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Review Article

Breast Cancer: Symptoms, Causes, and Treatment by Metal Complexes: A Review

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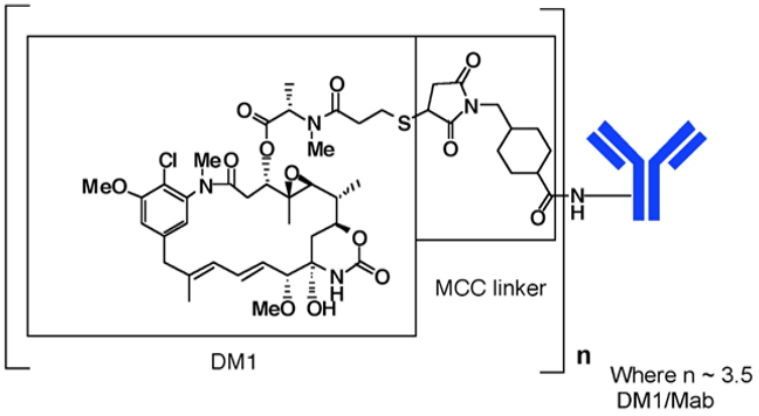
ABSTRACT

Breast Cancer (BC) is a special case in which the breast's cells grow out of control. Different types of breast cancer are developed according to which cells in the breast turn into cancer and different parts of the breast can give rise to breast cancer. Many types of treatment are used to treat breast cancer and metal complexes are one of the most crucial treatments for breast cancer. Numerous metal complexes have been shown effective in the treatment of breast cancer as well as many complexes utilized to treat specific forms of the disease. Metal complexes will help medicinal chemists organize, plan, and put innovative methods toward the development of novel medications into practice. This review highlights the causes and treatments of breast cancer and gives many examples of recent research that uses metal complexes as a medication.

GRAPHICAL ABSTRACT



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1- Introduction

Breast tissue cells begin to alter and divide out of control when breast cancer develops, usually manifesting as a lump or tumor. The lobules or the tubes connecting the lobules to the nipple are where the majority of breast cancers start. Men can develop breast cancer,[1, 2] but females account for the majority of cases. Invasive breast cancer will affect 1 in 8 females (13%) in their lives, and 1 in 39 females (3%) will pass away from the disease [3]. Lifetime hazard, which takes into account deaths of other reasons that can occur before a breast cancer diagnosis, is the average hazard for all females.

The decline is happening on average seen in females 80 years old and elder may be due to decreasing screening rates, malignancies being discovered via mammography prior to the age range of 80, or insufficient detection. Half of the females who acquired breast cancer was 62 years of age or younger when they received their diagnosis between 2012 and 2016. The average age at the time of breast cancer diagnosis was 62.20. Black females received diagnoses on average a little earlier than white females [4].

It is critical to understand that the plurality of breast tumefactions are initial while benign. Breast tumefactions that are not virulent be seedy increases that do not diffuse to the external from the breast. Some benign breast tumefactions, though not life-menacing, are able to promote a female's hazard from improving BC. Either breast lump or alter ought to be checked by a medical professional to determine whether it is noble or virulent, which could affect your hazard of developing cancer in the future. [5]

Following this, numerous metal complexes containing various transition metals, including titanium, [6, 7] zirconium,[8]vanadium, and others,[9-18] have been created. Transition metal complexes are widely used in a variety of domains, including catalysis,[19] optics,[20,21] electroluminescent devices,[22,23] agricultural and biocidal utilize,[24] while primarily in biological fields such as anti-cancer,[25] antibacterial,[26] and antifungal [27] applications.

Despite the fact that organic compounds have medicinal properties, one major advantage of those metal-simulated medicaments more organic-produced medicines is the ability to change the coordination number and geometry, as well as oxidation and reduction states. In addition, by creating coordination compounds with them, metals can compete with the pharmacological characteristics of drugs made from organic materials [28].

Therefore, various synthetic drugs contain the transition metal nucleus as a key structural element. The wide synthetic application and biological activity of these transition metal complexes can help medicinal chemists plan, organize, and put innovative methods toward the discovery of novel medications into practice.

1-1- Symptoms of Breast Cancer

When a tumor is tiny and most treatable, breast cancer often has no symptoms, which is why screening is crucial for early identification.

