Poster 145P

Trastuzumab deruxtecan in pretreated patients with HER2-expressing solid tumors: exploratory biomarker analysis of DESTINY-PanTumor02 Part 1

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Objective

- To explore the relationship between oncogenic biomarkers and clinical outcomes in pretreated patients with locally advanced, metastatic, or unresectable solid tumors who had received trastuzumab deruxtecan (T-DXd) in Part 1 of DESTINY-PanTumor02

Conclusions

- T-DXd showed clinically meaningful antitumor activity across multiple biomarker subgroups
  - Consistent with the primary analysis,[1] the greatest antitumor activity was observed for patients with human epidermal growth factor receptor 2 (HER2) immunohistochemistry (IHC) 3+ tumors by central testing
  - The lowest confirmed investigator-assessed objective response rate (ORR; 11.6%) was observed for patients with *H/K/NRAS* alterations; however, this subgroup had a limited sample size (n=43) and included only four (9.3%) patients with HER2 IHC 3+ tumors (by central testing)
- Overall, these data further demonstrate clinically meaningful activity for T-DXd in HER2-expressing tumors and HER2 IHC 3+ expression as the greatest predictor of treatment response, consistent with treatment recommendations for T-DXd in pretreated patients with HER2-positive (IHC 3+) tumors[2–4]

Plain language summary

Why did we perform this research?

Trastuzumab deruxtecan (T-DXd) is an antibody-drug conjugate, which is a chemotherapy with a linker (together called deruxtecan) joined to an antibody (trastuzumab). T-DXd binds to human epidermal growth factor receptor 2 (HER2) on the surface of cancer cells. Once inside the cell, it releases the chemotherapy to kill these cells.[1,2] Based, in part, on results from Part 1 of the DESTINY-PanTumor02 clinical study,[3] T-DXd is a recommended treatment in multiple countries for people with solid tumors that have the highest level of HER2 (HER2-positive, also known as immunohistochemistry [IHC] 3+) that have spread or cannot be completely removed with surgery and who have received prior systemic treatment and/or have no satisfactory alternative treatment options available.[4–6] People with advanced cancer can have different tumor features. Studying how tumors with different characteristics respond to T-DXd may help better understand which patients are most likely to benefit from the treatment.

How did we perform this research?

Using final data from DESTINY-PanTumor02 Part 1, this analysis determined which participants had changes in genes linked to cancer (*HER2, BRCA1/2, PIK3CA, H/K/NRAS*) by analyzing DNA from their tumors that was found in their blood (called circulating tumor DNA). Response to T-DXd treatment was evaluated according to the presence or absence of specific markers of disease that were identified in the circulating tumor DNA of the participants.

What were the findings of this research?

Circulating tumor DNA was evaluated in 260 out of 267 participants with solid tumors in the study. Consistent with previous results,[3] those with HER2 IHC 3+ tumors had the greatest response to T-DXd treatment compared with the other tumor characteristics evaluated. Treatment responses were observed across all subgroups of participants.

What are the implications of this research?

These findings further support the use of T-DXd as a treatment for people with HER2-positive (IHC 3+) solid tumors that have spread or cannot be completely removed with surgery and who have received prior systemic treatment and/or have no satisfactory alternative treatment options available. However, owing to the low number of participants present across the subgroups, more research is needed to better understand how changes in cancer-associated genes might affect T-DXd treatment response.

Where can I access more information?

For information about DESTINY-PanTumor02, please visit www.clinicaltrials.gov/study/NCT04482309. Primary results from DESTINY-PanTumor02 Part 1 have been published in the *Journal of Clinical Oncology* at https://ascopubs.org/doi/10.1200/JCO.23.02005. You can also speak to your doctor about this and other clinical studies.

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Poster presented at ESMO 2025 by Professor Do-Youn Oh. Corresponding author email address: ohdoyoun@snu.ac.kr

