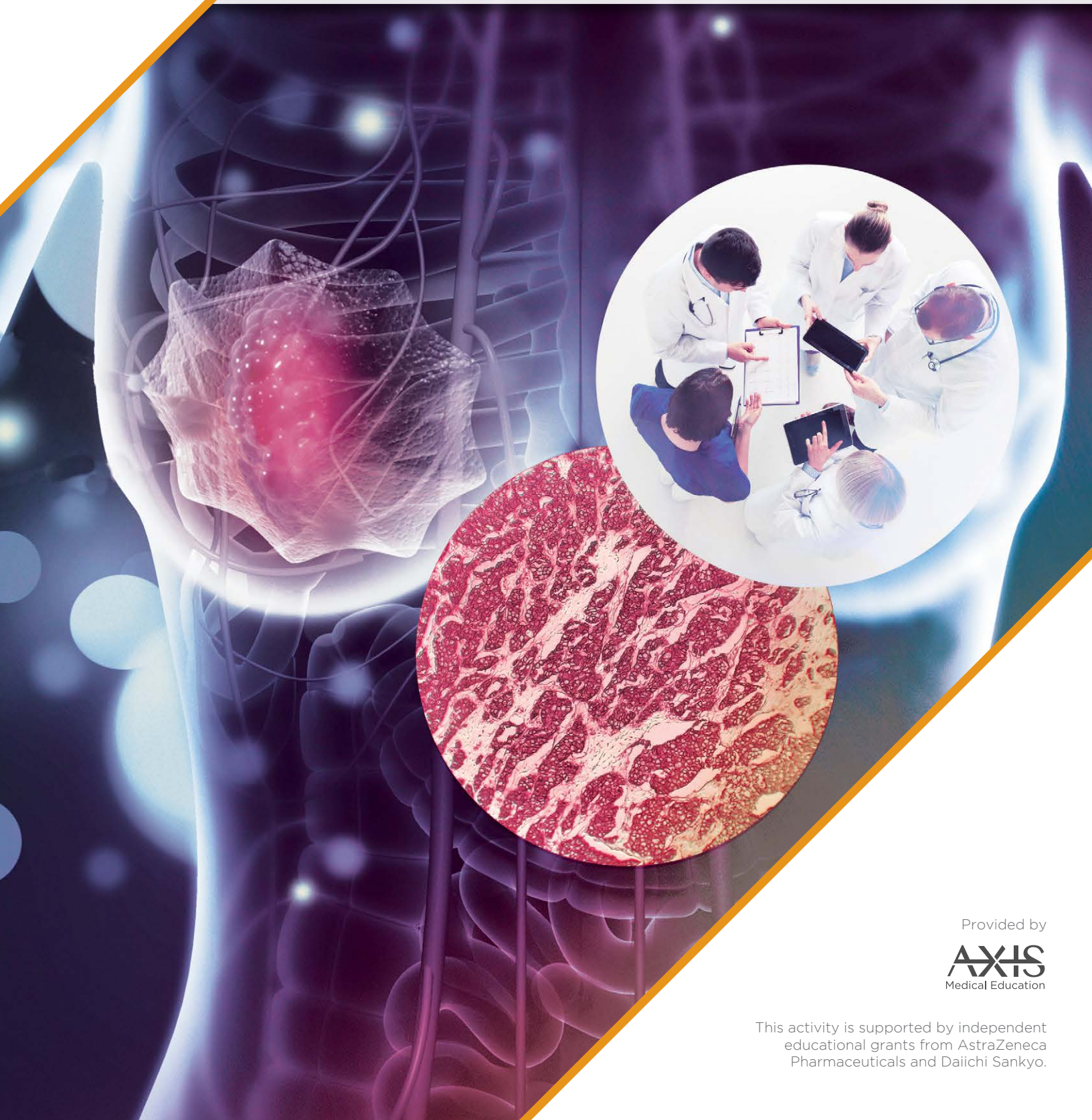


# Redefining Treatment Across the Spectrum of HR+/HER2-Expressing Metastatic Breast Cancer

This transcript has been edited for style and clarity and includes all slides from the presentation.