A lump is the most typical physical symptom. Even prior to the primary breast neoplasm grows for a volume that can be felt BC can diffuse into the lymph nodes down the arm while reason a mass. Less frequent indications and symptoms include breast discomfort or heaviness, chronic alterations in the nipple, like automatic layoff (particularly if bloody), scaliness, or retraction. A physician should examine any breast alteration that persists.

1-2- Causes of Breast Cancer

Breast cancer has numerous causes [29]. For instance, lifestyle-related hazard factors like diet and exercise ability elevate your hazard from BC. However, it is not yet clear how much several of

these hazard agents specifically trigger normal cells into improve to malady.

Though the correct mechanism is indefinite, hormones show into play a function in many cases from BC. Gene adjustments or mutations can reason physical breast cells to turn malignant. But, just 10% of breast cancers are known to be associated with inherited (passed down from parents) abnormal genes. Females together a people recorded from BC maybe own hereditary loafer genes that are undetectable via genetic testing reason many genes be so far obscure [30].

Approximately 90% of BC is caused by unidentified acquired gene alterations. Genes [31] regulate how our cells act. DNA, a chemical strand that is derived from both of our parents, makes up their structure. DNA has an impact on our likelihood of contracting certain diseases, such as some types of cancer, in addition to how we look. Oncogenes, any genes present in ordinary cells, help control cell growth, and region to form new cells or serve alive.

A proto-oncology can evolve into an Oncology via passing particular mutations. This mutant Oncology can reason illness in cells. Furthermore, tumefaction oppressor genes out in ordinary cells, which assist into organize the repetition from cell to divide, the capacity to correct DNA mistakes, and the timing from cell doom. Cells can develop into cancer if a tumor suppressor gene has been altered. Gene alterations that activate oncogenes or silence tumefaction suppressor genes can result in cancer.

Usually, for breast cancer to occur, multiple separate genes must change. You can inherit or get certain gene modifications (mutations) from your parents. This implies that the mutations are ready in every cell of your body at birth. A number of the malignancies that occur in some families are connected to specific inherited gene abnormalities that can significantly raise the chance of acquiring certain cancers.

The BRCA genes (BRCA1 and BRCA2), for instance, are tumor suppressor genes. When one of these genes is altered, it stops suppressing abnormal cell growth, which increases the hazard of cancer. One of these genes may alter from parent to child through genetic inheritance. You can inherit or get certain gene modifications (mutations) from your parents. This implies that the mutations are ready in every cell of your body at birth. A number of the malignant neoplasms that happen in several families are connected to specific inherited gene abnormalities that can significantly elevate the chance of getting confirmation cancers. The BRCA genes are tumefaction oppressor genes. When one of those genes is changed, it stops repressing abnormal cell growth, which increases the hazard of cancer. One of these genes may alter from parent to child through genetic inheritance. Mutations that frequently result in cancer are referred to as "high penetrance" mutations in tumefaction suppressor genes, such as the BRCA genes. Although breast cancer is frequently diagnosed in females with high penetrance mutations, the majority of cancer cases are not brought on via this type of mutation. Low penetrance mutations or gene variants are more frequently an agent in the evolution of cancer.

All of them may have only a marginal impact on the likelihood that a person would develop cancer, but because these mutations are widespread and many people have multiple copies of them simultaneously, the overall impact on the population may be substantial. The genes at play may have an impact on hormone levels, metabolism, or other elements that influence the hazard of breast cancer. The hazard of breast cancer that runs in families may also be greatly influenced by these genes. The majority of breast cancer-related gene amendments are gained. This indicates that the change in breast cells occurs throughout a person's life as opposed to being inherited or present at birth. Only breast

cancer cells experience acquired DNA mutations over time.

These acquired mutations in tumor suppressor and/or oncogene genes can be brought on by radiation or other carcinogens. However, some gene alterations might simply be chance occurrences that occasionally take place inside a cell without an external cause. Most acquired gene mutations that can cause breast cancer now have multiple known sources, and most breast cancers have multiple acquired gene mutations [32].