Introduction

- T-DXd is a HER2-directed antibody-drug conjugate that is approved in multiple countries for the treatment of HER2-positive, HER2-low, and HER2-ultralow breast cancer; HER2-positive gastric or gastroesophageal junction adenocarcinoma; and *HER2*-mutant non-small cell lung cancer[2–5]
- Primary results (data cutoff June 8, 2023) from Part 1 of the open-label, Phase 2 DESTINY-PanTumor02 study demonstrated durable and clinically meaningful antitumor activity of T-DXd in pretreated patients with advanced HER2-expressing solid tumors[1]
  - In all patients, investigator-assessed confirmed ORR was 37.1% (95% confidence interval [CI] 31.3, 43.2), investigator-assessed median progression-free survival (PFS) was 6.9 months (95% CI 5.6, 8.0), and median overall survival (OS) was 13.4 months (95% CI 11.9, 15.5)[1]
  - The greatest benefit was observed in patients with HER2 IHC 3+ tumors by central testing (investigator-assessed confirmed ORR 61.3% [95% CI 49.4, 72.4]; investigator-assessed median PFS 11.9 months [95% CI 8.2, 13.0])[1]
- Based, in part, on results from Part 1 of DESTINY-PanTumor02, T-DXd has also been approved in multiple countries for the treatment of adult patients with unresectable or metastatic HER2-positive (IHC 3+) solid tumors who have received prior treatment and/or have no satisfactory alternative therapies[2–4]
- Given the diverse clinical characteristics of patients with advanced solid tumors, this poster explores the relationship between oncogenic biomarkers and clinical outcomes in patients from DESTINY-PanTumor02 Part 1

Methods

Patient population

- Adults (aged ≥18 years) with histologically confirmed locally advanced, metastatic, or unresectable solid tumors (excluding breast, colorectal, gastric, and non-small cell lung cancer)
- Disease progression following ≥1 prior systemic treatment or without alternative treatment options
  - Prior HER2-directed therapy was allowed
- HER2-expressing tumors with IHC 3+/2+ scored using current American Society of Clinical Oncology / College of American Pathology guidelines for scoring HER2 in gastric cancer[6]
- HER2 expression at enrollment was based on local testing; however, if local testing was not available, enrollment was determined by central HER2 testing

Testing methods

- Prospective and retrospective central HER2 testing was performed using HER2 HercepTest (DAKO) and scored according to gastric-specific criteria[6]
- *HER2* amplification by in situ hybridization (ISH) was evaluated centrally using the VENTANA HER2 Dual ISH (Roche) assay
- Oncogenic or likely oncogenic gene alterations, defined by OncoKB,[7,8] in baseline circulating tumor DNA (ctDNA) were detected using the GuardantOMNI assay (Guardant Health)
- Tumor mutational burden was determined according to Guardant Health criteria, as described by Si et al.[9]

Study type

Open label, multicenter, multicohort, Phase 2

Treatment

T-DXd 5.4 mg/kg IV Q3W

Trial registration

NCT04482309

Data cutoff

October 10, 2024

Endpoints

Primary:
- Confirmed ORR*

Secondary:
- DOR*
- DCR*
- PFS*
- OS
- Safety and tolerability

Exploratory:
- Subgroup analyses by HER2 status
- Subgroup analyses by biomarker status

*Per RECIST 1.1, as assessed by investigator
DCR, disease control rate; DOR, duration of response; HER2, human epidermal growth factor receptor 2; IV, intravenous; ORR, objective response rate; OS, overall survival; PFS, progression-free survival; Q3W, every 3 weeks; RECIST, Response Evaluation Criteria in Solid Tumours; T-DXd, trastuzumab deruxtecan

Results

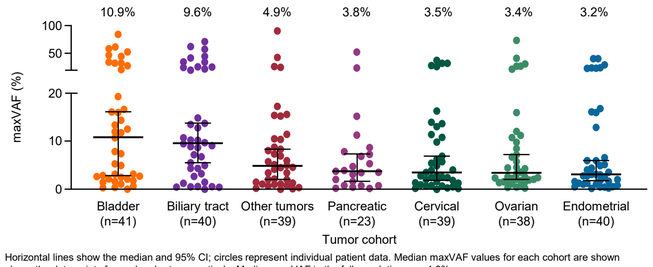
- At final data cutoff (October 10, 2024), 267 patients across seven tumor cohorts (biliary tract [n=41], bladder [n=41], cervical [n=40], endometrial [n=40], ovarian [n=40], pancreatic [n=25], and other tumors [n=40]) had received T-DXd
  - Of the 260 (97.4%) patients with evaluable baseline ctDNA, 28.1% had HER2 IHC 3+ and 46.5% had IHC 2+ tumors by central testing (**Table 1**)
- Median (range) follow up for all patients was 12.98 (0.4–47.7) months