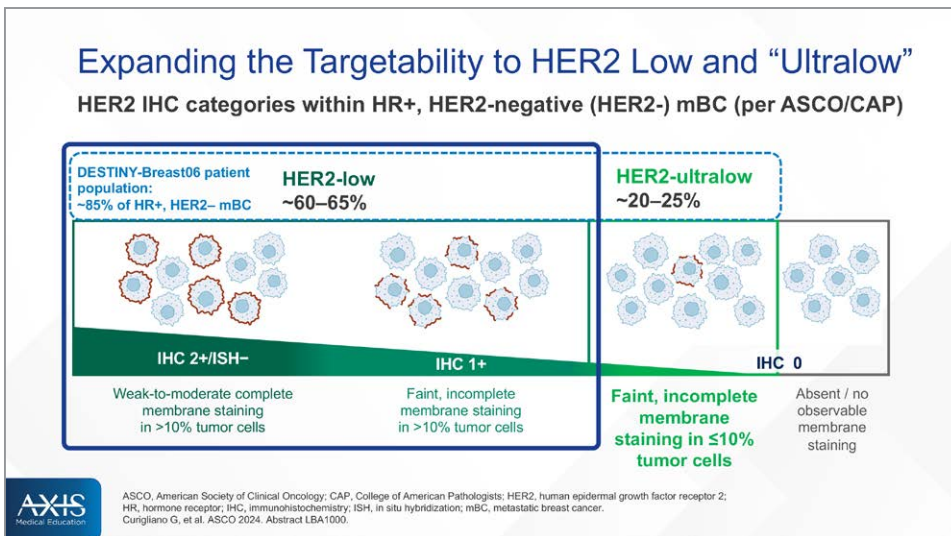


# Redefining Treatment Across the Spectrum of HR+/HER2-Expressing Metastatic Breast Cancer

Sara M. Tolaney, MD, MPH



► **Sara M. Tolaney, MD, MPH:** My name is Sara Tolaney. I'm a breast medical oncologist at Dana-Farber Cancer Institute and Chief of our Breast Oncology program here. Today I'll be reviewing ADC-directed therapies in metastatic hormone receptor-positive breast cancer. So, let's begin.



► So, when thinking about HER2 expression, we've seen our definitions of HER2 expression really evolve particularly over the past year, and this has really changed the way we think about which patients can be eligible for HER2-directed therapies. Initially, we had thought of HER2 being either positive or negative, and then we've evolved into classifying it as either HER2-positive, HER2-low, or negative.

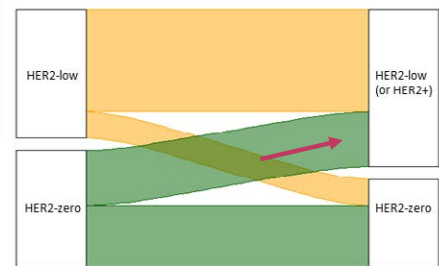
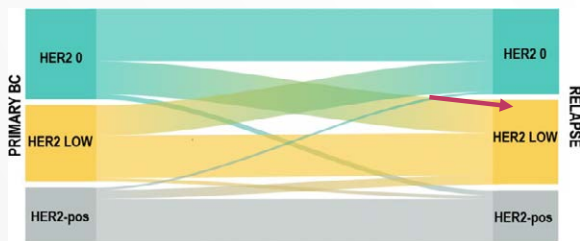
And now, we've started to introduce this idea of HER2-ultralow. And so, in essence, the way that we have divided HER2 expression has grouped patients into being what I call HER2-null, meaning having absolutely no HER2 expression on the cell surface, or HER2-ultralow, which means you have 1 to 10% faint staining of HER2 on the cell, or HER2-low, which means you have HER2 IHC 1+ or 2+ staining

and are not FISH amplified or HER2-positive where you're HER2 IHC 3+ or 2+ and FISH amplified. And so, you can see that this has become far more complex and, in fact, really has evolved, and our pathologists are now trying to figure out how to read HER2 because HER2-ultralow was not something initially included in ASCO-CAP guidelines.

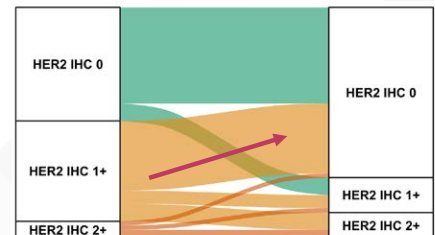


## HER2 Low Is Unstable

- Multiple studies have confirmed the instability of HER2-low expression between primary and metastatic tumors
- The reason is unclear, but may be multifactorial: (pre)analytical factors, HER2 expression heterogeneity, biologic evolution of the disease



Matched paired primary-met TNBC



HER2, human epidermal growth factor receptor 2; IHC, immunohistochemistry; TNBC, triple-negative breast cancer.  
 1. Tarantino P, et al *Eur J Cancer*. 2022;163:35-43. 2. Miglietta F, et al. *NPJ Breast Cancer*. 2021;7(1):137.  
 3. Garrido-Castro A, et al. SABCs 2022. Abstract HER2-10.

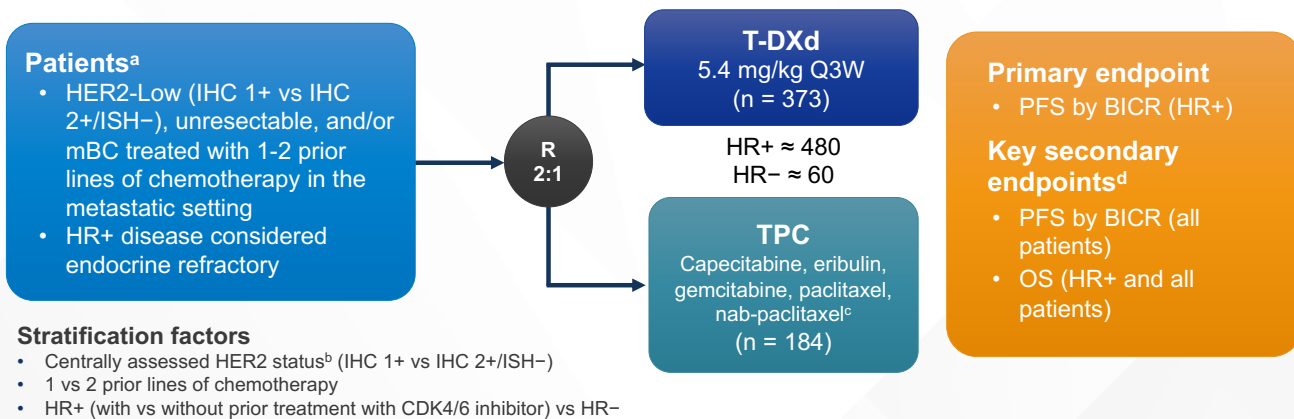
► I think another challenge is that HER2 expression is not stable. So, for example, if you look at expression on a primary tumor, and you compare expression to a metastatic tumor, you can see that they're not always consistent. You can have a primary tumor that's HER2-low and a metastatic recurrence that's HER2-0. You can have a primary tumor that's HER2-0 that becomes HER2-low, and so I think this is a big challenge for us because it means that HER2 expression is constantly evolving over time, even in the metastatic setting. If you were to biopsy patients over time, you can see sometimes it's low, sometimes it's 0, and can go back to low again, telling us that this is a dynamic expression.