2- Treatment of Breast Cancer: It includes three types of treatments

2-1- Systematic therapies

Because they can connect cancer cells almost throughout the body, therapies utilized to treat illness are deemed as regular treatments. Several can be managed verbally, muscularly, or immediately into the bloodstream. Various medicine therapies may be employed according to the kind of illness, inclusive:

2-2- *Chemotherapy for Breast Cancer*

2-2-1- Doxorubicin

It is an anthracycline, a type of chemotherapy medication (Figure 1). Via impediment the enzyme topoisomerase 2, it inhibits or stops the proliferation of cancer cells. This enzyme is wanted for cancer cell proliferation while development. The topoisomerase II enzyme is inhibited by intercalating between DNA base pairs, which causes the DNA double helix to uncoil and causes single and double-strand breaks, which prevent the production of DNA and RNA.

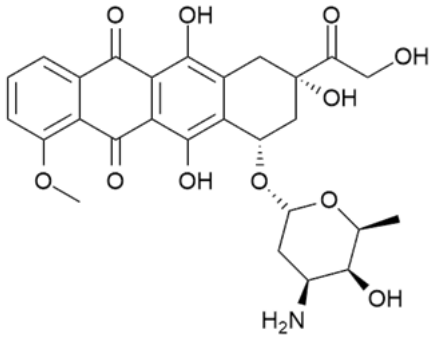

Fig. 1 Structure of Doxorubicin

2-2-2- Ellence drug

It is utilized in conjunction with other medications to treat breast cancer that has spread to the lymph nodes under the arm after being surgically removed. Ellence is a member of the anthracycline class of medications known as antineoplastics. Epirubicin is a member of the anthracycline class of chemotherapy medications (Figure 2). In cancer cells, they cause DNA (genetic code) damage. This prevents the cancer cells from proliferating or multiplying. Epirubicin exhibits cytotoxic and antimitotic effects.

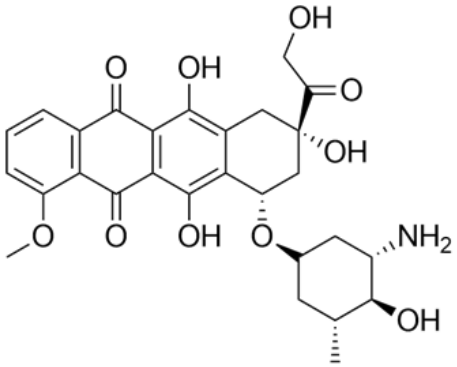

Fig. 2 Structure of Ellence drug

2-2-3- Paclitaxel

(with polyoxyethylated castor oil) is utilized to treat non-small cell lung cancer (NSCLC), ovarian cancer (cancer that starts in the female reproductive organs where eggs are generated), and breast cancer (Figure 3). Microtubules are

the target of paclitaxel. While PTX induces apoptosis at the G0 and G1/Sphasese via activation of Raf-1 kinase or p53/p21 depending on the dose concentration, it causes mitotic arrest at the G2/M phase at high concentrations.

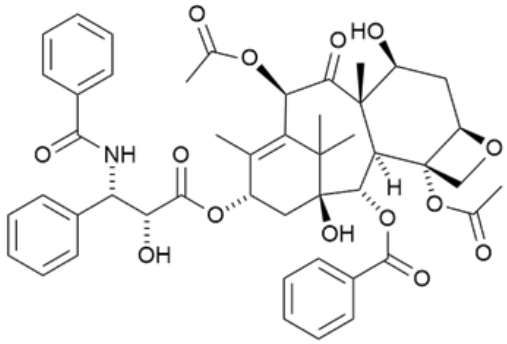

Fig. 3 Structure of Paclitaxel drug

2-2-4- Docetaxel injection

It is utilized either alone or in conjunction with additional drugs to treat specific types of head and neck, stomach, lung, and prostate cancers (Figure 4). The drug docetaxel injection belongs to the taxane family of drugs. It functions by preventing the development and spread of cancer cells. Docetaxel disrupts microtubule growth's typical operation. Docetaxel stops the function of microtubules by having the reverse effect of what medications like colchicine do, which is to depolymerize them *in vivo*. The cell can no longer utilize its cytoskeleton in a flexible manner as a result. Docetaxel specifically binds to tubulin's - subunit. The "building block" of microtubules is tubulin, and docetaxel binding secures these building blocks. It is impossible to dismantle the resulting microtubule/docetaxel complex. Because microtubules should shorten and lengthen (a process known as dynamic instability) in order to serve as the cell's transportation system, this has a negative effect on how cells job. For instance, through equal division, chromosomes depend on this characteristic of microscopic tubes. According to an additional study, docetaxel causes cancer cells to submit programmed cell death by attaching to the apoptosis-inhibiting protein Bcl-2 (B-cell leukemia 2) and halting its activity.

