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Meet AZN Management:

WCLC and ESMO 2024

Investor event

16 September 2024

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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AstraZeneca @ WCLC and ESMO 2024

Agenda

AstraZeneca ambition in oncology	–	**Pascal Soriot**, Chief Executive Officer
Key oncology themes at WCLC and ESMO	–	**Susan Galbraith**, EVP, Oncology R&D
Dato-DXd – patient selection	–	**Susan Galbraith**, EVP, Oncology R&D
Expanding into new frontiers: bladder cancer	–	**Prof. Thomas Powles**, Professor of Genitourinary Oncology; Director, Barts Cancer Centre **Sunil Verma,** SVP, Global Head, Oncology Franchise
Q&A session I	–	**Moderated by: Dave Fredrickson**, EVP, Oncology Business
Driving the ADC revolution	–	**Matt Hellmann,** VP, Early Oncology Development
Leading in next-generation IO	–	**Cristian Massacesi**, Chief Medical Officer, Chief Development Officer Oncology
Closing remarks & Q&A session II	–	**Moderated by: Dave Fredrickson**, EVP, Oncology Business

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Speakers and panelists

ASTRAZENECA LEADERSHIP

Pascal Soriot
CHIEF EXECUTIVE OFFICER

Dave Fredrickson
EXECUTIVE VICE PRESIDENT, ONCOLOGY BUSINESS

Susan Galbraith
EXECUTIVE VICE PRESIDENT, ONCOLOGY R&D

Cristian Massacesi
CHIEF MEDICAL OFFICER & ONCOLOGY CHIEF DEVELOPMENT OFFICER

Matt Hellmann
VP, EARLY ONCOLOGY DEVELOPMENT

Sunil Verma
SVP, GLOBAL HEAD, ONCOLOGY FRANCHISE

KEY EXTERNAL EXPERTS

Prof. Thomas Powles
UROLOGY MEDICAL ONCOLOGIST, UNIVERSITY OF LONDON AND DIRECTOR OF BARTS CANCER CENTRE

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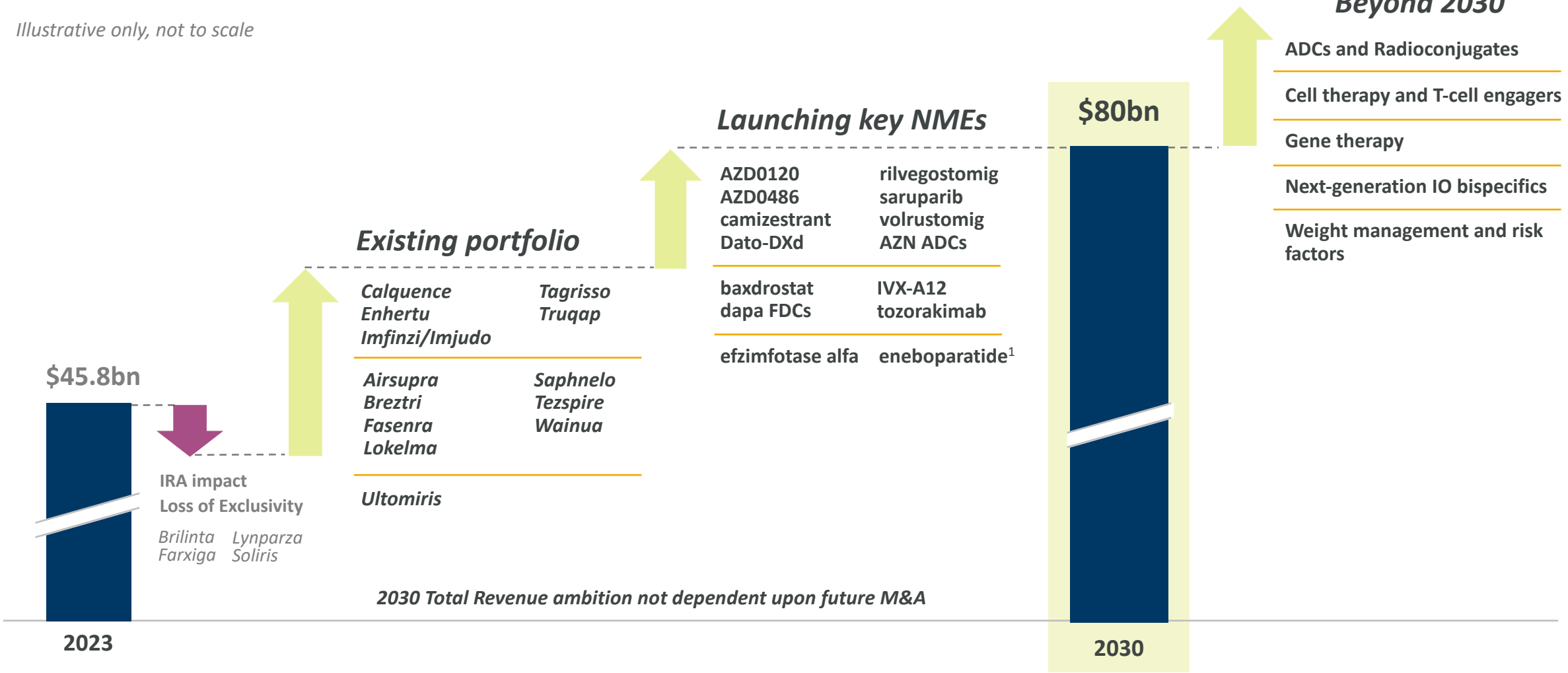
Furthering our Oncology ambition

Pascal Soriot
Chief Executive Officer

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

Working on “today, tomorrow and the day after"

Illustrative only, not to scale



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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Investing in disruptive categories to drive 2030+ growth

Weight management and risk factors	ADCs and Radioconjugates	Next-gen IO bispecifics	Cell therapy and T-cell engagers	Gene therapy and gene editing
Establish and lead in new weight management paradigm	Replace systemic chemotherapy and radiotherapy	Replace existing PD-1/ PD-L1 inhibitors	Develop scalable cell therapies and T-cell engagers across therapy areas	Make cure possible for a range of rare diseases
oGLP-1 mono and FDCs **Long-acting amylin** **GLP-1/glucagon**	**Six clinical-stage ADCs** **FPI-2265**[1]	**volrustomig** (PD-1/CTLA-4) **rilvegostomig** (PD-1/TIGIT)	**AZD0120** (BCMA/CD19) **Solid tumour cells** **AZD0486** (CD19/CD3 TCE)	**sAAVy and AAV capsid** **TALEN technology**

1. Fusion Pharmaceuticals acquisition remains subject to customary external clearances; all clinical development plans mentioned herein subject to deal closure. Collaboration partners: Compugen (rilvegostomig).

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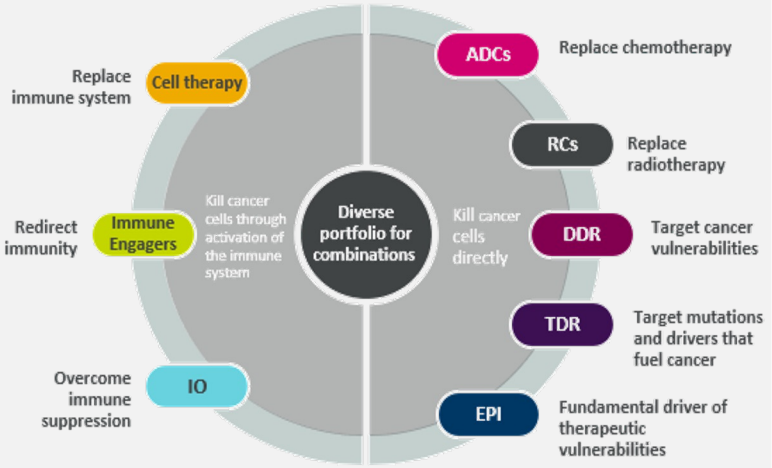
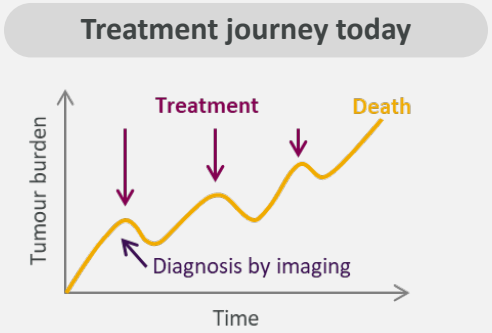
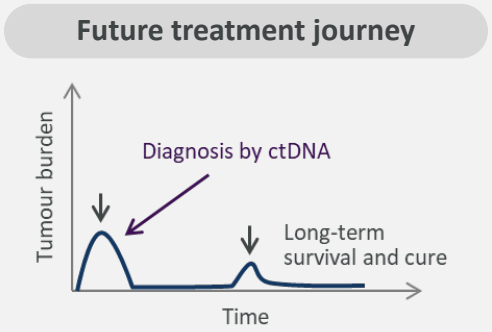
AstraZeneca – ambition in oncology

Attack cancer from multiple angles

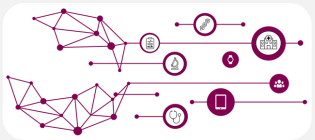


Treat earlier and smarter





Lead with innovative technology



Data and AI in clinical trials

HER2

TROP2

Accelerating computational pathology and QCS

Improving patient outcomes through digital health

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17 positive oncology readouts since beginning of 2023

DESTINY-PanTumor02
Enhertu
multiple HER2-expressing tumours

AEGEAN
Imfinzi
resectable NSCLC

ADAURA
Tagrisso
OS benefit in adjuvant NSCLC

DUO-O
Imfinzi | ***Lynparza***
advanced ovarian cancer

FLAURA-2
Tagrisso
combination regimen in 1L *EGFR*m NSCLC

MATTERHORN
Imfinzi
first IO + CTx to show benefit in GC/GEJ

HIMALAYA
Imfinzi + Imjudo
longest survival at 5 years in HCC

TROPION-Lung01
Dato-DXd
PFS improvement in 2-3L NSCLC

TROPION-Breast01
Dato-DXd
PFS improvement HR+/HER2low/– mBC

EMERALD-1
Imfinzi
first IO+TACE to show clinical benefit in HCC

LAURA
Tagrisso
expanding in early-stage *EGFR*m NSCLC

ADRIATIC
Imfinzi
potential first IO regimen in LS-SCLC

DESTINY-Breast06
Enhertu
moving into CTx-naïve mBC and HER2-ultralow

ECHO
Calquence
first BTKi to show OS trend in 1L MCL

NIAGARA
Imfinzi
first IO to extend survival in MIBC

AMPLIFY
Calquence
1L CLL with fixed-duration treatment

PACIFIC-5
Imfinzi
u/r Stg. III NSCLC sequential CRT+IO

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AstraZeneca presence at WCLC and ESMO 2024

Susan Galbraith

Executive Vice President, Oncology R&D

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>130 abstracts acceptances

74 poster presentations

41 oral presentations

5 Plenary presentations

2024 World Conference on Lung Cancer

SEPTEMBER 7-10, 2024
SAN DIEGO, CA USA

- **QCS-NMR for prediction of efficacy of Dato-DXd (PL02.11)** Presidential plenary
- **NeoCOAST-2 (PL02.07)** Presidential plenary
- TROPION-Lung01 OS (OA08.03)
- volrustomig in 1L NSCLC (OA11.04)
- ARTEMIDE-01 (OA11.03)

- **NIAGARA (LBA5)** Presidential plenary
- GEMINI-Gastric (1422P)
- BLUESTAR (606O)
- FONTANA (754P)

Eight simultaneous publications during WCLC and ESMO, including 3 publications in *NEJM*

1. Also known as C-CAR031. PROs = patient reported outcomes.
Collaboration partners: Daiichi Sankyo (*Enhertu*, Dato-DXd).

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WCLC and ESMO 2024 data mark key steps in delivering oncology ambition

Treat earlier and smarter

NIAGARA

Imfinzi + CTx

First perioperative IO regimen to extend OS in MIBC
Part of broader *Imfinzi* bladder cancer programme

NeoCOAST-2

Dato-DXd + *Imfinzi*

Unprecedented pCR observed in early-stage NSCLC
Underscores potential for Dato-DXd + IO + CTx

Attack cancer from multiple angles

BISPECIFICS

volrustomig

PD-1/CTLA-4

Follow-up demonstrates potential in NSCLC, particularly PD-L1 TC<1%

rilvegostomig

PD-1/TIGIT

Efficacy in CPI-naïve NSCLC | Ability to combine with CTx in gastric cancer

IN HOUSE ADCs

AZD8205

B7H4 TOP1i

Promising efficacy across multiple tumour and B7H4 expression levels

AZD5335

FRα TOP1i

Pronounced efficacy in platinum-resistant relapsed ovarian cancer

Lead with innovative technology

TROP2 QCS biomarker

QCS-NMR

Predictive for response to Dato-DXd in TROPION-Lung01

IHC alone not predictive for Dato-DXd response

QCS-NMR quantifies TROP2 expression on membrane and in cytoplasm

Potential to apply QCS methodology to other tumor types and targets

1. Also known as C-CAR031.
Collaboration partners: Daiichi Sankyo (*Enhertu*, Dato-DXd).

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Dato-DXd – patient selection

Susan Galbraith
Executive Vice President, Oncology R&D

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Why is Dato-DXd different from other TROP2 ADCs?

Internalisation is a critical component of Dato-DXd mechanism of action

Dato-DXd has a differentiated design compared to other TROP2-directed ADCs

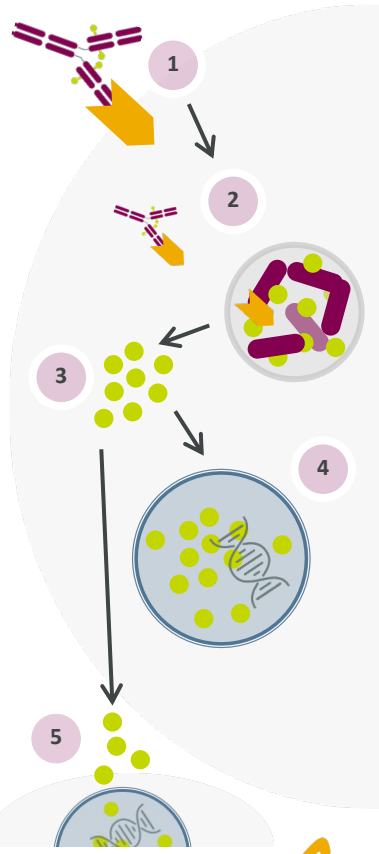
Enzymatic cleavage of plasma-stable linker

Benefits of Dato-DXd design

Plasma stable linker - 5-day half life - Low free payload exposure	→ Lower bone marrow toxicity	→ Ability to combine - IO - Platinum CTx - *Tagrisso*

Plasma-stable linker plays important role in understanding Dato-DXd mechanism of action[1]

1. Dato-DXd mAb component binds to TROP2 on cell membrane
2. Dato-DXd is internalised and plasma-stable linker is cleaved
3. TOPO1i payload (DXd) is released into cytoplasm of cell
4. Payload enters cell nucleus, inhibiting TOPO1, inducing cell death
5. Bystander antitumour effect – elimination of neighbouring tumour cells



1. Okajima D, et al. Mol Cancer Ther. 2021 Dec; 20(12):2329-2340.

Collaboration partner: Daiichi Sankyo (Dato-DXd).