And so, when thinking about HER2 expression and what we really need to keep in mind regarding how we should think about this, I think it's really key to realize that deciding on HER2 expression is not necessarily so clear, that we do need to realize that it is evolving. And so, for example, if you had a patient who's always not had any HER2 expression, you can re-biopsy them to see if they've acquired some HER2 expression because it does change, and it can then make them eligible for HER2-directed therapy with T-DXd. So very important to think about that if someone's always been consistently HER2-0, to think about re-biopsying to see if they could be eligible.

The other challenge is trying to understand ultralow, which I think is going to have to be a discussion individually with pathologists. So, at our institution, for example, we did have a discussion, and now our pathologists are starting to read cases as being either HER2-null, HER2-ultralow, HER2-low, or HER2-positive. And so, this has, again, allowed us to understand which patients may be ultralow, but if your pathologist isn't reading it that way, then you won't have that information. And so important to think about these discussions upfront so that you can understand what information is accessible to you.

# DESTINY-Breast04: Study Design

## An Open-Label, Multicenter, Phase 3 Study (NCT03734029)



<sup>a</sup>If patients had HR+ mBC, prior endocrine therapy was required. <sup>b</sup>Performed on adequate archived or recent tumor biopsy per ASCO/CAP guidelines using the VENTANA HER2/neu (4B5) investigational use only (IUO) Assay system. <sup>c</sup>TPC was administered according to the label. <sup>d</sup>Other secondary endpoints included ORR (BICR and investigator), DOR (BICR), PFS (investigator), and safety; efficacy in the HR- cohort was an exploratory endpoint. BICR, blinded independent central review; CDK, cyclin-dependent kinase; HER2, human epidermal growth factor receptor 2; HR, hormone receptor; IHC, immunohistochemistry; ISH, in situ hybridization; mBC, metastatic breast cancer; OS, overall survival; PFS, progression-free survival; Q3W, every three weeks; T-DXd, trastuzumab deruxtecan; TPC, treatment of physician's choice. Modi S, et al. *N Engl J Med.* 2022;387(1):9-20.



► So next, we're going to move on to ADC efficacy in hormone receptor-positive breast cancer. And I think our understanding of how HER2-directed ADCs [work] has dramatically evolved over the last few years, initially, we saw that T-DXd worked very well in cases of patients who had metastatic HER2-positive breast cancer; this came initially from DESTINY-Breast01. And then, in DESTINY-Breast03, we saw head-to-head T-DXd compared to T-DM1 in patients in the second- and third-line setting, and, in fact, saw really unprecedented results where PFS with T-DXd was four times as long as it was with T-DM1. And it was also

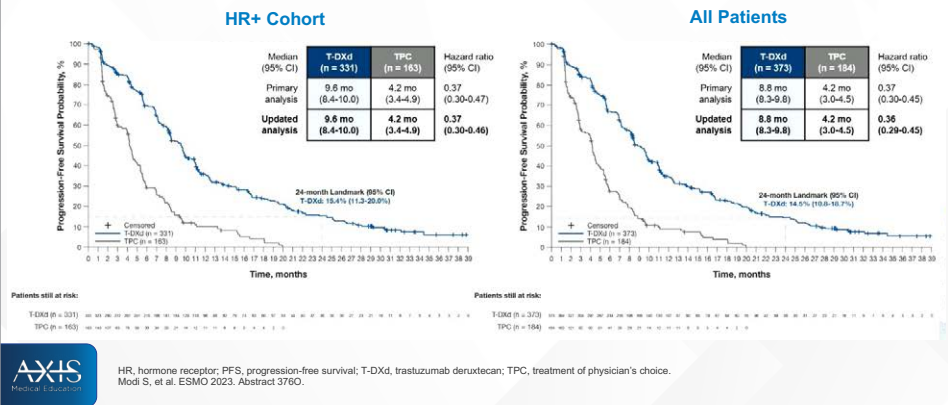
associated with the significant improvement in overall survival.

