name	eribulin mesylate
Brand name	Halaven Injection
Chemical formula	C40H59NO11
Molecular weight	729.908 g·mol−1
Mechanism of action	Eribulin's antimitotic MOA is achieved by binding to high-affinity sites on β-tubulin at the exposed (plus) ends of growing microtubules, which inhibits microtubule polymerization (or growth).
Route of administration	Intravenous [31]

8.Everolimus

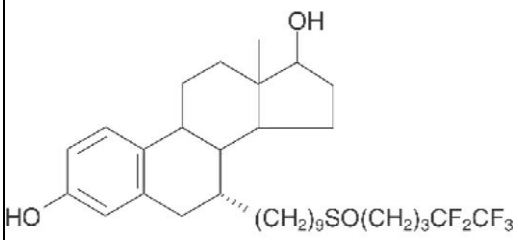
Structure	
Generic name	Everolimus
Brand name	Afinitor, Zortress, Afinitor-Disperz
Chemical formula	C53H83NO14
Molecular weight	958.22 g/mol
Mechanism of action	Everolimus selectively inhibits mTORC1 with minimal effect on mTORC2. This inhibition can activate AKT through disruption of the mTORC1 negative feedback loop, influencing cell growth, proliferation, and survival
Route of administration	Intravenous [32]

9.Ixabepilone

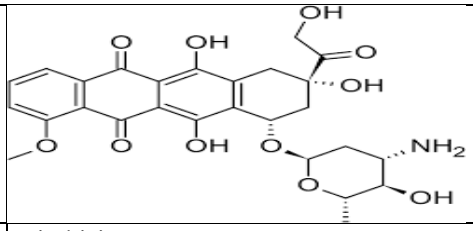
Structure	
Generic name	Ixabepilone
Brand name	Ixempra

Chemical formula	C27H42N2O5S
Molecular weight	506.70 g·mol−
Mechanism of action	Ixabepilone is an anti-cancer medication. It works by damaging the genetic material (DNA) of the cancer cells and stops their growth and multiplication.
Route of administration	Oral [33]

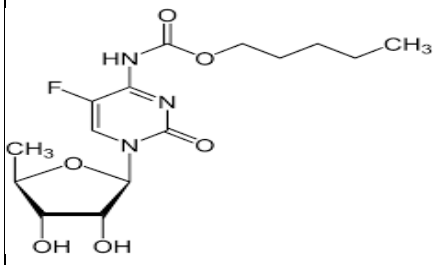
10.Fulvestrant

Structure	
Generic name	fulvestrant
Brand name	Faslodex
Chemical formula	C32H47F5O3S
Molecular weight	06.78 g·mol−1
Mechanism of action	Fulvestrant blocks the action of estrogen on breast cancer cells. This can lower or stop the growth of some breast cancer cells that need estrogen to grow.
Route of administration	Intramuscular [34]

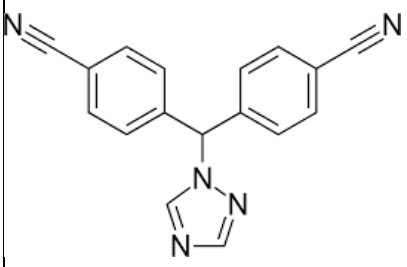
11.Epirubicin

Structure	
Generic name	Epirubicin
Brand name	
Chemical formula	C27H29NO11
Molecular weight	543.525 g·mol−1
Mechanism of action	Epirubicin is an anti-cancer drug that inhibits the enzyme topoisomerase II and generates free radicals, causing DNA damage that slows and kills breast cancer cells.
Route of administration	Intravenous [35]

12.Capecitabine

Structure	
Generic name	Capecitabine
Brand name	Xeloda
Chemical formula	C15H22FN3O6
Molecular weight	359.354 g·mol−1
Mechanism of action	Capecitabine is converted into 5- fluorouracil. These chemical hampers the synthesis of DNA and RNA in the cancer cells, thereby interferes with their growth. This slows down the growth of cancer cells and kill them.
Route of administration	Oral

13.Letrozole

Structure	
Generic name	Letrozole
Brand name	Femara
Chemical formula	C17H11N5
Molecular weight	285.310 g·mol−1
Mechanism of action	Letrozole is a non-steroidal, type II aromatase inhibitor that competitively blocks the CYP19A1 enzyme, preventing the conversion of androgens to estrogen
Route of administration	Oral

Conclusions

In this review, we aimed to summarize and update the current knowledge about breast cancer with an emphasis on its current epidemiology, risk factors, classification, prognostic biomarkers, and available treatment strategies. Since both the morbidity and mortality rates of breast cancer have significantly increased over the past decades, it is an urgent need to provide the most effective prevention taking into account that modifiable risk factors might be crucial in providing the reduction of breast cancer incidents. So far, mammography and sonography is the most common screening test enabling quite an early detection of breast cancer. The continuous search for prognostic biomarkers and targets for the potential biological therapies has significantly contributed to the improvement of management and clinical outcomes of breast cancer patients.

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