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Novel biomarker solution – QCS-NMR for Dato-DXd

QCS measures features linked to biological effect unlike conventional IHC

Challenges of conventional IHC assessed by visual scoring for Dato-DXd

TROP2 is highly expressed across tumour and normal tissue

TROP2 has variable patterns of expression in membrane and cytoplasm

Total TROP2 IHC does not reflect subset of TROP2 to which Dato-DXd binds/internalises

	QCS	IHC
Distinguish tumour vs normal	✓	✓
Quantify membrane vs cytoplasm	✓	✗
Associate with internalisation	✓	✗
Associate with cytotoxicity	✓	✗
Prevalence associates with histology	✓	✗
Associate with clinical efficacy	✓	✗

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.
Collaboration partner: Daiichi Sankyo (Dato-DXd).

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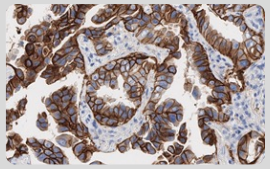
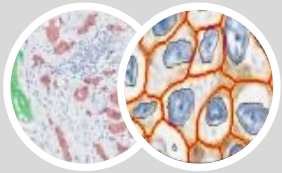
TROP2 QCS-NMR – what is Quantitative Continuous Scoring (QCS)?

1 IHC with TROP2 Assay



2 Whole Slide Imaging

3 Automated Image Analysis (QCS)

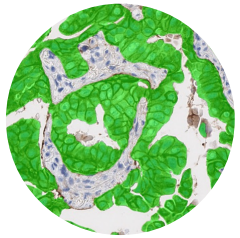


4 Patient Biomarker Status Determination

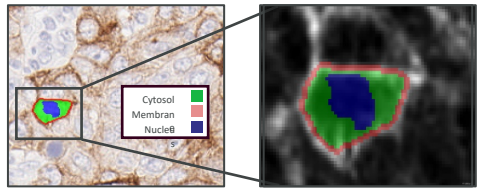
\+ ≥75% of tumour cells with TROP2 NMR ≤0.56

\- <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).
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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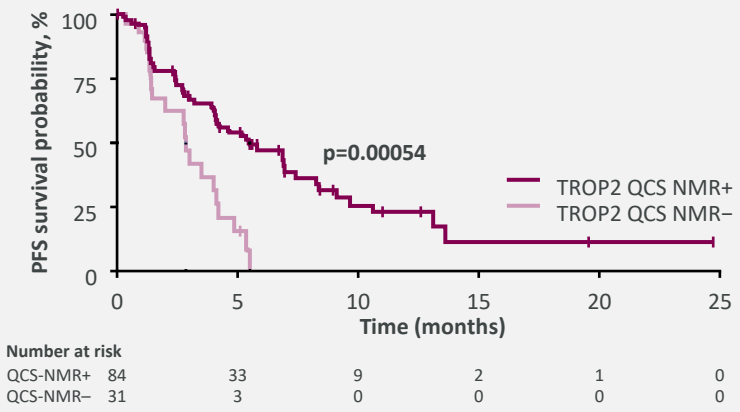
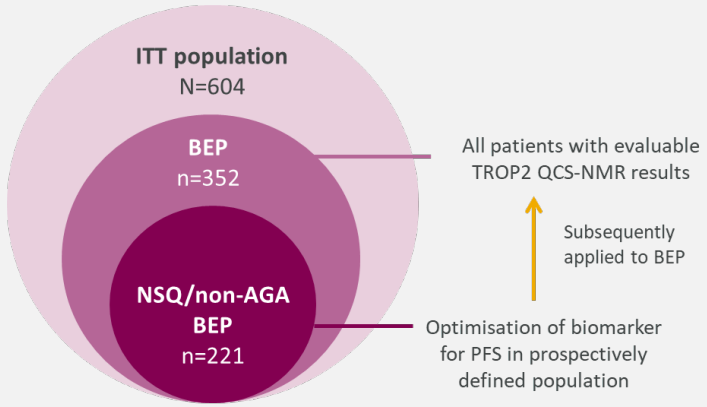
TROP2 QCS-NMR – cut-points were optimised for PFS in TROPION-Lung01 NSQ/Non-AGA patients



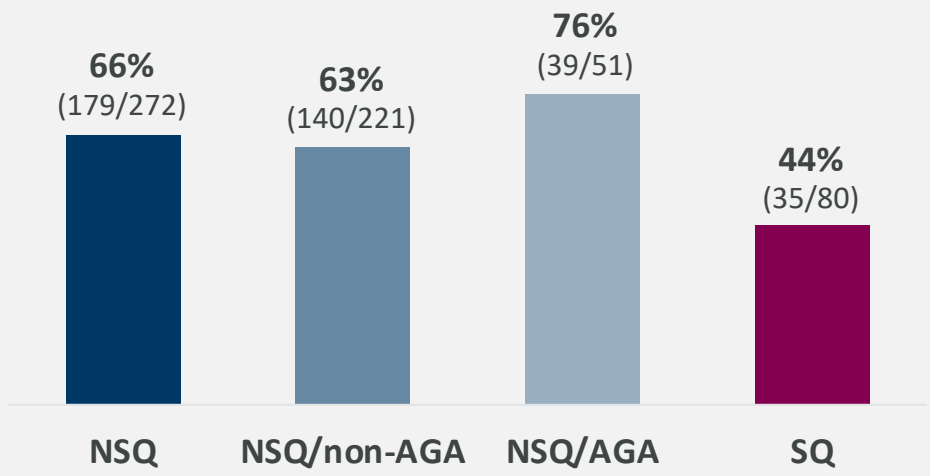
QCS-NMR biomarker identified in TROPION-PanTumor01



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

Then tested in full BEP population

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



Histology subgroup, BEP (n=352)

NSCLC prevalence

NSQ/non-AGA 43% | AGA 30% | SQ 27%

1. Spitzmueller A, Schmidt G, Triltsch N & Kapil A, 2023: A scoring method for an anti-TROP2 antibody-drug conjugate therapy. International Patent Application No. PCT/IB2023/052428. 2. 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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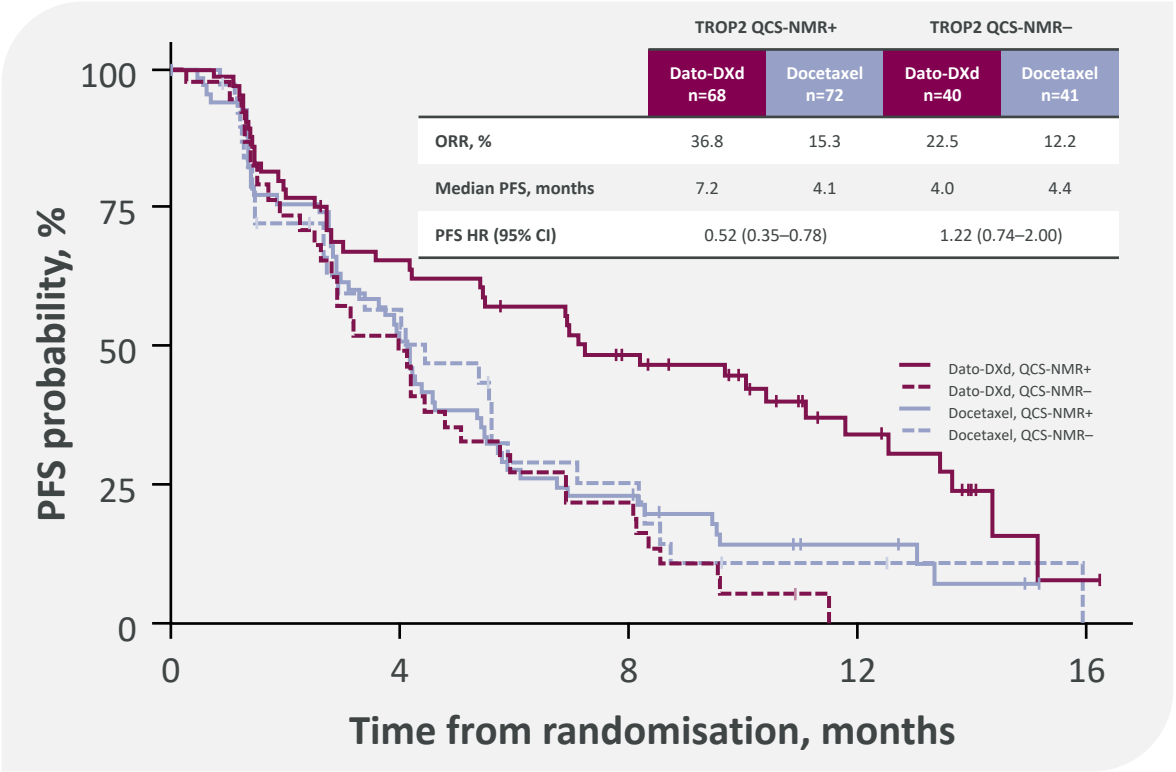
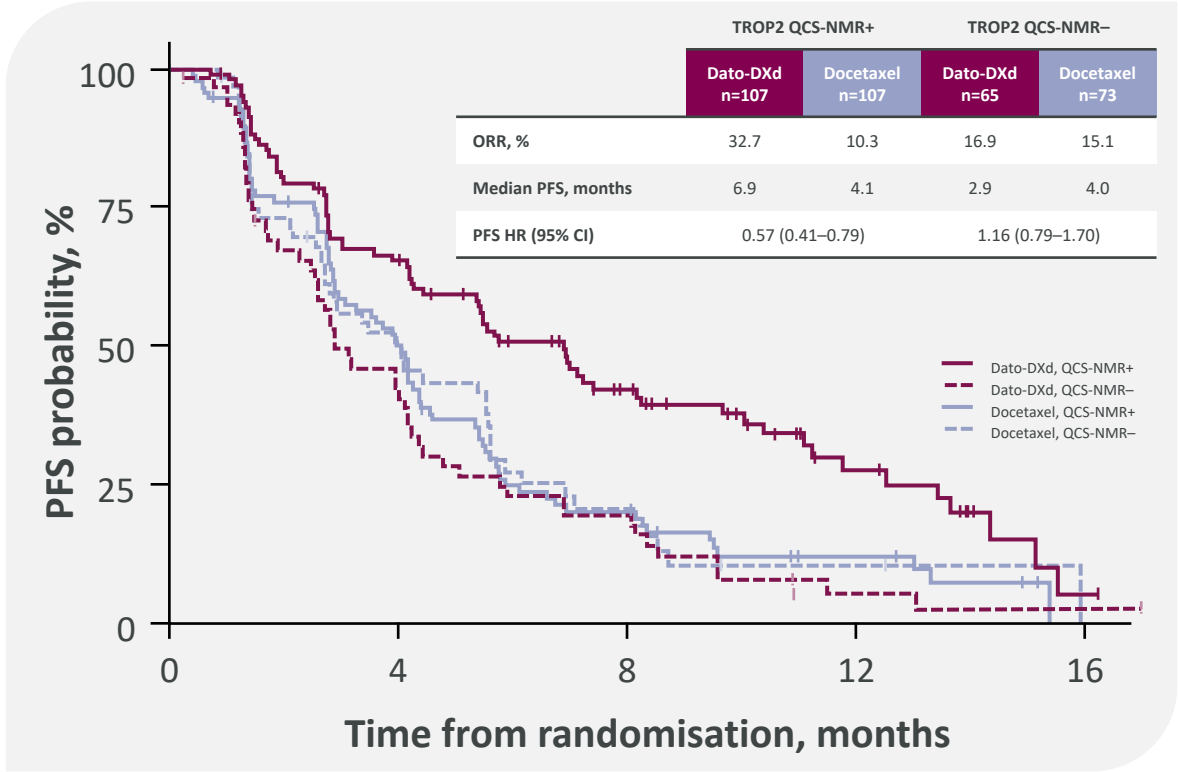
TROP2 QCS-NMR – TROP2 QCS-NMR status was predictive of efficacy in TROPION-Lung01

Non-squamous/non-AGA BEP (n=221)



Overall BEP (n=352)



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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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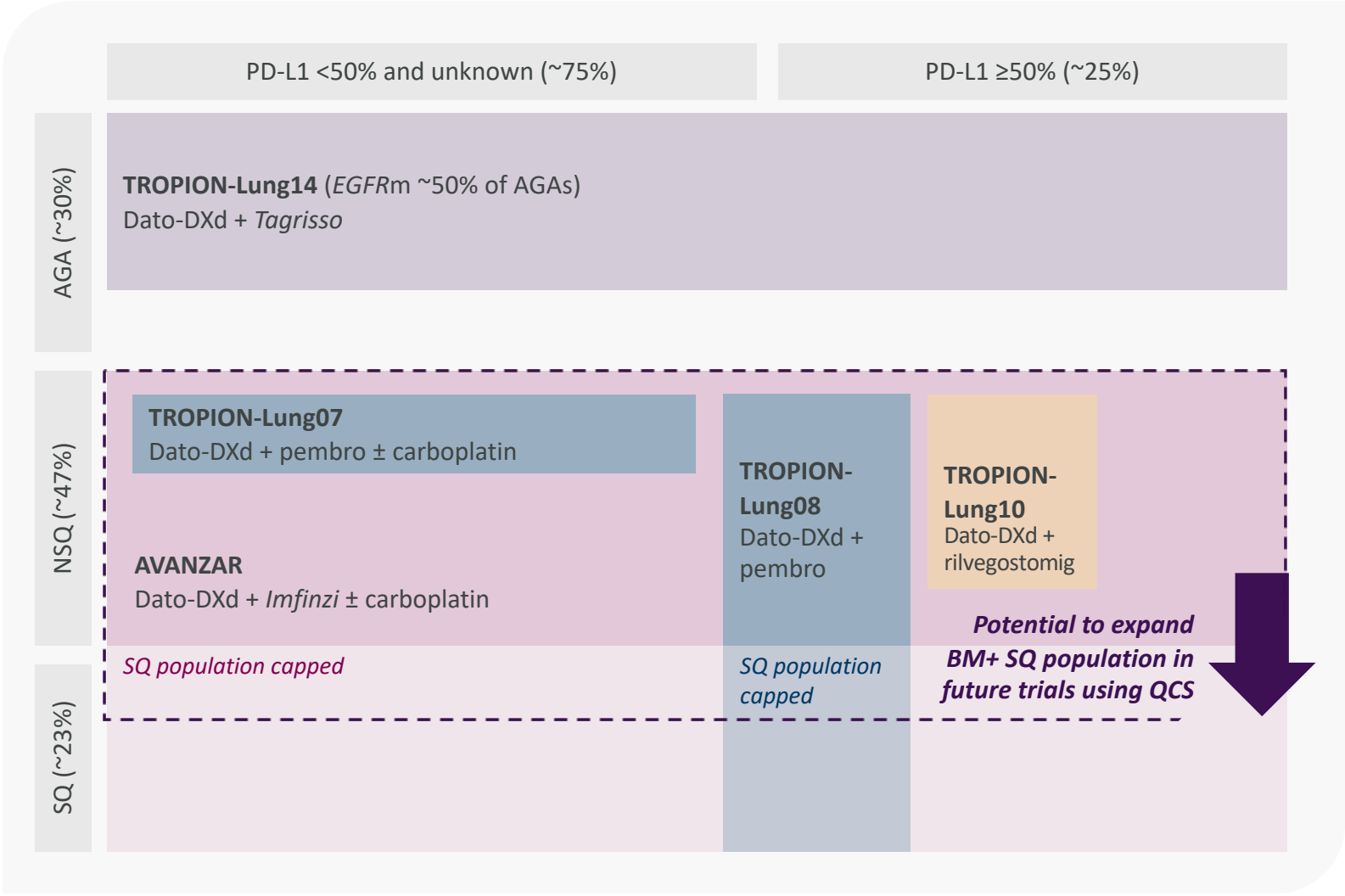
Five 1L Dato-DXd trials ongoing, representing significant share of potential $5bn+ PYR[1]