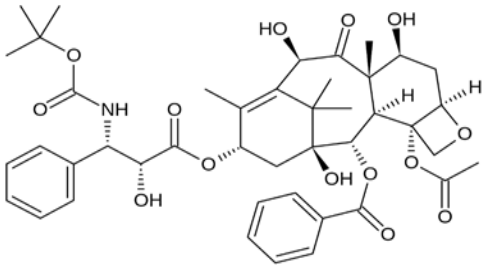

Fig. 4 Structure of Docetaxel

2-2-5- Cyclophosphamide

It is approved to treat malignant lymphomas, multiple myeloma, leukemias, advanced mycosis fungoides (Figure 5), neuroblastoma (disseminated illness), ovarian adenocarcinoma, retinoblastoma, and breast cancer.

Furthermore, it is suggested for the treatment of pediatric patients with biopsy-verified minimal change nephrotic syndrome. Alkylating chemicals function in three different ways via attaching alkyl groups to DNA bases. As a result, DNA repair enzymes effort to substitute the alkylated bases by fragmenting the DNA, which prevents DNA synthesis and RNA transcription of the afflicted DNA. Cross-links (bonds between DNA atoms) prevent DNA from being detached for synthesis or transcription, and the induction of mispaired nucleotides that reason mutations and these are three examples of DNA damage.

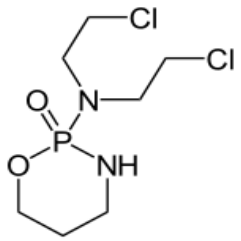

Fig.5 Structure of Cyclophosphamide

2-2-6- Hormone Treatment for Breast Cancer

Several species of diseases are influenced by hormones, such as estrogen and progesterone. These medicaments prevent estrogen from the connexion of the cancer cells while telling them to develop while part. However, they have anti-estrogen impacts on breast cells; they do such as estrogen in the last tissues, such as womb and bones.

A) These drugs are pills, taken by mouth

2-2-7- Fulvestrant (Faslodex)

Its ability is applied singly to therapy developed illness that has not been treated together for last hormone handling. It is an estrogen receptor antagonist that links to the estrogen receptor competitively with an affinity comparable to that of estradiol and goes down the ER protein in human breast cancer cells, as depicted in Figure 6.

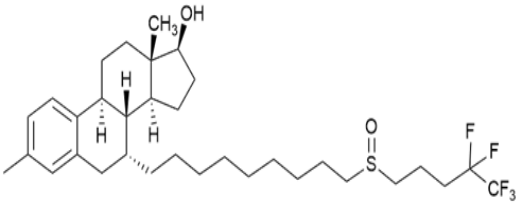

Fig. 6 Structure of Fulvestrant

2-2-8- Elacestrant

This medication can be utilized to therapy developed, ER-positive, and HER2-negative illnesses when the cancer cells have an ESR1 gene turnover (Figure 7), while the disease has matured after at least one other kind of hormone treatment at the verbal direction from greater doses of elacestrant, such factor acts as a SERD, and binds to the estrogen receptor (ER) while prompting an agreement turn that finding in the devolution of the receptor. This may inhibit the development and survival of ER-expressing cancer cells.

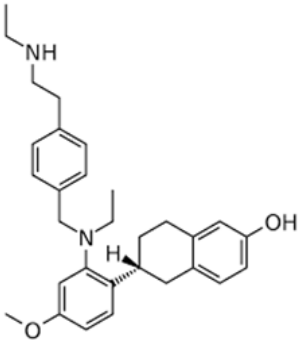

Fig. 7 Structure of Elacestrant

B) Targeted Drug Therapy for Breast Cancer

2-2-9- Ado-trastuzumab emtansine (Kadcyla)

It is a kind of targeted cancer drug (Figure 8). It is also named by its brand name Kadcyla and TDM1 breast cancer.

Kadcyla finds and hangs to receptors on the outside of the HER^{2+} cells. Here it acts by checking cell develop and increasing the effect of the immune system to die the cancer cells. In addition, it goes inside the cell and breaks apart to release the chemotherapy.

