

ANTIBODY-DRUG CONJUGATES IN BREAST CANCER: BENCH TO BEDSIDE INSIGHTS ON MECHANISM, EFFICACY, AND SAFETY

¹Pooja Kumari, ²Kunwar Aditi, ³*Dr. Najiya Fatima

¹Pharm D Scholar, Department of Pharmacy Practice, RBVRR Women's College of Pharmacy, Osmania University, Barkatpura, Hyderabad, Telangana, India.

²Pharm D Scholar, Department of Pharmacy Practice, RBVRR Women's College of Pharmacy, Osmania University, Barkatpura, Hyderabad, Telangana, India.

³*Assistant Professor, Department of Pharmacy Practice, RBVRR Women's College of Pharmacy, Osmania University, Barkatpura, Hyderabad, Telangana, India.

Article Info: Received: 17 August 2026, Revised: 07 September 2026, Accepted: 27 September 2026

***Corresponding Author: Dr. Najiya Fatima**

Assistant Professor, Department of Pharmacy Practice, RBVRR Women's College of Pharmacy, Osmania University, Barkatpura, Hyderabad, Telangana, India.

DOI: <https://doi.org/10.5281/zenodo.23066781>



Citation:

¹Pooja Kumari, ²Kunwar Aditi, ³*Dr. Najiya Fatima. (2026). Antibody-Drug Conjugates In Breast Cancer: Bench To Bedside Insights On Mechanism, Efficacy, And Safety. International Journal of Clinical and Pharmaceutical Innovations, 1(7), 36-48.

ABSTRACT

Antibody Drug Conjugates (ADCs) are a novel type of chemotherapy that treats breast cancer. It has three components where monoclonal antibodies are combined with cytotoxic payloads via a linker mechanism. In comparison with other conventional approaches, the latter approach is highly dependent upon high specificity and selectivity, which makes it suitable for addressing the needs of breast cancer therapy. Antibody drug conjugates (ADCs) focusing on HER2 and TROP-2 antigens have been developed to overcome the needs regarding breast cancer therapy. A comprehensive literature review was conducted by analyzing the PubMed/Medline, Scopus, and ClinicalTrials.gov databases to collect information on clinical trials, regulation, and translational medicine related to ADCs. Other sources have been explored to become acquainted with FDA-approved ADCs, new therapies, toxicities, and management, along with mechanisms of chemo resistance in breast cancer therapy. This article discusses information about FDA-approved ADCs, emerging therapies, toxicities, and mechanisms of therapy resistance in breast cancer. Precision oncology in breast cancer treatment has been proven due to the absence of side effects, up to now. To enhance their applicability and efficacy, further research is necessary in the case of their molecular structure and combinations.

KEYWORDS: Antibody-drug conjugates, Breast cancer, HER2, TROP-2, Drug resistance, Precision oncology.

INTRODUCTION

Breast cancer is a malignancy of the breast tissue that causes unregulated development of the cells that comprise the breast tissue and, if left untreated, spreads to other regions of the body. The global growth of breast cancer in population is putting stress on healthcare systems. Among all the cancer cases in women, one in four women is diagnosed with breast

cancer. Despite improved treatments available, around 15% of women diagnosed with breast cancer die due to the disease.^[1, 2]

Chemotherapy used to be the main choice before ADCs arrived. It worked well against tumors yet hit healthy cells too, bringing many undesired reactions. Monoclonal antibodies came next, aiming better at

cancer in some people. Yet their success often faded when tumors adapted or blocked access. Poor delivery meant medicines failed where needed most. Over time, damage built up, helping cancers return stronger than before.^[3]

To overcome the challenges with traditional therapies, the researchers developed new therapies, one among them is development of ADC which are stepping into the spotlight and are now recognized as a powerful approach in treating breast cancer. These agents are designed by combining a specific antibody with a potent cytotoxic drug with the help of a chemical linker, and each of these components has individual roles to perform in combating breast cancer. The antibody directs the ADC by identifying specific targets present on cancer cells, the linker stabilizes the bond between the antibody and payload, and the active payload is the main component that contains the drug and shows therapeutic effect.^[2]

Around the year 1900-1908, Paul Ehrlich, a scientist, came up with an idea once thought impossible—chemotherapy that targets only bad cells, leaving good ones undisturbed, unlike conventional therapy. He named it "magic bullet," sharp and exact in how it worked. Not long before, researchers found certain markers on cell surfaces that pulled antibodies toward them, almost like metal to a magnet. Due to that finding, targeted therapies in cancer treatment became possible. During the 1980s, researchers tested the first version of what they now call ADCs. The approval followed years later, when gemtuzumab ozogamicin made its way into medical use, which works by specially designed proteins that identify and bind to specific markers that are present on the surface of tumor cells, and upon attachment, they release the cytotoxic payload

(drug) directly into the cell, leading to the death of the tumor.^[4]

So far, studies have shown that this approach increases treatment effectiveness with only the usual adverse effects associated with standard chemotherapy. That means ADCs perform better in the treatment of both high and low HER2 types of breast cancer, opening additional paths for therapy. There are a few challenges associated with the use of ADCs: finding the right patients, managing and overcoming adverse effects, and resistance. Researchers are currently working on the development of ADC designs, adjusting linkers to improve stability, to reduce toxicity, and searching for previously overlooked targets.^[3]

This review aims to explore the role of ADCs in breast cancer, their mechanism, toxicities, limitations, and resistance mechanisms.

ADCs UNDER TRIAL

About 190 ADCs are undergoing clinical testing, and the development of ADCs is expected to increase their therapeutic reach to about 15 ADCs in the market.⁵ refer to Fig. 1 for information about ADCs currently in clinical trial.

Finding more tumor-specific drugs, enhancing linker stability, and improving conjugation techniques to increase effectiveness and reduce or prevent adverse effects are current advancements. Dual payload techniques are next generation advancements that can boost tumor cell death and fight medication resistance and offer a framework that enables patient choices and treatment modification. These advances show increasing focus on ADCs' expansion for the development of targeted cancer therapy.^[3]

ADCs CURRENTLY IN CLINICAL TRIALS

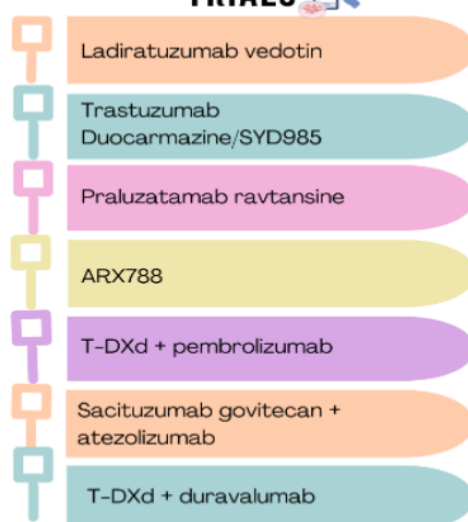


Fig. 1: The new drugs that are under development for breast cancer therapy.^[6,7]

COMPONENTS OF ADCs

ADCs are a complex structure having a monoclonal antibody, a linker, and a cytotoxic payload as shown

in Fig. 2. Their success depends upon the precise integration of these components together rather than showing their individual roles.^[2]

