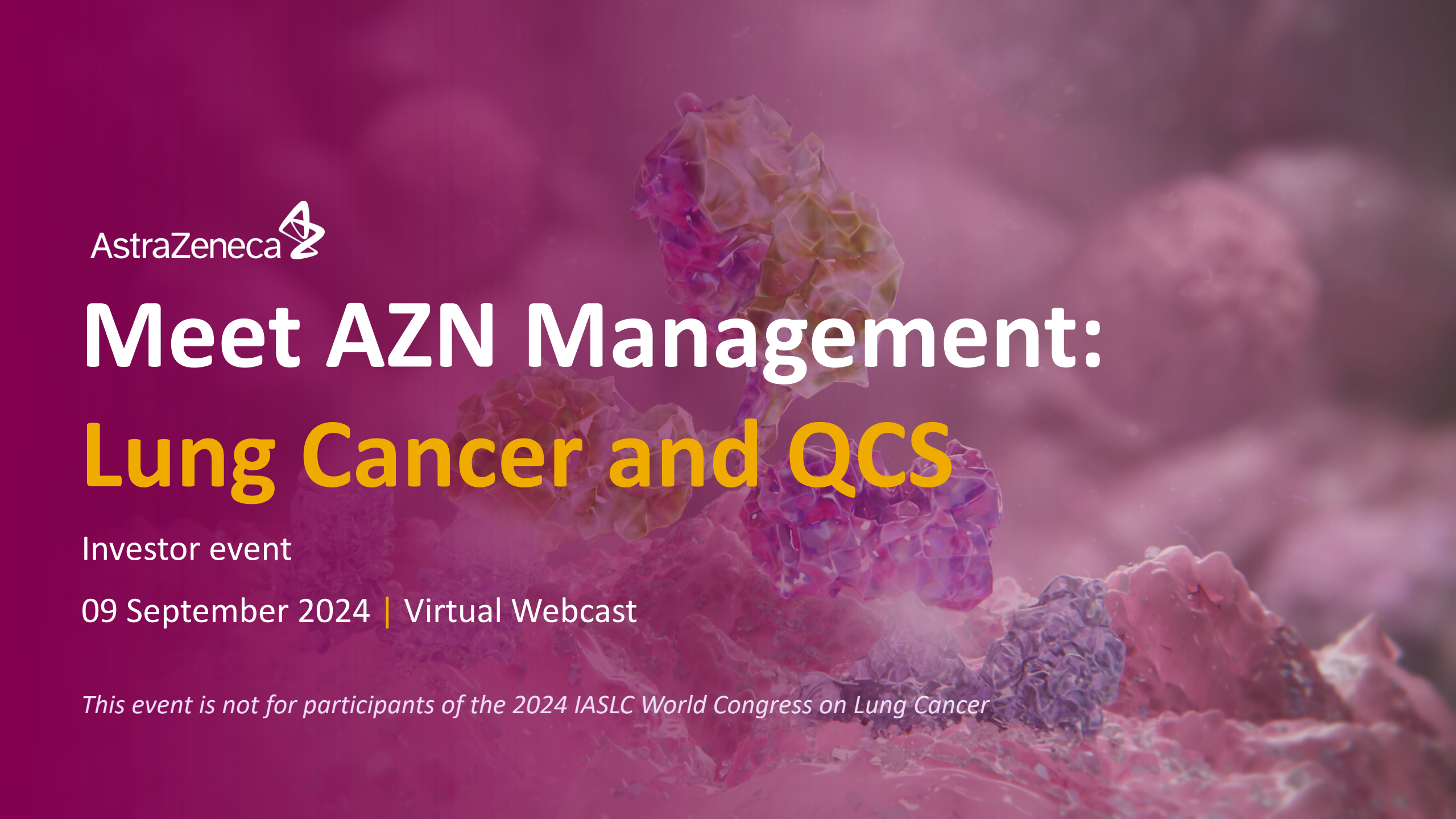

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Meet AZN Management:

Lung Cancer and QCS

Investor event

09 September 2024 | Virtual Webcast

This event is not for participants of the 2024 IASLC World Congress on Lung Cancer

Forward-looking statements

In order, among other things, to utilise the 'safe harbour' provisions of the US Private Securities Litigation Reform Act of 1995, AstraZeneca (hereafter 'the Group') provides the following cautionary statement: This document contains certain forward-looking statements with respect to the operations, performance and financial condition of the Group, including, among other things, statements about expected revenues, margins, earnings per share or other financial or other measures. Although the Group believes its expectations are based on reasonable assumptions, any forward-looking statements, by their very nature, involve risks and uncertainties and may be influenced by factors that could cause actual outcomes and results to be materially different from those predicted. The forward-looking statements reflect knowledge and information available at the date of preparation of this document and the Group undertakes no obligation to update these forward-looking statements. The Group identifies the forward-looking statements by using the words 'anticipates', 'believes', 'expects', 'intends' and similar expressions in such statements. Important factors that could cause actual results to differ materially from those contained in forward-looking statements, certain of which are beyond the Group's control, include, among other things: the risk of failure or delay in delivery of pipeline or launch of new medicines; the risk of failure to meet regulatory or ethical requirements for medicine development or approval; the risk of failures or delays in the quality or execution of the Group's commercial strategies; the risk of pricing, affordability, access and competitive pressures; the risk of failure to maintain supply of compliant, quality medicines; the risk of illegal trade in the Group's medicines; the impact of reliance on third-party goods and services; the risk of failure in information technology or cybersecurity; the risk of failure of critical processes; the risk of failure to collect and manage data in line with legal and regulatory requirements and strategic objectives; the risk of failure to attract, develop, engage and retain a diverse, talented and capable workforce; the risk of failure to meet regulatory or ethical expectations on environmental impact, including climate change; the risk of the safety and efficacy of marketed medicines being questioned; the risk of adverse outcome of litigation and/or governmental investigations; intellectual property-related risks to the Group's products; the risk of failure to achieve strategic plans or meet targets or expectations; the risk of failure in financial control or the occurrence of fraud; the risk of unexpected deterioration in the Group's financial position; the impact that global and/or geopolitical events may have or continue to have on these risks, on the Group's ability to continue to mitigate these risks, and on the Group's operations, financial results or financial condition. Nothing in this document, or any related presentation/webcast, should be construed as a profit forecast.

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Investor Deep Dive | Lung Cancer and QCS

Agenda

AstraZeneca ambition in Lung Cancer	-	**Dave Fredrickson**, *EVP, Oncology Business*
Advancing ADC leadership | Improving patient selection	-	**Susan Galbraith**, *EVP, Oncology R&D*
Leading in next-generation IO	-	**Susan Galbraith**, *EVP, Oncology R&D*
Summary and concluding remarks	-	**Dave Fredrickson**, *EVP, Oncology Business*
Q&A session		

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AstraZeneca in Lung Cancer

Speakers and panelists

Dave Fredrickson
EXECUTIVE VICE PRESIDENT,
ONCOLOGY BUSINESS

Susan Galbraith
EXECUTIVE VICE PRESIDENT,
ONCOLOGY R&D

Q&A only

Sunil Verma
SVP, GLOBAL HEAD, ONCOLOGY
FRANCHISE

Matt Hellmann
VP, EARLY ONCOLOGY DEVELOPMENT

Leora Horn
VP, LATE CLINICAL DEVELOPMENT
AND GLOBAL CLINICAL STRATEGY
LEAD, LUNG CANCER

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AstraZeneca ambition in Lung Cancer

Dave Fredrickson

Executive Vice President, Oncology Business

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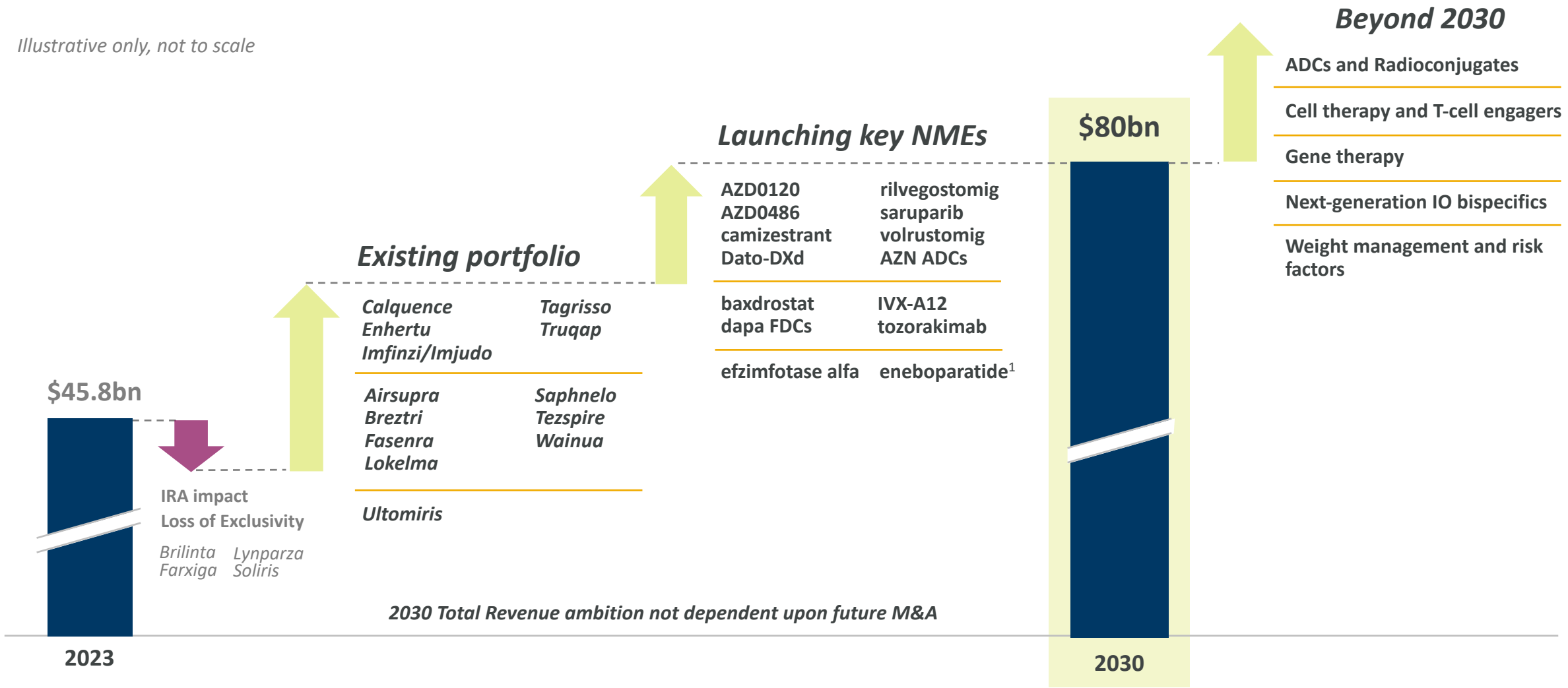
Ambition – $80bn Total Revenue by 2030 & sustained 2030+ growth

Working on “today, tomorrow and the day after"



Note: Ambition to achieve $80bn in Total Revenue by 2030 is risk-adjusted, based on latest long-range plan – see slide 3 for details.
Medicines and assets listed reflect key contributors to 2030 Total Revenue ambition; however, this list is not exhaustive. Medicines and assets listed in alphabetical order and sorted by therapy area.
1. Amolyt Pharma acquisition remains subject to customary external clearances; all clinical development plans mentioned herein subject to deal closure.
Collaboration partners: Daiichi Sankyo (*Enhertu*, Dato-DXd), Amgen (*Tezspire*), Ionis (*Wainua*), Compugen (rilvegostomig), Merck & Co., Inc. (*Lynparza).*

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Pioneering in lung cancer – important step in achieving our strategic ambitions

Established leadership in early- and late-stage lung cancer

TAGRISSO®
Transforming care in all stages of *EGFR*m NSCLC

IMFINZI®
Enabling benefit across both early and late NSCLC and SCLC

IMJUDO™
Deepening IO benefit in metastatic NSCLC

ENHERTU®
First targeted option for patients with HER2 mutations or overexpression

NMEs in late-stage development

Investigating novel modalities and combinations

Dato-DXd (TROP2 ADC)	Replace conventional chemotherapy
volrustomig (PD-1/CTLA-4) **rilvegostomig** (PD-1/TIGIT)	Deepen response vs traditional checkpoint inhibitors with potential for extended survival
ceralasertib (ATR inhibitor)	Address IO resistance