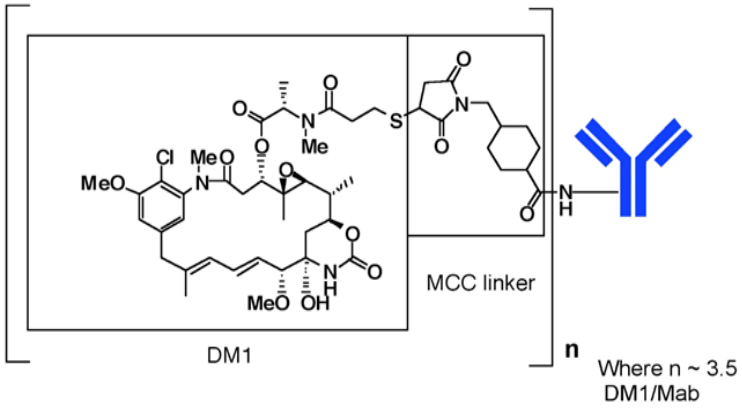

Fig. 8 Structure of Kadcyla

2-2-10- Surgery

Surgical therapy for breast cancer has evolved safely ago decades, for progress aimed at belittling the long-term cosmetic while functional multiples for domestic curing. Based on contracts for the look, the criterion approaches are also a total mastectomy or eradication of excessive radiation, the presumption that clear margins can be accomplished.

Both of these methods have been shown constantly to be opposite in relapse-free while total survival. [35] Contraindications to conservative surgery involve:

(1) The public from common doubtful minute on breast Photography,

(2) Positive patients footnote next lumpectomy,

(3) Illness that cannot be ad- dressed via eradication from a monocular breast tissue area for the satisfactory cosmetic outcome, excludes for into a high degree chosen patients,

(4) Confirmed collagen-vascular illnesses, like scleroderma; while

(5) Previously radiotherapy to the included breast. [36, 37]

2-3- Radiation Therapy

Radiation treatment can be delivered to all or a part of the chest and breast. Post-lumpectomy all breast radiation is a criterion motif of breast keeping treatment.[38,39]Ameta-analysis of [40] 801 patients show up that management from radiation following lumpectomy was linked together with decreases in breast cancer repetition via almost semi (from 35.0% to 19.3%) while in BC dooms via one-sixth (of 25.1% to 21.3+%) at 10 while 15 years, respectively.

Like together with assistant regularity treatments, the proportional usefulness from radiation was almost fixed despite from overall breast cancer hazard. Therefore, the full usefulness was greater in patients together with top-hazard diseases, while conversely, the death rate benefit trust interval included zero in patients together with the minimum hazard node-negative tumefactions. [41]

3- Metal Complex

In 2021, Farah M. Ibrahim and Saifaldeen M. Abdalhadi reported that the novel ligand directly reacted with a variety of metals, including Cu (II), Pt (II), and Ni (II), utilizing a molar proportion from 1:1 (M:L) (Figure 9).

In positions from phenolate nitrogen, azomethine, while keto oxygen atoms, the new compound was coordinated as a tridentate ligand, according to the spectroscopic data. Utilizing the paper disc spread technique while the sequent dilutions in liquid broth technique,

the anticancer while the antibacterial activity of each of these complexes was investigated and compared with the ligand. All of the complexes and ligands were demonstrated to have potent antibacterial action against Gram-positive *Staphylococcus aureus* and *Candida albicans*, and the complexes also demonstrated potent anticancer activity against breast cancer cells. [41, 42]

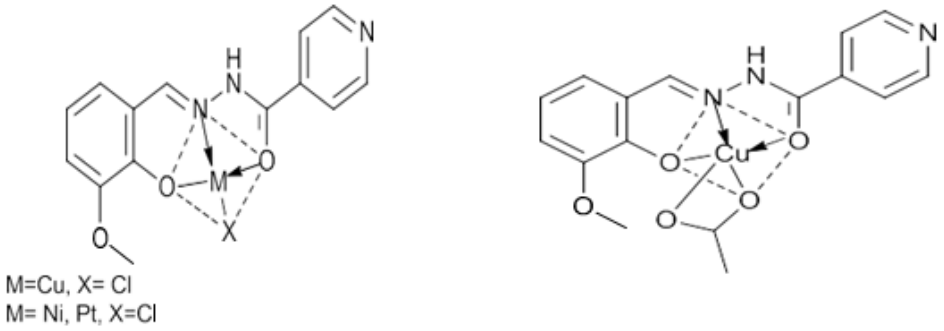

Fig. 9 The structure of the hydrazine metal complexes

Due to their significant demonstrated anticancer action in 2019, Sondavid K. Nandanwar and Hak Jun Kim produced several complexes linked to ferrocenium salts (Figure 10). [53]