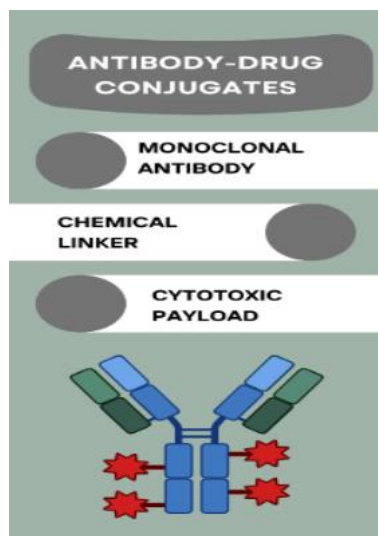


Fig. 2: Components of ADCs.^[2]

ANTIBODY

Monoclonal antibodies enable selective binding of ADCs to specific antigens present on cancer cells, making them one of the important components of ADCs.^[8, 9]

For effective ADCs, antibodies must have strong target affinity towards the binding site, good internalization into the target cell, show minimal immunogenicity, and have a long circulating half-life for better therapeutic outcomes. Early ADCs used murine antibodies, which had high immunogenicity that often resulted in adverse immune reactions. To overcome this problem, modern ADCs have shifted towards chimeric, humanized ADCs with improved safety profiles, homogeneity, tumor penetration, and reduced immunogenicity. Among all five types of immunoglobulin classes (IgA, IgD, IgE, IgG, and IgM), IgG is the most common type used in ADCs due to its stability and capacity to trigger immune effectors, whereas IgG3 is avoided due to its short half-life.^[8,9]

PAYLOAD

The payload is one of the important elements of ADC that contains the highly potent cytotoxic agents that cannot be used alone due to their tendency to cause toxicity. The commonly used anti-tumour agents are from the class of microtubule inhibitors, DNA-damaging agents, and topoisomerase inhibitors. Selection of the payload depends on potency, stability, and compatibility with linkers. This approach improves the treatment outcomes. Newer payload approaches include the use of radionucleotides, immune modulators, and the use of dual payload designs. Drug-to-antibody ratio (DAR) is another factor that needs to be considered, which affects the stability and efficacy of ADC. Most of the ADCs use a DAR of 2-4, while a few drugs, such as trastuzumab

deruxtecan and Sacituzumab govitecan, remain effective with a higher DAR.^[8,9]

THE LINKER

Linkers are the components that connect the antibody and the payload, which regulate ADC stability and drug release. Ideal linkers stay stable in the blood but release the payload inside tumour cells. They are of two types: cleavable linkers and non-cleavable linkers. Cleavable linkers (example-protease-sensitive, acid-sensitive bonds) show high toxicity, are less stable when compared to non-cleavable linkers, are resistant to hydrolysis (stable), respond to tumour-specific conditions, and produce a bystander effect, which shows extra benefits of therapy. Non-cleavable linkers (Example- thioether) release the payload only after antibody degradation, but show limited bystander activity compared to cleavable linkers.^[8,9]

MECHANISM BY WHICH ADCS WORK

1. Target recognition, binding, and internalisation of ADCs

The antibody has the capability to recognise its target site, then attaches to it selectively, providing a site-specific action, followed by uptake by the cell through a process called endocytosis.^[8,10]

2. Intracellular processes

Following internalization into the target cell by clathrin- or caveolin-mediated uptake, the ADCs form early endosomes, mature into late endosomes, and then bind lysosomes. which are then exposed to proteolytic enzymes, reductive redox reactions, and the cell's acidic intracellular environment. These intracellular processes prepare the ADCs before payload release.^[8,9,10]

3. Release of the payload within the target cell

In response to the above changes, the ADCs release the payload (drug) in two ways: ADCs with cleavable linkers release the cytotoxic payload directly, ADCs with non-cleavable linkers require antibody destruction to release the drug within the target cell.^[10]

4. Cytotoxic activity shown by the drug

Once the payload is released, the cytotoxic effect of the drug is observed either by damaging the DNA or microtubules, leading to tumour cell death.^[10]

5. Bystander effect

Some payloads diffuse into cells surrounding the tumour cells, get internalized, and the payload is released and

shows cytotoxic effect, overall producing a bystander effect that collectively enhances the efficacy of ADCs against tumours that have low antigen expression as shown in Fig. 3.^[8,9,10]

6. Immune-mediated effects

Apart from causing direct cytotoxicity, ADCs are also involved in mechanisms such as phagocytosis, which further contribute to anti-tumour activity, improving the therapeutic outcomes.^[8,10]

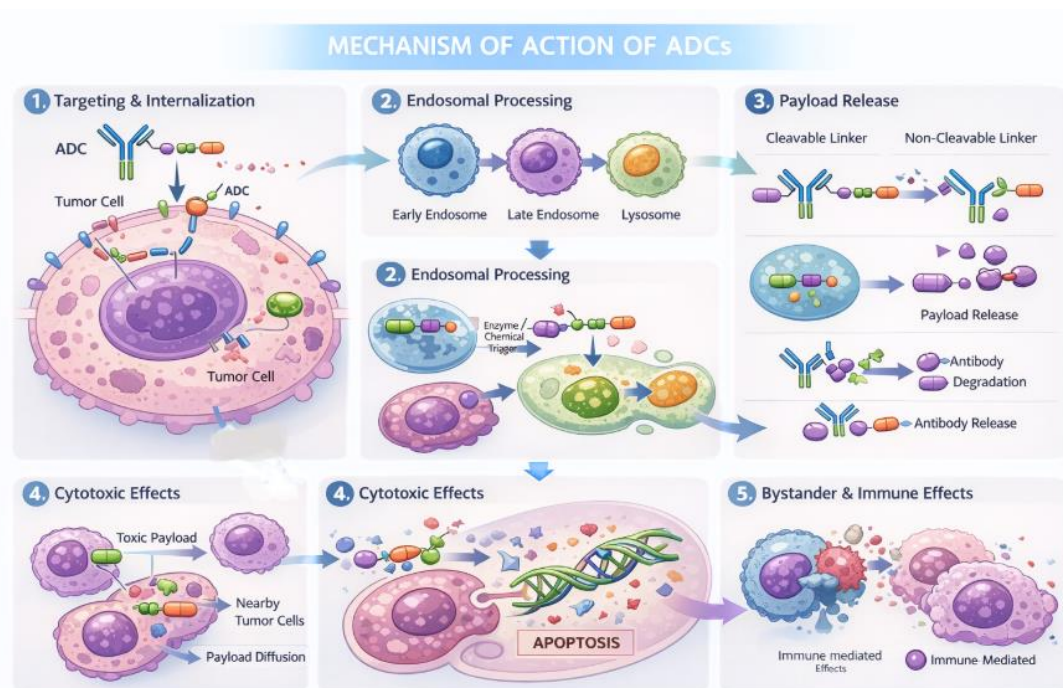


Fig. 3: Mechanism of Action of ADCs.^[8,9,10]

COMPARISON OF STANDARD CHEMOTHERAPY WITH ANTIBODY DRUG CONJUGATES

The oldest and most preferred method for treating breast cancer is standard chemotherapy, which lowers the risk of relapse by 30% in cases of the early stages of breast cancer. Anthracyclines alone or in combination with taxane-based regimens are found to be effective for treating high-risk HER2-positive and triple-negative breast cancer, even though their use is restricted.^[11]

However, unlike ADCs, chemotherapy and hormonal therapies have shown many undesirable and dangerous adverse effects over time, including the development of secondary cancers, but are still used widely due to the

lack of other therapies in treating breast cancer. Compared to conventional therapies, antibody-drug conjugates (ADCs) are emerging as newer agents to reduce the limitations of systemic chemotherapy, showing better efficacy and prolonging the patient's survival.^[12]