Robust 1L programme informed by learnings from TROPION-Lung01

Building on *EGFR*m evidence base

- TROPION-Lung14

Enriching for NSQ, capping SQ patients

- AVANZAR
- TROPION-Lung08

Novel IO combinations

- AVANZAR (Dato-DXd + *Imfinzi* + CTx)
- TROPION-Lung10 (Dato-DXd + rilve)

Prospective QCS-NMR biomarker validation

- AVANZAR | TROPION-Lung10

1. Non-risk adjusted Peak Year Revenue.
Collaboration partner: Daiichi Sankyo (Dato-DXd).

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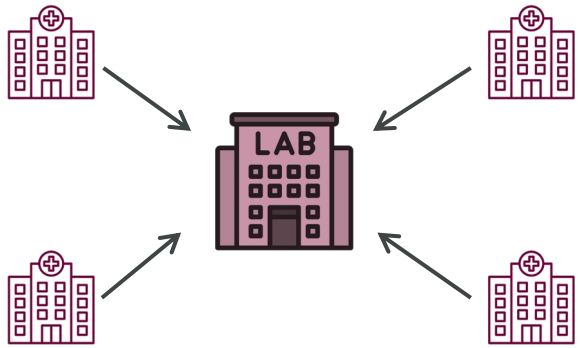
Potential real-world implementation of QCS follows same path as other companion diagnostics

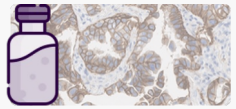
Centralised testing model



Institutions send 3 slides or blocks to central labs for analysis

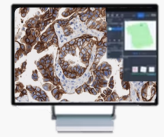
Future option for decentralised testing model

TROP2 IHC Assay



Slide scanner

Image analysis system



Institutions can elect to bring testing in-house with approved assay, slide scanner and image analysis system

AstraZeneca is partnering to develop and commercialise a TROP2-QCS companion diagnostic

NOTE: Graphics are illustrative only and do not intend to accurately reflect technology systems.

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NIAGARA

Prof. Thomas Powles

Urology Medical Oncologist, University of London
and Director of Barts Cancer Centre

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High unmet need remains in muscle-invasive bladder cancer (MIBC)

- Neoadjuvant cisplatin-based chemotherapy with radical cystectomy improves overall survival vs radical cystectomy alone and has been the recommended treatment for MIBC for the past 40 years[2-4], however, ~50% of patients experience recurrence within 3 years[5,6]
- In the setting of MIBC, immune checkpoint inhibitors as adjuvant monotherapy have demonstrated improved disease-free survival in Phase III trials in patients at high risk of recurrence after surgery (CheckMate 274,[7,8] AMBASSADOR[9])
- Perioperative immune checkpoint inhibitors could improve long-term clinical outcomes by priming anti-tumour immunity before surgery and eradicating micrometastatic disease after surgery[10]
- Perioperative *Imfinzi* was shown to be safe and efficacious in a Phase II study of MIBC[11]

NIAGARA is the first global Phase III study to evaluate a perioperative immune checkpoint inhibitor, *Imfinzi*, combined with neoadjuvant chemotherapy in cisplatin-eligible patients with MIBC

1. Yin M, et al. Oncologist 2016;21:708–715. 2. Witjes, et al. EAU Guidelines on Muscle-Invasive and Metastatic Bladder Cancer. European Association of Urology. 2024. 3. Powles T, et al. Ann Oncol. 2022;33:P244–P258. 4. Holzbeierlein J, et al. J Urol. 2024;212:3–10. 5. Pfister C, et al. J Clin Oncol. 2022;40:2013–2022. 6. Lee Y, et al. J Can Res Clin Oncol. 2021;147:3421–3429. 7. Bajorin DF, et al. N Engl J Med. 2021;384:2102–2114. 8. Opdivo SmPC. EMA. Accessed 20 Aug 2024: https://www.ema.europa.eu/en/medicines/human/EPAR/opdivo. 9. Apolo AB, et al. J Clin Oncol. 2024;42(suppl 4; abstr LBA531). 10. Vansteenkiste J, et al. Ann Oncol. 2019:30:1244–1253. 11. Cathomas R, et al. J Clin Oncol. 2023;41:5131–1539.

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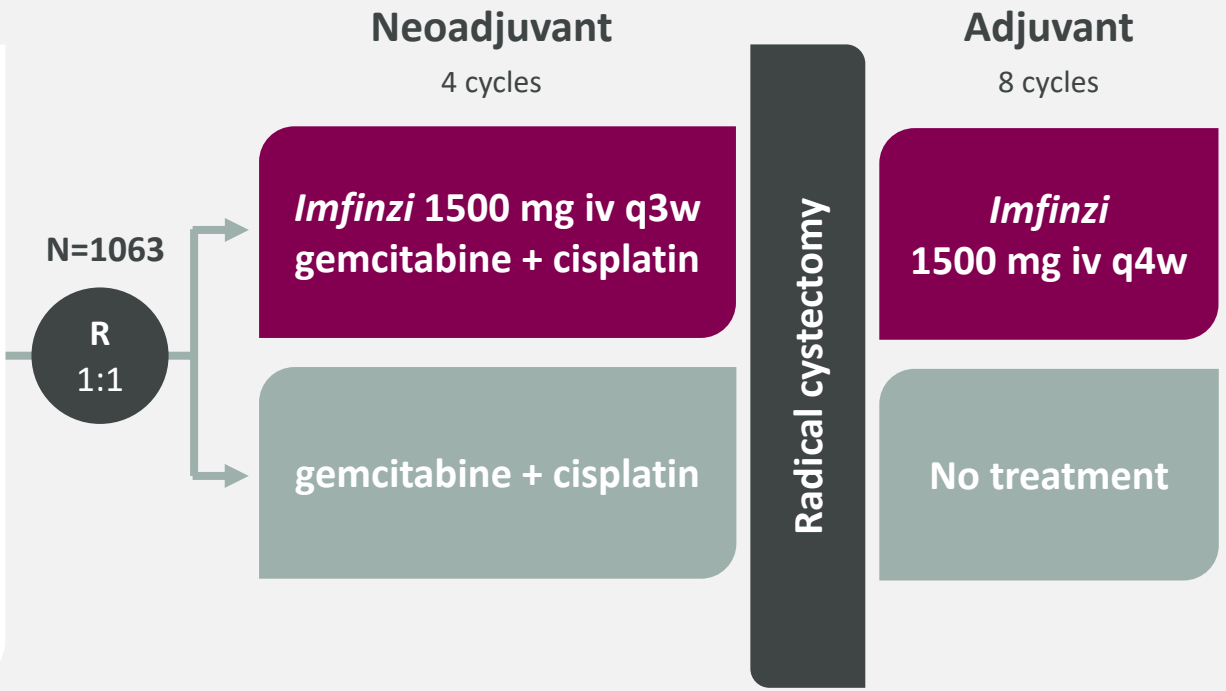
NIAGARA is Phase III trial of patients with cisplatin-eligible MIBC who were planned for radical cystectomy

- Cisplatin-eligible MIBC (cT2–T4aN0/1M0)
- UC or UC with divergent differentiation or histologic subtypes
- Evaluated and confirmed for RC
- CrCl of ≥40 mL/min



Stratified by:
- Clinical tumour stage (T2N0 vs >T2N0)
- Renal function (CrCl ≥60 mL/min vs ≥40–<60 mL/min)
- PD-L1 status (high vs low/negative expression)

Endpoints

Dual primary endpoints:
- EFS using BICR (per RECIST v1.1) or central pathology review if a biopsy was required for a suspected new lesion
- pCR by blinded central pathology review

Key secondary endpoints:
- OS

Powles TB et al. Abstract LBA5 presented at the European Society of Medical Oncology 2024.

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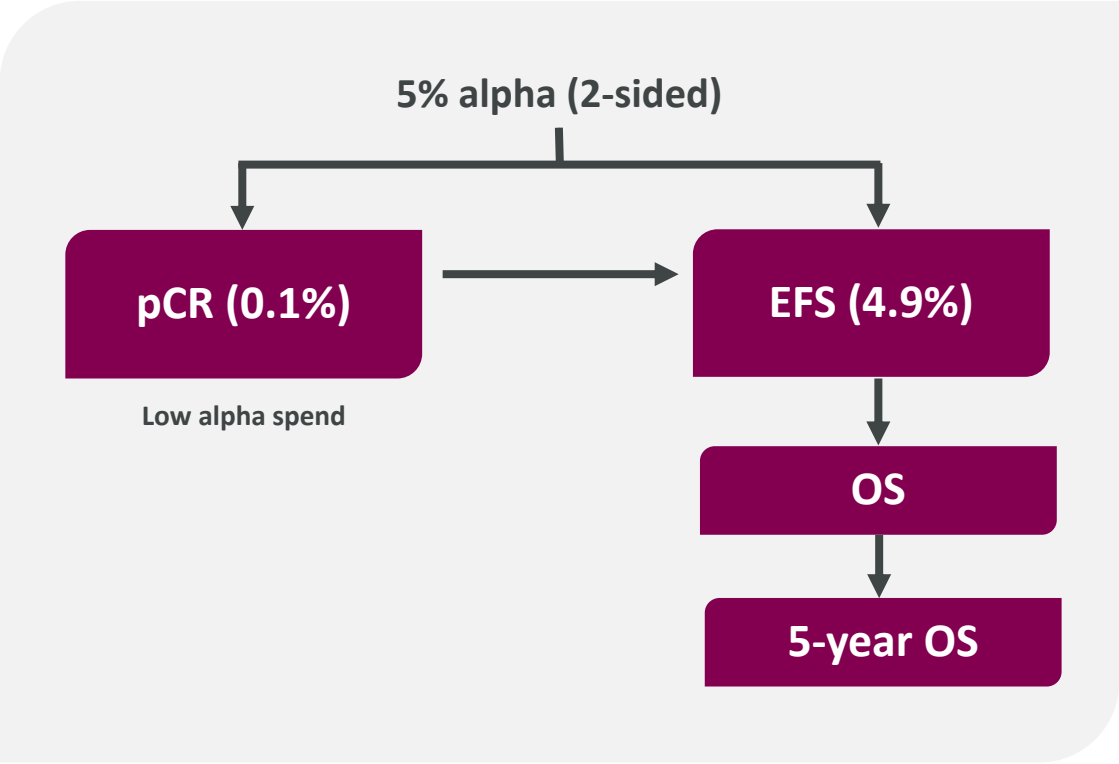
NIAGARA was designed with a multiple testing procedure across key primary and secondary endpoints

- MTP with alpha-exhaustive recycling and gatekeeping strategy used across dual primary endpoints and then secondary endpoints of OS and 5-year OS
- One pCR analysis was planned ~6 months after the last patient was randomised
- In this planned interim analysis, the estimated number of events across the 2 arms was 410 for EFS and 288 for OS
  - The actual numbers of events were 433 for EFS and 305 for OS



Study considered positive if either of the dual primary endpoints were met

Powles TB et al. Abstract LBA5 presented at the European Society of Medical Oncology 2024.

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Baseline characteristics were generally well balanced

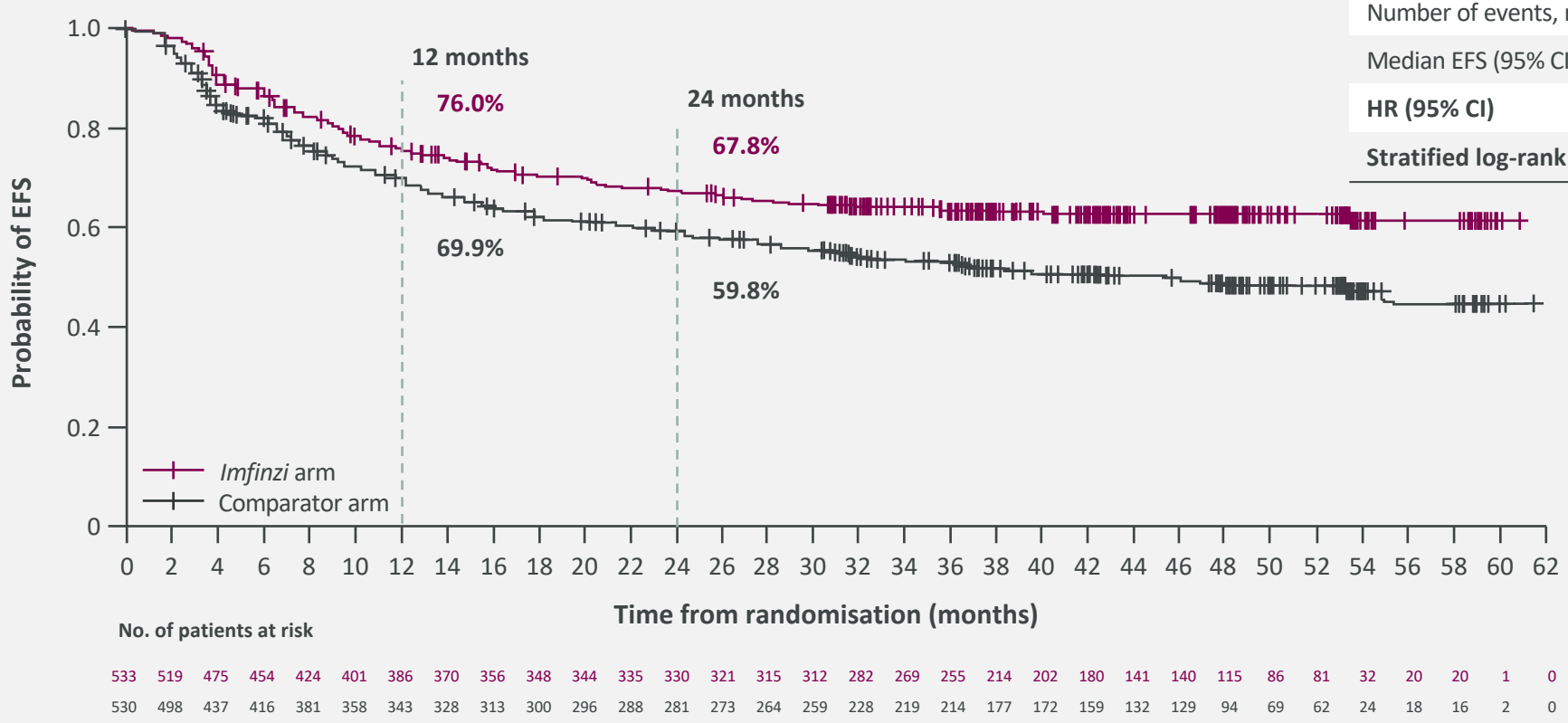
Characteristics		*Imfinzi* arm N=533	Comparator arm N=530
Age	Median, years (range)	65 (34–84)	66 (32–83)
Sex, %	Male	82	82
Race, %	White	66	68
	Asian	29	27
	Black/Other	2	1
	Not reported	3	4
ECOG PS, %	0	78	78
	1	22	22
Smoker, %	Yes (current or former)	71	75
Renal function, %	CrCl ≥60 mL/min	81	81
	CrCl ≥40–<60 mL/min	19	19

Characteristics		*Imfinzi* arm N=533	Comparator arm N=530
Tumour stage[1], %	T2N0	40	40
	>T2N0	60	60
PD-L1 expression[2], %	High	73	73
	Low/negative	27	27
Histology, %	UC	86	83
	UC with divergent differentiation or histologic subtypes	14	17
Regional lymph nodes, %	N0	95	94
	N1	5	6

1. The study design capped recruitment of patients with tumour stage T2 at 40% and CrCl of <60 mL/min to 20%. 2. Assessed with the VENTANA PD-L1 (SP263) Assay using the TC/IC25% algorithm; high PD-L1 expression was defined as ≥25% of TCs with any membrane staining or ICs staining for PD-L1 at any intensity. Data cutoff 29 Apr 2024.