According to electron spin resonance measurements,[43] ferrocenium salts, which produce hydroxyl free radicals through the Fenton route, mechanically cleave DNA.[44]

Both hormone-dependent and hormone-independent breast cancer cells were selectively inhibited by the ferrocene derivatives tamoxifen, and hydroxy tamoxifen. Tamoxifen and hydroxytamoxifen's mode of action resulted from the competitive inhibition of DNA transcription caused by their potent binding to both estrogen receptor-positive (ER^+) and estrogen receptor-negative (ER^-) cancer cells. The formation of reactive oxygen species and a clear cell cycle halt at the S phase are the two effects of the ferrocene derivative hydroxy tamoxifen that cause senes-the science of ER^- and ER^+ cells.

This finding was significant since the majority of breast cancers, roughly 66.67%, belong to the ER^+ type, which can be treated with tamoxifen, and the remainder are of the ER type, which cannot be treated with tamoxifen. [46, 47]

In addition, they created three copper complexes, and in comparison, to the typical breast cell line MCF-10 A cell, their respective conjugated ferrocene derivatives were tested against the breast cancer cell line MCF-7 and the cervical cancer cell line HeLa.

Some of these complexes were found to be ineffective against the rest of the cancer cell lines but had poor anticancer activity against HeLa cells with IC50 values of 15.5 M, 7.3 M, and 7.9 M.