TARGET ANTIGENS FOR BREAST CANCER ADC DEVELOPMENT

Subsequent research identified specific cell surface markers for targeting the cancer cells; they are HER2, HER3, CD228, CD205, CD197, CD174, CD25, c-MET, Globo H, NECTIN-4, CRIPTO, PTK7, IGF-1R, ROR2, integrin beta-6, and SLC39A6 as illustrated in Fig. 4.^[6]

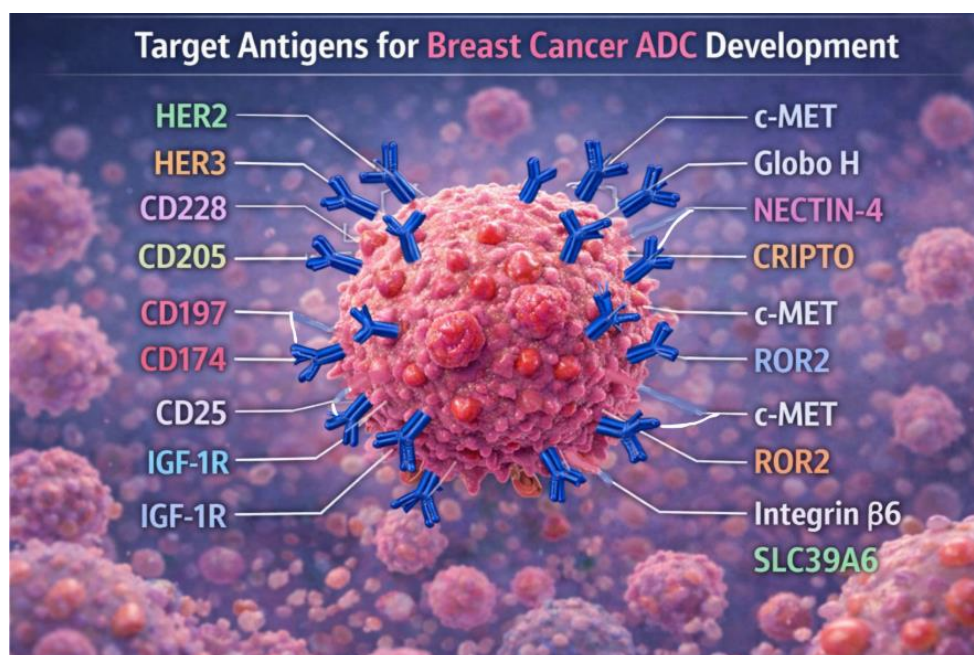


Fig. 4: Target Antigens For Breast Cancer Adc Development.^[6]

EFFICACY OF ADC THERAPY IN BREAST CANCER

HER2-POSITIVE BREAST CANCER

Found on the outer layer of cells, HER2 belongs to a group of proteins known as the EGFR receptor family. Though most receptors in this group need external signals to turn on, those pairing with HER2 skip this step entirely. Pairing up with another ERBB family member triggers HER2's internal switch through self-modification and changes nearby components too. Once switched on, this receptor revs up division signals and movement within the cell. Unchecked activity here speeds up growth, fosters tumor formation, and may spread cancer to distant sites.^[13]

Back in 2013 came the debut of a new therapy: ADO-trastuzumab emtansine (T-DM1), marking the first antibody-drug conjugate cleared for HER2-positive breast cancer. Not long afterward, an updated version followed - trastuzumab deruxtecan (T-DXd) - stronger by design, arriving on the scene to help those facing advanced stages after earlier treatments had been tried.^[2]

TRIPLE NEGATIVE BREAST CANCER (TNBC)

TNBC makes up between fifteen and twenty percent of global breast cancer diagnoses, standing out because of its rapid progression and frequent occurrence alongside challenging outcomes and few treatment paths. A diagnosis emerges when tumor cells show absence of three critical biomarkers. Because standard chemo often brings difficult reactions, newer methods like antibody-linked drugs have taken shape in tackling this disease. One after another, treatments including sacituzumab govitecan and trastuzumab deruxtecan appear promising within clinical settings.^[14, 15]

FDA-APPROVED ADCs IN BREAST CANCER

1. TRASTUZUMAB EMTANSINE (T-DM1)

INDICATION: For use in the treatment of HER2-positive breast cancer.^[16]

COMPOSITION OF T-DM1: Antibody-Trastuzumab (T); **Payload-** Emtansine (DM1); **Linker-**Stable thioether linker.^[16]

TARGET RECEPTOR: HER2 receptor.^[16, 17]

PROGRESSION-FREE SURVIVAL (PFS) WHEN ON T-DM1: 14 Months.^[18]

METHOD OF FUNCTIONING: T-DM1 combines with HER2 receptor overexpressing cancer cells and becomes internalized into the target cell by a process called endocytosis and releases DM1 intracellularly this leads to cancer cell death. T-DM1 ADC improves targeting and also reduces systemic toxicity.^[16]

CLINICAL DATA

FOR USE OF T-DM1 IN TREATMENT OF EARLY-STAGE BREAST CANCER

- **KATHERINE trial:** T-DM1 is used as a standard adjuvant therapy for treating HER2-positive breast cancer. According to this trial, reduced recurrence risk of breast tumor was seen in patients with existing disease who were earlier on neoadjuvant therapy.^[16]

YEAR IN WHICH T-DM1 WAS APPROVED FOR TREATING EARLY-STAGE BREAST CANCER: 2019.^[19]

- **ATEMPT trial:** It was found that the drug showed three-year disease-free survival with fewer side effects such as neuropathy and alopecia observed.^[16]

- **KRISTINE trial:** In this trial, a combination of T-DM1 with pertuzumab was observed when given as a neoadjuvant therapy in patients having early stage breast cancer; a lower pathologic complete response (pCR) was observed, and some patients may achieve disease free survival without receiving full chemotherapy.^[20]

CLINICAL EVIDENCE FOR USE OF T-DM1 AGAINST ADVANCED OR METASTATIC BREAST CANCER

- **EMILIA trial:** This trial highlights that T-DM1 has shown improved efficacy along with improved safety when compared to use with standard therapy.^[16]

YEAR OF APPROVAL OF T-DM1 FOR TREATING ADVANCED OR METASTATIC BREAST CANCER: 2013^[19]

- **TDM4450g study:** This trial reports that the ADC T-DM1 was well tolerated by the patients along with a few common undesirable clinical presentations such as nausea, fatigue, headache, and hepatic enzyme elevation.^[16]
- **TH3RESA trial:** The study reports higher progression-free survival and overall survival rates in pretreated breast cancer patients.^[16]

- **ESMO 2019:** According to this trial, the drug showed efficacy after dual HER2-target therapy failure.^[16]
- **NCCN guidelines:** These guidelines suggest that T-DM1 can be used for advanced or metastatic types of breast cancer as a second line of treatment.^[16]

OTHER EVIDENCE OF ADC T-DM1

- **WSG-ADAPT-TP trial:** Evidence from this study suggests trastuzumab-based regimens showed long-term survival.^[16]
- **KAMELEON study:** The study showed that the drug was effective in some non-breast HER2-positive tumors.^[16]

FDA RECOMMENDED DOSE FOR ADO-TRASTUZUMAB EMTANSINE FOR TREATING EARLY BREAST CANCER: The suitable dosage regimen according to the FDA for use in the treatment of early breast cancer is 3.6 mg/kg IV infusion on day 1 and once every 21 days (3 weeks) for 14 cycles.^[21]