And so, it really established T-DXd as the standard in metastatic HER2-positive disease. But we had never seen a HER2 ADC be utilized outside of HER2-positive breast cancer. However, we had seen a signal in early phase work that T-DXd did seem to have activity even in tumors that weren't truly HER2-positive but just had a little bit of HER2 expression. And so, then we saw data evolve from DESTINY-Breast04; this was a registration trial comparing T-DXd to treatment of physician's choice chemotherapy in patients who

had tumors that were HER2-low, and the primary endpoint was focused on those patients who also had hormone receptor-positive disease. There was a small subset of patients included who had triple-negative breast cancer that was also HER2-low, and the study was designed for patients who had received one or two lines of chemotherapy in the metastatic setting. And the primary endpoint was to compare the progression-free survival of T-DXd compared to treatment of physician's choice chemotherapy in those patients with hormone receptor-positive HER2-low breast cancer.

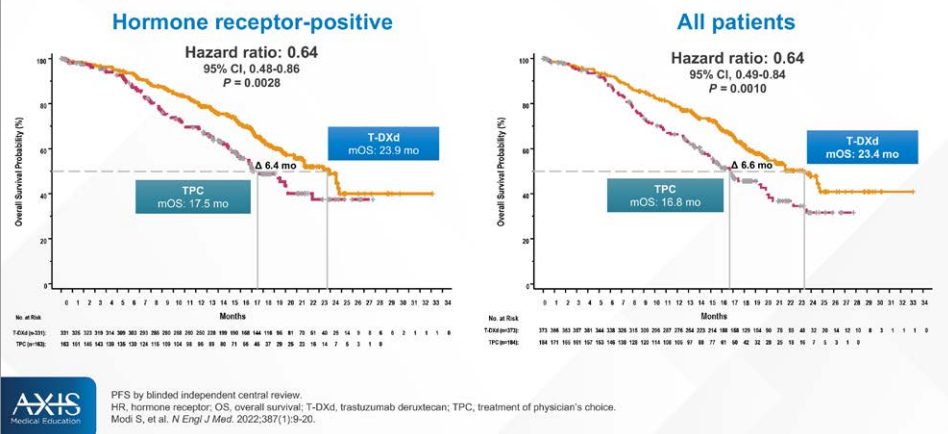
## DESTINY-Breast04: Updated PFS Analysis

Median of 2 prior lines of ET and 1 prior line of chemo



► And we saw that T-DXd was associated with a significant improvement in progression-free survival, going from around 4 to almost 10 months. This was true both in the hormone receptor-positive population but also in the ITT population, which added in that triple-negative subgroup of patients.

## DESTINY-Breast04: OS in HR+ and All Patients

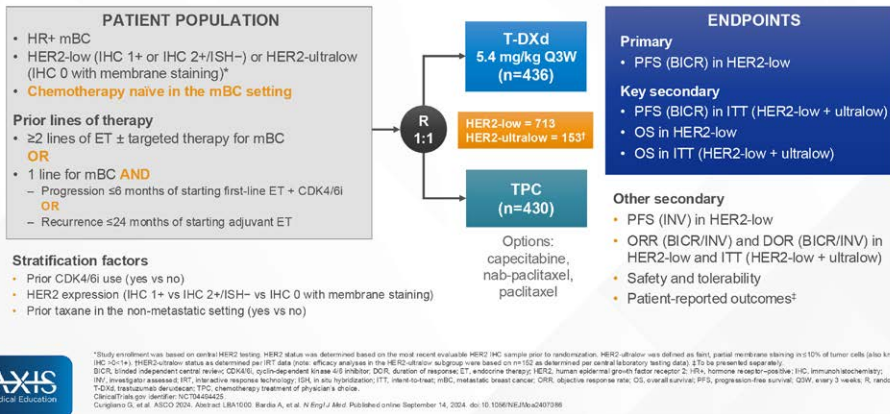


► It also resulted in a significant improvement in overall survival, again favoring T-DXd over standard chemotherapy, where OS improved from about 17 months to 24 months in the hormone receptor-positive population with a hazard ratio that was consistent with 0.64 and was also consistent in the ITT population with the same hazard ratio. So, really suggesting that T-DXd was better both in terms of PFS and OS. This trial, however, was restricted to patients who had already received some chemotherapy for metastatic disease and, again, was restricted to those patients who had HER2-low disease.

So, the question remained as, could we move T-DXd earlier? Could it be a drug for patients who had never seen any chemotherapy for metastatic hormone receptor-positive breast cancer? And could it even work in patients who had just a teeny tiny bit of HER2 expression? So, those cases where patients had HER2-ultralow expression, again, 1 to 10% faint staining.

## DESTINY-Breast06: Study Design

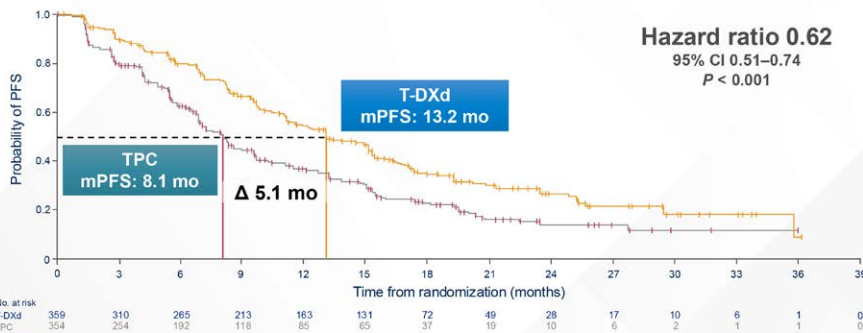
DESTINY-Breast06: a phase 3, randomized, multicenter, open-label study (NCT04494425)



► And so, DESTINY-Breast06 looked to answer this question. It took patients who had progressed on endocrine therapy in the metastatic setting, either one or two lines of endocrine treatment, but had not received any chemotherapy, and randomized them to get T-DXd or treatment of physician's choice chemotherapy. And the study was primarily designed to assess PFS in HER2-low disease, but also included an exploratory group of 150 patients who had HER2-ultralow disease that were included in a secondary analysis with ITT.