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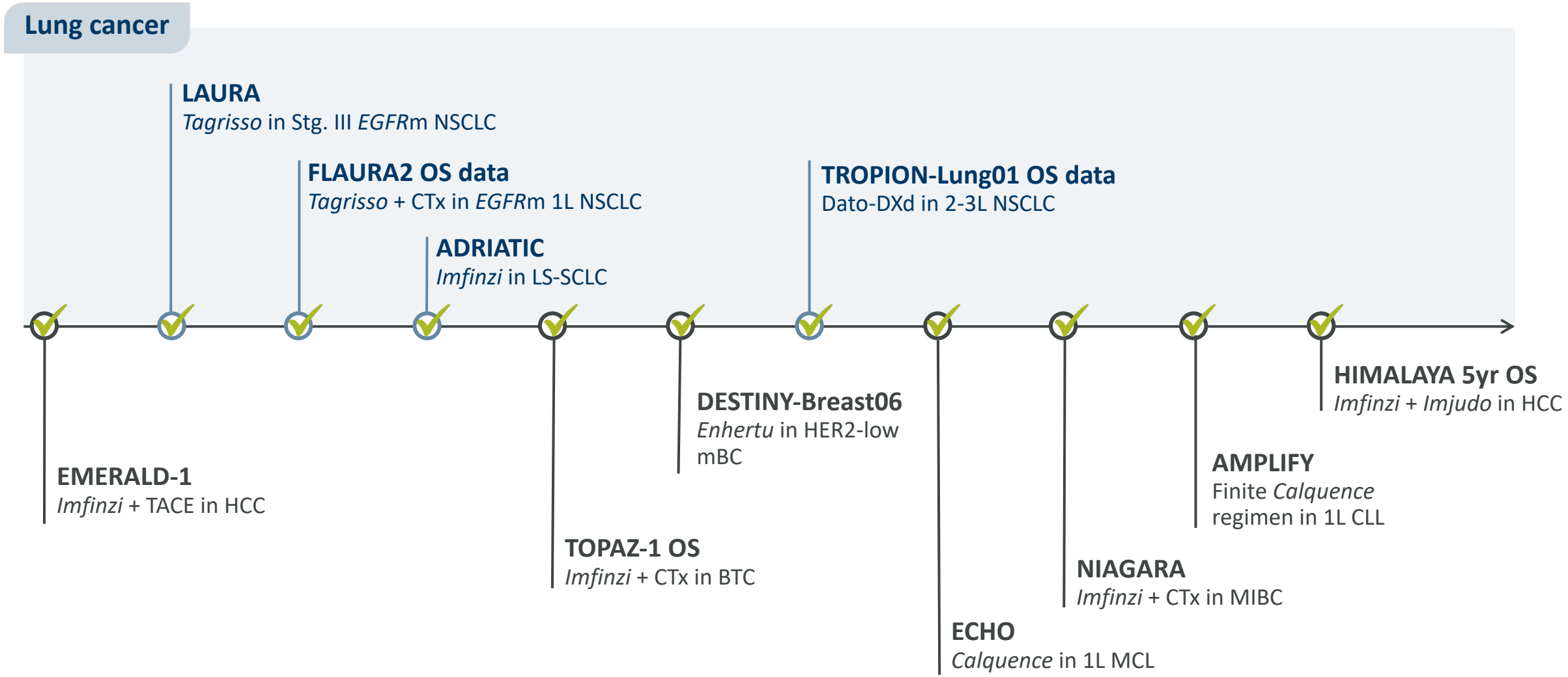
Strong Phase III pipeline momentum already in 2024



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Advancing ADC leadership

Susan Galbraith

Executive Vice President, Oncology R&D

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Dato-DXd strategy in lung cancer

1

Demonstrate efficacy over chemotherapy in late-line setting

- **TROPION-Lung01** Phase III improved PFS in 2-3L NSCLC vs docetaxel[1]

2

Advance novel combinations across early and 1L advanced setting

- Phase II **NeoCOAST-2** IO early-stage combination[2], newly announced **TROPION-Lung12**
- Phase III **AVANZAR, TROPION-Lung07, -08, -10** ongoing in 1L mNSCLC

3

Build on strong evidence in *EGFR*m with *Tagrisso* combination approach

- Phase II **TROPION-Lung05** in AGA[3]
- Phase III **TROPION-Lung14, -15** ongoing, HLR >2025

4

Improve patient selection with QCS technology

- **QCS-NMR predictive for PFS** in TROPION-Lung01[4]
- Differential expression by histology

1. Ahn M-J, et al. Abstract 509MO presented at the European Society of Medical Oncology 2023. 2. Cascone T et al. Abstract #PL02.07 presented at the 2024 World Conference on Lung Cancer.
3. Kitazono S et al. Abstract 518MO presented at the European Society of Medical Oncology 2023. 4. Garassino MC et al. Abstract #PL02.11 presented at the 2024 World Conference on Lung Cancer.
Collaboration partner: Daiichi Sankyo (Dato-DXd).

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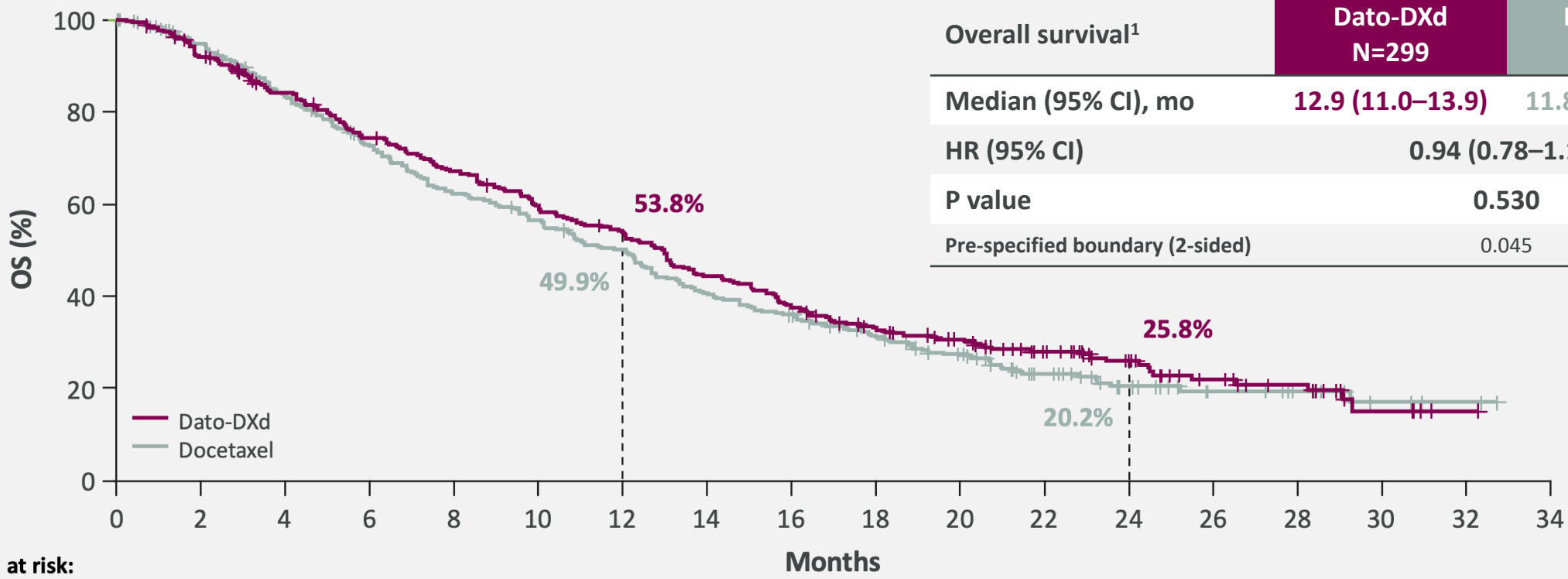
Survival results numerically favoured Dato-DXd in overall population but did not reach statistical significance



Overall survival[1]	Dato-DXd N=299	Docetaxel N=305
Median (95% CI), mo	12.9 (11.0–13.9)	11.8 (10.0–12.8)
HR (95% CI)	0.94 (0.78–1.14)	
P value	0.530	
Pre-specified boundary (2-sided)	0.045	

No. at risk:

Dato-DXd	299	272	242	213	190	168	151	124	106	84	71	51	35	22	16	5	1	0
Docetaxel	305	273	239	205	175	157	138	112	98	81	63	41	26	15	11	4	2	0

1. Median (95% CI) OS follow-up was 23.1 (22.0, 24.8) months for Dato-DXd and 23.1 (21.7, 24.2) months for docetaxel. At primary OS analysis (data cutoff: March 1, 2024), 433 OS events (IF) were observed.
Sands J et al. Abstract #OA08.03 presented at the 2024 World Conference on Lung Cancer.
Collaboration partner: Daiichi Sankyo (Dato-DXd).

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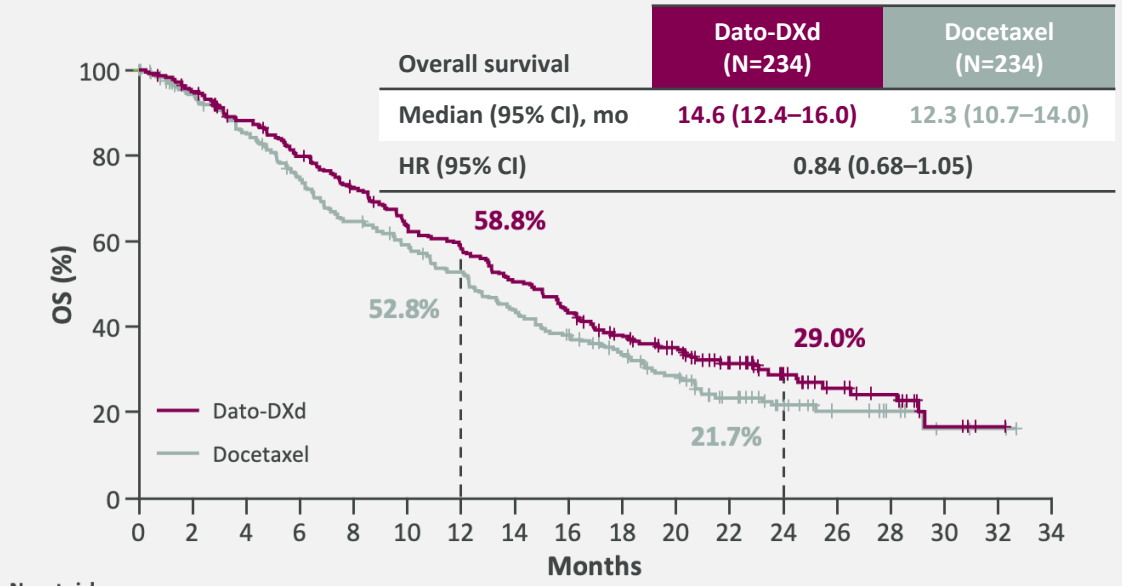
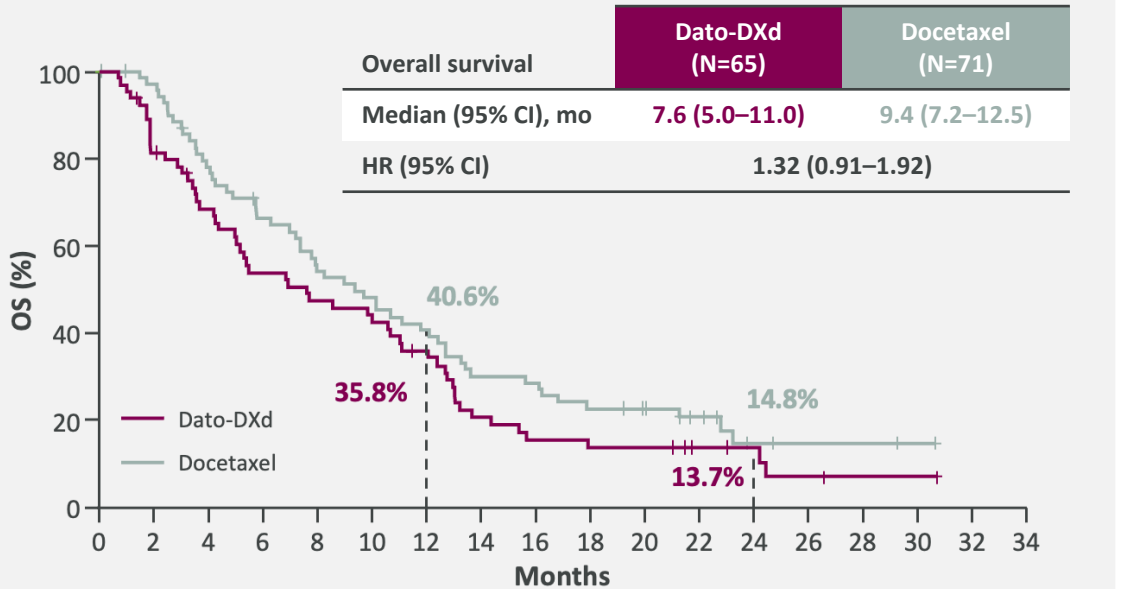
Clinically meaningful OS improvement seen in NSQ population

Overall survival in non-squamous population



Overall survival in squamous population



- No new adjudicated drug-related ILD events or deaths occurred since the PFS database lock. Compelling benefit:risk profile in non-squamous population with 1.7% rate of ILD related death (n=4/232) compared with squamous population (4.6% ILD related deaths [n=3/65])[2]

Data cutoff: March 1, 2024. 1. Based on the number of patients in the respective actionable genomic alteration subsets. Values were calculated based on patient data in the electronic case report forms.

1. Sands J et al. Abstract #OA08.03 presented at the 2024 World Conference on Lung Cancer. 2. Ahn M-J, et al. Abstract 509MO presented at the European Society of Medical Oncology 2023

Collaboration partner: Daiichi Sankyo (Dato-DXd).