YEAR IN WHICH T-DM1 WAS APPROVED FOR USE BY FDA: 2013^[2]

ADVERSE EVENTS OF T-DM1: Thrombocytopenia, mild hepatic enzyme elevation, anemia, hypokalemia, neutropenia, muscle and joint pain, and headache as depicted in Fig. 5.^[17, 18, 21, 22]

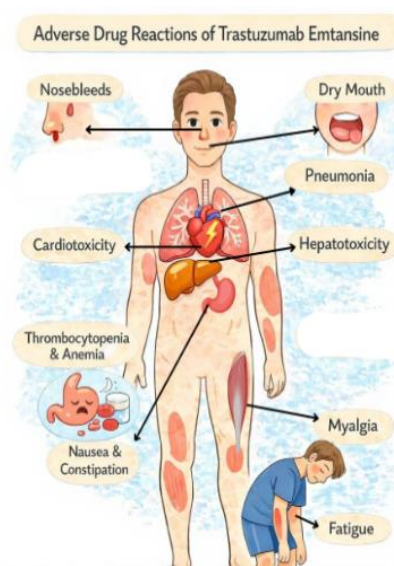


Fig 5: Adverse drug reactions of Trastuzumab Emtansine.^[17, 18, 21, 22]

2. TRASTUZUMAB DERUXTECAN (T-DXd)

INDICATION FOR USE OF T-DXd: HER2 positive and HER2-low metastatic breast cancer.^[17]

COMPOSITION OF T-DXd: **Antibody-** Trastuzumab (T); **Payload-** deruxtecan (DXd); **Linker used-** mc-Gly-Gly-Phe-Gly chain.^[17]

RECEPTOR OF TARGET: HER2 receptor.^[16,17]

METHOD OF FUNCTIONING

T-DXd binds to HER2 receptor overexpressing tumor cells at their cell surface and enters the cell by a process called endocytosis and releases DXd intracellularly after undergoing separation from the antibody and shows lethal action on targeted cells, indicating site-specific action of the drug. It also shows the bystander effect, due to which it penetrates into surrounding cancer cells and kills them.^[16]

CLINICAL STUDIES PERFORMED TO PROVE EFFECTIVENESS OF T-DXd

- **DESTINY-Breast02:** This shows a comparison trial conducted in comparison of T-DXd with T-DM1 in patients with metastatic HER2-positive breast cancer who were using T-DM1 ADC for treatment and were now given T-DXd. This study concludes that T-DXd performed better than standard physician-selected treatments, delaying disease progression in patients to whom T-DM1 was given earlier as treatment.^[19]

YEAR IN WHICH T-DXd WAS APPROVED FOR USE IN METASTATIC BREAST CANCER IN PLACE OF T-DM1: 2022^[19]

- **DESTINY-Breast03:** This study is a comparison study that uses T-DXd along with T-DM1. Patients who were using T-DXd ADC as a second-line treatment were now given T-DXd for treating metastatic HER2-positive breast cancer. The results indicate that using T-DXd has reduced the likelihood of disease progression.^[19]

YEAR OF APPROVAL OF USE OF T-DXd AGAINST T-DM1 FOR METASTATIC BREAST CANCER: 2022^[19]

- **TRIO-US B-12 TALENT study:** This study provides evidence that pathologic complete response (pCR) was limited with drug use but showed strong radiographic tumour shrinkage with HER low breast cancer. This study suggests the need for improved biomarkers for achieving maximum benefit.^[20]
- **SOLTI VALENTINE Trial:** According to this trial, treatment with HER3-DXd showed good antitumor activity with very few high-grade adverse events compared with standard chemotherapy, indicating the need for more such targeted therapies.^[20]
- **CLINICAL STUDIES FOR DUAL THERAPIES INVOLVING T-DXd**

T-dxd with immune checkpoint inhibitors (durvalumab, nivolumab, and pembrolizumab): enhances tumor activity by enhancing each other's activity.^[17]

- **BEGONIA:** A study evaluating the use of T-DXd and durvalumab as combination therapy has shown an overall response rate of 57% and a progression-free survival of ~1 year in patients with

HR-negative, HER2-low advanced and metastatic types of breast cancer.^[17]

- **DS8201-A-U105:** This study showed promising activity in treating metastatic breast cancer and other cancers, such as urothelial cancers, with a response of up to 65.6%, highlighting additional benefits with the use of this combination when T-DXd was used in combination with nivolumab.^[17]
- **DS8201-A-U106:** This clinical study uses T-DXd with pembrolizumab and shows a good response in metastatic breast cancer (HER2-positive and HER2-mutant cancer) and a few types of lung cancers, supporting the use of combination regimens for treatment.^[17]

FDA DOSING FOR FAM-TRASTUZUMAB DERUXTECAN-NXKI FOR USE IN THE TREATMENT OF HER2 POSITIVE BREAST CANCER (ENHERTU)

The FDA dose was an IV infusion of 5.4 mg/kg once every 3 weeks (=21-day cycle).^[23]

ADVERSE EVENTS OF FAM-TRASTUZUMAB DERUXTECAN-NXKI

Interstitial lung disease, embryofetal toxicity, neutropenia, left ventricular dysfunction, nausea, anemia, other hematological abnormalities, hepatic enzyme abnormalities, decreased appetite, fatigue, diarrhea, hypokalaemia, cough as depicted in Fig. 6.^[23]

FDA DOSING RECOMMENDED FOR FAM-TRASTUZUMAB DERUXTECAN-NXKI AND PERTUZUMAB IN METASTATIC HER2 POSITIVE BREAST CANCER

Recommended dose is fam-trastuzumab deruxtecan-nxki (5.4 mg/kg IV d1, followed by pertuzumab (840 mg d1 for one cycle, then 420 mg) then fam-trastuzumab deruxtecan-nxki (5.4 mg/kg IV d1) and pertuzumab (420 mg IV d1) every 21 days.^[24]

ADVERSE EVENTS

Anemia, interstitial lung disease, leukopenia, neutropenia, thrombocytopenia, and organizing pneumonia as depicted in Fig. 6.^[16]

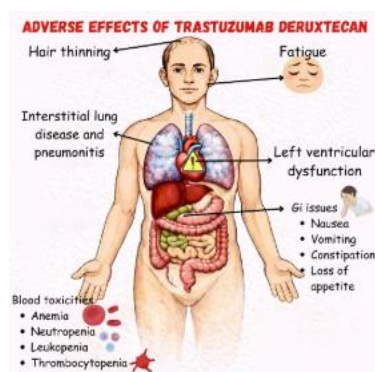


Fig 6: Adverse drug reactions of Trastuzumab Deruxtecan.^[16]

3. DATOPOTAMAB DERUXTECAN (Dato-DXd)

INDICATION FOR USE: Triple-negative breast cancer, HER2-negative, HR-positive metastatic breast cancer.^[17,20]

COMPOSITION: Antibody-Datopotamab (Dato); Payload-deruxtecan (DXd); Linker used- mc-Gly-Gly-Phe-Gly chain.^[17]

TARGET: TROP-2^[17]

MECHANISM OF ACTION: Datopotamab (Monoclonal antibody) combines with TROP-2 expressing cancer cells, gets internalized, leads to DNA damage, tumor growth inhibition, and finally causes programmed cell death of the cancer cell with very little damage to normal cells.^[25]