## PFS (BICR) in HER2-Low: Primary Endpoint

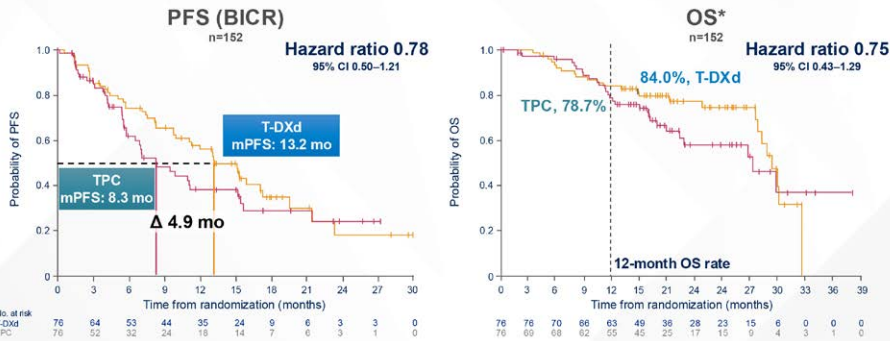
Median of 2 prior lines of ET, 90% with prior CDK4/6i, no prior chemo, 85% had visceral disease, 70% relapsed



► And so, when you looked at PFS in a HER2-low group, you saw a significant improvement going from 8 to 13 months. So, really suggesting that T-DXd's outperforming either capecitabine or taxane-based therapy as a first chemotherapy in metastatic hormone receptor-positive breast cancer.



## PFS and OS in HER2-Ultralow: Prespecified Exploratory Analyses



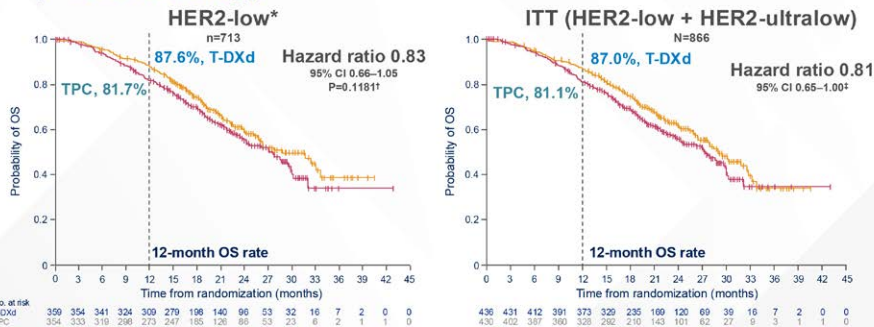
PFS improvement with T-DXd vs TPC in HER2-ultralow was consistent with results in HER2-low

▶ When looking at the ultralow patients, again, remember this is a small subset, it's only about 150 patients, and it was an exploratory endpoint, but you can see that the PFS looks really identical to the HER2-low population where, again, the PFS improved from about 8 to 13 months with a hazard ratio in this case that was around 0.8.



\*34.9% maturity (of total N for population) at this first interim analysis; median duration of follow up was 18.8 months.  
BICR, blinded independent central review; CI, confidence interval; HER2, human epidermal growth factor receptor 2; mPFS, (median) progression-free survival; OS, overall survival; PFS, progression-free survival; T-DXd, trastuzumab deruxtecan; TPC, chemotherapy treatment of physician's choice.  
Curigliano G, et al. ASCO 2024. Abstract LBA1000. Bardia A, et al. *N Engl J Med*. Published online September 14, 2024. doi:10.1056/NEJMoa2407086

## OS in HER2-Low and ITT: Key Secondary Endpoints (~40% Maturity)



20.1% of patients in the TPC group received T-DXd post treatment discontinuation (HER2-low)

17.9% of patients in the TPC group received T-DXd post treatment discontinuation (ITT)



†39.6% maturity (of total N for population) at this first interim analysis; median duration of follow up was 18.6 months (HER2-low). †P-value of <0.0046 required for statistical significance.  
†No test of significance was performed in line with the multiple testing procedure; median duration of follow up was 18.2 months (ITT).  
CI, confidence interval; HER2, human epidermal growth factor receptor 2; ITT, intent-to-treat; OS, overall survival; T-DXd, trastuzumab deruxtecan; TPC, chemotherapy treatment of physician's choice.  
Curigliano G, et al. ASCO 2024. Abstract LBA1000. Bardia A, et al. *N Engl J Med*. Published online September 14, 2024. doi:10.1056/NEJMoa2407086

▶ The overall survival data is still immature, and while numerically there is a trend towards T-DXd having a better OS, it is still not yet statistically significant with a hazard ratio of 0.83. So, these data from DESTINY-Breast06 really established T-DXd as a first-line chemotherapy option for patients with hormone receptor-positive HER2-low disease and potentially suggests also, this could be an option for those patients who have HER2-ultralow breast cancer as well.