Improving patient selection

Susan Galbraith

Executive Vice President, Oncology R&D

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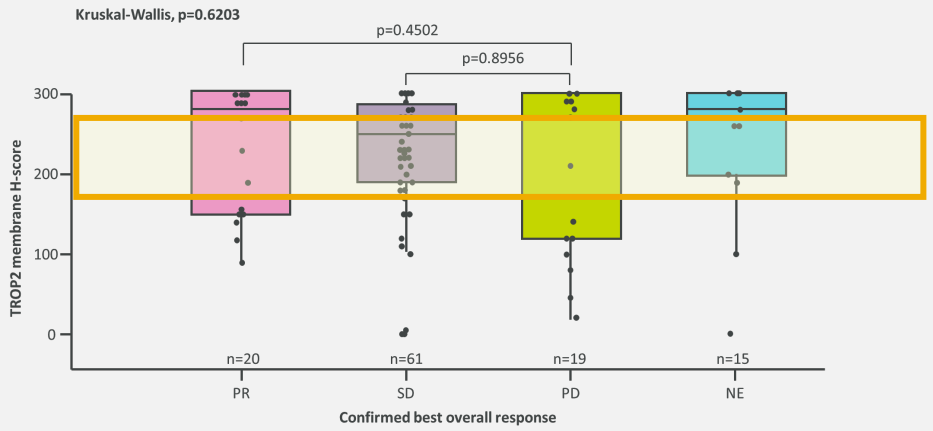
Why isn’t IHC sufficient to predict Dato-DXd benefit?

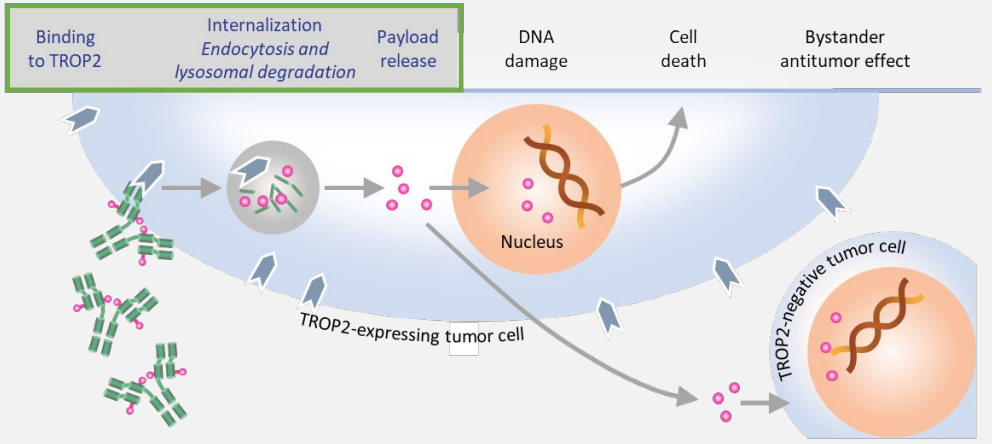
Conventional IHC scoring has not predicted response to TROP2-directed ADCs in NSCLC[1,2]



High TROP2 expression and overlapping expression levels across response categories

Dato-DXd is a TROP2 directed ADC with highly plasma-stable linker[3,4]

- Dato-DXd binds to membrane TROP2 and needs to be internalised for cytotoxic payload to be released
- Not all TROP2 protein on the cell surface ends up in the cytoplasm
- Assessment of both cell membrane and cytoplasm is needed to predict benefit from Dato-DXd



1. Shimizu T, et al. J Clin Oncol 2023;41:4678–87. 2. Heist RS, et al. J Clin Oncol 2017;35:2790–7. 3. Dent R, et al. Future Oncol 2023, 19;2349–59. 4. Okajima D, et al. Mol Cancer Ther 2021, 20:2329–40.
Collaboration partner: Daiichi Sankyo (Dato-DXd).

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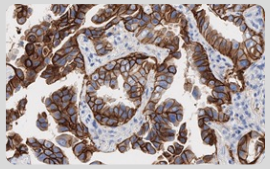
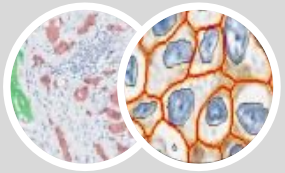
What is Quantitative Continuous Scoring (QCS)?

1 IHC with TROP2 Assay



2 Whole Slide Imaging

3 Automated Image Analysis (QCS)

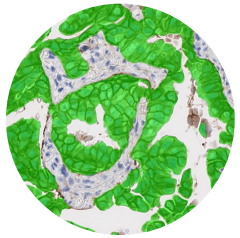


4 Patient Biomarker Status Determination

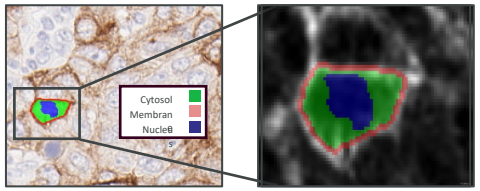
\+ ≥75% of tumour cells with TROP2 NMR ≤0.56

\- <75% of tumour cells with TROP2 NMR ≤0.56[1]

Differentiates tumour from non-tumour



Measures OD in each tumour cell



Membrane and cytoplasm optical density (OD)

Calculates TROP2 NMR for every tumour cell

$$\left[\frac{\text{Membrane OD}}{\text{Membrane OD + Cytoplasm OD}}\right]$$

Lower NMR → higher cytoplasm proportion

QCS is a novel, fully-supervised computational pathology approach that quantifies and locates targets like TROP2

1. Or >25% of cells with an NMR >0.56. OD, optical density (a measure of staining intensity).

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What is TROP2 QCS-NMR for Dato-DXd?

Normalised Membrane Ratio (NMR) predictive for Dato-DXd response in TROPION-PanTumor01

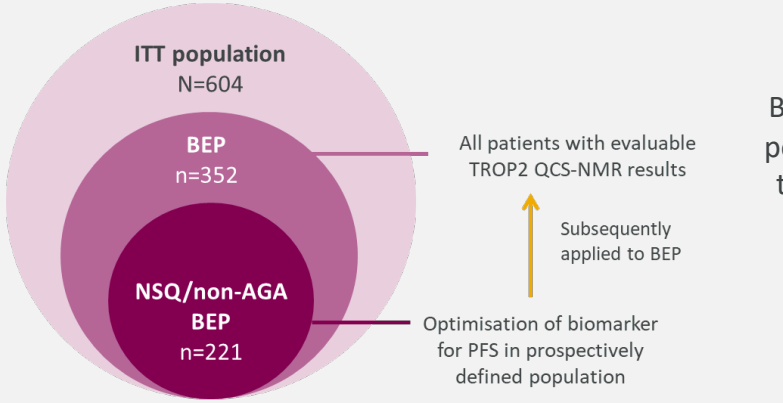
$$\left[\frac{\text{Membrane OD}}{\text{Membrane OD + Cytoplasm OD}}\right] = \text{Normalised Membrane Ratio (NMR)}$$

Higher cytoplasm staining	Lower Normalised Membrane Ratio	Higher response to Dato-DXd

Dato-DXd must be internalised to release cytotoxic payload, lower NMR predictive of response

Cut-point for TROP2 QCS-NMR biomarker positivity using samples from TROPION-Lung01

QCS-NMR cut-points optimised for PFS in TROPION-Lung01 NSQ/Non-AGA patients



Biomarker evaluable population (BEP)[2] are those patients with available tissue samples for QCS determination

Patients considered biomarker positive if ≥75% tumour cells have TROP2 QCS-NMR ≤0.56

1.Optical density is a measure of staining intensity. 2. Biomarker evaluable population (BEP) are those patients with available tissue samples for QCS determination; in safety analysis, excludes patients who were randomised but did not receive treatment. Collaboration partner: Daiichi Sankyo (Dato-DXd).

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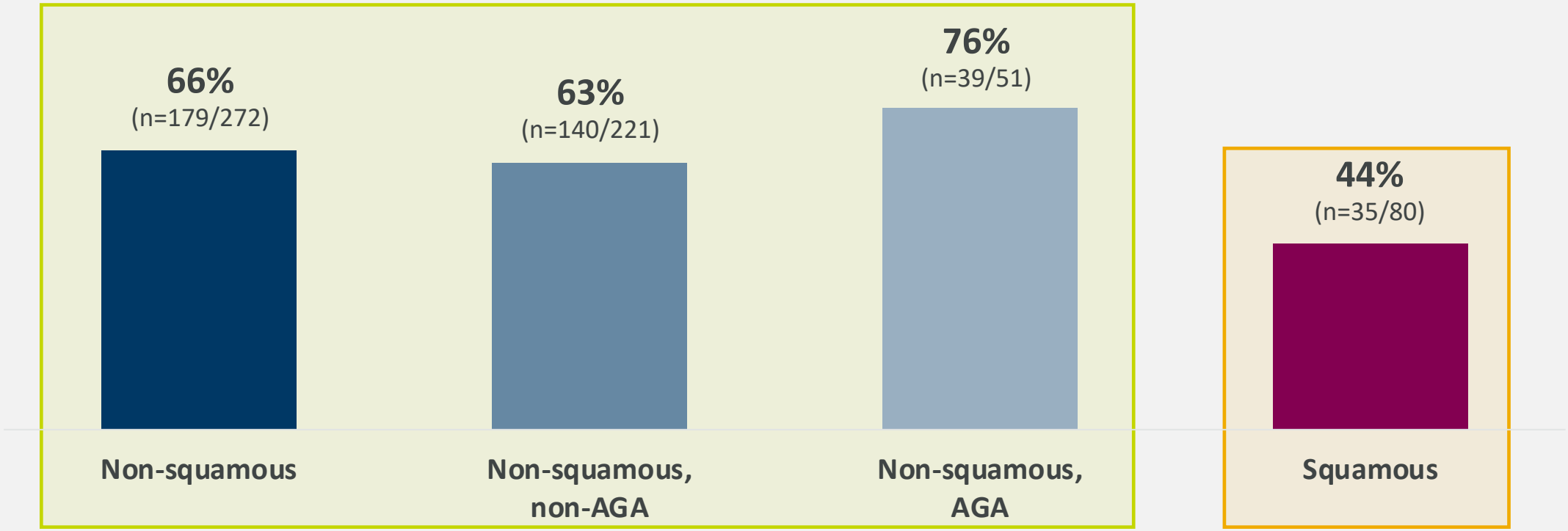
Approximately two-thirds of non-squamous patients were TROP2 QCS-NMR+



Histology subgroup, BEP (n=352)

- Overall/Grade ≥3 adverse event rates with Dato-DXd were similar regardless of TROP2 QCS-NMR status

Patients with *KRAS* mutations in the absence of other known actionable genomic alterations were included in non-AGA subgroup.
Garassino MC et al. Abstract #PL02.11 presented at the 2024 World Conference on Lung Cancer.
Collaboration partner: Daiichi Sankyo (Dato-DXd).

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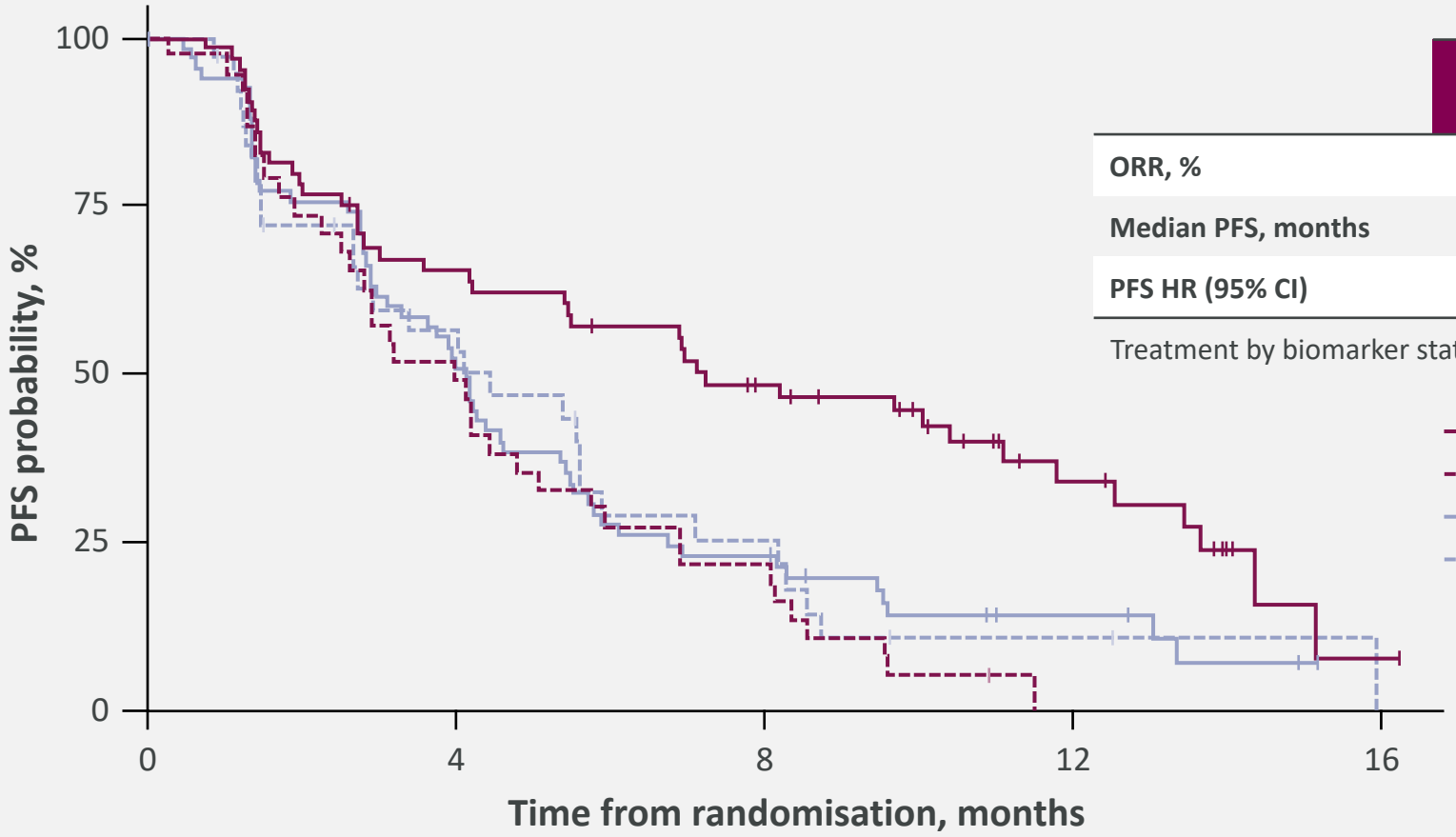
TROP2 QCS-NMR status was predictive of efficacy in the non-squamous/non-AGA BEP (n=221)