Table 1. Baseline demographics and clinical characteristics

	All patients (N=267)	ctDNA evaluable (n=260)	ctDNA not evaluable (n=7)*
Median age, years (range)	62 (23–85)	62 (23–85)	48 (30–68)
Sex, female, n (%)	178 (66.7)	173 (66.5)	5 (71.4)
Race, n (%)			
White	163 (61.0)	160 (61.5)	3 (42.9)
Black or African American	6 (2.2)	6 (2.3)	0
Asian	87 (32.6)	84 (32.3)	3 (42.9)
Other	6 (2.2)	5 (1.9)	1 (14.3)
Not reported	5 (1.9)	5 (1.9)	0
ECOG performance status, n (%)†			
0	126 (47.2)	121 (46.5)	5 (71.4)
1	140 (52.4)	138 (53.1)	2 (28.6)
No. of prior lines of therapy, n (%)			
0	3 (1.1)	3 (1.2)	0
1	71 (26.6)	68 (26.2)	3 (42.9)
2	84 (31.5)	82 (31.5)	2 (28.6)
3	55 (20.6)	55 (21.2)	0
4	21 (7.9)	19 (7.3)	2 (28.6)
≥5	33 (12.4)	33 (12.7)	0
HER2 testing at enrollment, n (%)			
Local	202 (75.7)	197 (75.8)	5 (71.4)
Central	65 (24.3)	63 (24.2)	2 (28.6)
HER2 IHC status at enrollment, n (%)			
IHC 3+	111 (41.6)	107 (41.2)	4 (57.1)
IHC 2+	151 (56.6)	148 (56.9)	3 (42.9)
IHC 1+	5 (1.9)	5 (1.9)	0
HER2 IHC status by central testing, n (%)			
IHC 3+	75 (28.1)	73 (28.1)	2 (28.6)
IHC 2+	125 (46.8)	121 (46.5)	4 (57.1)
IHC 1+	25 (9.4)	25 (9.6)	0
IHC 0	30 (11.2)	29 (11.2)	1 (14.3)
Unknown	12 (4.5)	12 (4.6)	0
Tissue *HER2* amplification, n (%)			
ISH+	78 (29.2)	76 (29.2)	2 (28.6)
ISH−	145 (54.3)	143 (55.0)	2 (28.6)
ISH unknown	44 (16.5)	41 (15.8)	3 (42.9)
Plasma *HER2* amplification, n (%)‡			
Detected	48 (18.0)	48 (18.5)	–
ND	212 (79.4)	212 (81.5)	–
***HER2* mutations, n (%)‡**			
Detected	19 (7.1)	19 (7.3)	–
ND	241 (90.3)	241 (92.7)	–
***PIK3CA* alterations, n (%)‡**			
Detected	46 (17.2)	46 (17.7)	–
ND	214 (80.1)	214 (82.3)	–
***H/K/NRAS* alterations, n (%)‡**			
Detected	43 (16.1)	43 (16.5)	–
ND	217 (81.3)	217 (83.5)	–
***BRCA1/2* mutations, n (%)‡**			
Detected	23 (8.6)	23 (8.8)	–
ND	237 (88.8)	237 (91.2)	–

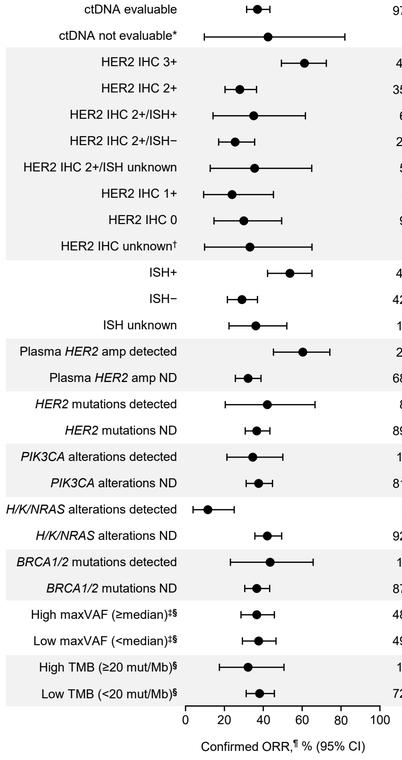
*Five patients were considered to have non-shedding tumors (no mutations detected, very low-frequency mutations, or only variants of uncertain significance detected), and the remaining two patients were not profiled; †one patient with ovarian cancer (included in the ctDNA evaluable set) had an ECOG performance score of 2; ‡detected in ctDNA
ctDNA, circulating tumor DNA; ECOG, Eastern Cooperative Oncology Group; HER2, human epidermal growth factor receptor 2; IHC, immunohistochemistry; ND, not detected