CLINICAL TRIALS CONDUCTED FOR USE AS COMBINATION THERAPY IN TREATING TRIPLE NEGATIVE BREAST CANCER

- **BEGONIA:** Dato-DXd with Durvalumab was evaluated in combination in this study, and the overall response rate observed was around 79% in patients having PD-L1-low advanced or metastatic TNBC.^[17]
- **I-SPY 2.2:** Another combination study with the same combination as used in the BEGONIA trial

Dato-DXd with Durvalumab, produced a pathological complete response rate of 54% in patients having high risk early-stage breast cancer.^[17]

- **TROPION-Breast04:** This study is assessing neoadjuvant Dato-DXd with or without Durvalumab in patients diagnosed with early stage TNBC or HR-low/HER2-negative disease, followed by adjuvant Durvalumab.^[17]
- **TROPION-Breast05:** This study compares the use of neoadjuvant Dato-DXd along with Durvalumab against chemotherapy with Pembrolizumab in patients having PD-L1-high metastatic type of TNBC.^[17]

FDA RECOMMENDED DOSING FOR DATOPOTAMAB DERUXTECAN-dlnk FOR METASTATIC, HR POSITIVE, HER2-NEGATIVE BREAST CANCER: 6mg/kg (for patients greater than 90kg, maximum 540 mg) IV infusion on day 1 followed by once every 3 weeks (or 21-day cycle).^[26]

ADVERSE EVENTS: Hematological abnormalities, stomatitis, nausea, vomiting, weakness, alopecia, dryness in the eyes, constipation, hepatic enzyme abnormalities, keratitis as depicted in Fig. 7.^[17,26]

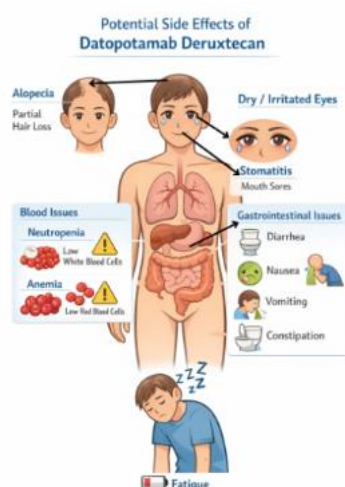


Fig 7: Adverse drug reactions of Daptomycin Deruxtecan.^[17,26]

4. SACITUZUMAB GOVITECAN (SG)

INDICATION FOR USE: Triple negative breast cancer (TNBC), HER2-negative or HR-positive breast cancer.^[20]

COMPOSITION: Antibody- Anti-TROP-2 antibody
Payload- SN-38(active metabolite of irinotecan)

Linker used- Cleavable CLA2 linker.^[27]

TARGET: TROP-2^[27]

MECHANISM OF ACTION: Anti-TROP-2 antibody of SG identifies and binds to TROP-2 receptor which is taken up by the cancer cell which is then transported to lysosomes, where the linker is cleaved, releasing the payload (drug) SN-38. This drug damages the cancer cells' DNA by inhibiting topoisomerase 1, causing the death of cancer cells. Additionally, it also shows the

bystander effect, where the drug diffuses into surrounding tumor cells and kills them.^[27]

CLINICAL EVIDENCE FOR USE OF SG IN TREATMENT OF TNBC

- **The NeoSTAR trial:** In this trial it was seen that neoadjuvant SG, when used for treatment of TNBC, showed 30% pathologic complete response (pCR), with a good safety profile, and improved results.^[20]
- **IMMU-132-01:** In this clinical study, pre-treated triple negative breast cancer patients were given SG at 10mg/kg, which produced a ~30% response rate with progression-free survival of 55 months, along with overall survival of 13 months.^[28]

YEAR OF APPROVAL OF SG FOR TREATMENT OF PRE TREATED TNBC: 2019^[19]

- **TROPiCS-02:** This study focused on endocrine-resistant HR-positive/HER2-negative breast cancer. The use of SG in such patients has demonstrated improved response rates, progression free survival, and overall survival.^[28]

YEAR OF APPROVAL OF SG FOR ENDOCRINE-RESISTANT HR-POSITIVE/HER2-NEGATIVE BREAST CANCER: 2023^[19]

- **ASCENT:** This study demonstrates the use of SG in patients with triple-negative breast cancer. Clinical benefit was observed in HER2-negative/HER2-low patient subgroups. SG showed improved outcomes along with progression free survival of 5.6 vs 1.7 months and overall survival of 12.1 vs 6.7 months.^[28]

YEAR OF APPROVAL OF SG FOR TNBC, HER2 NEGATIVE /HER2 LOW PATIENT GROUPS: 2020^[19]

FDA RECOMMENDED DOSING REGIMEN FOR USE OF SG IN TREATING TNBC: 10mg per kg once weekly cycle (day 1 followed by day 7).^[29]

ADVERSE EVENTS OF SACITUZUMAB GOVITECAN

Alopecia, nausea, vomiting, rash, loss of appetite, constipation, diarrhoea, abdominal cramps as depicted in Fig. 8.^[29]

FDA RECOMMENDED DOSE FOR SACITUZUMAB GOVITECAN-hziy FOR HR POSITIVE TYPE OF BREAST CANCER: 10mg per kg IV infusion once weekly cycle (day 1 followed by day 7).^[30]

ADVERSE EFFECTS ASSOCIATED WITH SACITUZUMAB GOVITECAN-hziy: Hematological abnormalities, nausea, weakness, alopecia, reduced albumin.^[28,30]

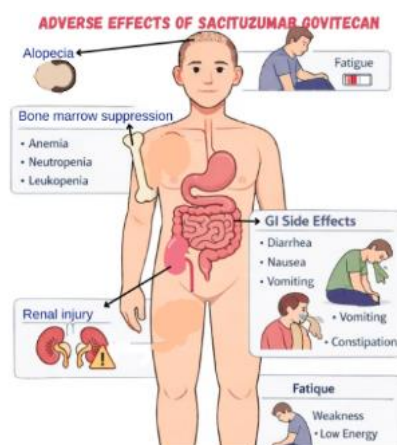


Fig 8: Adverse drug reactions of Sacituzumab Govitecan.^[28,30]

TOXICITIES ASSOCIATED WITH ADCs

ADCs were developed to reduce the toxicity caused by systemic chemotherapeutic agents. But ADCs can also cause toxicities related to payload and how the target antigen is expressed in normal tissues. When the target antigen is normally present on healthy tissues, it may trigger unexpected tissue-specific toxicities, known as on-target toxicity. For example, CD44v6-targeted ADC

bivatuzumab mertansine led to serious skin toxicity due to CD44v6 expression on normal skin. Despite this, many ADC-related side effects are off-target toxicities. Common examples include bone marrow suppression, peripheral neuropathy, hepatotoxicity, anemia, and neuropathy with MMAE-based ADCs and liver toxicity and thrombocytopenia with DM1-based ADCs.^[31]

ADC (Drug)	Toxicities associated	Dose related?	Manageable?
Datopotamab Deruxtecan (Dato-DXd)	GI- Diarrhea, nausea, vomiting, constipation Blood- Neutropenia, anemia Others- stomatitis, alopecia, fatigue, dryness in eye ^[32]	Yes ^[33]	Yes, dose reduction of Datopotamab deruxtecan From an initial dose of 6mg per kg once every 3 weeks to a reduction to a 4mg per kg dose once every 3 weeks, reduces the frequency of dose related toxicities of the drug. ^[33]
Trastuzumab Deruxtecan (T-DXd)	Black box warning- Interstitial lung disease Lung- Pneumonitis	Yes ^[34]	Yes, the standard dose is 5.4mg per kg body weight -6.4mg per kg body weight IV infusion once every three