	TROP2 QCS-NMR+		TROP2 QCS-NMR–	
	Dato-DXd n=68	Docetaxel n=72	Dato-DXd n=40	Docetaxel n=41
ORR, %	36.8	15.3	22.5	12.2
Median PFS, months	7.2	4.1	4.0	4.4
PFS HR (95% CI)	0.52 (0.35–0.78)		1.22 (0.74–2.00)	

Treatment by biomarker status interaction: p=0.0098

Dato-DXd, QCS-NMR+
Dato-DXd, QCS-NMR–
Docetaxel, QCS-NMR+
Docetaxel, QCS-NMR–

Garassino MC et al. Abstract #PL02.11 presented at the 2024 World Conference on Lung Cancer.
Collaboration partner: Daiichi Sankyo (Dato-DXd).

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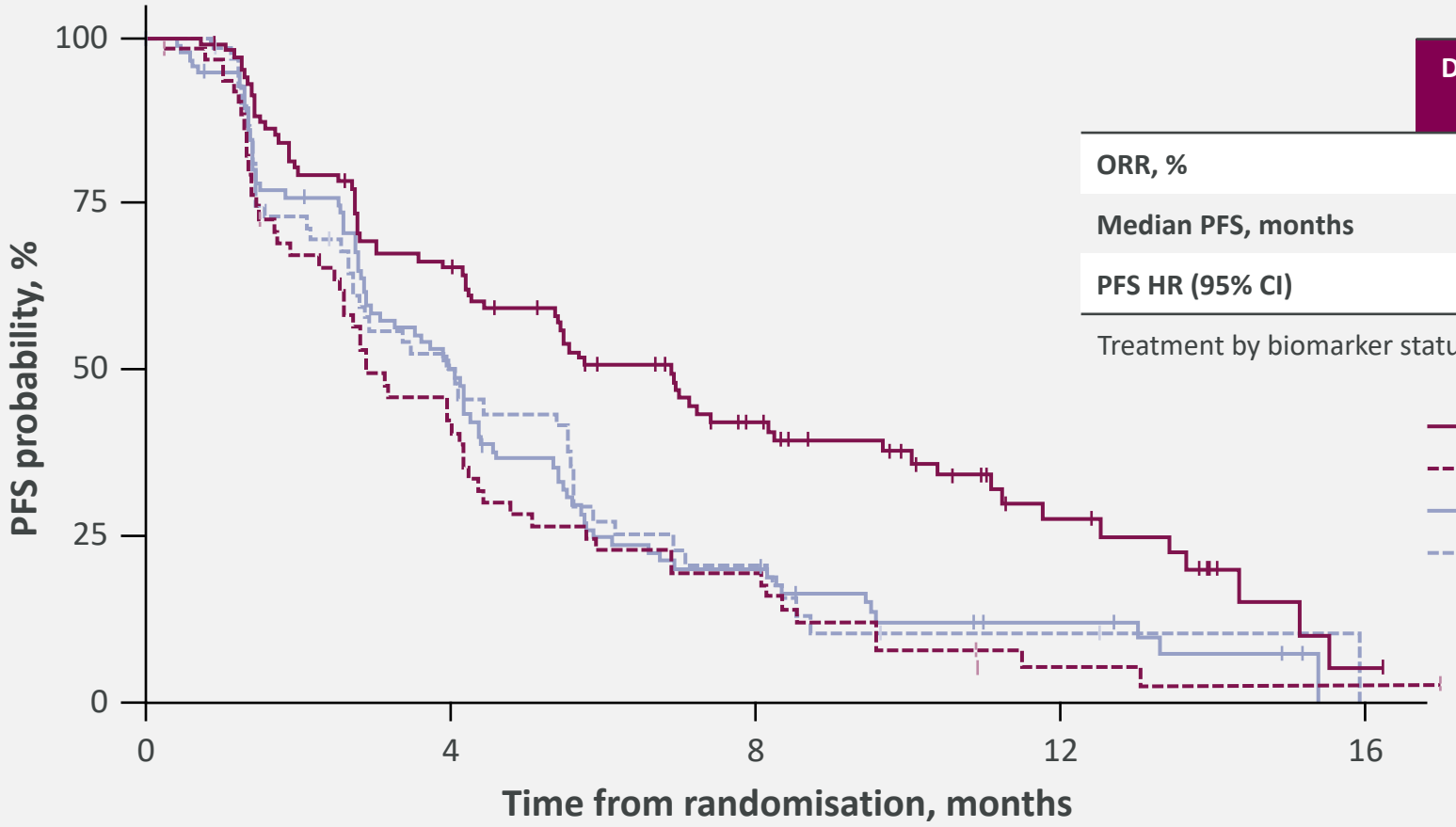
TROP2 QCS-NMR status was predictive of efficacy in the broader overall BEP (n=352)



	TROP2 QCS-NMR+		TROP2 QCS-NMR–	
	Dato-DXd n=107	Docetaxel n=107	Dato-DXd n=65	Docetaxel n=73
ORR, %	32.7	10.3	16.9	15.1
Median PFS, months	6.9	4.1	2.9	4.0
PFS HR (95% CI)	0.57 (0.41–0.79)		1.16 (0.79–1.70)	

Treatment by biomarker status interaction: p=0.0063

Garassino MC et al. Abstract #PL02.11 presented at the 2024 World Conference on Lung Cancer.
Collaboration partner: Daiichi Sankyo (Dato-DXd).

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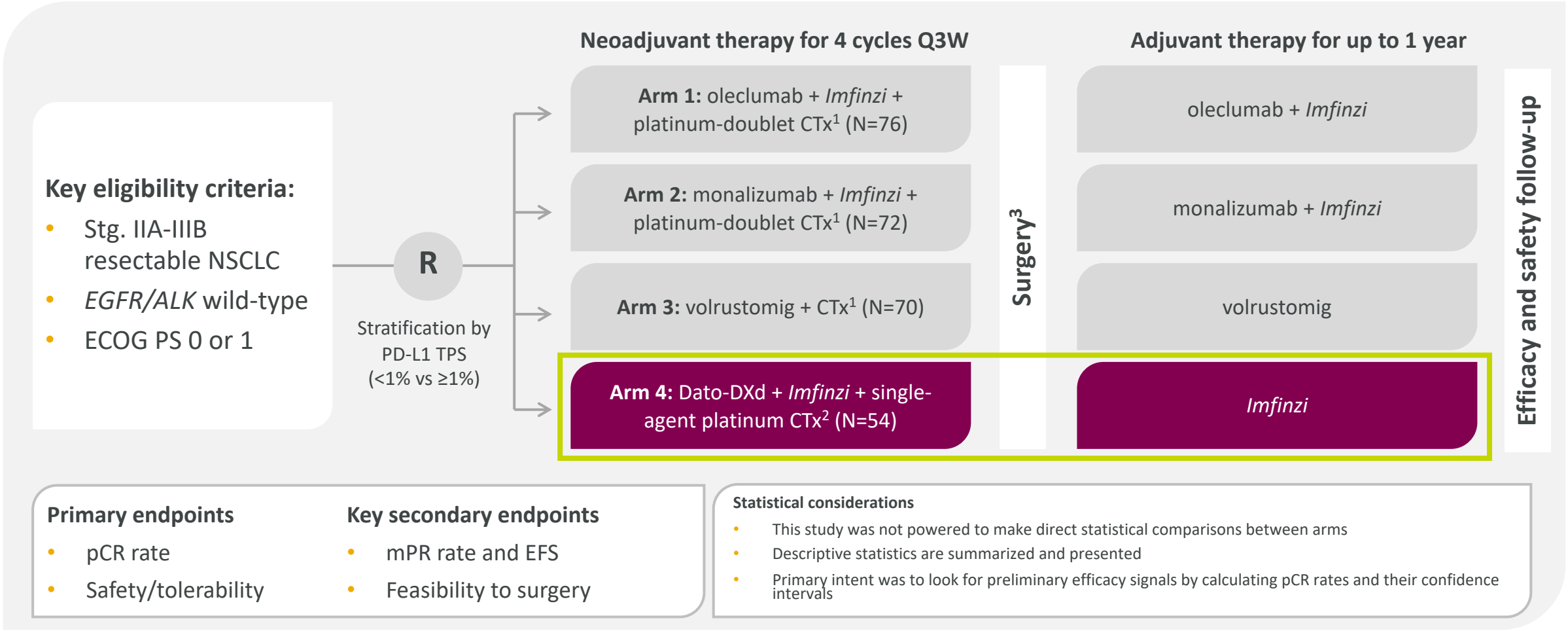
NeoCOAST-2 investigates Dato-DXd combination with *Imfinzi* in NSCLC in platform trial



Primary endpoints
- pCR rate
- Safety/tolerability

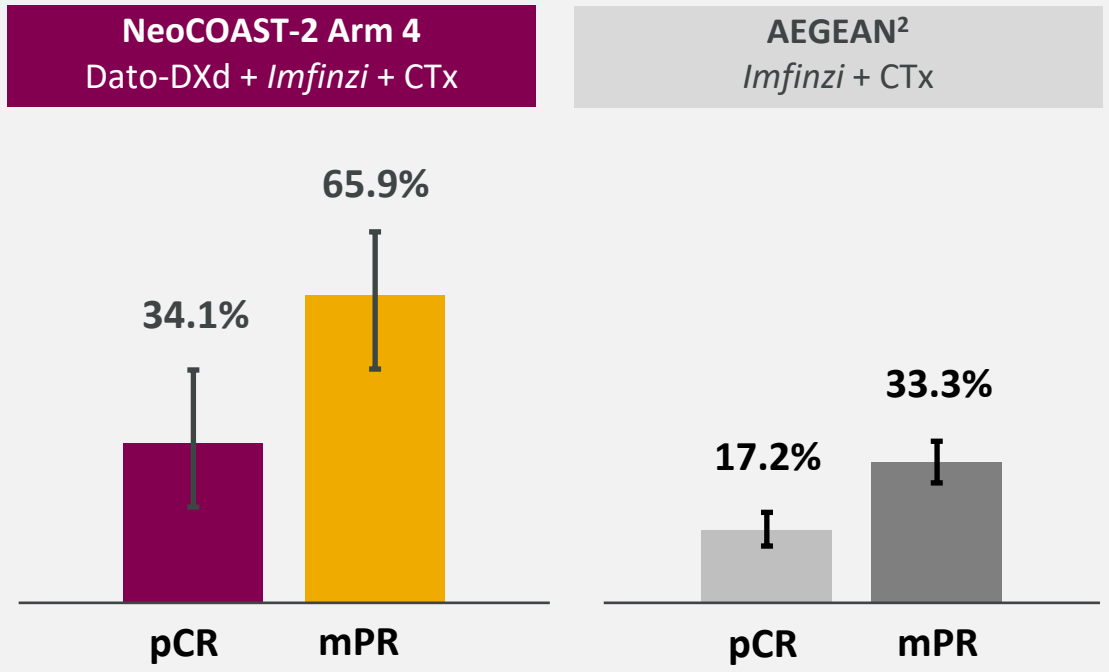
Key secondary endpoints
- mPR rate and EFS
- Feasibility to surgery

Statistical considerations
- This study was not powered to make direct statistical comparisons between arms
- Descriptive statistics are summarized and presented
- Primary intent was to look for preliminary efficacy signals by calculating pCR rates and their confidence intervals

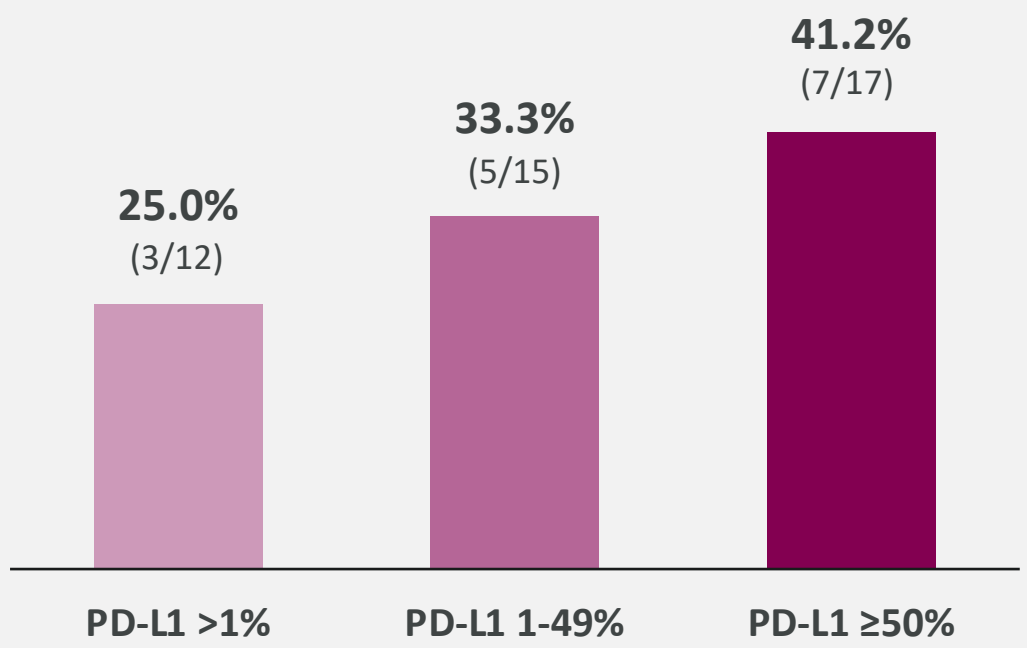
1. Carboplatin + paclitaxel for squamous tumour histology, pemetrexed + cisplatin or carboplatin for non-squamous tumour histology. 2. Physician's choice of carboplatin or cisplatin. 3. Within 40 days of the last dose of neoadjuvant treatment.
Cascone T et al. Abstract #PL02.07 presented at the 2024 World Conference on Lung Cancer.
Collaboration partner: Daiichi Sankyo (Dato-DXd).