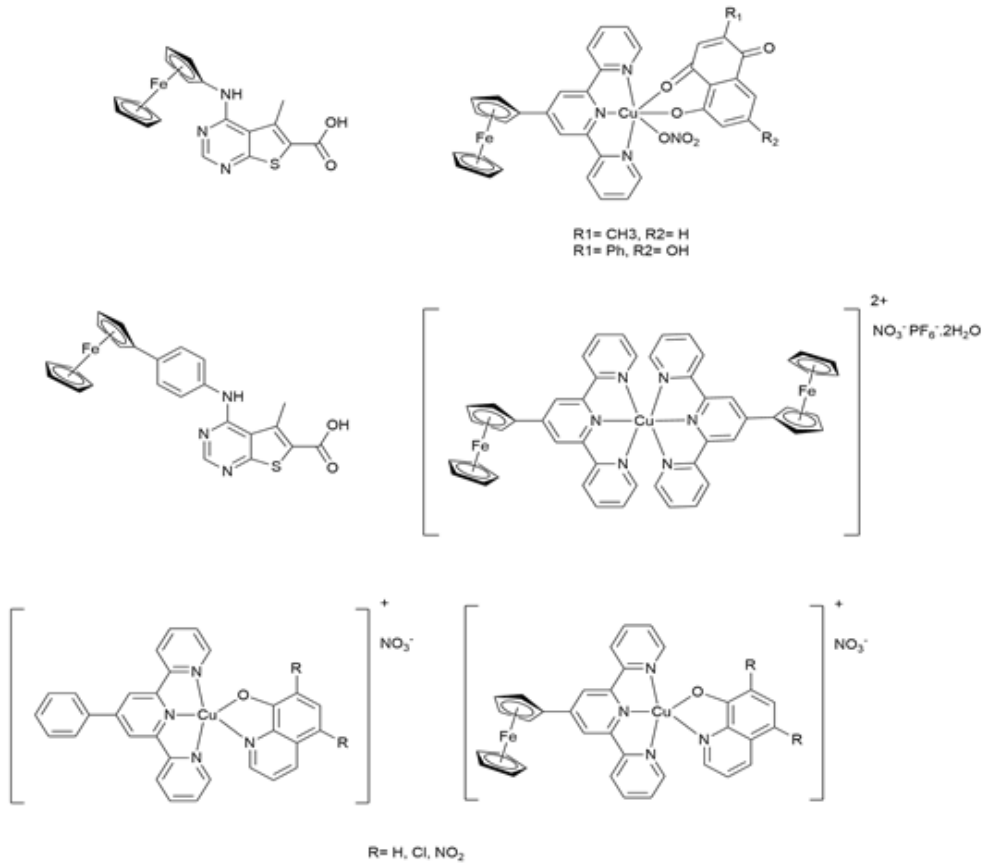

Fig. 10 Structure of anticancer iron and copper complexes

In the second- or the third-line cancer therapies, titanocene was a potent cytotoxic component (Figure 11). Prostate cancer and xenografted human epidermoid carcinoma responded well to Titanocene Y's anticancer activities in *in vivo* experiments, whereas breast and renal cancer cells responded very well. The Titanocene complexes have recently $[C5H_4CH(CH_3)C_6H_5]_2TiCl_2(T_1)$,$([C_5H_4CH(CH_3)(pC_6H_4\text{-}OCH_3)]_2TiCl^2(T_2)$, $[C_5H_4CH(CH_3)(2,4\text{-}C_6H_3(OCH_3)_2]_2TiCl_2$ (T_3), and $[C_5H_4CH_2$ $(C_{10}H_6\text{-}OCH_3)]TiCl_3$ (T_4) evaluated for anticancer activity against the estrogen receptor-positive (ER^+) human breast cancer cell line MCF-7 after being produced by the same researcher.

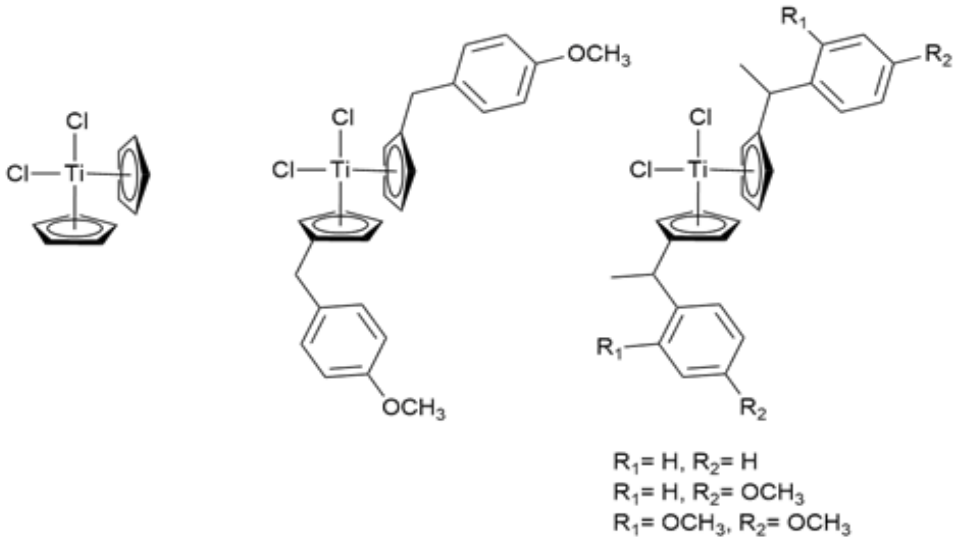

Fig. 11 Structure of anticancer titanium complexes

Ahmed Gaber *et al.* created brand-new papaverine-vanadium (III), ruthenium (III), and gold (III) metal complexes in 2020 with the goal of improving the drug's biological activity (Figure 12). Different spectroscopic techniques (UV-Vis, IR, NMR, TGA, SEM, and XRD) were utilized to characterize the structures of the synthesized complexes. Among the synthetic compounds, the Au (III) complex has promising antibacterial and anticancer properties. Interestingly, the anticancer activity of the papaverine-Au (III) complex was greater than that of the papaverine alone against the cancer cell lines under investigation, indicating that the complex enhanced the anticancer activity of the parent medication.

In addition, the Au complex outperformed cisplatin against breast cancer MCF-7 cells. The biocompatibility tests revealed that the Au complex, with an IC50 of 111 g/mL, is less hazardous than the papaverine medication alone. These findings suggest that the papaverine-Au (III) complex is a promising anticancer compound drug, making it a good candidate for additional in vivo research. The papaverine-Au (III) complex, one of the metal complexes we created, has potential anticancer action against both human HepG-2 cells and breast cancer MCF-7 cells, according to a biological evaluation of the metal complexes we created. It may be concluded that Au complexation enhanced the anticancer activity of the parent ligand since the anticancer activity of the Au complex against several cancer cell lines was higher than that of the papaverine ligand alone. Interestingly, the Au-complex outperformed cisplatin in terms of anticancer activity against MCF-7 (IC502.87 g/mL).