	GI- nausea, vomiting, constipation, diarrhoea, loss of appetite Blood- anaemia, neutropenia, leukopenia, thrombocytopenia Others- fatigue, alopecia, embryo fetal toxicity, left ventricular dysfunction. ^[34]		weeks (21 day cycle). Dose reductions can be made to minimize toxicities associated with therapy. Reduce the dosage to 4.4 mg per kg body weight to 5.4 mg per kg body weight IV infusion once every three weeks (21 day cycle). If toxicity is still seen without any changes, lower the dose even further to 3.2 mg per kg body weight to 4.4 mg per kg body weight IV infusion once every three weeks (21-day cycle). If toxicities appear again then stop taking the medication. ^[34,35]
Trastuzumab Emtansine (T-DM1)	Black box warning- Hepatotoxicity GI-nausea, constipation Blood-Thrombocytopenia, anemia Liver- Abnormal transaminase enzymes Heart- cardiotoxicity Lung- Pneumonia Others- Fatigue, myalgia, dry mouth, Nose bleeding. ^[34,36]	Yes ^[34]	Yes, the normal dose given is 3.6mg per kg IV dose once every three weeks. If any adverse events are observed, the dose should be reduced up to 2mg/kg IV dose once every 3 weeks and then to 2.4mg per kg IV dose once every 3 weeks. If toxicities appear again then stop the drug use immediately. ^[34,37,38]
Sacituzumab Govitecan (SG)	Black box warning-Severe neutropenia, diarrhoea GI- Nausea, vomiting, diarrhoea, constipation, Blood- anemia, neutropenia, leukopenia Renal- renal injury Others- alopecia, fatigue, infection. ^[34,36]	Yes ^[37]	Yes, most of the adverse events are manageable. The standard dose for Sacituzumab govitecan is 8mg per kg body weight –10mg per kg body weight IV on days 1 and 8 of a 21 day cycle (3 week cycle). First reduction of doses is 7.5 mg per kg body weight IV dose on days 1 and 8 of a 21 day cycle (25%reduction), Then, a 5mg per kg body weight IV dose on days 1 and 8 of a 21 day cycle, and if still ADRs are reported, then stop the drug. ^[37,39]

RESISTANCE PATTERNS WITH ADCs

Despite advantages, resistance mechanisms continue to limit the outcome of the treatment. Although the exact mechanisms of ADC resistance are yet unknown, a few early research studies indicates that tumor cells evade the ADCs at multiple stages. A few of the mechanisms are:

- Reduced expression of target antigen
- Cytotoxic payloads are pumped back out of the tumour cells
- Failure to release the payload.^[31]

BIOMARKERS USED IN BREAST CANCER

TROP-2 RECEPTOR

A protein called Trophoblast cell surface antigen-2 (TROP-2) is an integral membrane protein that was first discovered on trophoblastic cells. Its expression is either absent or very limited, normally on the cell surface. whereas it is highly expressed in many types of cancers of epithelial cells, including breast cancer (especially TNBC), where it helps the tumor to proliferate, migrate, invade, develop resistance, and undergo apoptosis. TROP-2 expression also increases tumor cell aggressiveness by activating a few signalling pathways targeting cell motility and survival. It also serves as a biomarker. Sacituzumab govitecan is

currently approved by the FDA for the treatment of metastatic TNBC.^[7,40]

HER2 RECEPTOR

Human epidermal growth factor receptor or HER is a part of the family of ErbB which includes four types of receptors: HER1 (ErbB1), HER2 (ErbB2), HER3 (ErbB3), and HER4 (ErbB4). HER2 receptors get triggered by a few signalling cascades and gets activated. It controls tumor cell proliferation, their maturity, and survival. Twenty out of a hundred breast tumors contain a higher number of HER2 receptors expressed on their surface; the more these receptors are expressed, the more aggressive the tumor cells become, which makes treatment ineffective.^[41]

Higher the HER2 expression higher is the therapeutic efficacy of a drug.^[7]

HER2-targeted therapies show effective efficacy against breast and gastric cancers, their role in other cancers such as ovarian cancer, is under investigation.^[41]

The higher the HER2 expression, the higher the therapeutic efficacy of a drug.^[7]

MDR1 EXPRESSION

Where cancer cells develop resistance to ADC drugs due to various mechanisms (usually more than one mechanism), such as efflux and reduced drug levels within the cell, is called MDR1 (Multidrug resistance protein).^[7,42,43,44]

CANCER HETEROGENECITY

Current evidence suggests that specific type of cancer cell expresses various antigens on it which are useful for predicting the efficacy of ADC.^[7,42,43,44]

FUTURE DIRECTIONS

The ADC design is becoming a more prominent way of thinking about future development strategies. Enhancement of tumor specificity and internalization efficiency by optimizing antibody–antigen interactions, potentially minimizing off-target toxicity while increasing intracellular payload delivery (Davis et al., 2025). Improvements in linker design continue to be at the heart of this effort, and we can expect that next generation cleavable linkers will be developed, which possess plasma stability while allowing for effective drug delivery within the tumor cell microenvironment (Xing et al., 2024). These refinements are designed to augment intratumoral cytotoxicity while minimizing circulating effects on the immune system.

Moreover, novel conjugation methods that would allow site-specific and controlled payload coupling may have the potential to favourably affect pharmacokinetics, heterogeneity, and reproducibility of therapeutic outcome. However, beyond structure optimization, combination with optimisation of biomarker refinement and resistance profiling will also be crucial to maintain the clinical benefit.

Finally, the evolution of ADC based treatment strategies depends on the confluence of molecular engineering, translational research, and precision oncology, which provides more effective treatment along with patient specific personalized therapy.^[45,46,47]

LIMITATIONS

Despite many benefits, ADC use in breast cancer is associated with many limitations. Due to its complex manufacturing, its cost is high, and its accessibility is limited in low-income families. ADCs are associated with adverse events such as hepatotoxicity, myelosuppression, interstitial lung disease, and GI-related and hematological issues. They are usually less than standard chemotherapy but are also clinically important. The cancer cells develop various resistance mechanisms leading to failure of therapy. Due to its complex structure, it should be developed with precision, and the selection of the linker becomes important as it is responsible for drug release and maintaining toxicity. There are many problems involved in patient selection for this treatment. Appropriate patients must receive accurate biomarker testing and then be

properly identified to receive benefit from this treatment approach. The ADCs are an emerging therapy for the treatment of breast cancer, and therefore, there is limited information available about the long-term effects, post marketing data of these treatments, as well as data on survival outcomes.^[7,31, 32, 37]

CONCLUSION

Antibody-drug conjugates have helped bypass the barriers that were associated with traditional chemotherapy which offered limited selectivity and substantial toxicity. By linking targeted antibodies with highly potent cytotoxic payloads, ADCs have demonstrated that precision delivery can translate into meaningful improvements in progression-free and overall survival across multiple breast cancer subtypes. Clinical success of these agents that targets HER2 and TROP2 reflects not only advancements in molecular engineering and deeper understanding of tumor biology. Although ADCs show promising outcomes, they are associated with multiple limitations like resistance mechanisms, tumor heterogeneity and toxicity profiles which continue to shape clinical decision making. Optimizations of biomarker-driven patient selection and refining ADC design-through improved linker stability, site specific conjugation and novel payloads are critical to ensure long-term sustainability. Looking Ahead the continued integration of translational research with well-designed clinical trials will determine how far this therapeutic class can evolve. As new ADCs emerge the goal is not only improved efficacy, but also individualized and durable treatment approaches for treatment of breast cancer.