Promising efficacy observed in Dato-DXd + *Imfinzi* + CTx compared with doublet CTx containing regimens

Encouraging pCR and mPR rates in Dato-DXd arm[1]



Higher pCR rates with increasing PD-L1 expression[3]



1. mITT population includes all randomised patients with confirmed NSCLC histology who received at least 1 dose of study treatment and had data available at the data cut-off, including those who were unable to receive or complete surgery. Some patients who underwent surgery did not have pathology results available at data cut-off. 2. Heymach JV, et al. New Engl J Med 2023;389:1672–84. 3. Based on the modified intention-to-treat population which includes all randomised patients with confirmed NSCLC histology who received at least 1 dose of study treatment and had data available at the DCO, including those who were unable to receive or complete surgery. Baseline PD-L1 status is assessed using central (Ventana SP263) or local testing (Ventana SP263, pharmDx 28-8, or pharmDx 22C3). Central results are reported for Arm 4, 13/44 (30%) patients. Local results are reported for all other patients.

Cascone T et al. Abstract #PL02.07 presented at the 2024 World Conference on Lung Cancer. Collaboration partner: Daiichi Sankyo (Dato-DXd).

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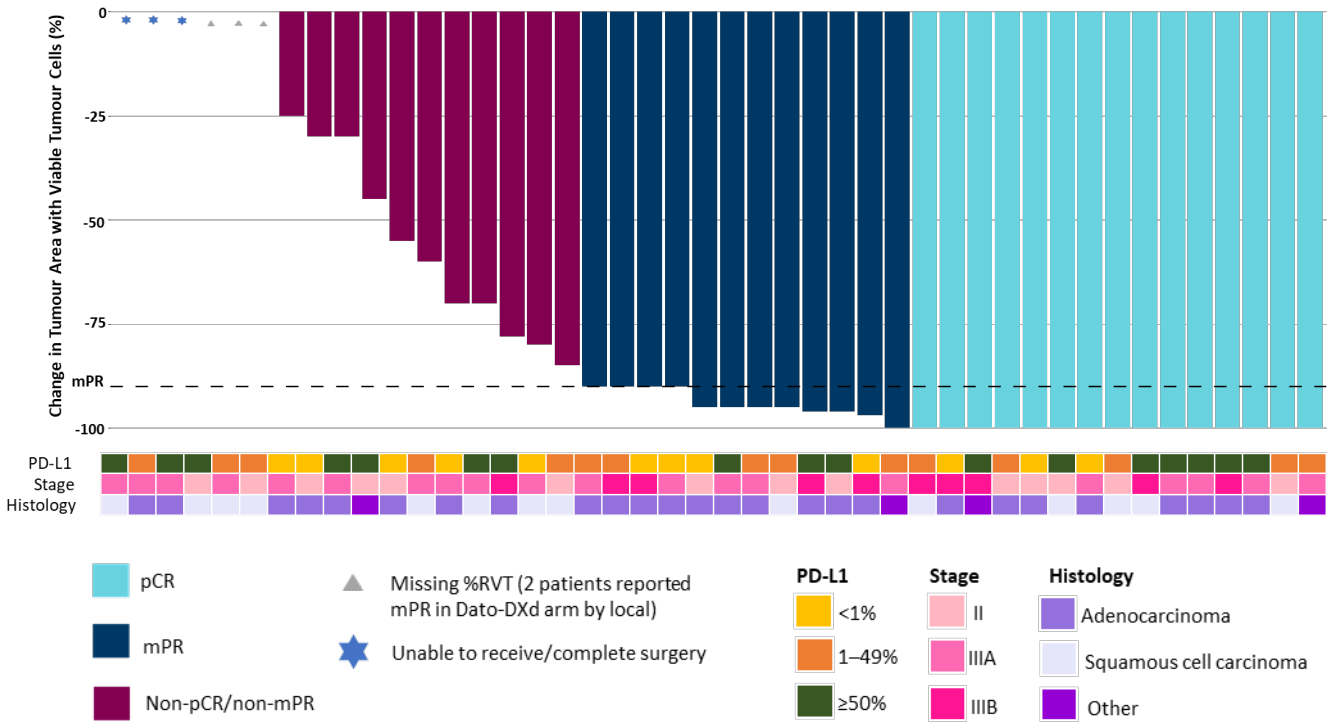
Dato-DXd + *Imfinzi* + CTx demonstrated strong and consistent benefit with manageable safety profile

Change in tumour area across PD-L1, stage and histology subgroups



Manageable safety profile

n (%)	Neoadjuvant N=54	Post-surgery N=46	Adjuvant N=25
Any TEAE	53 (98.1)	24 (52.2)	11 (44.0)
Any TRAE	52 (96.3)	6 (13.0)	5 (20.0)
Grade ≥3 TEAE	13 (24.1)	4 (8.7)	1 (4.0)
Grade ≥3 TRAE	10 (18.5)	0	0
AE leading to discontinuation	4 (7.4)	0	0
SAE	10 (18.5)	7 (15.2)	1 (4.0)
Any SAE with outcome of death	0	1 (2.2)[1]	0

Encouraging Grade ≥3 TEAE and discontinuation rates vs doublet CTx arms and historical IO + doublet CTx trials

The median (range) of number of adjuvant cycles completed per protocol in Arm 4 is 2 (1–6) as of data cut-off. Patients with multiple occurrences in the same category are counted once per category regardless of the number of occurrences.

1. Due to idiopathic pulmonary fibrosis unrelated to treatment, unrelated per principal investigator, independent adjudication is pending.

Cascone T et al. Abstract #PL02.07 presented at the 2024 World Conference on Lung Cancer.

Collaboration partner: Daiichi Sankyo (Dato-DXd).

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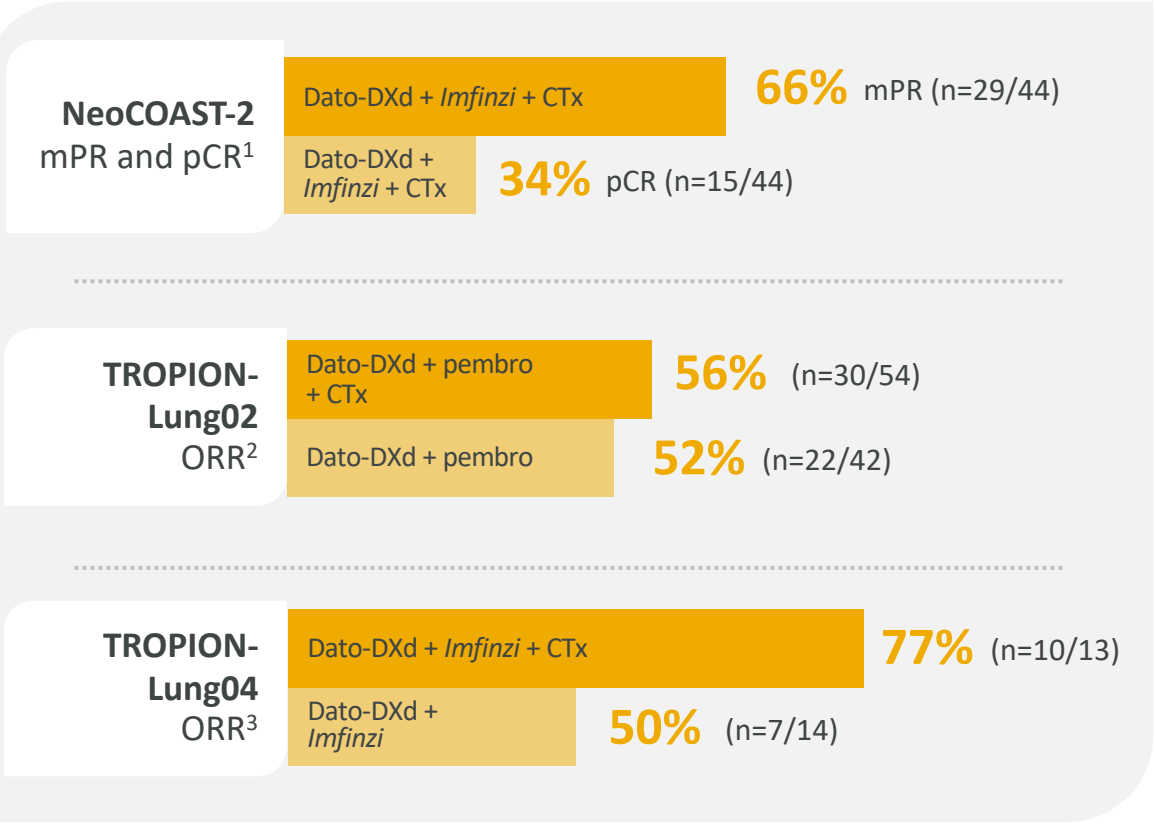
Early phase data drives confidence in 1L NSCLC trials

Early efficacy data reinforces potential in Phase III combination trials



AE profile enables combination with IO and CTx

Lower bone marrow toxicity confers better combinability with platinum chemotherapy

Convenient dosing aligns with chemotherapy – one i.v. infusion per cycle

Data monitoring committees have not raised any concern with ILD or any other safety finding in the 1L trials

1. Cascone T et al. Abstract #PL02.07 presented at the 2024 World Conference on Lung Cancer. 2. Levy BP et al. Abstract 8617 presented at American Society of Clinical Oncology 2024. 3. Papadopoulos KP et al. Abstract OA05.06 presented at World Conference on Lung Cancer 2023. Collaboration partner: Daiichi Sankyo (Dato-DXd).

AstraZeneca

Learnings from recent Dato-DXd datasets

TROPION-Lung01 final OS[1]	• Dato-DXd showed clinically meaningful median overall survival improvement of 2.3 months vs docetaxel in previously treated patients • Builds on previously met dual primary endpoint of progression-free survival
TROP2 QCS-NMR as a biomarker for Dato-DXd[2]	• Results support potential of TROP2, as measured by QCS-NMR, as a predictive biomarker for Dato-DXd and build confidence in AVANZAR and TROPION-Lung10 • Employing QCS across ADC portfolio with the goal of developing predictive biomarkers to enhance patient selection and improve outcomes for patients
NeoCOAST2.0[3]	• Reinforces confidence in 1L NSCLC alongside TROPION-Lung02 and TROPION-Lung04 • Demonstrates potential for neoadjuvant Dato-DXd plus *Imfinzi* and CTx in patients with early-stage non-small cell lung cancer

1. Sands J et al. Abstract #OA08.03 presented at the 2024 World Conference on Lung Cancer. 2. Garassino MC et al. Abstract #PL02.11 presented at the 2024 World Conference on Lung Cancer. 3. Cascone T et al. Abstract #PL02.07 presented at the 2024 World Conference on Lung Cancer.
Collaboration partner: Daiichi Sankyo (Dato-DXd).