## TROPiCS-02 Phase 3 trial: Expanding the Benefit of Sacituzumab Govitecan to HR+ Disease

**Metastatic or locally recurrent inoperable HR+/HER2- (IHC0, IHC1+, or IHC2+/ISH-) breast cancer that progressed after<sup>a,b</sup>:**

- At least 1 endocrine therapy, taxane, and CDK4/6 inhibitor in any setting
- At least 2, but no more than 4, lines of chemotherapy for metastatic disease
- Measurable disease by RECIST 1.1

N = 543

R  
1:1

Treatment was continued until progression or unacceptable toxicity

**Sacituzumab govitecan**  
10 mg/kg IV  
days 1 and 8, every 21 days  
n = 272

**Treatment of physician's choice<sup>c</sup>**  
(capecitabine, vinorelbine,  
gemcitabine, or eribulin)  
n = 271

**Stratification:**

- Visceral metastases (yes/no)
- Endocrine therapy in metastatic setting ≥ 6 months (yes/no)
- Prior lines of chemotherapies (2 vs 3/4)

**End points**

**Primary**

- PFS by BICR

**Secondary**

- OS
- ORR, DoR, CBR by LIR, and BICR
- PRO
- Safety

**Exploratory**

- OS by HER2 IHC status<sup>d</sup>

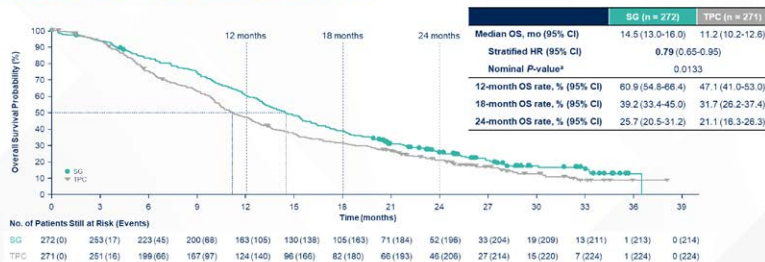
<sup>a</sup>ClinicalTrials.gov; NCT03611338. <sup>b</sup>Disease histology based on the ASCO/CAP criteria. <sup>c</sup>Single-agent standard-of-care treatment of physician's choice was specified prior to randomization by the investigator. <sup>d</sup>HER2-low was defined as IHC score of 1+, or score of 2+ with negative ISH result; HER2 IHC0 was defined as IHC score of 0. ASCO/CAP, American Society of Clinical Oncology/College of American Pathologists; BICR, blinded independent central review; CBR, clinical benefit rate; CDK, cyclin-dependent kinase; DoR, duration of response; HER2, human epidermal growth factor receptor 2; HR, hormone receptor; IHC, immunohistochemistry; ISH, in situ hybridization; IV, intravenously; LIR, local investigator review; ORR, objective response rate; OS, overall survival; PFS, progression-free survival; PRO, patient-reported outcomes; R, randomized; RECIST, Response Evaluation Criteria in Solid Tumors. Tolanev SM, et al. ASCO 2023. Abstract 1003. Ruigo HS, et al. Lancet. 2023;402(10411):1423-1433.

▶ We do, however, have other ADC options in addition to T-DXd for hormone receptor-positive breast cancer, and this includes sacituzumab govitecan. Sacituzumab govitecan is a TROP2-directed antibody-drug conjugate, so not targeting HER2 and has a TOPO1 payload, which is SN-38. This drug was studied in a more pretreated group of patients.

So, the TROPiCS-02 study looked at patients who had 2 to 4 prior lines of chemotherapy for metastatic hormone receptor-positive disease and randomized them to get sacituzumab or treatment of physician's choice chemotherapy.

## TROPiCS-02: OS

Median of 3 prior lines of chemo



SG continued to demonstrate improvement in OS vs TPC at longer follow-up, with 21% reduction in risk of death and a higher proportion of patients remaining alive at each landmark

▶ And found that sacituzumab was associated with a significant improvement in both progression-free survival as well as overall survival, with an OS hazard ratio of about 0.8.

And so, this really gives us another antibody-drug conjugate option for our patients with metastatic hormone receptor-positive breast cancer.

HR, hazard ratio; OS, overall survival; SG, sacituzumab govitecan; TPC, treatment of physician's choice. Tolanev SM, et al. ASCO 2023. Abstract 1003. Ruigo HS, et al. Lancet. 2023;402(10411):1423-1433.



## Impact of DESTINY-Breast06 on Treatment Sequencing



- 1L T-DXd preferred for patients with:
  - Symptomatic disease
  - Extensive visceral disease burden
  - Short PFS on AI+CDK4/6i
  - Relapse within 2 years on adjuvant endocrine therapy



1L, first line; AI, aromatase inhibitor; CDK, cyclin-dependent kinase; HER2, human epidermal growth factor receptor 2; HR, hormone receptor; MBC, metastatic breast cancer; PFS, progression-free survival; SG, sacituzumab govitecan; T-DXd, trastuzumab deruxtecan.