REFERENCES

1. Xiong X, Zheng LW, Ding Y, Chen YF, Cai YW, Wang LP, et al. Breast cancer: pathogenesis and treatments. *Signal Transduct Target Ther.*, 2025; 10: 49. doi:10.1038/s41392-024-02108-4.
2. Monteiro M, Nunes N, Junior A, Fede A, Bretas G, Souza C, et al. Antibody-Drug Conjugates in Breast Cancer: A Comprehensive Review of How to Selectively Deliver Payloads. *Breast cancer (Dove Med Press).*, 2024; 16: 51-70. doi:10.2147/BCTT.S448191
3. Precision for Medicine. Clinical trial trends: antibody-drug conjugates [Internet]. 2024. Available from: <https://www.precisionformedicine.com/blog/clinical-trial-trends-antibody-drug-conjugates>
4. Shastry M, Gupta A, Chandarlapaty S, Young M, Powles T, Hamilton E. Rise of antibody-drug conjugates: The present and future. *American Society of Clinical Oncology Educational Book*, 2023 May 1; 43. doi:10.1200/EDBK_390094
5. Ruan D, Wu H, Meng Q, Xu R. Development of antibody-drug conjugates in cancer: overview and prospects. *Cancer communications*, 2024 Dec 30; 44(1): 3–22. doi:10.1002/cac2.12517.

6. Novotech. Antibody–drug conjugates: Global clinical trial landscape (2024) [Internet]. 2024 Apr 19 [cited 2026 Jan 27]. Available from: <https://novotech-cro.com>
7. Larose ÉA, Hua X, Yu S, Pillai AT, Yi Z, Yu H. Antibody-drug conjugates in breast cancer treatment: resistance mechanisms and the role of therapeutic sequencing. *Cancer Drug Resist*, 2025 Mar 6; 8: 11. doi: 10.20517/cdr.2024.180.
8. Takakura T, Shimizu T, Yamamoto N. Antibody-drug conjugates in solid tumors; new strategy for cancer therapy. *Jpn J Clin Oncol*, 2024; 54(8): 837-846. doi:10.1093/jjco/hyae054.
9. Long R, Zuo H, Tang G, Zhang C, Yue X, Yang J, et al. Antibody-drug conjugates in cancer therapy: applications and future advances. *Frontiers in Immunology*, 2025 May 21; 16. doi:10.3389/fimmu.2025.1516419
10. Chen B, Zheng X, Wu J, Chen G, Yu J, Xu Y, et al. Antibody–drug conjugates in cancer therapy: current landscape, challenges, and future directions. *Mol Cancer*, 2025 Nov 3; 24: 279. doi:10.1186/s12943-025-02489-2.
11. Wang J, Wu SG. Breast Cancer: An Overview of Current Therapeutic Strategies, Challenges, and Perspectives. *Breast Cancer* (Dove Med Press), 2023 Oct 20; 15: 721-730. doi: 10.2147/BCTT.S432526.
12. Teama H, Elnewishy A, Noureldin M, Elnewishy O. Comparative efficacy and safety of antibody-drug conjugates versus standard chemotherapy in metastatic breast cancer: a comprehensive systematic review and meta-analysis. *Cureus.*, 2025; 17(12): e98924. doi:10.7759/cureus.98924.
13. Li X, Zhang X, Yin S, Nie J. Challenges and prospects in HER2-positive breast cancer-targeted therapy. *Crit Rev Oncol Hematol*, 2025; 207: 104624. doi:10.1016/j.critrevonc.2025.104624.
14. Dri A, Arpino G, Bianchini G, Curigliano G, Danesi R, De Laurentiis M, et al. Breaking barriers in triple negative breast cancer (TNBC) – Unleashing the power of antibody-drug conjugates (ADCs). *Cancer Treatment Reviews*, 2024 Feb 1; 123: 102672. doi:10.1016/j.ctrv.2023.102672
15. Nagayama A, Vidula N, Ellisen L, Bardia A. Novel antibody-drug conjugates for triple negative breast cancer. *Ther Adv Med Oncol*, 2020 May 11; 12: 1758835920915980. doi: 10.1177/1758835920915980.
16. Liu K, Li M, Li Y, Li Y, Chen Z, Tang Y, et al. A review of the clinical efficacy of FDA-approved antibody–drug conjugates in human cancers. *Mol cancer*, 2024 Mar 23; 23(1): 62. doi:10.1186/s12943-024-01963-7.
17. Jang H, Chang JE. Strategic combinations of antibody-drug conjugates from 2023 to 2025: from dual therapies to innovative ADC-based regimens. *Pharmaceutics*, 2025; 17(12): 1581. doi:10.3390/pharmaceutics17121581
18. Gupta S, Singhal M, Nag S, Abbas W, Doval DC, Goyal H, et al. Safety and efficacy assessment of trastuzumab emtansine in Indian patients with HER2-positive unresectable locally advanced or metastatic breast cancer previously treated with trastuzumab and a taxane: an open-label, single-arm, phase IV study. *Asia Pac J Clin Oncol*, 2026; 22(2): 240-250. doi:10.1111/ajco.70007.
19. Hurwitz J, Haggstrom L, Lim E. Antibody–Drug Conjugates: Ushering in a New Era of Cancer Therapy. *Pharmaceutics*, 2023 Jul 26; 15(8): 2017. doi: 10.3390/pharmaceutics15082017.
20. Patel P, Giordano A, Giordano S, Schlam I, Tolaney SM, Tarantino P. Antibody-drug conjugates for treating early-stage breast cancer: current use, anticipated evolutions. *NPJ Breast Cancer*, 2025 Jul 30; 11: 81. doi:10.1038/s41523-025-00800-4
21. Center for Drug Evaluation and Research. FDA approves ado-trastuzumab emtansine for early breast cancer [Internet]. Silver Spring (MD): U.S. Food and Drug Administration.; 2019 [cited 2026 Jan 27]. Available from: <https://www.fda.gov/drugs/resource-s-information-approved-drugs/fda-approves-ado-trastuzumab-emtansine-early-breast-cancer>
22. Gupta A, Sansar B, Mishra BK, Khan A, Singh A, Upadhyay A, et al. Real-world data on trastuzumab emtansine (T-DM1) efficacy and safety: results of a single-centre retrospective study of HER2-positive metastatic breast cancer patients. *Sci Rep.*, 2025; 15(1): 18669. doi:10.1038/s41598-025-97923-2
23. U.S. Food and Drug Administration. ENHERTU (fam-trastuzumab deruxtecan-nxki) for injection: prescribing information [Internet]. Silver Spring (MD): U.S. Food and Drug Administration; 2021 Jan [cited 2026 Jan 27]. Available from: https://www.accessdata.fda.gov/drugsatfda_docs/label/2024/761139s028lbl.pdf
24. U.S. Food and Drug Administration. FDA approves fam-trastuzumab deruxtecan-nxki with pertuzumab for unresectable or metastatic HER2-positive breast cancer [Internet]. Silver Spring (MD): U.S. Food and Drug Administration [cited 2026 Jan 28]. Available from: <https://www.fda.gov/drugs/drug-approvals-and-databases/fda-approves-fam-trastuzumab-deruxtecan-nxki-pertuzumab-unresectable-or-metastatic-her2-positive>
25. Mobashir M, Tarique H, Habib I, Ahmad S, Inam A, Qamar I, et al. Datopotamab deruxtecan: a new era in targeted therapy for metastatic breast cancer. *Annals of Medicine & Surgery*, 2025 Sep 12; 87: 6942-6943. doi: 10.1097/MS9.0000000000003857
26. U.S. Food and Drug Administration. FDA approves datopotamab deruxtecan-dlnk for unresectable or metastatic, HR-positive, HER2-negative breast cancer [Internet]. Silver Spring (MD): U.S. Food and Drug Administration; 2025 Jan 17 [cited 2026 Jan 27]. Available from: <https://www.fda.gov/drugs/resources-information-approved-drugs/fda-approves-datopotamab-deruxtecan-dlnk-unresectable-or-metastatic-hr-positive-her2-negative-breast>