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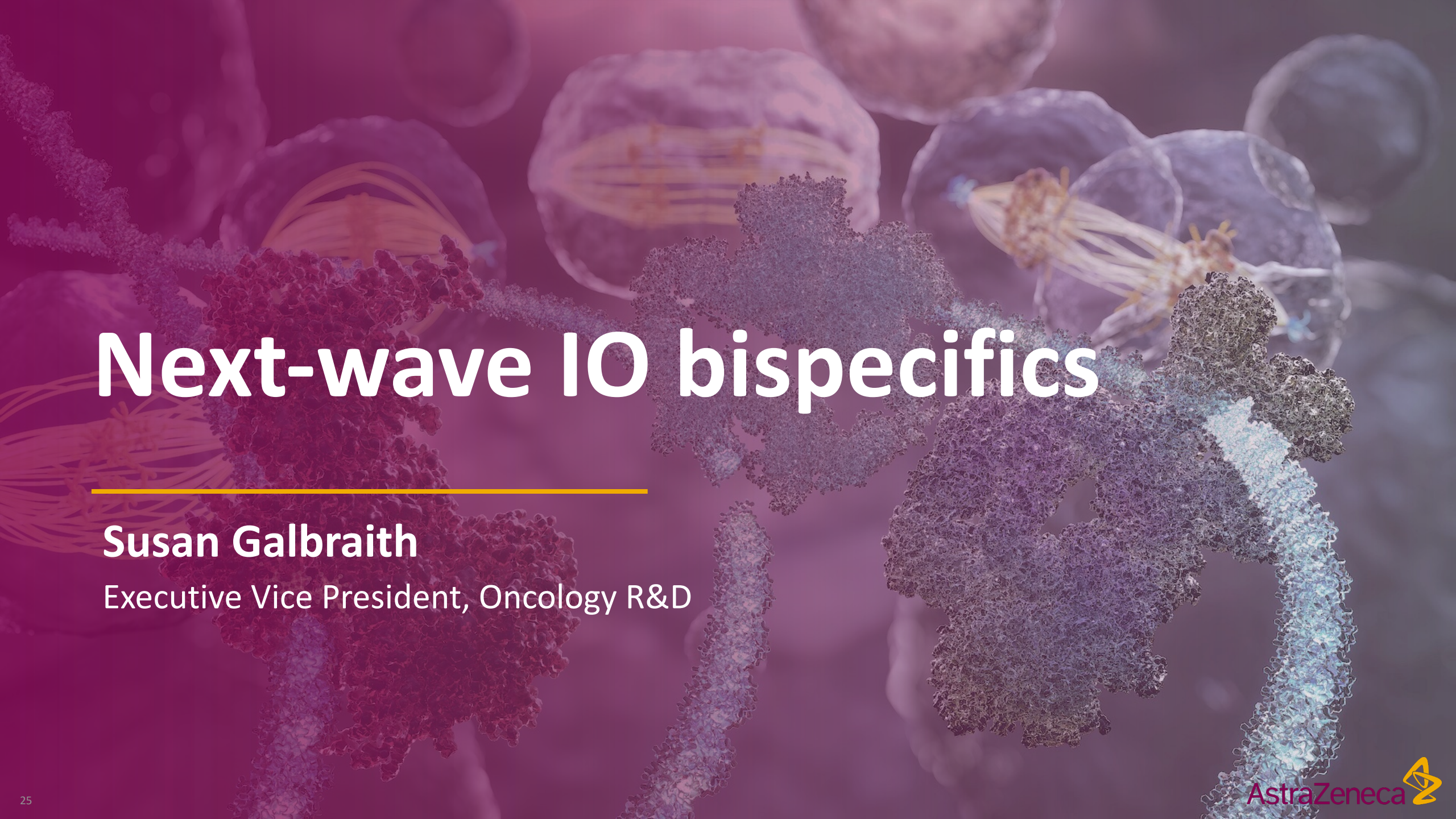


Next-wave IO bispecifics

Susan Galbraith

Executive Vice President, Oncology R&D

AstraZeneca

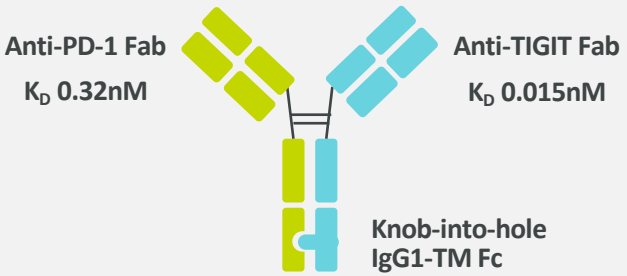
Leading in next-wave IO bispecifics

Rilvegostomig (PD-1/TIGIT) and volrustomig (PD-1/CTLA-4) unique bispecific design

Unique bispecific mechanism of action

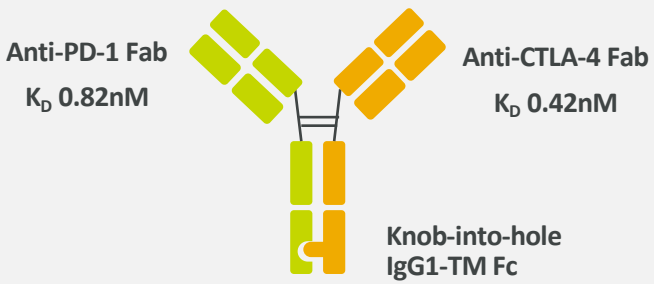
Cooperative binding in the presence of both checkpoint inhibitors

rilvegostomig



Fc attenuated triple-mutant IgG1 avoids unselective depletion by Fc-mediated ADCC/ADCP

volrustomig



Designed to fully inhibit PD-1 while preferentially inhibiting CTLA-4 on activated T cells

Growing Phase III programme

Across tumour types both monotherapy and combinations

- **ARTEMIDE-Biliary01** – rilve + CTx – BTC
- **TROPION-Lung10** – rilve + Dato-DXd – 1L NSCLC
- **DESTINY-BTC01** – rilve + *Enhertu* – HER2+ BTC
- **TROPION-Lung12** – rilve + Dato-DXd – Stg. I NSCLC
- **eVOLVE-Lung02** – volru + CTx – 1L NSCLC PD-L1 <50%
- **eVOLVE-meso** – volru + CTx – mesothelioma
- **eVOLVE-cervical** – volru – high-risk LA cervical
- **eVOLVE-HNSCC** – volru – LA u/r HNSCC

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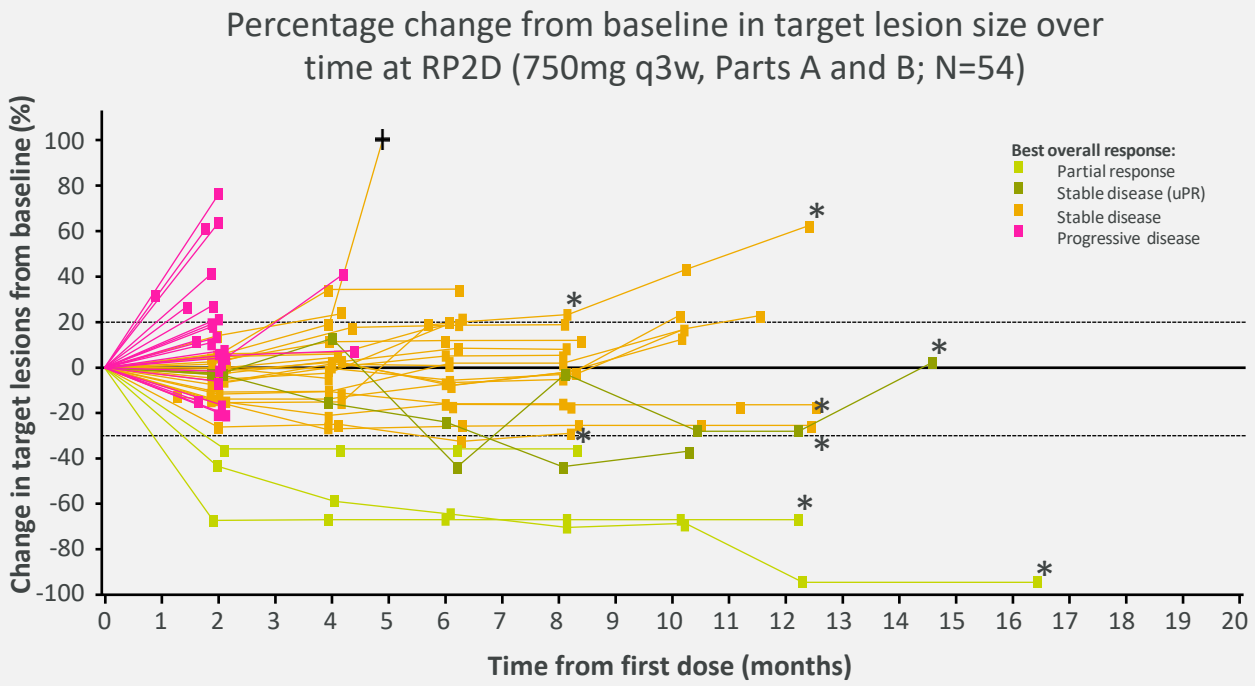
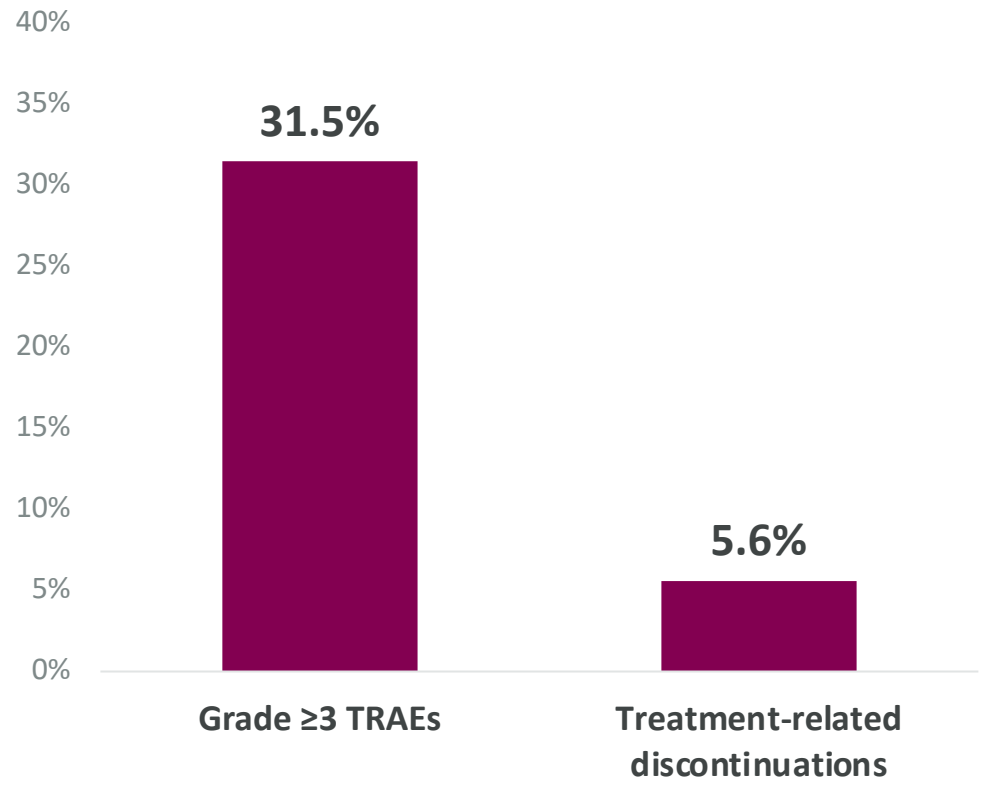
Rilvegostomig – promising anti-tumour activity and encouraging safety in mNSCLC in CPI pre-treated patients

ESMO 2023 | ARTEMIDE-01





+Percentage change from baseline in tumour lesion size exceeds +100%. Spider plot truncated for the patient at this point.
*Represents patient still on treatment (n=8/54).

Brandão M et al. Abstract 1446P presented at the European Society of Medical Oncology 2024.
Collaboration partner: Compugen (rilvegostomig).

Rilvegostomig – well tolerated in CPI-naïve mNSCLC

Patients in were CPI-naïve in Parts C and D

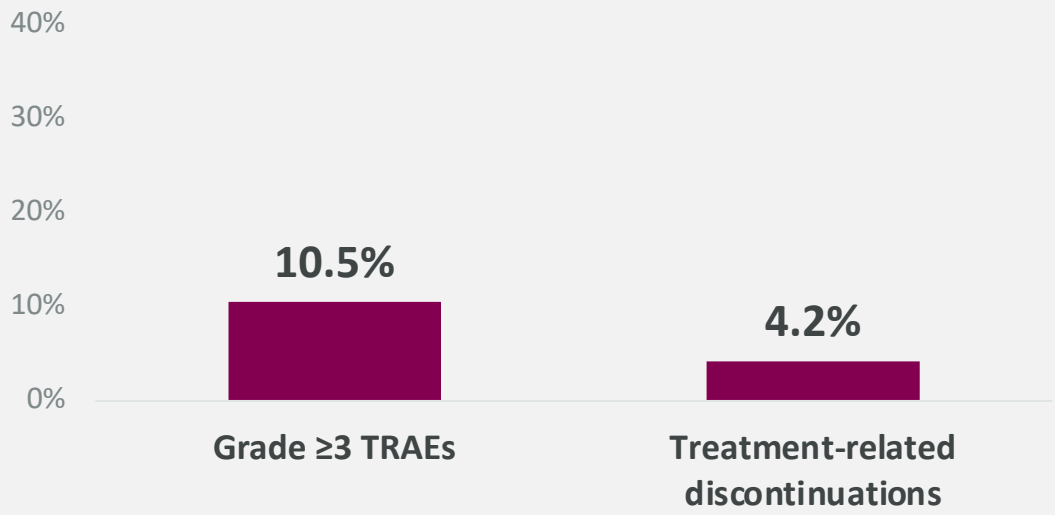
N=95 received ≥1 dose of rilvegostomig and had ≥9 weeks follow-up

16.7% had prior chemotherapy for metastatic disease[1]

13.5% had liver metastases[1]

21.9% had brain metastases[1]

Rilvegostomig was generally well tolerated



No evident differences between 750 mg and 1500 mg in safety profile

1. Of all enrolled patients (n=96).
Hiltermann, TJN et al. Abstract OA11.03 presented at the 2024 World Conference on Lung Cancer.
Collaboration partner: Compugen (rilvegostomig).