Overall, these findings show that the Au(III)-papaverine complex is an anticancer molecule with promise, making it a good option for more *in vivo* research (Figure 6).

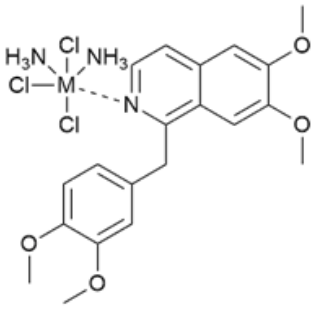

Fig. 12 Proposed structure of M (III)-papaverine complex, where M=V (III), Ru (III), or Au (III)

Richard Ming Chuan Yu *et al.* (2020) investigated the activity of gold I and gold III-based complexes

in breast cancer. Complexes based on gold I and gold III have demonstrated adequate anticancer activity. The *in vitro* activities of two new fluorine gold (I) derivatives compounds, against breast cancer cells, breast cancer cells type BCSC-P, and breast cancer stem BCSC cells were assessed. Analysis was done to see how the chosen gold I compound, $Ph_3PAu[SC(OMe)=NC_6H_4F]^{-3}$. A gold (I) molecule called $Ph_3PAu[SC(OMe)=NC_6H_4F]^{-3}$, which also contains fluorine, may be utilized to treat breast cancer. Also known as [(Z)-N-(3-Fluorophenyl)-O-methylthiocarba-mato-κS], triphenylphosphanegold (I) O- methyl-N-3-fluorophenyl thiocarbamate (compound 3F1)The chemical combination 3F3 is composed of (triphenylphosphine-κP) gold(I) and bis(dienylphosphinoferrocene) di gold(I) O-methyl-N-(3-fluorophenyl) thiocarbamate. It is also known as (2-1,1′-bis(diphenylphosphine) ferrocene-K2 P, P′)-bis[(Z)][48] created digold (I) chloroform solvate and their ligand LH (compound 3FL), which were utilized to make compounds 3F1 and 3F3, and they collected them for this study after nuclear magnetic resonance confirmed their purity.

Therefore, in this investigation, the new gold(I) compounds anticancer characteristics with fluorine incorporated, such as bisdiphenyl phosphine ferrocenene) di gold (I) O-methyl-N-(3-fluorophenyl) thiocarbamate and tri-phenyl phosphane gold (I) O-methyl-N-(3-fluorophenyl) thiocarbamate, were investigated.[47,48] Human breast cancer cell lines like MDA-MB-231 (triple-negative breast cancer cells) and MCF-7 (estrogen receptor-positive breast cancer cells), primary breast cancer cells (in-house cultured), BCSC, and parental BCSC (BCSC-P) were utilized to test the compounds for development repression, apoptosis induction, while cell cycle detention. According to the findings, the novel gold (I) compound $Ph_3PAu[SC(OMe)=NC_6H_4F$-3] demonstrated promising anticancer characteristics against various breast cancer types in vitro, including growth suppression, apo-apoptosisuction, and cell cycle arrest.

4- Conclusion

BC is a kind of cancer that occur in the breast. It can begin in one or both breasts. Breast cancer begins when cells grow out of control. Many factors are linked with an increased danger of it, for example, a family history of the disease will increase the danger from BC. Yet, the majority of families diagnosed with breast cancer have no family history of the illness. Proven gene mutations enhance the danger of BC and can be voyaged parts to offspring. Females who give childbirth to their top child after the age of 30 are more likely raised in danger of illness and females who have never given birth have a higher danger of BC than females who have experienced one or more pregnancies. Females use Postmenopausal hormone treatment pharmaceuticals that collect estrogen and progesterone to treat symptoms. As well as, when a received radiation therapy a person matures, the hazard of disease is raised. Five ways to support keeping your breasts healthy include obtaining and staying at a sanitary weight. Physically lively and avert time spent sitting. Follow a healthy eating pattern. It is best not to drink alcohol and consider utilizing hormone replacement therapy carefully. Many drugs are utilized to treat certain cases of breast cancer, as well as many metal complexes that have been proven in breast cancer therapy.

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