Powles TB et al. Abstract LBA5 presented at the European Society of Medical Oncology 2024.

Imfinzi regimen improved event-free survival vs comparator arm



	Imfinzi arm N=533	Comparator arm N=530
Number of events, n (%)	187 (35.1)	246 (46.4)
Median EFS (95% CI), months	NR (NR–NR)	46.1 (32.2–NR)
HR (95% CI)	**0.68 (0.56–0.82)**	
Stratified log-rank *P* value[1]	**<0.0001**	

Re-analysis of pCR showed nominal statistical significance in favour of the *Imfinzi* arm

Median follow-up in censored patients: 42.3 months (range, 0.03–61.3). EFS was assessed using RECIST v1.1. EFS is defined as the time from randomisation to the first: 1) progressive disease that precluded RC; 2) recurrence after RC; 3) date of expected surgery in patients who did not undergo RC; 4) death from any cause.1. The threshold to declare statistical significance was based on a Lan-DeMets alpha spending function with O'Brien-Fleming boundary – with the observed number of events, the boundary for declaring statistical significance was 0.04123 for a 4.9% overall 2-sided alpha.

Data cutoff 29 Apr 2024.

Powles TB et al. Abstract LBA5 presented at the European Society of Medical Oncology 2024.

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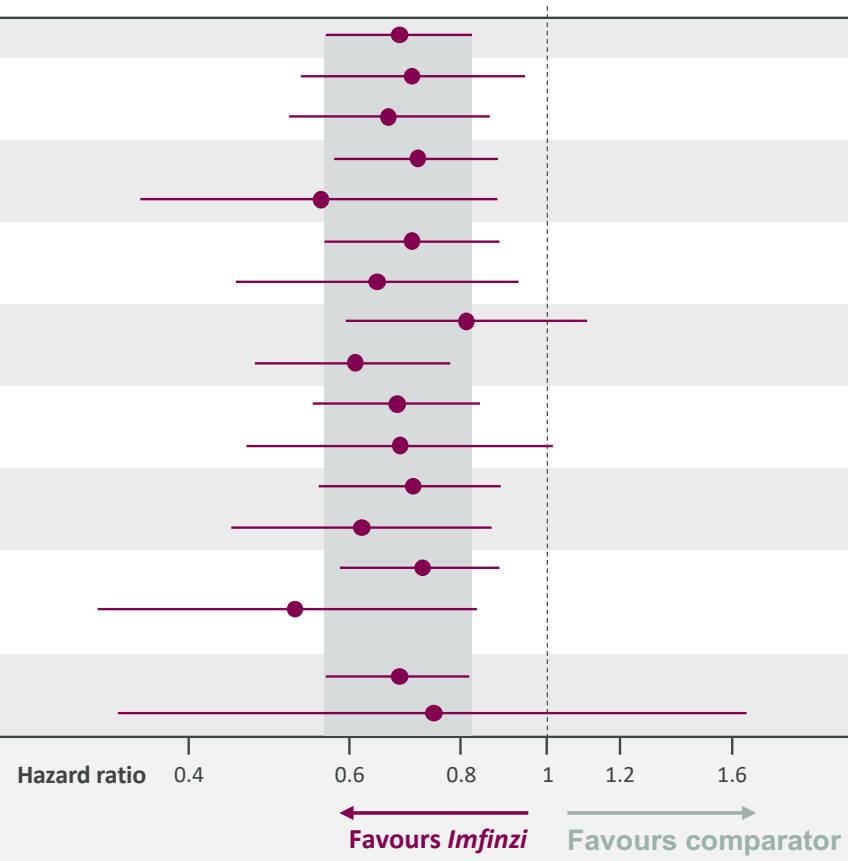
Consistent benefit for EFS across key subgroups

Subgroup	Category	N	Hazard ratio (95% CI)
All patients		1063	0.68 (0.56–0.82)
Age at randomisation	<65 years	499	0.71 (0.53–0.94)
	≥65 years	564	0.67 (0.52–0.86)
Sex	Male	870	0.71 (0.58–0.88)
	Female	193	0.56 (0.35–0.88)
Race	White	712	0.71 (0.56–0.89)
	Non-White	315	0.65 (0.45–0.92)
Tumour stage at baseline	T2N0	428	0.81 (0.60–1.10)
	>T2N0	635	0.61 (0.48–0.78)
Renal function at baseline	CrCl ≥60 mL/min	862	0.68 (0.54–0.84)
	CrCl ≥40–<60 mL/min	201	0.69 (0.46–1.01)
PD-L1 expression at baseline[1]	High	777	0.70 (0.56–0.88)
	Low/negative	286	0.62 (0.44–0.87)
Histology at baseline	UC	898	0.72 (0.59–0.89)
	UC with divergent differentiation or histologic subtypes	165	0.52 (0.32–0.84)
Lymph node positive at baseline	N0	1005	0.68 (0.56–0.83)
	N1	58	0.75 (0.33–1.64)



EFS was assessed by blinded independent central review or by central pathology review, using RECIST v1.1. The plot is of hazard ratio and 95% CI. Tan-coloured band represents the 95% CI for the overall (all patients) hazard ratio. The subgroup analyses were performed using an unstratified Cox proportional hazard model, with treatment as only covariate and ties handled by Efron approach. 1. Assessed using the VENTANA PD-L1 (SP263) Assay using the TC/IC25% algorithm; high PD-L1 expression was defined as ≥25% of TCs with any membrane staining or ICs staining for PD-L1 at any intensity. Due to observed inconsistencies between central laboratories in PD-L1 IC prevalence, but not TC prevalence, in the PD-L1 TC/IC25% algorithm, additional analyses of EFS by TC expression levels of 1% and 25% were performed and the results were consistent with those in the intent-to-treat population. Data cutoff 29 Apr 2024.

Powles TB et al. Abstract LBA5 presented at the European Society of Medical Oncology 2024.

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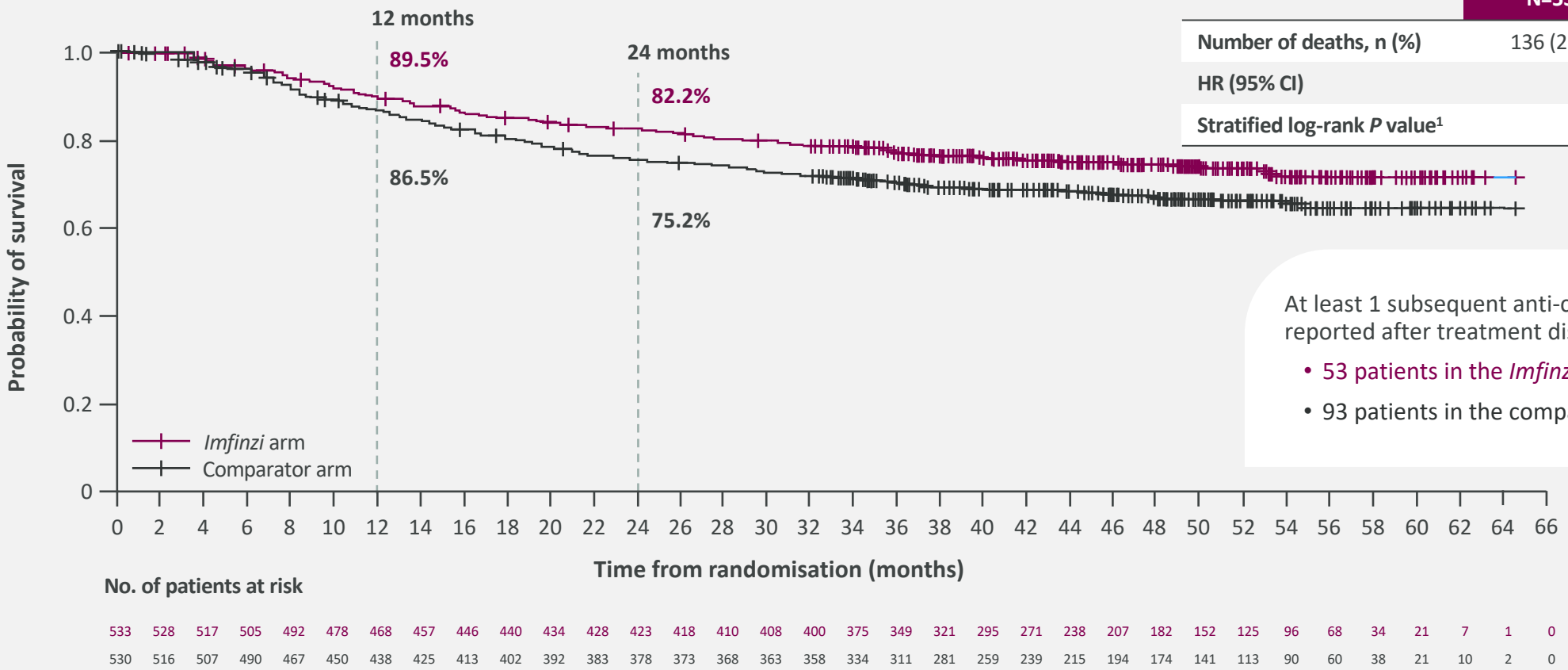
Demonstrated improvement in overall survival with *Imfinzi* arm



	Imfinzi arm N=533	Comparator arm N=530
Number of deaths, n (%)	136 (25.5)	169 (31.9)
HR (95% CI)	0.75 (0.59–0.93)	
Stratified log-rank *P* value[1]	0.0106	

At least 1 subsequent anti-cancer therapy was reported after treatment discontinuation for:

- 53 patients in the *Imfinzi* arm
- 93 patients in the comparator arm

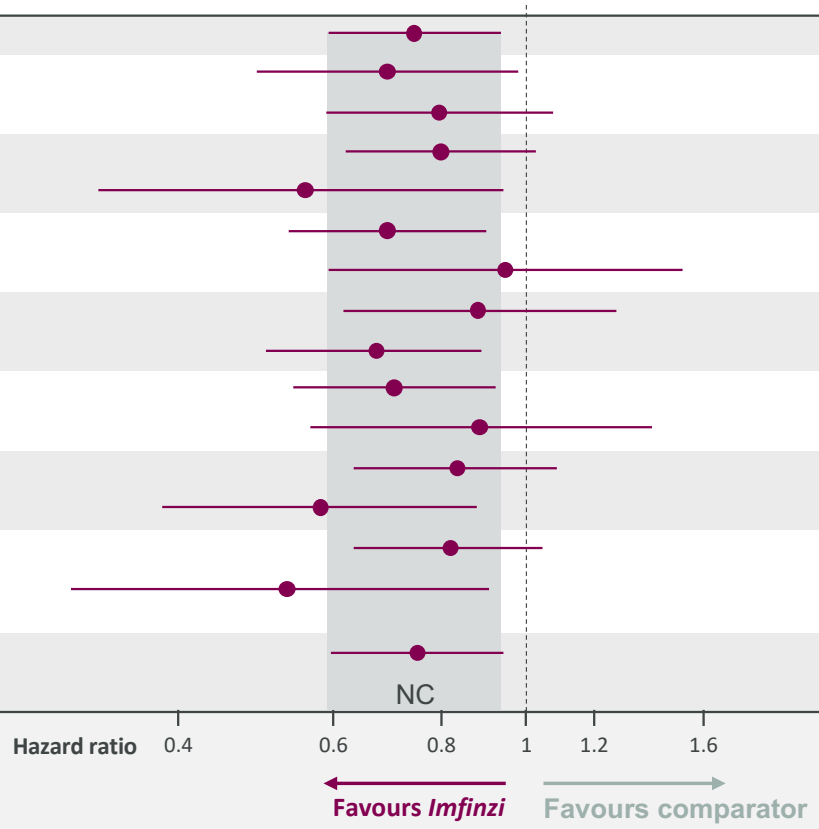
Median follow-up in censored patients: 46.3 months (range, 0.03–64.7). OS is the time from the date of randomisation until death due to any cause regardless of whether the patient withdraws from randomised therapy or receives another anti-cancer therapy. 1. The threshold for statistical significance was based on a Lan-DeMets alpha spending function with O'Brien-Fleming boundary – with the observed number of events, the boundary for declaring statistical significance was 0.01543 for a 4.9% overall 2-sided alpha. Data cutoff 29 Apr 2024.

Powles TB et al. Abstract LBA5 presented at the European Society of Medical Oncology 2024.

AstraZeneca

Consistent OS benefit across subgroups

Subgroup	Category	N	Hazard ratio (95% CI)
All patients		1063	0.75 (0.59–0.93)
Age at randomisation	<65 years	499	0.70 (0.49–0.98)
	≥65 years	564	0.80 (0.59–1.07)
Sex	Male	870	0.80 (0.62–1.02)
	Female	193	0.56 (0.32–0.94)
Race	White	712	0.70 (0.53–0.90)
	Non-White	315	0.94 (0.59–1.51)
Tumour stage at baseline	T2N0	428	0.89 (0.61–1.28)
	>T2N0	635	0.67 (0.50–0.89)
Renal function at baseline	CrCl ≥60 mL/min	862	0.70 (0.54–0.91)
	CrCl ≥40–<60 mL/min	201	0.89 (0.56–1.40)
PD-L1 expression at baseline[1]	High	777	0.83 (0.63–1.09)
	Low/negative	286	0.58 (0.38– 0.88)
Histology at baseline	UC	898	0.81 (0.63–1.04)
	UC with divergent differentiation or histologic subtypes	165	0.53 (0.30–0.91)
Lymph node positive at baseline	N0	1005	0.75 (0.59–0.94)
	N1	58	NC (NC–NC)



The plot is of hazard ratio and 95% CI. Tan-coloured band represents the 95% CI for the overall (all patients) hazard ratio. The subgroup analyses were performed using an unstratified Cox proportional hazard model, with treatment as only covariate and ties handled by Efron approach. 1. Assessed using the VENTANA PD-L1 (SP263) Assay using the TC/IC25% algorithm; high PD-L1 expression was defined as ≥25% of TCs with any membrane staining or ICs staining for PD-L1 at any intensity.

Data cutoff 29 Apr 2024.

Powles TB et al. Abstract LBA5 presented at the European Society of Medical Oncology 2024.