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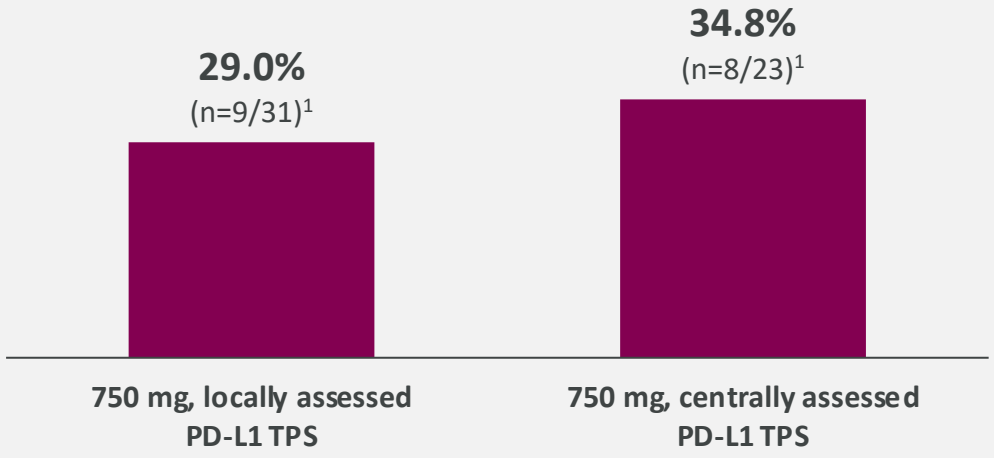
Rilvegostomig – encouraging preliminary response rates in CPI-naïve mNSCLC in both PD-L1 TPS ≥50% and 1-49% subsets

Robust ORR in PD-L1 TPS 1–49%



Stronger ORR in 750 mg cohort for PD-L1 TPS ≥50%

At data cutoff, 53.7% of patients remained on treatment with response ongoing in 28 (80%) confirmed responders

1. All responses confirmed. 2. 4 pending confirmation. 3. 3 pending confirmation.
Hiltermann, TJN et al. Abstract OA11.03 presented at the 2024 World Conference on Lung Cancer.
Collaboration partner: Compugen (rilvegostomig).

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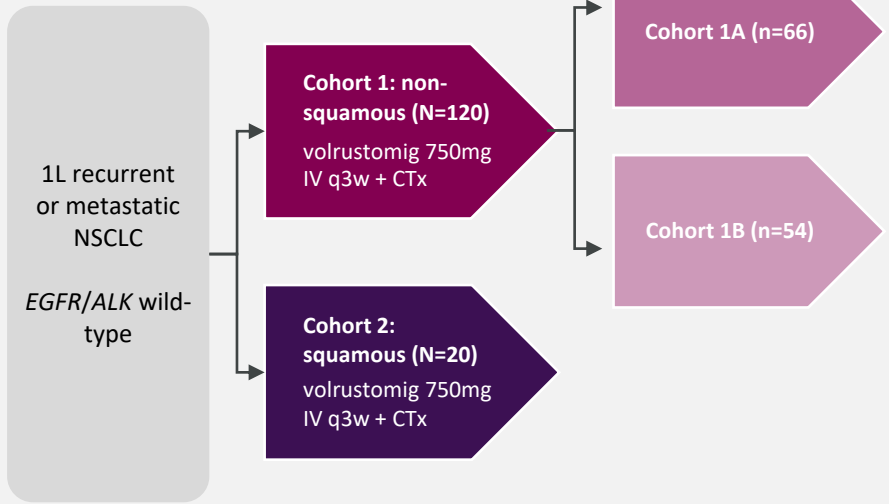
Volrustomig – promising efficacy in 1L NSQ NSCLC patients at ESMO 2022

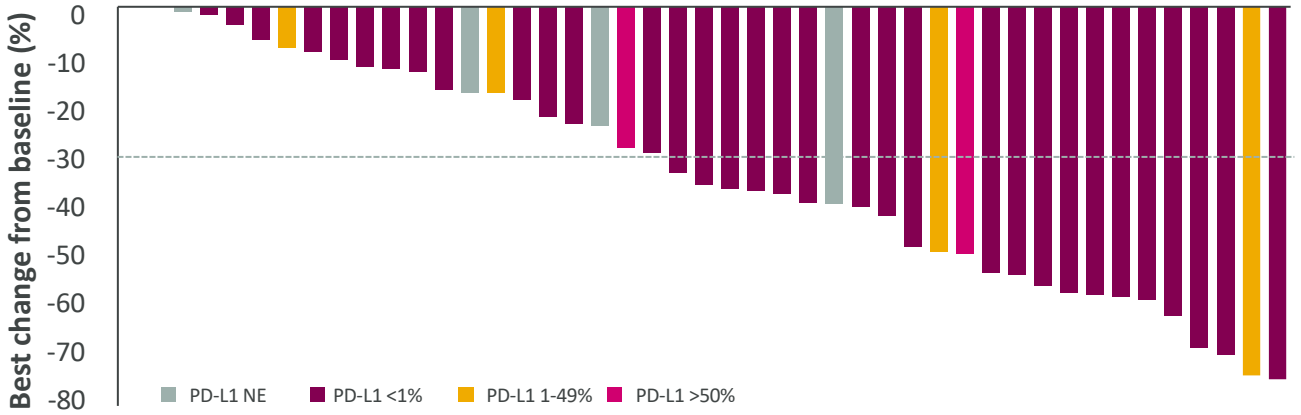
Phase I/II volrustomig

First-in-human in NSCLC[1,2]



- **Primary endpoints:** ORR per RECIST v1.1 (non-squamous); safety (squamous)
- **Key secondary endpoints:** Safety, DCR, DoR, PFS
- **Key exploratory endpoints:** Receptor occupancy, pharmacodynamic biomarkers of CTLA-4 blockade

ESMO 2022 | Strong efficacy in combination with CTx, especially in PD-L1 negative NSCLC[1]



Population (N=50)	≥30% reduction in target lesions (%)	ORR (%)
ITT (n=49)	49	40.8
PD-L1<1% (n=36)	55.6	44.4

- Overall safety of volrustomig 750 mg + CTx improved vs 1500 mg + CTx

1. al. Abstract LBA56 presented at European Society of Medical Oncology 2022. 2. Spigel DR et al. Abstract OA11.04 presented at the 2024 World Conference on Lung Cancer.

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Volrustomig – large proportion of PD-L1 expression <1% enrolled, reflecting unmet need in this population

Population of high unmet need

63.6% **PD-L1 tumour cell expression <1%**

140 patients enrolled with median age of 68.0 years

73.6%
male

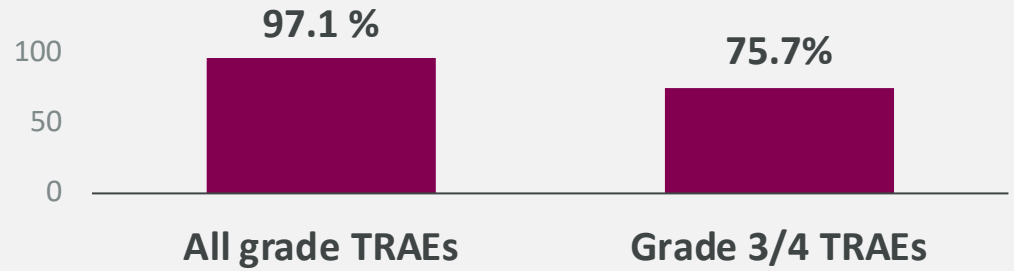
67.9%
ECOG PS 1

15.7%
liver metastases

15.0%
brain metastases

Manageable safety profile for volrustomig + CTx

Median number of cycles: 6 (range 1-39)



7 treatment-related deaths

2 volrustomig-related immune AEs | 5 CTx-related

6 in Cohort 1A (n=66) | 0 in Cohort 1B (n=54) | 1 in Cohort 2 (n=20)

Learnings applied across later cohorts improved safety management

Spigel DR et al. Abstract OA11.04 presented at the 2024 World Conference on Lung Cancer.

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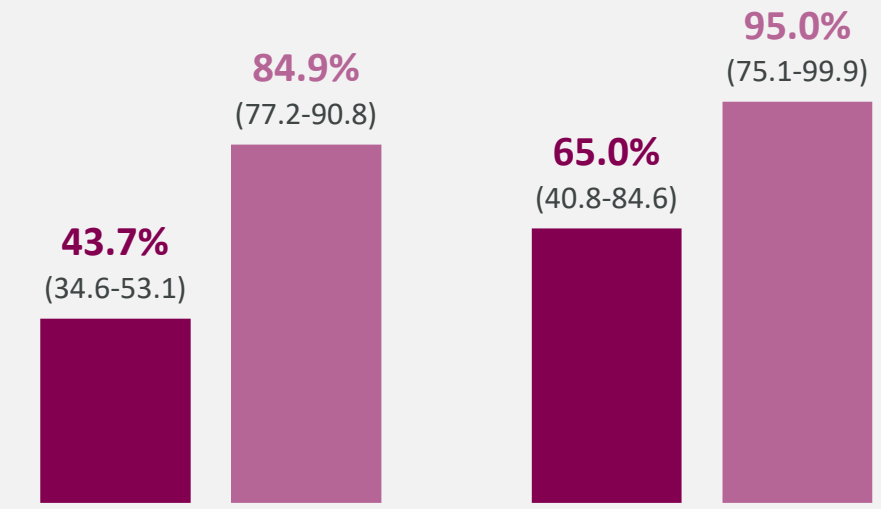
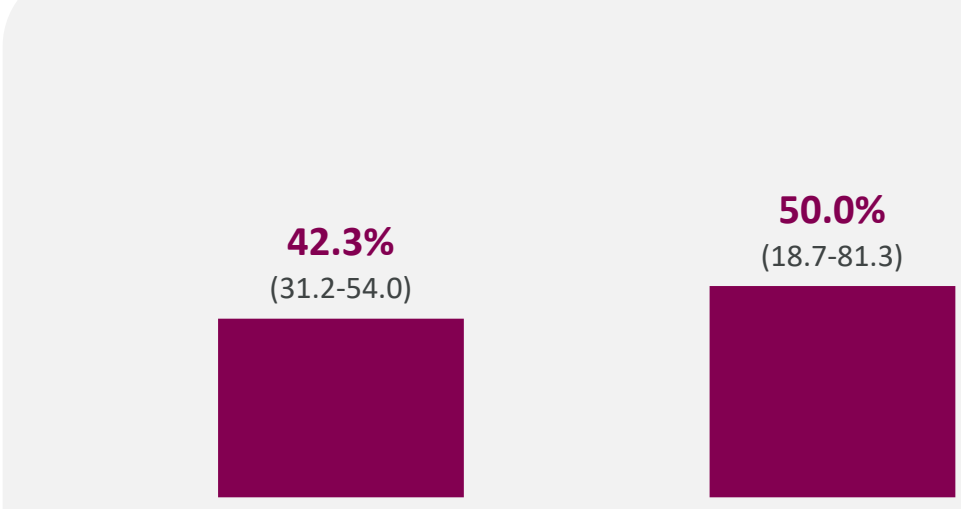
Volrustomig – promising clinical activity, especially in patients with tumour PD-L1 TC <1%

Nearly all patients achieved disease control



Encouraging ORR in PD-L1 TC <1%



Greater T-cell proliferation and memory T-cell activation with volrustomig 750mg + CTx vs anti-PD-1 + CTx

Median follow-up in Cohort 1 (non-squamous) was 8.9 months, in Cohort 2 (17.6 months)
1. One patient in Cohort 1 was excluded due to squamous histology.
Spigel DR et al. Abstract OA11.04 presented at the 2024 World Conference on Lung Cancer.

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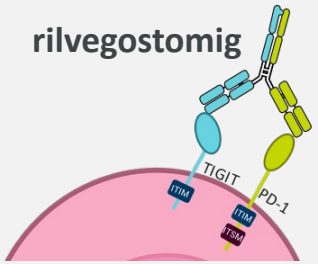
Lung cancer data reinforce potential for next-wave IO

Unique mechanism of action

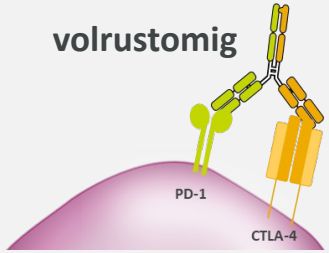
Bispecific design allows for cooperative binding

rilvegostomig



Synchronised, coordinated PD-1 /TIGIT blockade preserves and enhances immune effector cell function

volrustomig



Synchronised, coordinated PD-1/ CTLA-4 blockade increases T-cell activation and proliferation with manageable safety

Encouraging efficacy in early phase trials

- Encouraging ORR rates and PFS for rilvegostomig in CPI-naïve patients in Phase I/II ARTEMIDE-01[1]
- Rilvegostomig + CTx Gastric data to be presented at ESMO 2024
- Durable responses and encouraging PFS curves for volrustomig in FIH Phase I/II NSCLC[2]

Phase III trials enhanced based on early-phase learnings

- Rilvegostomig Phase III trials designed in known PDx-sensitive populations
- Volrustomig Phase III trials designed in CTLA-4-sensitive populations with:
  - >400 patients enrolled across four Phase III trials
  - Updated treatment management guidelines

1. Hiltermann, TJN et al. Abstract OA11.03 presented at the 2024 World Conference on Lung Cancer.2. Spigel DR et al. Abstract OA11.04 presented at the 2024 World Conference on Lung Cancer.
Collaboration partner: Compugen (rilvegostomig).