► And so, in my mind, I typically will think about treating patients who have HER2-ultralow or low metastatic hormone receptor-positive breast cancer with T-DXd either as their first chemotherapy based on the DESTINY-Breast06 results or as their second chemotherapy. And I make that decision about whether to use it first or second based on clinical characteristics.

And that does take into account understanding if they've had symptomatic disease, in which case maybe you want to turn to T-DXd given the higher response rate. Someone who has extensive visceral involvement. Someone

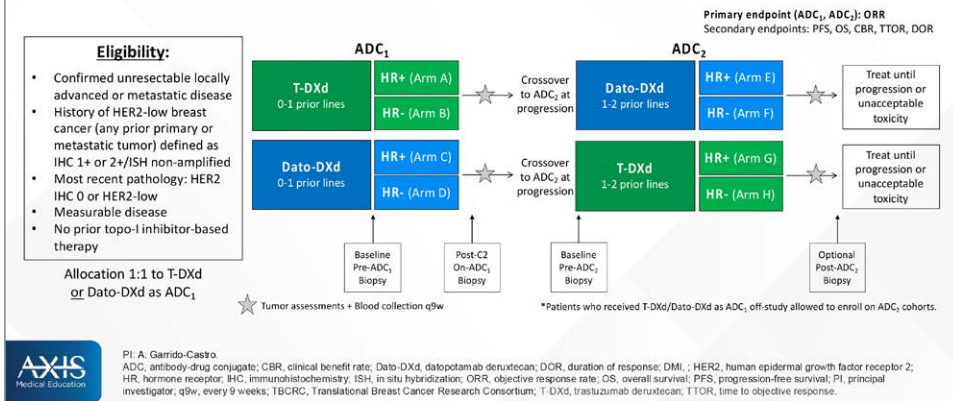
who maybe rapidly progressed through upfront AI/CDK in less than 6 months. For someone who had rapid recurrence on adjuvant endocrine therapy and then blew through their first lines of endocrine treatment quite quickly. Those are patients that I think have quite aggressive disease, and those are patients for whom I would really think about T-DXd as the first chemotherapy option for that patient.

And so, again, just to put this into framework for all of our options for hormone receptor-positive disease, we do have T-DXd also as a second-line option. Obviously, we saw that in DESTINY-Breast04, and we don't yet know if it matters if

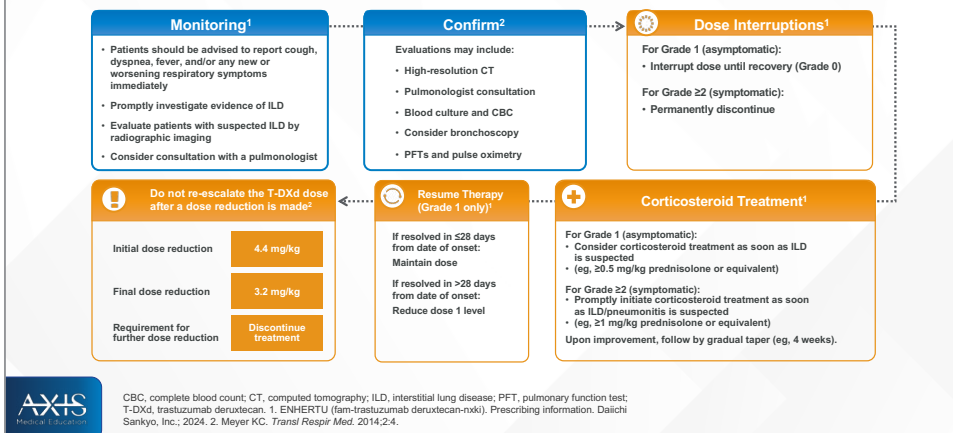
you give T-DXd first or second because we don't yet have mature overall survival data from DESTINY-Breast 06. And there are going to be lots of patients who cross over from the control arm in DB06 to get T-DXd. And so this could help us understand if it matters giving T-DXd earlier versus later. But for now, again, I would prioritize it for people with more aggressive disease. And then remember sacituzumab govitecan could be used subsequently to T-DXd, or if someone does not have any HER2 expression should be the first ADC that that patient receives based on TROPiCS-02.

## TBCRC 064: Treatment of ADC-Refractory Breast Cancer with Dato-DXd or T-DXd (TRADE-DXd)

Understanding how these ADCs work in sequence is going to take us more prospective data. There is a trial called TRADE-DXd, which should be activating soon, which will be sequencing T-DXd and DATO-DXd, either one first, then the other, or vice versa, and this will help us understand how these agents work one after the other.



## Management Strategies for ILD/Pneumonitis With T-DXd



So, while these ADCs have been highly effective, we do know that they can be associated with some toxicities that we do have to be mindful of. T-DXd, for example, has been associated with a risk of interstitial lung disease that's about 10 to 15%, and we have even seen a death rate of about 1% from ILD.