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Imfinzi regimen generally well tolerated

Overall study period (unless otherwise stated)	***Imfinzi* arm N=530**	**Comparator arm N=526**
AEs of any cause, n (%)	527 (99)	525 (100)
Maximum grade 3 or 4	368 (69)	355 (68)
Serious AEs	326 (62)	287 (55)
Outcome of death	27 (5)	29 (6)
Leading to discontinuation of study treatment	112 (21)	80 (15)
Leading to discontinuation of neoadjuvant durvalumab	50 (9)	---
Leading to discontinuation of NAC	72 (14)	80 (15)
Leading to patient not undergoing RC	6 (1)	7 (1)
Leading to delay in surgery[1]	9 (2)	6 (1)
Leading to discontinuation of adjuvant durvalumab	30/383[2] (8)	---
AEs possibly related to any treatment, n (%)[3]	502 (95)	487 (93)
Maximum grade 3 or 4 (treatment related)	215 (41)	215 (41)
Outcome of death (treatment related)	3 (0.6)	3 (0.6)
Any-grade immune-mediated AEs	111 (21)	16 (3)

The safety population includes all patients who received treatment. 1. Recommended timeframe for RC was within 56 days after the last dose of NAC. 2. In patients who started adjuvant durvalumab. 3. Investigator-assessed causality. The overall study period includes AEs that occurred between the first dose of study treatment, and whichever occurred first: 1) 90 days after the last dose of treatment, surgery, or last adjuvant visit; 2) date of first dose of subsequent anti-cancer therapy; or 3) data cutoff date.

Data cutoff 29 Apr 2024.

Powles TB et al. Abstract LBA5 presented at the European Society of Medical Oncology 2024.

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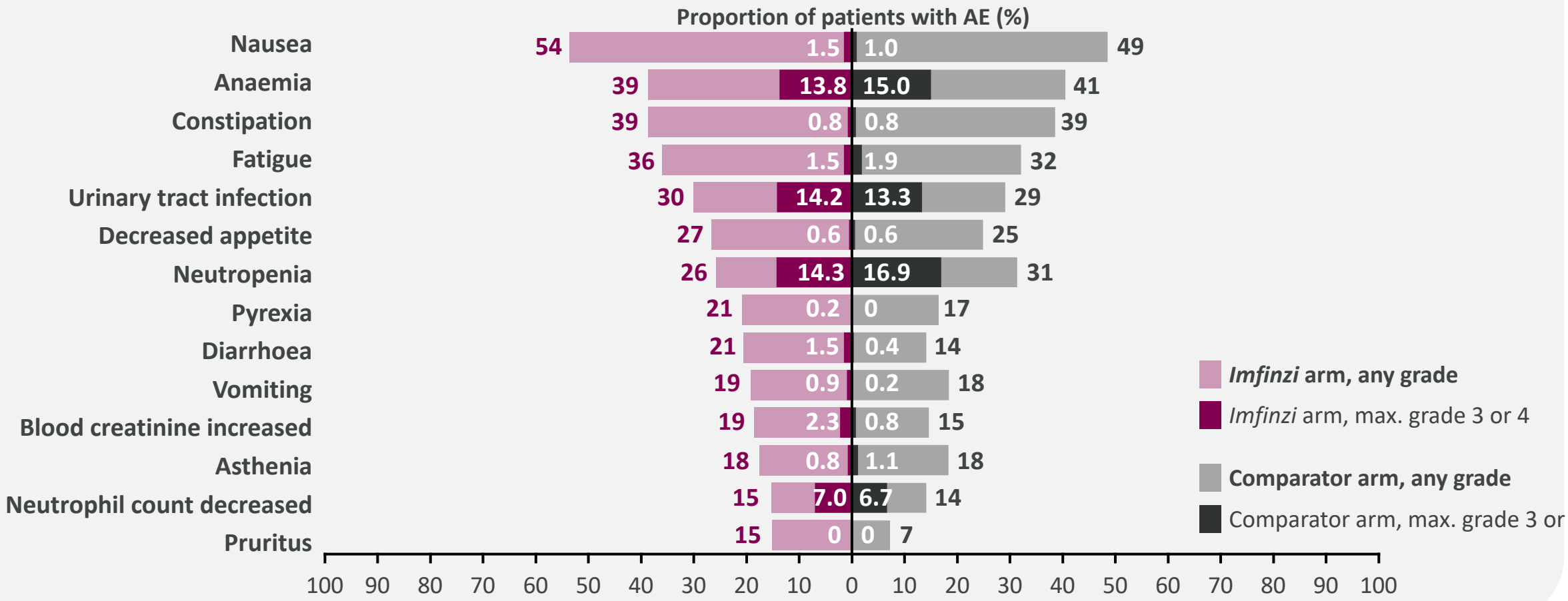
Common AEs in line with known profile for the regimen



All-causality AEs reported for ≥15% of patients in the safety population from either arm in the overall study period are shown. The overall period includes AEs that occurred between the first dose of study treatment, and whichever occurred first: 1) 90 days after the last dose of treatment, surgery, or last adjuvant visit; 2) date of first dose of subsequent anti-cancer therapy; or 3) data cutoff date. Data cutoff 29 Apr 2024.

Powles TB et al. Abstract LBA5 presented at the European Society of Medical Oncology 2024.

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Conclusions

- NIAGARA is the first Phase 3 perioperative immunotherapy study in MIBC and has demonstrated a statistically significant and clinically meaningful improvement in EFS and OS
- EFS and OS benefits with *Imfinzi* were consistent across subgroups
- The pCR results and the significant OS benefit support the perioperative approach
- Addition of perioperative *Imfinzi* to neoadjuvant chemotherapy was tolerable and manageable, with no new safety signals
- Neoadjuvant *Imfinzi* did not delay surgery and did not impact the ability of patients to undergo/complete surgery

NIAGARA supports perioperative *Imfinzi* + neoadjuvant chemotherapy as a potential new treatment option for patients with cisplatin-eligible MIBC

Powles TB et al. Abstract LBA5 presented at the European Society of Medical Oncology 2024.

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AstraZeneca in bladder cancer

Sunil Verma

SVP, Global Head, Oncology Franchise

AstraZeneca

NIAGARA establishes AstraZeneca as a key player in bladder cancer

$1bn+*

NIAGARA addresses significant need in MIBC

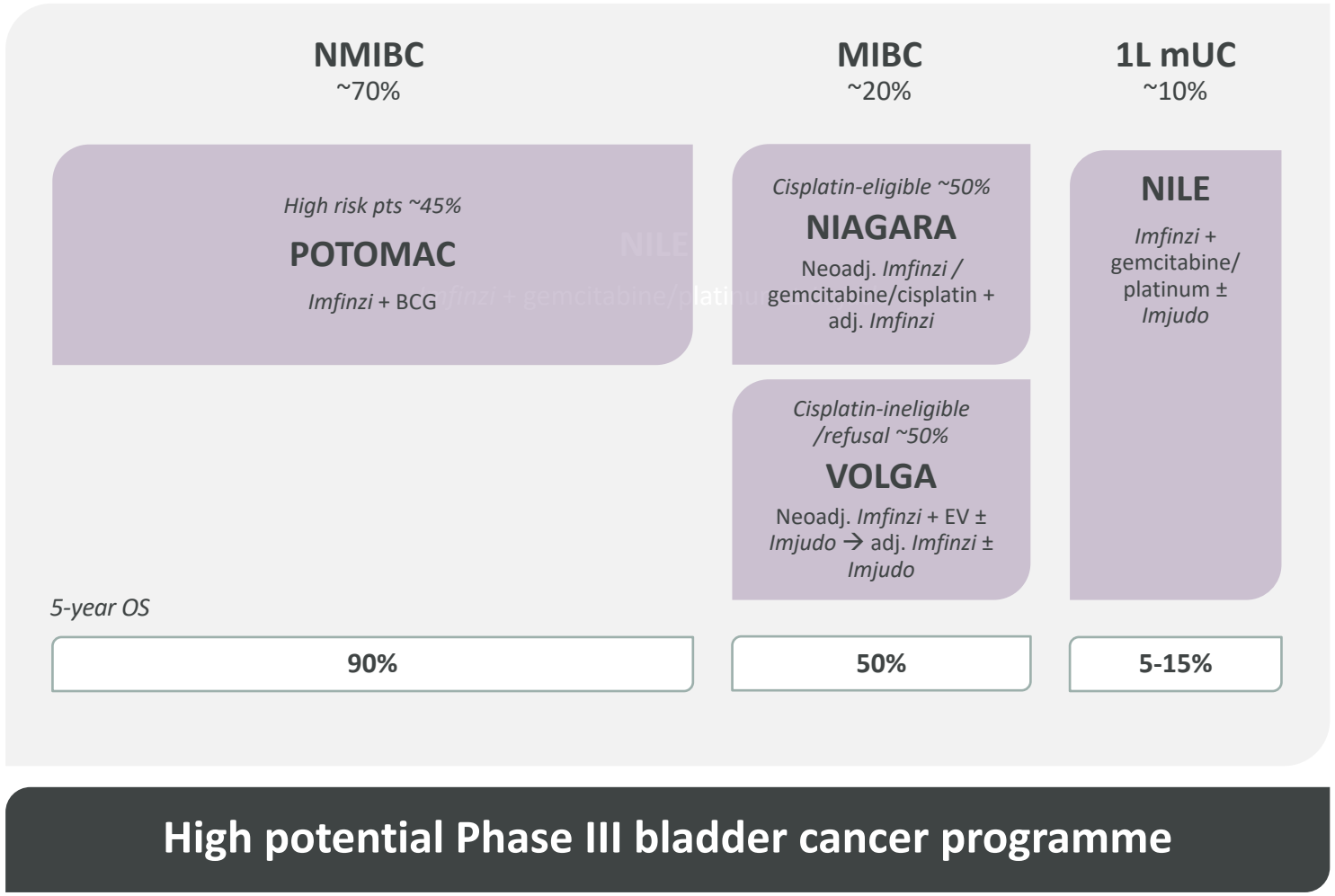
~120k MIBC patients across G8

Current SoC

Radical cystectomy

Neoadj. CTx *cisplatin-based* → **High risk pts: adjuvant IO**

- ~5-8% survival benefit with cisplatin-based neoadjuvant CTx vs no neoadjuvant CTx
  - ~50% patients are cisplatin-eligible
- ~50% of patients recur within 5 years of radical cystecomy



High potential Phase III bladder cancer programme

*Non-risk adjusted peak year revenue opportunity for *Imfinzi* bladder programme.

For cisplatin ineligible patients, some guidelines recommend gemcitabine ± paclitaxel (NCCN bladder cancer guidelines V1 2022).

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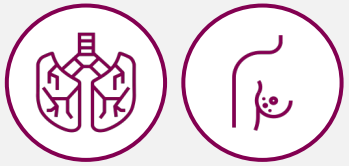
Multiple reasons for confidence in NIAGARA success

IO and CTx approach demonstrated efficacy in early-stage disease

- PACIFIC
- AEGEAN
- ADRIATIC
- MATTERHORN
- NIAGARA

HCPs confident using perioperative IO

Imfinzi well characterised safety profile



Experience from other indications e.g. lung, breast

Straightforward implementation by MDT anticipated

HCP and patient confidence in upfront systemic treatment and overall treatment plan

Role for both oncologists and surgeons in treatment

AZN established relationships with GU community

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Q&A Session I

AstraZeneca @ WCLC and ESMO 2024

ASTRAZENECA LEADERSHIP

Pascal Soriot
CHIEF EXECUTIVE OFFICER

Dave Fredrickson
EXECUTIVE VICE PRESIDENT, ONCOLOGY BUSINESS

Susan Galbraith
EXECUTIVE VICE PRESIDENT, ONCOLOGY R&D

Cristian Massacesi
CHIEF MEDICAL OFFICER & ONCOLOGY CHIEF DEVELOPMENT OFFICER

Matt Hellmann
VP, EARLY ONCOLOGY DEVELOPMENT

Sunil Verma
SVP, GLOBAL HEAD, ONCOLOGY FRANCHISE

KEY EXTERNAL EXPERTS

Prof. Thomas Powles
UROLOGY MEDICAL ONCOLOGIST, UNIVERSITY OF LONDON AND DIRECTOR OF BARTS CANCER CENTRE

AstraZeneca

Driving the ADC revolution

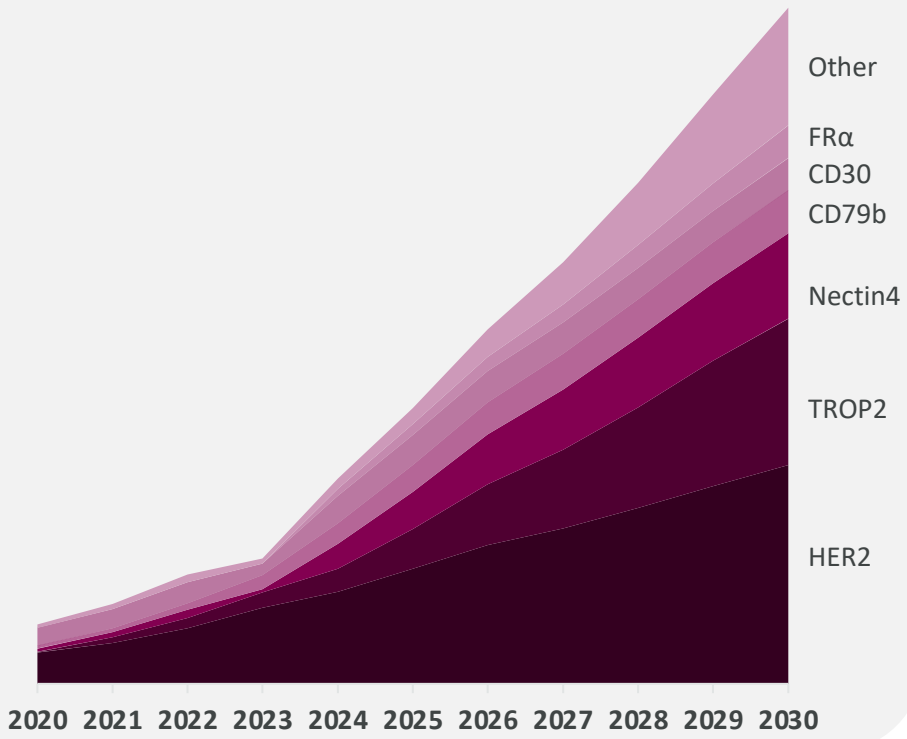
Matt Hellmann

VP, Early Oncology Development

AstraZeneca

Expanding leadership position in growing ADC market

Growing global ADC market, estimated externally to exceed $45bn by 2030[1]



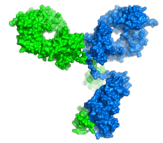
Well positioned to lead with different payloads and opportunity for combinations across portfolio