Figure 1. maxVAF by tumor cohort



Horizontal lines show the median and 95% CI; circles represent individual patient data. Median maxVAF values for each cohort are shown above the data points for each cohort, respectively. Median maxVAF in the full population was 4.8%
CI, confidence interval; maxVAF, maximum variant allele frequency

Figure 2. Investigator-assessed confirmed ORRs by HER2 status and biomarker subgroups



	Response/total, n/n (%) [95% CI]	HER2 IHC 3+ tumors, n/N (%)
All patients	100/267 (37.5) [31.6, 43.6]	75/267 (28.1)
ctDNA evaluable	97/260 (37.3) [31.4, 43.5]	73/260 (28.1)
ctDNA not evaluable*	3/7 (42.9) [9.9, 81.6]	2/7 (28.6)
HER2 IHC 3+	46/75 (61.3) [49.4, 72.4]	75/75 (100)
HER2 IHC 2+	35/125 (28.0) [20.3, 36.7]	0/125 (0)
HER2 IHC 2+/ISH+	6/17 (35.3) [14.2, 61.7]	0/17 (0)
HER2 IHC 2+/ISH−	24/94 (25.5) [17.1, 35.6]	0/94 (0)
HER2 IHC 2+/ISH unknown	5/14 (35.7) [12.8, 64.9]	0/14 (0)
HER2 IHC 1+	6/25 (24.0) [9.4, 45.1]	0/25 (0)
HER2 IHC 0	9/30 (30.0) [14.7, 49.4]	0/30 (0)
HER2 IHC unknown†	4/12 (33.3) [9.9, 65.1]	0/12 (0)
ISH+	42/78 (53.8) [42.2, 65.2]	53/78 (67.9)
ISH−	42/145 (29.0) [21.7, 37.1]	15/145 (10.3)
ISH unknown	16/44 (36.4) [22.4, 52.2]	7/44 (15.9)
Plasma *HER2* amp detected	29/48 (60.4) [45.3, 74.2]	30/48 (62.5)
Plasma *HER2* amp ND	68/212 (32.1) [25.8, 38.8]	43/212 (20.3)
HER2 mutations detected	8/19 (42.1) [20.3, 66.5]	10/19 (52.6)
HER2 mutations ND	89/241 (36.9) [30.8, 43.4]	63/241 (26.1)
PIK3CA alterations detected	16/46 (34.8) [21.4, 50.2]	10/46 (21.7)
PIK3CA alterations ND	81/214 (37.9) [31.3, 44.7]	63/214 (29.4)
H/K/NRAS alterations detected	5/43 (11.6) [3.9, 25.1]	4/43 (9.3)
H/K/NRAS alterations ND	92/217 (42.4) [35.7, 49.3]	69/217 (31.8)
BRCA1/2 mutations detected	10/23 (43.5) [23.2, 65.5]	5/23 (21.7)
BRCA1/2 mutations ND	87/237 (36.7) [30.6, 43.2]	68/237 (28.7)
High maxVAF (≥median)‡§	48/130 (36.9) [28.6, 45.8]	37/130 (28.5)
Low maxVAF (<median)‡§	49/130 (37.7) [29.3, 46.6]	36/130 (27.7)
High TMB (≥20 mut/Mb)§	11/34 (32.4) [17.4, 50.5]	12/34 (35.3)
Low TMB (<20 mut/Mb)§	72/188 (38.3) [31.3, 45.7]	54/188 (28.7)

HER2 status was determined by central testing
*Five patients were considered to have non-shedding tumors (no mutations detected, very low-frequency mutations, or only variants of uncertain significance detected), and the remaining two patients were not profiled; †unknown central IHC test results include patients whose samples were unevaluable (for various technical reasons) and may include patients for whom a sample was not provided for central testing; ‡median maxVAF = 4.8%; §not adjusted by ctDNA abundance; ¶per RECIST 1.1
Amp, amplification; CI, confidence interval; ctDNA, circulating tumor DNA; HER2, human epidermal growth factor receptor 2; IHC, immunohistochemistry; ISH, in situ hybridization; maxVAF, maximum variant allele frequency; ND, not detected; ORR, objective response rate; RECIST, Response Evaluation Criteria in Solid Tumours; TMB, tumor mutational burden