And so, it's really important to make all patients aware of this potential risk upfront, so I do warn patients that if they get any new upper respiratory-type symptoms,

they need to inform me right away. And if so, I would obtain a high-resolution chest CT to see if there are ground-glass changes that are present, suggestive of drug-induced ILD. You do need to rule out infectious etiologies because certainly this could be other things and not necessarily related to T-DXd. But, if in the end you conclude it is drug-related ILD, usually that means that if it's symptomatic you have to permanently discontinue T-DXd and treat with steroids. Whereas if you just happen to get a

scan for, let's say, restaging purposes, and the patient had no symptoms, but you saw ground-glass changes, that would be grade 1 ILD, in which case you would hold drug. I often do give steroids in that situation, though not mandatory, and then reimaging in 3 to 4 weeks, see if ground-glass changes have resolved. If so, then reinstate therapy, and I usually reinstate with a dose modification, particularly if it's been longer than 4 weeks since onset of that ILD.

## Potential Management Approaches for Neutropenia and Diarrhea With SG

- Neutropenia

- Withhold SG for ANC < 1500/mm<sup>3</sup> or neutropenic fever
- Monitor blood counts periodically during treatment
- Consider G-CSF for secondary prophylaxis
- Begin anti-infective treatment in patients with febrile neutropenia immediately

- Diarrhea

- Monitor patients and give fluids/electrolytes as needed
- Evaluate for infectious causes and if negative, begin loperamide
- For severe diarrhea, withhold SG until diarrhea is ≤ grade 1 and reduce subsequent doses



ANC, absolute neutrophil count; G-CSF, granulocyte colony stimulating factor; SG, sacituzumab govitecan. TRODELVY (sacituzumab govitecan-hziy). Prescribing information. Gilead Sciences; 2023.

▶ Sacituzumab, on the other hand, does have a high risk of neutropenia, and so it is important to carefully monitor neutrophil counts in these patients. If someone does have neutropenia, we do hold the sacituzumab, and then, when improved, reinstate therapy

either with a dose reduction or with utilization of growth factor.

There are some patients for whom you may think about prophylactic growth factor. For example, if someone had a required growth factor with

their prior therapy or has very low baseline counts, that may be someone you want to use upfront growth factor support. But otherwise, they typically will use drug and only initiate growth factor as needed.



## Key Takeaways

- The definition of HER2 status in mBC is evolving and HER2 heterogeneity is commonly observed
- Antibody-drug conjugates have changed the treatment landscape for metastatic HR+/HER2-expressing mBC
- We will need to better understand how to optimally select patients for ADC therapy, and in whom these agents can be effectively sequenced
- Monitoring and managing adverse events associated with ADCs are critical to achieving optimal patient outcomes



ADC, antibody-drug conjugate; HER2, human epidermal growth factor receptor 2; HR, hormone receptor; mBC, metastatic breast cancer.

▶ I did want to just conclude with a few key takeaways. So, I think the definition of HER2 expression in metastatic breast cancer has clearly evolved, and now it's really important to understand that we do need to know if someone has a little bit of HER2 expression. Before, we didn't really think about this if they weren't HER2-positive, but now we're going to even have to think about if they have HER2-ultralow expression. Important to remember that HER2 expression is dynamic. And so, you can re-biopsy

and sometimes find that the patient actually has some expression, so do think about that over time.

And while we do have choices for ADCs now, with both T-DXd and sacituzumab, this is likely to going to get more complex as there will be likely more ADCs that come into this space, such as datopotamab deruxtecan, in which case sequencing will get even more complicated. And so, I think we really do need better prospective studies to help us understand how

to optimally sequence these agents. And even, potentially more critically, how to manage toxicities is really very important for our patients, particularly with ILD risk for T-DXd. Really critical to be monitoring this carefully and to never re-expose someone who's had symptomatic ILD to T-DXd, again, given the potential risks that can associated with this.

With that, we will conclude today's activity, so thank you so much for your participation.

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AXIS Medical Education, Inc. is a full-service continuing education company that designs and implements live, web-based, and print-based educational activities for healthcare professionals. AXIS provides convenient opportunities to engage learners based on their individual learning preferences through a full spectrum of educational offerings.

The executive leadership of AXIS combines 75 years of experience in adult learning theory, curriculum design/implementation/assessment, continuing education accreditation standards, and medical meeting planning and logistics. Our team has a deep understanding of the governing guidelines overseeing the medical education industry to ensure compliant delivery of all activities.

AXIS employs an experienced team of medical and scientific experts, medical writers, project managers, meeting planners, and logistics professionals. This team is dedicated to meeting the unmet educational needs of healthcare professionals, with the goal of improving patient outcomes.

AXIS believes that partnerships are crucial in our mission to deliver timely, relevant, and high-quality medical education to healthcare professionals. To that end, AXIS partners with other organizations and accredited providers to offer added expertise and assist in expanding access to our educational interventions. AXIS also partners with numerous patient advocacy organizations to provide recommended patient education and caregiver resources in specific disease areas. AXIS finds value in these partnerships because they complement our core clinical curriculum with validated and relevant supplemental resources for busy clinicians and their patients.

The mission of AXIS is to enhance the knowledge, skills, competence, and performance of the interprofessional healthcare team to ensure patients receive quality care, resulting in improved patient outcomes. We engage healthcare professionals in fair-balanced, scientifically rigorous, expert-led certified educational activities designed to foster lifelong learning that is applicable to clinical practice and patient-centered care.

To learn more and to see our current educational offerings, visit us online at [www.AXISMedEd.com](http://www.AXISMedEd.com).