Payloads
match disease biology

TOPO1 | MTIs | + alternative MoAs

Antibody engineering
with differentiated novel mAbs and chemistry

nnAA/ss conjugation



bispecific mAb

Novel targets
via surface proteomics

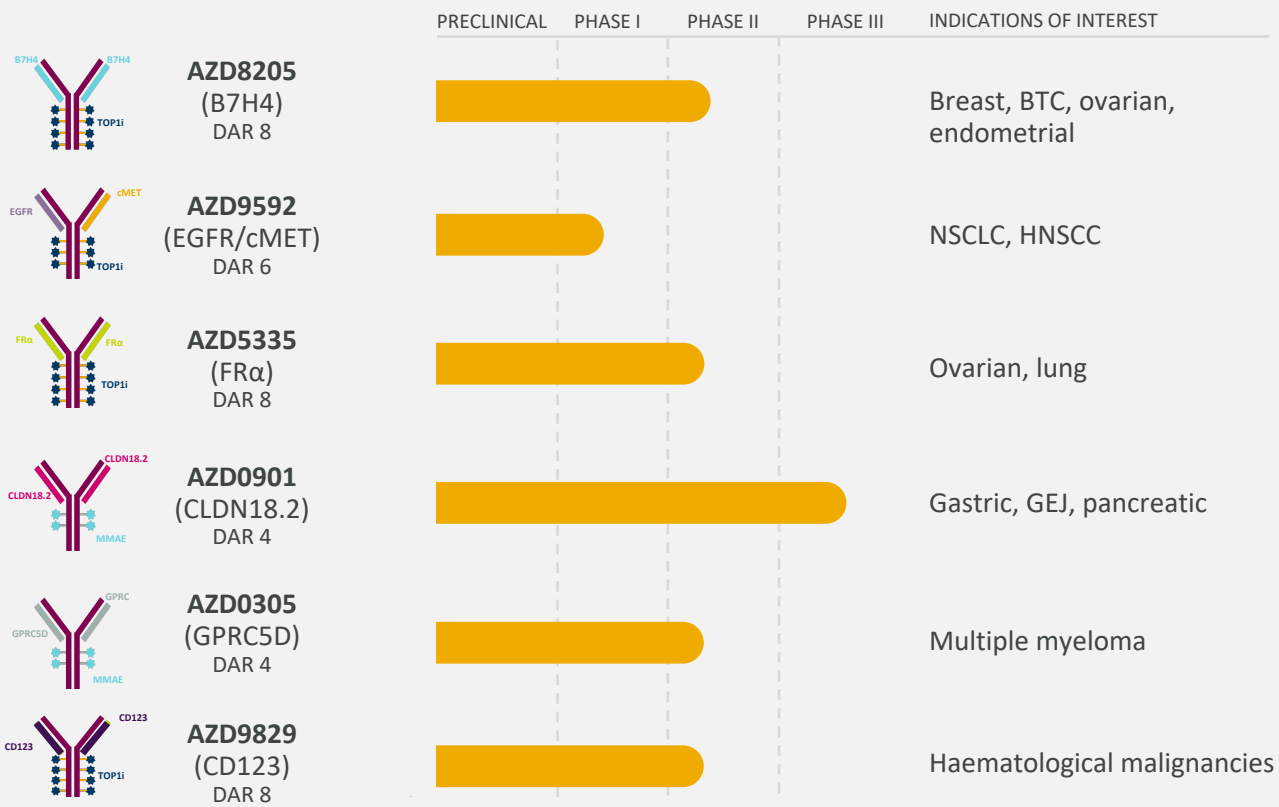
Tumour + normal + PDx models + Tox species

1. Q2 2024 Global Data (external data source), accessed September 2024.

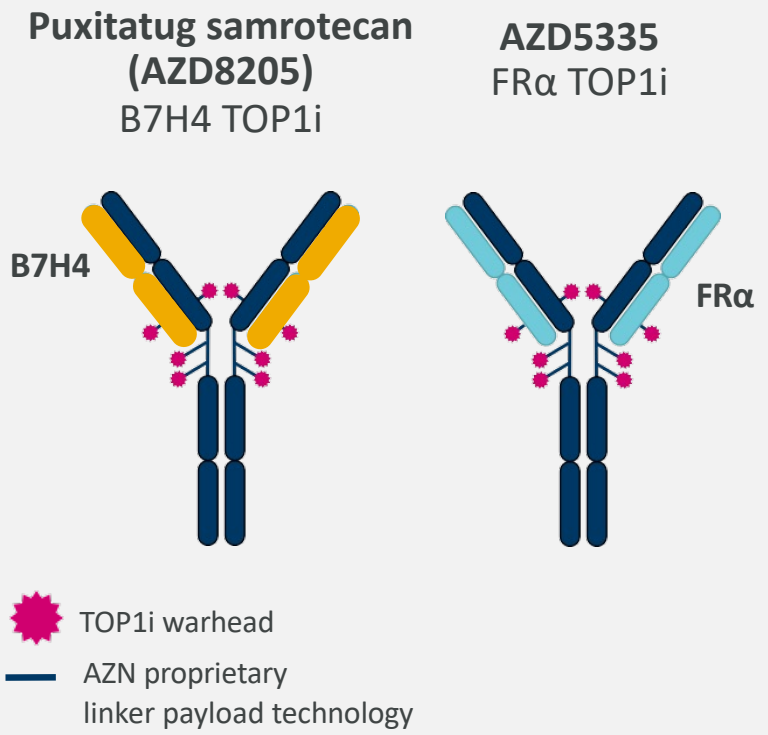
AstraZeneca

AstraZeneca ADC programme designed to address unmet need across multiple tumour types

Six wholly owned clinical-stage ADCs



Novel ADCs at ESMO 2024

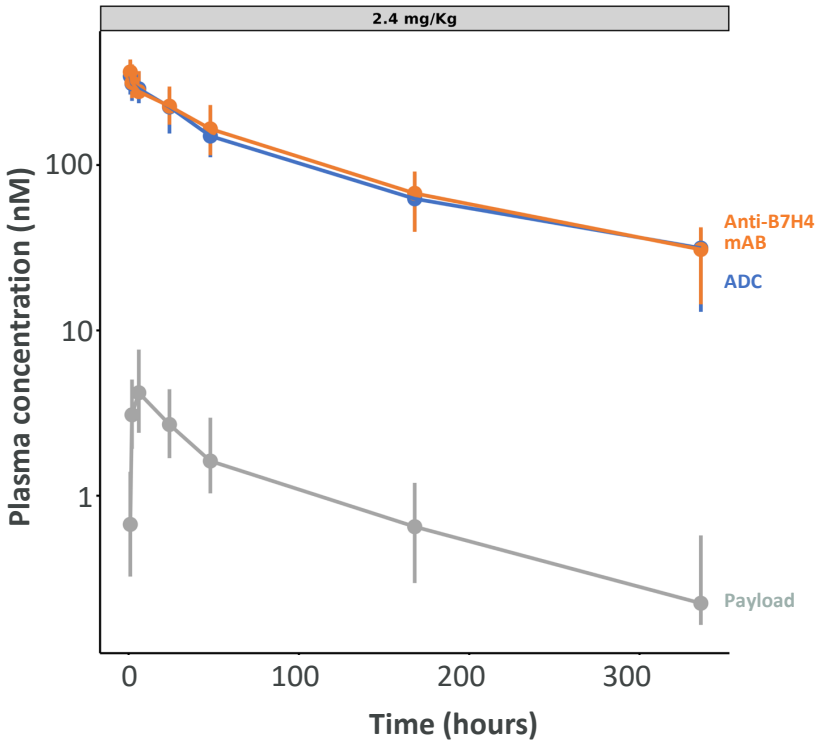


1. Meric-Bernstam F et al. Abstract 606O presented at the European Society of Medical Oncology 2024. 2. Shapira-Frommer R et al. Abstract 754P presented at the European Society of Medical Oncology 2024.

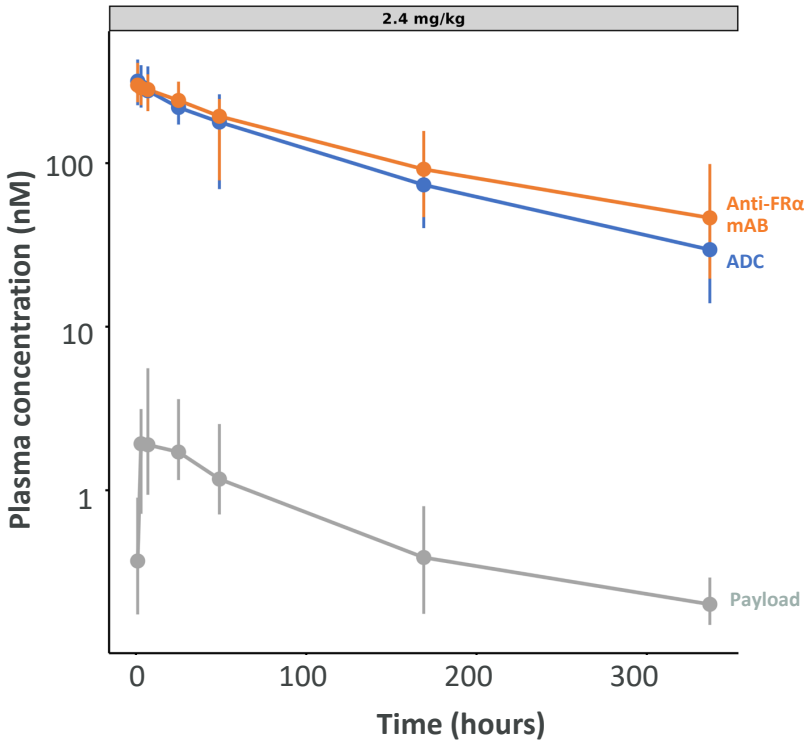
AstraZeneca

PK profiles of AZD8205 and AZD5335 demonstrate stability of linker in AstraZeneca platform and support Q3W dosing

AZD8205[1]



AZD5335[2]



Similar ADC and mAb profiles confirm AstraZeneca platform linker-payload stability

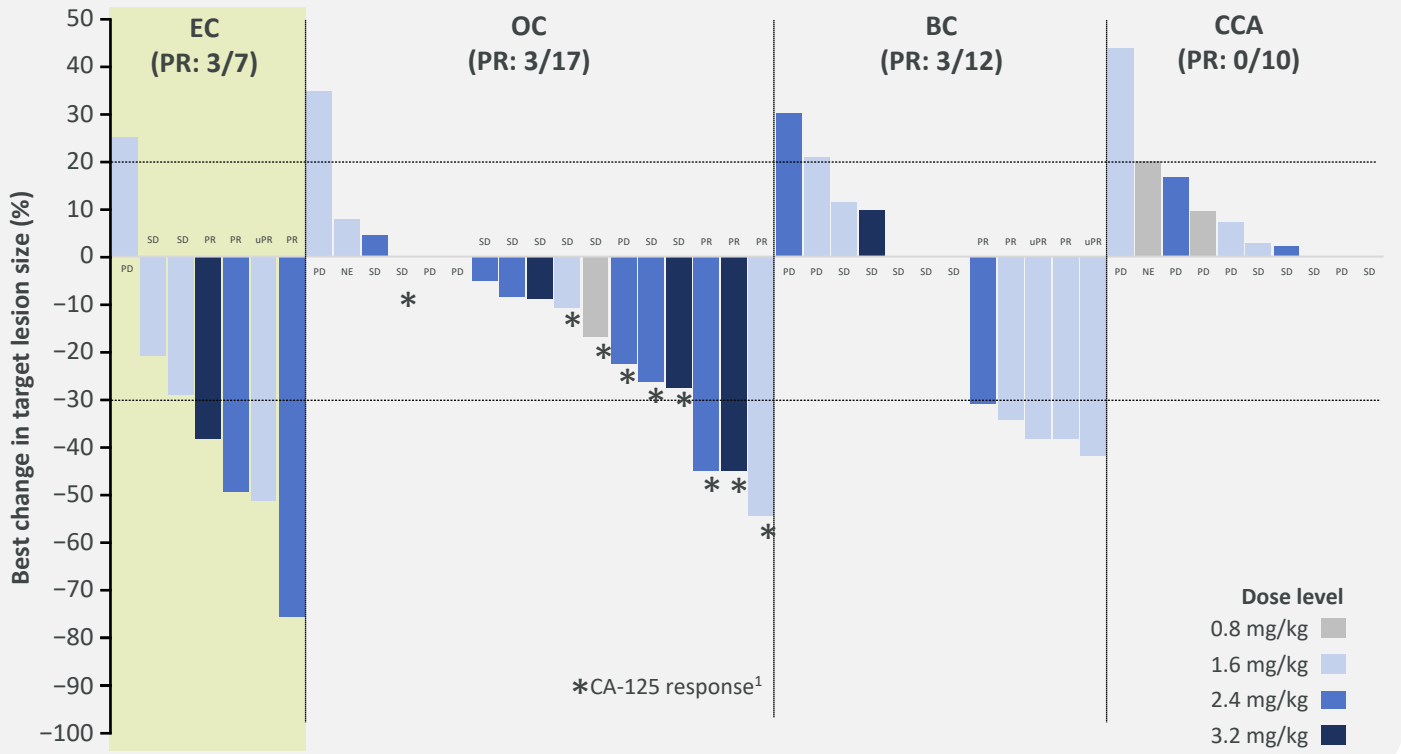
Profile supports Q3W dosing for both AZD8205 and AZD5335

1. Meric-Bernstam F et al. Abstract 606O presented at the European Society of Medical Oncology 2024. 2. Shapira-Frommer R et al. Abstract 754P presented at the European Society of Medical Oncology 2024.

AstraZeneca

AZD8205 – promising efficacy in heavily pre-treated patients with ovarian, breast and endometrial cancer

20.5% ORR with doses ≥1.6 mg/kg across B7-H4 levels



- B7H4 exciting new target highly expressed in range of solid tumours
- Preliminary efficacy across B7H4 expression and dose levels
- Toxicity as expected for warhead with no observed stomatitis
- At 1.6-2.4 mg/kg dose, manageable haematological toxicity
- Phase II dose optimisation, rilvegostomig combination and QCS analyses ongoing

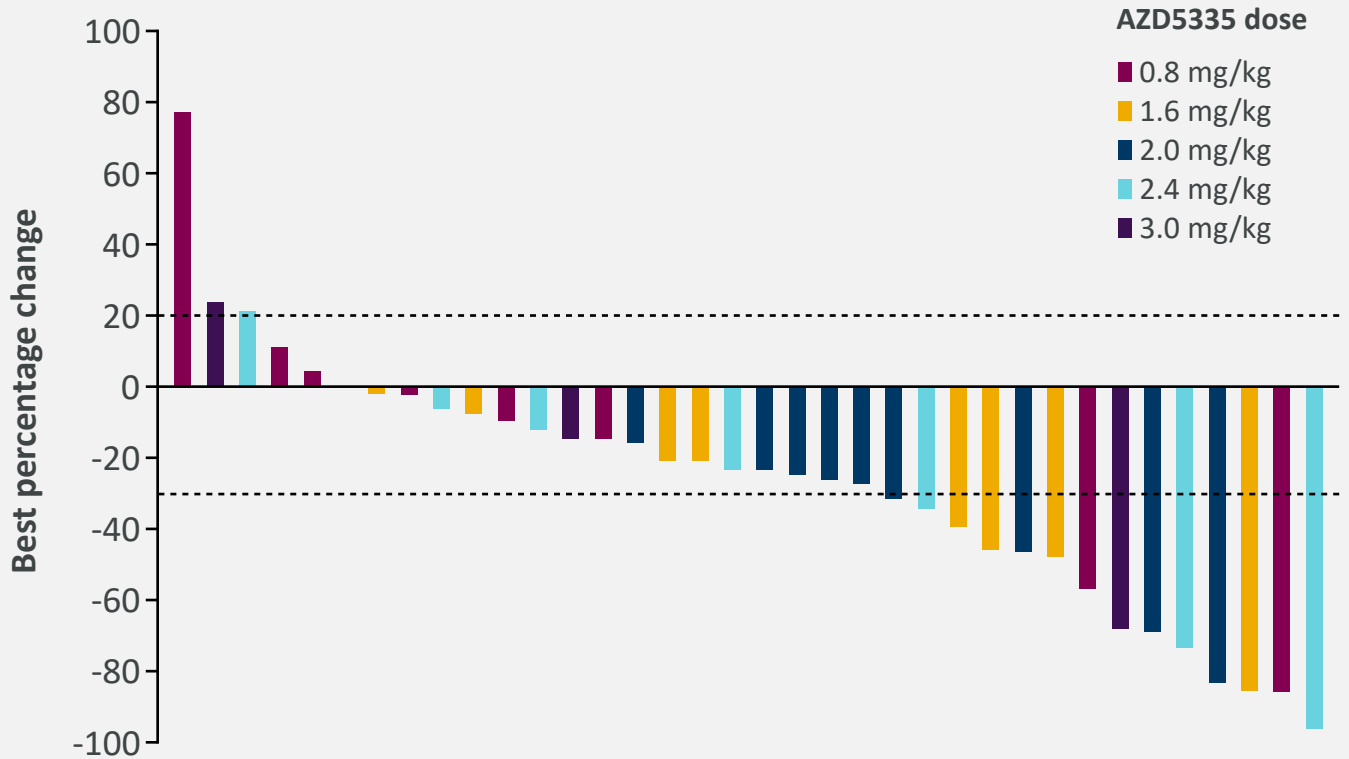
All doses are IV Q3W. Response based on RECIST v1.1 (response and progression defined as -30% and +20% change from baseline, respectively). 1. Patients with baseline CA-125 value and ≥1 post-baseline CA-125 value were included. According to the GCIG criteria, patients can be evaluated for an investigator-assessed response based on RECIST v1.1 only if they have a baseline sample that is ≥2x the upper limit of normal, obtained within 2 weeks prior to starting treatment. CA-125 response is defined as a ≥50% reduction in CA-125 levels from a pretreatment sample. The response must be confirmed and maintained for ≥28 days.