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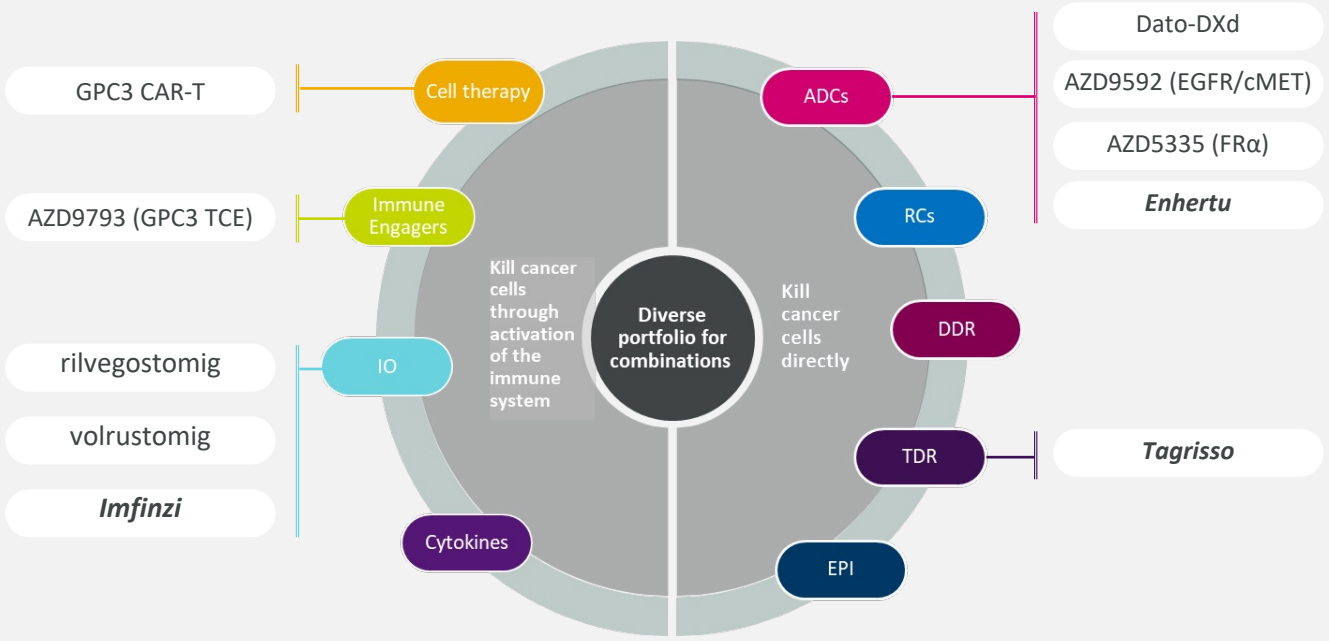
Summary

Dave Fredrickson

Executive Vice President, Oncology Business

AstraZeneca in lung cancer – proven execution

Attacking lung cancer from multiple angles and with novel combinations



Ambition to treat >50% lung cancer patients with AstraZeneca medicine by 2030

Proven ability to deliver companion diagnostics across portfolio

TAGRISSO® *EGFR*m testing rates

85%	**>80%**
Stage IV	Adjuvant

Lynparza® olaparib	BRCAm / HRD
IMFINZI®	PD-L1
ENHERTU®	HER2+, HER2-low, *HER2*m
Truqap™	*PIK3CA, AKT1, PTEN* alt.

Internal estimates. 1. Market share in the US.

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Accelerating catalyst path into 2025

Strong cadence of oncology read outs anticipated through 2025 with four pivotal trials in lung cancer

Lung cancer

Dato-DXd
AVANZAR – 1L NSCLC

Tagrisso
SAFFRON – 2L *EGFR*m NSCLC

Enhertu
DESTINY-Lung04 – *HER2*m NSCLC

ceralasertib
LATIFY – post-IO NSCLC

Imfinzi
POTOMAC – NMIBC

Enhertu
DESTINY-Breast11 – high-risk HER2+ early BC

Enhertu
DESTINY-Breast05 – high-risk HER2+ early BC

Imfinzi
MATTERHORN – GC/GEJC

Imfinzi
VOLGA – MIBC

Enhertu
DESTINY-Breast09 – HER2+ 1L mBC

camizestrant
SERENA-6 – 1L HR+ HER2- mBC

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Join us in Barcelona for the Meet AstraZeneca Management Event at ESMO

Monday 16 September, 20:00 CEST

Register and find more details at:
https://www.astrazeneca.com/investor-relations.html

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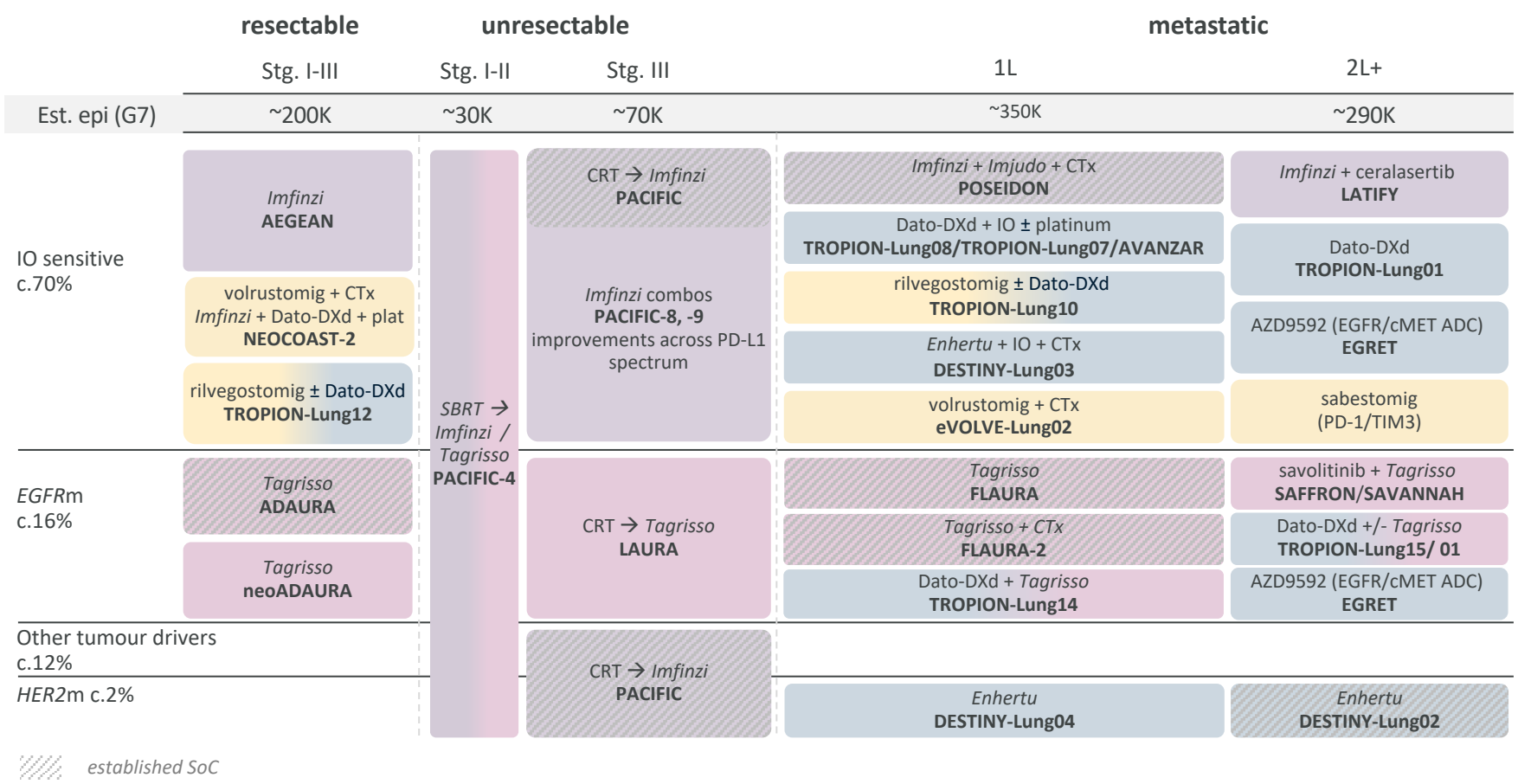
Appendix

AstraZeneca in Lung Cancer

Ambition for >50% of lung cancer patients to be eligible for AZN medicine by 2030



Leading the future of lung cancer treatment

- Establishing *Tagrisso* as backbone TKI in *EGFR*m
- *Imfinzi* leading IO in unresectable
- Advancing best-in-class ADCs to replace systemic chemotherapy
- Delivering next-wave bispecifics to improve on PD1/PD-L1
- Developing novel combinations, including IO + ADCs
- Investing behind new technologies and platforms, including cell therapy and testing/screening

Collaboration partners: Daiichi Sankyo (*Enhertu*, Dato-DXd), Compugen (rilvegostomig).

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Glossary

1/2/3L	1st/2nd/3rd line
AGA	actionable genomic alterations
AE	adverse event
ALT	alanine transaminase
alt.	alteration
ALK	anaplastic lymphoma kinase
ADC	antibody drug conjugate
ADCC	antibody-dependent cellular cytotoxicity
ADCP	antibody-dependent cellular phagocytosis
AST	aspartate transaminase
AZN	AstraZeneca
ATR	ataxia telangiectasia-mutated and Rad3-related
BTC	biliary tract cancer
BM	biomarker
BEP	biomarker evaluable population
BC	breast cancer
BRCAm	BReast CAncer gene mutation
CPI	checkpoint inhibitor
CTx	chemotherapy
CAR-T	chimeric antigen receptor T-cell therapy
CLL	chronic lymphocytic leukemia
CI	confidence interval
CTLA-4	cytotoxic T-lymphocyte-associated antigen 4
DCR	disease control rate
DDR	DNA damage response
DLT	dose limiting toxicities
DoR	duration of response
ECOG PS	Eastern Cooperative Oncology Group performance status
EGFR(m)	epidermal growth factor receptor (mutated)
EPI	epigenetics
ESMO	European Society for Medical Oncology
EFS	event-free survival
q3w	every 3 weeks
FIH	first-in-human
FRα	folate receptor alpha
Fab	fragment antigen-binding
GC	gastric cancer
GEJC	gastroesophageal junction adenocarcinoma
GPC3	glypican-3
HR	hazard ratio
HNSCC	head and neck squamous cell carcinoma
HCC	hepatocellular carcinoma
HLR	high level results
HRD	homologous recombination deficiency
HR+	hormone receptor positive
HER2	human epidermal growth factor receptor 2
IASLC	International Association for the Study of Lung Cancer
IgG1-TM	immunoglobulin G1 triple mutation
IHC	immunohistochemistry
IO	immunooncology
IRA	Inflation Reduction Act
ITT	intent-to-treat
LS-SCLC	limited stage small cell lung cancer
LA	locally advanced
mPR	major pathologic response
MCL	mantle cell lymphoma
m	median
M&A	mergers and acquisitions
cMET	mesenchymal-epithelial transition factor
mBC	metastatic breast cancer
mTPI	modified toxicity probability interval
mo	months
MIBC	muscle-invasive bladder cancer
NME	new molecular entity
NC	non-calculable
NMIBC	non-muscle invasive bladder cancer
NSCLC	non-small cell lung cancer
NSQ	non-squamous
NMOD	normalised membrane optical density
NMR	normalised membrane ratio
no.	number
ORR	objective response rate
OS	overall survival
pCR	pathologic complete response
PDx	PD-(L)1 inhibitor
PTEN	phosphatase and tensin homolog
PIK3CA	phosphatidylinositol-4,5-bisphosphate 3-kinase catalytic subunit alpha
PD-(L)1	programmed cell death (ligand) 1
PFS	progression-free survival
AKT	protein kinase B
QCS	quantitative continuous scoring
RC	radioconjugate
RP2D	recommended Phase II dose
R&D	Research & Development
RECIST	Response Evaluation Criteria In Solid Tumors
rilve	rilvegostomig
SAE	serious adverse event
SCLC	small cell lung cnacer
SQ	squamous
stg.	stage
SoC	standard-of-care
TIGIT	T cell immunoreceptor with immunoglobulin and ITIM domain
TCE	T-cell engager
TACE	transarterial chemoembolisation
TEAE	treatment-emergent adverse event
TRAE	treatment-related AE
TROP2	trophoblast antigen 2
TC	tumour cell
TDR	tumour drivers and resistance
TPS	tumour proportion score
u/r	unresectable
volru	volrustomig
WCLC	World Conference on Lung Cancer

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