- Biliary tract and bladder tumors showed the highest amount of shedding, with median (range) maximum variant allele frequency (maxVAF) values of 10.9% (0–85.1) in the bladder cohort and 9.6% (0–71.7) in the biliary tract cohort (**Figure 1**), which is generally consistent with published data[10,11]
- Investigator-assessed confirmed objective responses were observed across biomarker subgroups (**Figure 2**)
  - Although the presence of HER2-based biomarkers (by ctDNA analysis, ISH, or IHC) is associated with favorable ORRs, the highest response rates were observed for patients with HER2 IHC 3+ tumors by central testing
- Across all patients (N=267), investigator-assessed median progression-free survival (PFS; 95% CI) and median overall survival (OS; 95% CI) was 6.9 months (5.6, 8.0) and 13.4 months (11.9, 15.3), respectively
  - Survival outcomes by ctDNA biomarker status are shown in **Table 2**
- The lowest confirmed investigator-assessed ORR, investigator-assessed median PFS, and median OS was observed for patients with detected *H/K/NRAS* alterations (n=43); only four (9.3%) patients with detected *H/K/NRAS* alterations had HER2 IHC 3+ tumors (by central testing)

Table 2. Survival outcomes by ctDNA biomarker status

Characteristic		n	Median PFS,* months (95% CI)	Median OS, months (95% CI)
ctDNA	Evaluable	260	6.8 (5.5, 7.6)	13.4 (11.9, 15.1)
	Not evaluable†	7	17.4 (3.2, NE)	31.2 (4.0, NE)
Plasma *HER2* amplification	Detected	48	12.5 (6.9, 22.3)	21.3 (12.4, 26.5)
	ND	212	5.6 (4.8, 7.0)	12.7 (11.2, 13.8)
***HER2* mutations**	Detected	19	6.7 (3.5, 11.9)	13.5 (6.6, 19.3)
	ND	241	6.9 (5.4, 7.6)	13.4 (11.7, 15.5)
***PIK3CA* alterations**	Detected	46	4.6 (4.0, 8.8)	11.5 (7.6, 17.1)
	ND	214	6.9 (5.6, 8.0)	13.5 (12.2, 15.7)
***BRCA1/2* mutations**	Detected	23	5.3 (2.8, 8.2)	12.0 (6.2, 14.8)
	ND	237	6.9 (5.6, 8.0)	13.5 (11.9, 15.9)
***H/K/NRAS* alterations**	Detected	43	2.8 (1.5, 3.9)	4.9 (3.7, 11.2)
	ND	217	7.1 (6.3, 8.3)	14.2 (12.7, 17.2)
maxVAF‡	High (≥median)§	130	5.5 (4.2, 7.0)	11.7 (8.8, 13.2)
	Low (<median)§	130	7.6 (6.0, 9.7)	16.0 (13.4, 19.9)
TMB‡	High (≥20 mut/Mb)	34	6.0 (4.2, 7.1)	12.3 (6.0, 14.9)
	Low (<20 mut/Mb)	188	5.7 (4.7, 8.0)	12.6 (11.1, 14.6)
	Not evaluable¶	38	8.5 (7.0, 11.3)	21.9 (13.6, 25.3)

PFS defined as time from first dose until date of objective disease progression or death, regardless of discontinuation of treatment or receipt of another cancer therapy. OS defined as time from date of first dose until death due to any cause. *Assessed by the investigator per RECIST 1.1; †five patients were considered to have non-shedding tumors (no mutations detected, very low-frequency mutations, or only variants of uncertain significance detected), and the remaining two patients were not profiled; ‡not adjusted by ctDNA abundance; §median maxVAF = 4.8%; ¶based on Guardant Health criteria[9]
CI, confidence interval; ctDNA, circulating tumor DNA; maxVAF, maximum variant allele frequency; mut, mutations; ND, not detected; NE, not evaluable; OS, overall survival; PFS, progression-free survival; RECIST, Response Evaluation Criteria in Solid Tumours; TMB, tumor mutational burden

Limitations

- Interpretation of results across subgroups was limited by sample sizes and range of tumor types included in the study
- Results may be confounded by the heterogeneity in patient characteristics between subgroups, which may affect treatment response
  - In particular, the distribution of patients with HER2 IHC 3+ tumors across subgroups may confound interpretation of ORRs
- Plasma and tissue samples were not collected at the same time; changes in disease or potential treatment received between sample collection should be considered when interpreting these findings
- Owing to a lack of archival tissue samples available for next-generation sequencing, it could not be confirmed whether alterations observed in baseline ctDNA co-exist with HER2 IHC/ISH status within the same tumor sample
- Low-shedding tumors may limit the ability to detect copy number alterations in ctDNA

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Disclosures

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