Meric-Bernstam F et al. Abstract 606O presented at the European Society of Medical Oncology 2024.

AstraZeneca

AZD5335 – differentiated efficacy in heavily pretreated patients with platinum-resistant relapsed ovarian cancer

Overall ORR[1] of 34.2% with responses observed at all doses



- 55.6% cORR in FRα high at doses ≥1.6 mg/kg
- 41.7% ORR in FRα low at doses ≥1.6 mg/kg
- Manageable safety profile – no stomatitis, ILD, ocular toxicity or neuropathy
- Dose expansions at 1.6-2.4 mg/kg doses in FONTANA
- Future opportunities in ovarian and lung cancer

Three patients were non-evaluable for radiological response and are therefore not included. Response based on RECIST v1.1 (response and progression defined as -30% and +20% change from baseline, respectively).

1. Confirmed and unconfirmed

Shapira-Frommer R et al. Abstract 754P presented at the European Society of Medical Oncology 2024.

AstraZeneca

Next-generation IO bispecifics

Cristian Massacesi
Chief Medical Officer & Oncology Chief Development Officer

AstraZeneca

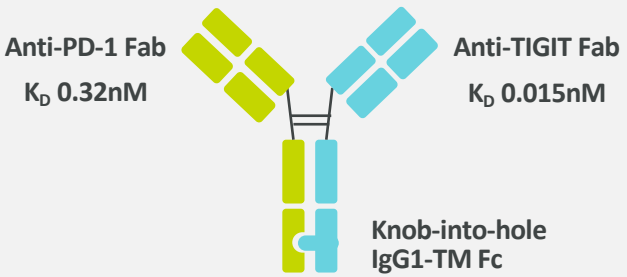
Leading in next-wave IO bispecifics

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

Unique bispecific mechanism of action

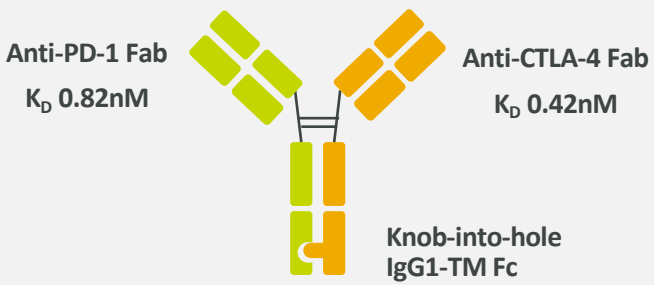
cooperative binding in the presence of both checkpoint inhibitors

rilvegostomig



Fc attenuated triple-mutant IgG1 potentially confers improved safety profile[1] with low discontinuation rate and limited Gr3+ AEs observed

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

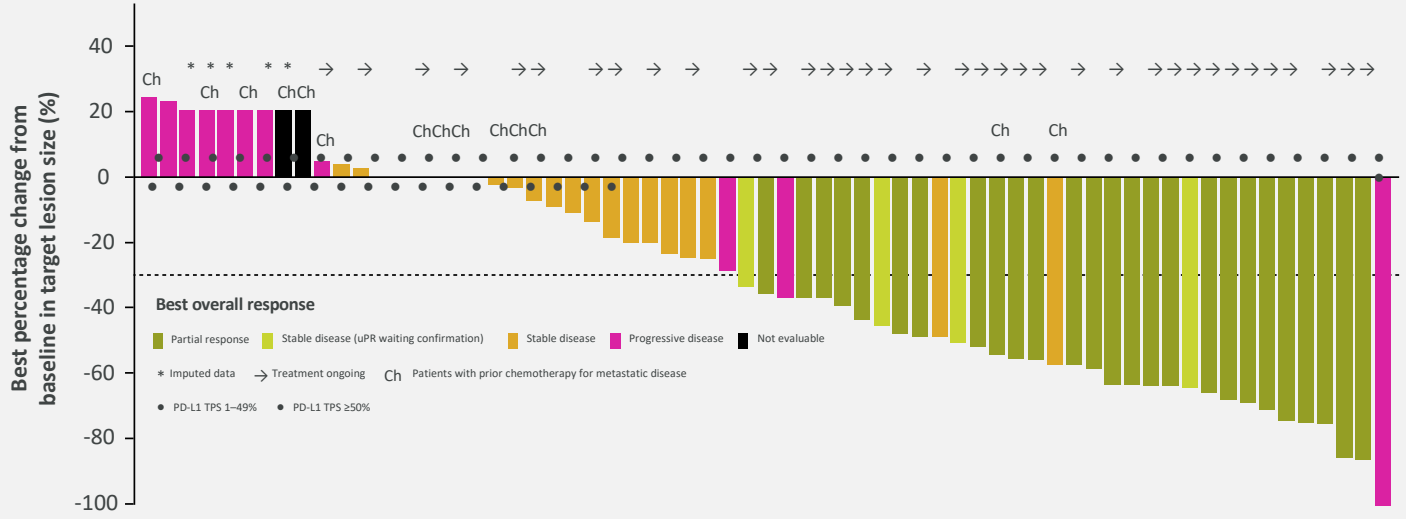
- **eVOLVE-Lung02** – volru + CTx – 1L NSCLC PD-L1 <50%
- **eVOLVE-meso** – volru + CTx – mesothelioma
- **eVOLVE-cervical** – volru – high-risk LA cervical
- **eVOLVE-HNSCC** – volru – LA u/r HNSCC

1. Verus Fc active TIGIT compounds

AstraZeneca

rilvegostomig – encouraging preliminary response rates and durable responses in CPI-naïve metastatic NSCLC

Responses observed across CPI-naïve NSCLC



n (%)	PD-L1 1–49% TPS (n=31)	PD-L1 ≥50% TPS (n=34)
ORR, confirmed + pending [95%CI]	**9 (29%)** [14.2, 48.0]	**21 (61.8%)** [43.6, 77.8]
Continuing treatment	12 (38.7%)	24 (70.6%)

Durable responses across both PD-L1 TPS ≥50% and 1-49% subgroups

rilvegostomig well tolerated – no Grade ≥4 TRAEs, low rates of discontinuation and Grade ≥3 imAEs

At data cutoff treatment was ongoing in 55% of patients on 750 mg dose[1]

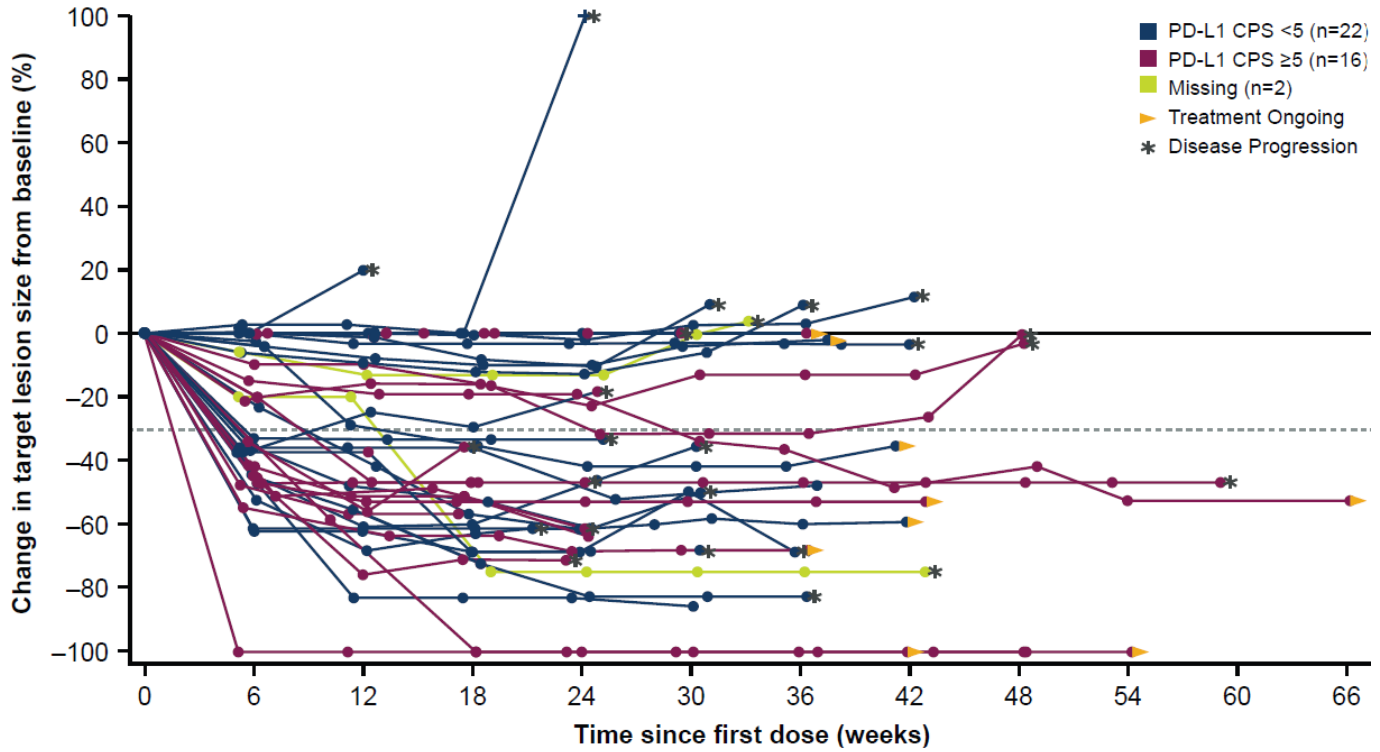
Supports 750mg Q3W dose for Phase III trials, including TROPION-Lung10

1. Data cut off: 7 July 2024. Treatment ongoing in 36 of 65 patients.
Hiltermann, TJN et al. Abstract OA11.03 presented at the 2024 World Conference on Lung Cancer.
Collaboration partner: Compugen (rilvegostomig).

AstraZeneca

rilvegostomig – differentiated safety profile enables CTx combination in gastric and GEJ cancers

Durable responses for rilvegostomig + CTx in locally advanced/ metastatic gastric and GEJ cancers



Favourable 62.5% cORR, mPFS of 8.3 months across ITT

In CPS ≥5 cORR was 81.3% and mPFS was 11.1 months

10% Grade ≥3 AEs related to rilvegostomig, 2.5% discontinuation rate of rilvegostomig

1. Data cut off: 4 July 2024. Treatment ongoing in 9 of 40 patients.
Rivera Herrero F et al. Abstract 1422P presented at the European Society of Medical Oncology 2024.
Collaboration partner: Compugen (rilvegostomig).

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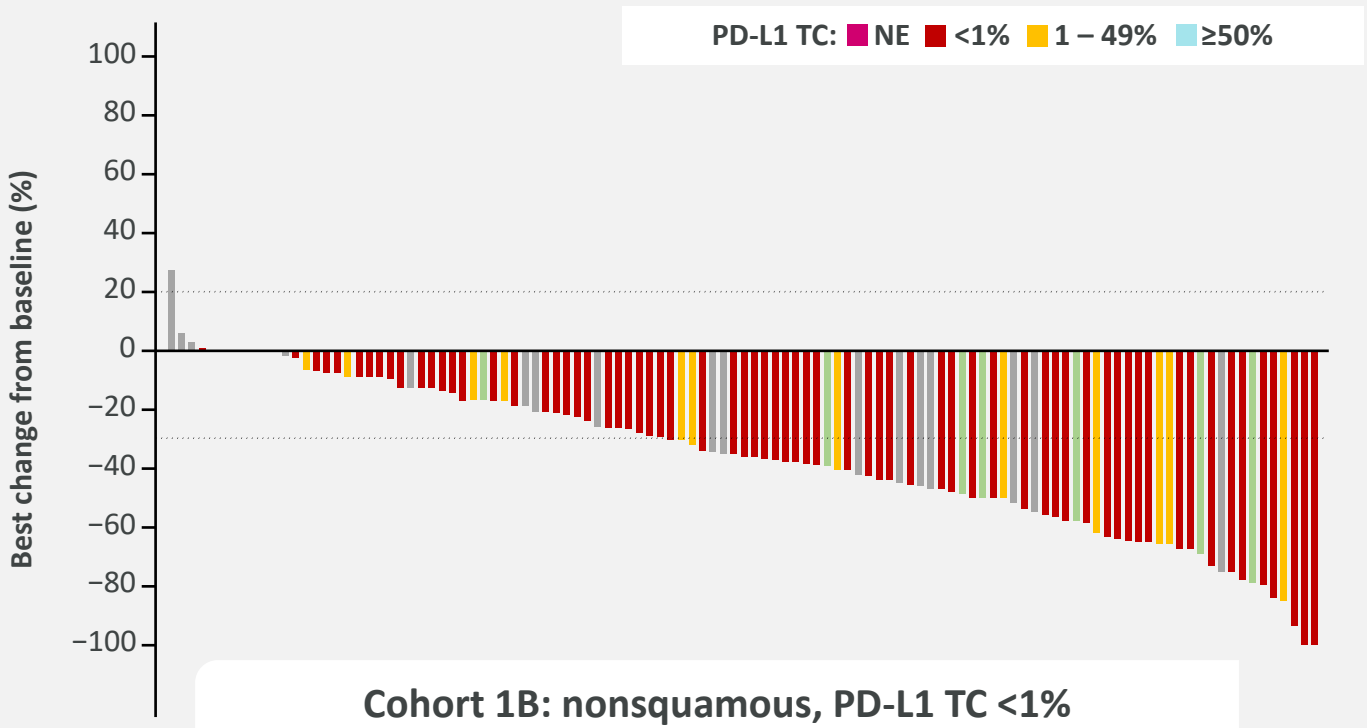
volrustomig + CTx – promising activity, particularly in PD-L1<1% population, a group with high unmet need