27. Tolaney SM, DeMichele A, Takano T, Rugo HS, Perou C, Lynce F, et al. OptimICE-RD: sacituzumab govitecan + pembrolizumab vs pembrolizumab ($\pm$ capecitabine) for residual triple-negative breast cancer; *Future Oncol*, 2024; 20(31): 2343-2355. doi:10.1080/14796694.2024.2357534.
28. Manna M, Brabant M, Greene R, Chamberlain MD, Kumar A, Alimohamed N, et al. Canadian Expert Recommendations on Safety Overview and Toxicity Management Strategies for Sacituzumab Govitecan Based on Use in Metastatic Triple-Negative Breast Cancer. *Current Oncology*, 2024; 31(9): 5694-5708. <https://doi.org/10.3390/curroncol31090422>
29. U.S. Food and Drug Administration. FDA grants regular approval to sacituzumab govitecan for triple-negative breast cancer [Internet]. Silver Spring (MD): U.S. Food and Drug Administration; 2021 Apr 7 [cited 2026 Jan 28]. Available from: <https://www.fda.gov/drugs/resources-information-approved-drugs/fda-grants-regular-approval-sacituzumab-govitecan-triple-negative-breast-cancer>
30. U.S. Food and Drug Administration. FDA approves sacituzumab govitecan-hziy for HR-positive breast cancer [Internet]. Silver Spring (MD): U.S. Food and Drug Administration; 2023 Feb 3 [cited 2026 Jan 28]. Available from: <https://www.fda.gov/drugs/resources-information-approved-drugs/fda-approves-sacituzumab-govitecan-hziy-hr-positive-breast-cancer>
31. Drago JZ, Modi S, Chandarlapaty S. Unlocking the potential of antibody-drug conjugates for cancer therapy. *Nat Rev Clin Oncol*, 2021 Jun; 18(6): 327-344. doi: 10.1038/s41571-021-00470-8.
32. Kang S, Kim SB. Toxicities and management strategies of emerging antibody-drug conjugates in breast cancer. *Ther Adv Med Oncol*, 2025 Mar 24; 17: 17588359251324889. doi: 10.1177/17588359251324889
33. Shimizu T, Sands J, Yoh K, Spira AI, Garon EB, Kitazono S, et al. First-in-Human, Phase I Dose-Escalation and Dose-Expansion Study of Trophoblast Cell-Surface Antigen 2-Directed Antibody-Drug Conjugate Datopotamab Deruxtecan in Non-Small-Cell Lung Cancer: TROPION-PanTumor01. *Journal of Clinical Oncology*, 2023 Oct 10; 41(29): 4678-4687. doi:10.1200/JCO.23.00059.
34. Nguyen TD, Bordeau BM, Balthasar JP. Mechanisms of ADC Toxicity and Strategies to Increase ADC Tolerability. *Cancers (Basel)*, 2023 Jan 24; 15(3): 713. doi:10.3390/cancers15030713.
35. ClinicalTrials.gov. A phase II, open-label study to evaluate the efficacy and safety of trastuzumab deruxtecan (T-DXd) for the treatment of unresectable and/or metastatic solid tumors harboring HER2 activating mutations regardless of tumor histology [Internet]. Bethesda (MD): U.S. National Library of Medicine; 2020 [cited 2026 Jan 27]. Identifier: NCT04639219. Available from: <https://www.astrazenecaclinicaltrials.com/study/D967MC00001/>
36. Cheng Y, Lu J, Zhang C, Yan W, Zhu P, Qin Q, Gong L. Overview of antibody-drug conjugates nonclinical and clinical toxicities and related contributing factors. *Antib Ther.*, 2025 Mar 18; 8(2): 124-144. doi: 10.1093/abt/tbaf004
37. D'Arienzo A, Verrazzo A, Pagliuca M, Napolitano F, Parola S, Viggiani M, et al. Toxicity profile of antibody-drug conjugates in breast cancer: practical considerations. *E Clinical Medicine*, 2023; 62: 102113. doi:10.1016/j.eclinm.2023.102113.
38. Peddi PF, Hurvitz SA. Ado-trastuzumab emtansine (T-DM1) in human epidermal growth factor receptor 2 (HER2)-positive metastatic breast cancer: latest evidence and clinical potential. *Ther Adv Med Oncol*, 2014 Sep; 6(5): 202-9. doi: 10.1177/1758834014539183.
39. Spring LM, Nakajima E, Hutchinson J, Viscosi E, Blouin G, Weekes C, et al. Sacituzumab govitecan for metastatic triple-negative breast cancer: clinical overview and management of potential toxicities. *Oncologist*, 2021; 26(10): 827-834. doi:10.1002/onco.13878
40. Hu Y, Zhu Y, Qi D, Tang C, Zhang W. Trop2-targeted therapy in breast cancer. *Biomarker Res.*, 2024; 12(1): 82. doi:10.1186/s40364-024-00633-6.
41. Iqbal N, Iqbal N. Human epidermal growth factor receptor 2 (HER2) in cancers: overexpression and therapeutic implications. *Mol Biol Int.*, 2014; 2014: 852748. doi:10.1155/2014/852748.
42. Yamazaki CM, Yamaguchi A, Anami Y, et al. Antibody-drug conjugates with dual payloads for combating breast tumor heterogeneity and drug resistance. *Nat Commun.*, 2021; 12: 3528. doi:10.1038/s41467-021-23793-7.
43. Chang HL, Schwettmann B, McArthur HL, Chan IS. Antibody-drug conjugates in breast cancer: overcoming resistance and boosting immune response. *J Clin Invest.*, 2023 Sep 15; 133(18): e172156. doi:10.1172/JCI172156
44. Chen N, Michaels E, Howard F, Nanda R. The evolving therapeutic landscape of antibody-drug conjugates in breast cancer. *Expert Rev Anticancer Ther.*, 2022; 22(12): 1325-1331. doi:10.1080/14737140.2022.2147510.
45. Davis AA, Hesse J, Pereira PMR, Ma CX. Novel treatment approaches utilizing antibody-drug conjugates in breast cancer. *NPJ Breast Cancer*, 2025; 11(1): 42. doi:10.1038/s41523-025-00743-w.
46. Xing M, Li Z, Cui Y, He M, Xing Y, Yang L, et al. Antibody-drug conjugates for breast cancer: a bibliometric study and clinical trial analysis. *Discov Oncol*, 2024; 15(1): 329. doi:10.1007/s12672-024-01192-w.