43.7% ORR in non-squamous patients with 84.9% DCR (n=119)



Cohort 1B: nonsquamous, PD-L1 TC <1%
ORR: 34.5% | **mPFS:** 7.0 months

Meaningful ORR, durable responses and encouraging PFS in PD-L1 TC <1%

volrustomig + CTx regimen had manageable safety profile

Ongoing Phase III eVOLVE-Lung02 in PD-L1 <50% NSCLC

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

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Closing remarks

Dave Fredrickson

Executive Vice President, Oncology Business

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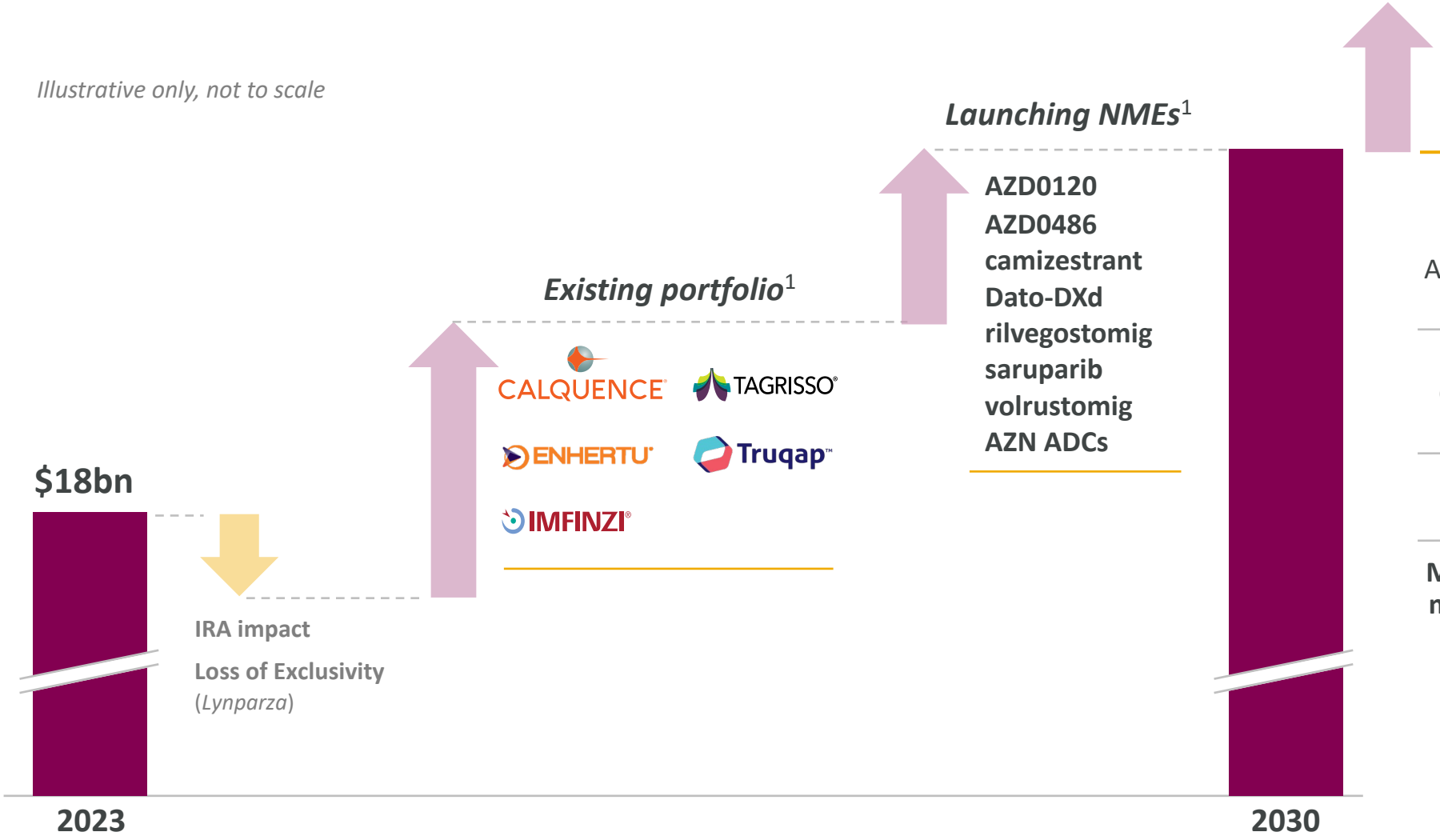
AstraZeneca – ambition in oncology

Illustrative only, not to scale



Beyond 2030

Sustained leadership in disruptive categories

ADCs • Radioconjugates • IO bispecifics • cell therapy • T-cell engagers

Broad portfolio with unique combination potential and precision medicine expertise

Global, differentiated commercial model and footprint

Multiple blockbuster medicines across major tumour types and haematology

1. includes select medicines and pipeline opportunities.
Collaboration partners: Daiichi Sankyo (*Enhertu*, Dato-DXd); Merck & Co., Inc. (*Lynparza*); Compugen (rilvegostomig).

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Significant oncology news flow across key medicines through 2025

2024

- ***Imfinzi*** **ADRIATIC** ASCO plenary (June 2024) ✓
- ***Tagrisso*** **LAURA** ASCO plenary (June'24) ✓
- ***Enhertu*** **DESTINY-Breast06** ASCO LBA (June 2024) ✓
- ***Calquence*** **ECHO** MCL updated data cut ✓
- **Dato-DXd TROPION-Lung01** regulatory decision
- **Dato-DXd TROPION-Breast02** 1L TNBC data readout
- ***Truqap*** **CAPitello-281** d*PTEN* prostate data readout
- **volrustomig + CTx AZ FIH Phase I/II** updated data cut ✓
- **rilvegostomig Phase I/II ARTEMIDE-01** 1L PD-L1>1% NSCLC update ✓
- **rilvegostomig + CTX Phase I/II GEMINI** 1L gastric data cut ✓
- ***Imfinzi*** **+ CTX Phase III NIAGARA** Cis-eligible MIBC ✓

2025

- **Dato-DXd TROPION-Breast01** regulatory decision
- **Dato-DXd +** ***Imfinzi*** **AVANZAR** 1L NSCLC data readout
- ***Imfinzi*** **+ ceralasertib LATIFY** 2L NSCLC data readout
- ***Imfinzi*** **MATTERHORN** early-stage gastric EFS data readout
- ***Imfinzi*** **EMERALD-2** adj. HCC data readout (>2025)
- ***Imfinzi*** **EMERALD-3** locoregional HCC data readout (>2025)
- ***Enhertu*** **DESTINY-Breast09** 1L HER2+ breast data readout
- ***Enhertu*** **DESTINY-Breast11** early-stage HER2+ breast data readout
- **camizestrant SERENA-4/6** 1L HR+ HER2- breast data readout (>2025, 2025)
- ***Enhertu*** **DESTINY-Lung04** 1L *HER2*m NSCLC data readout
- ***Calquence*** **AMPLIFY** 1L CLL data readout ✓IA
- ***Tagrisso*** **+ savolitinib SAFFRON** 2L MET+ *EGFR*m data readout

Multiple high value Phase III readouts over next 12-18 months extending leadership in oncology

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Q&A Session II

AstraZeneca @ WCLC and ESMO 2024

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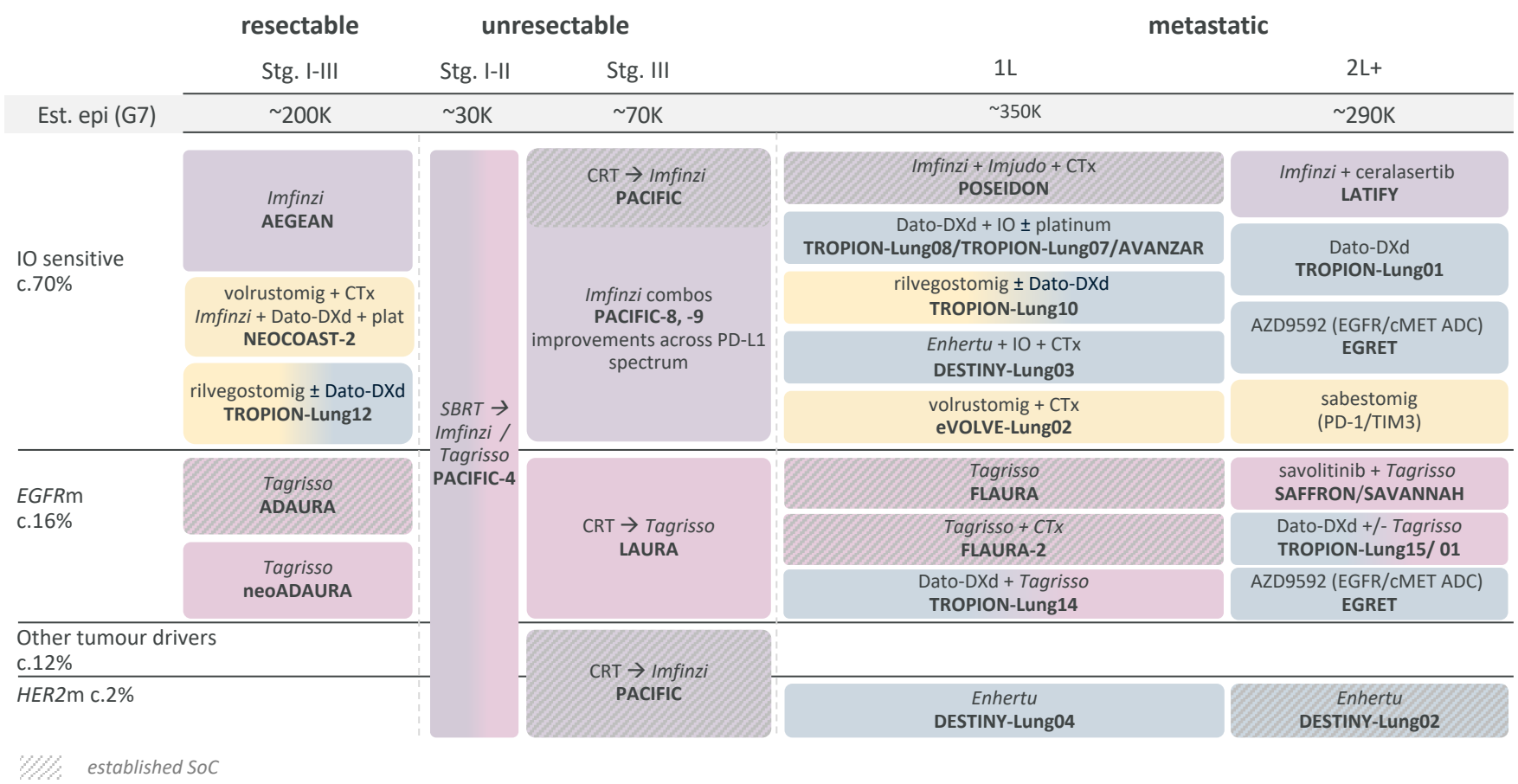
Appendix

Lung and breast tumour maps

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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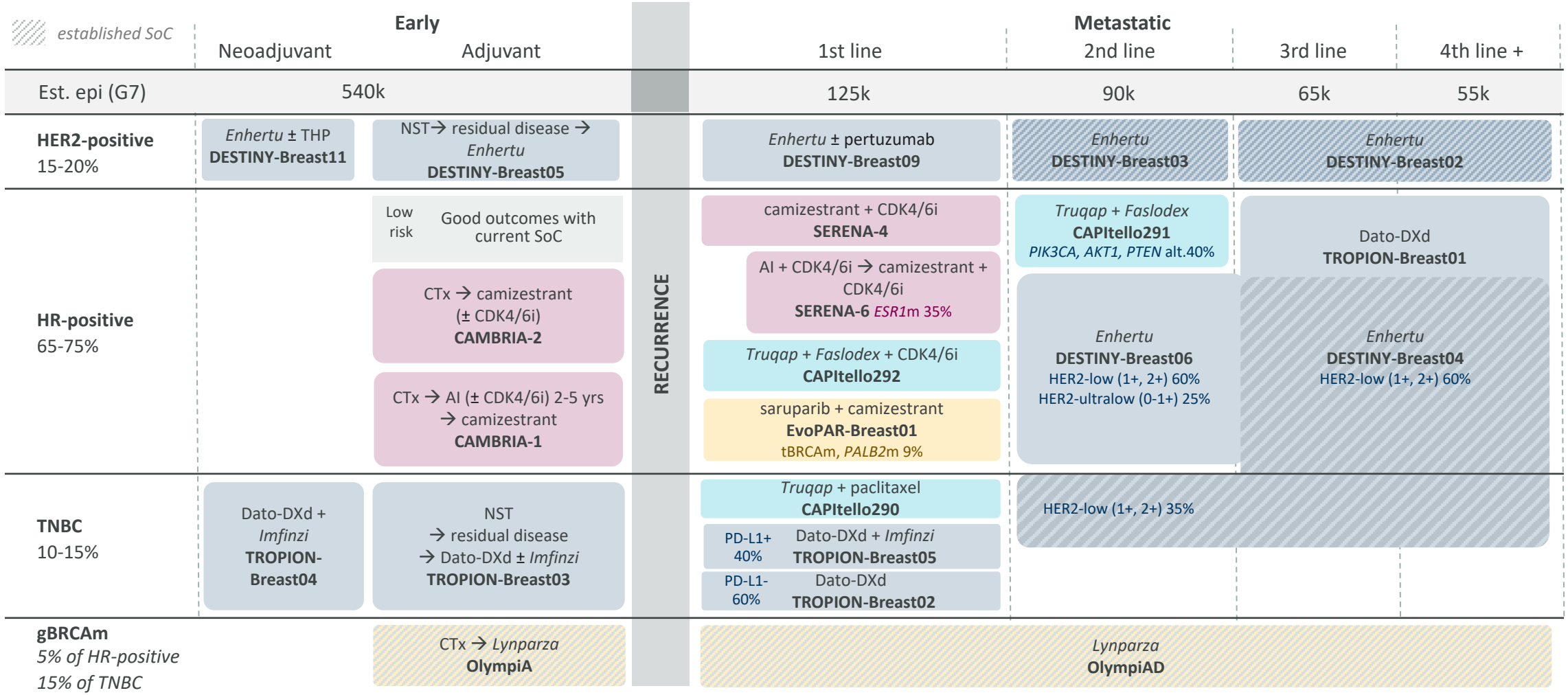
AstraZeneca in breast cancer

Ambition to eliminate breast cancer as a cause of death



All numbers are approximate. Illustrative settings and populations, not to scale.
Collaboration partners: Daiichi Sankyo (*Enhertu*, Dato-DXd), Merck & Co., Inc. (*Lynparza*).

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Glossary

1/2/3L	1st/2nd/3rd line
AAV	adeno-associated viruses
AGA	actionable genomic alterations
AE	adverse event
AI	artificial intelligence
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
BCMA	B-cell maturation antigen
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
FDCs	Fixed dose combinations
FRα	folate receptor alpha
Fab	fragment antigen-binding
GC	gastric cancer
GEJC	gastroesophageal junction adenocarcinoma
GLP-1	glucagon-like-peptide-1
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
QCS-NMR	quantitative continuous scoring-normalised membrane ratio
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
TOPO1	topoisomerase-1
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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