AstraZeneca

AstraZeneca at #AACR23

Highlights from key programmes presented at the American Association for Cancer Research (AACR) Annual Meeting 2023

14–19 April 2023

Oncology at AstraZeneca

We have the vision to **redefine cancer care** and, one day, eliminate cancer as a cause of death. It is through **persistent innovation that we have built an industry-leading pipeline** across multiple scientific platforms, with the aim of transforming outcomes and increasing the potential for cures.

"It's exciting to see our strategy to attack cancer from multiple angles come to life at AACR this year through data from our proprietary antibody drug conjugates, next generation cell therapies, and epigenetics molecules. Furthermore, results from the AEGEAN trial show the potential of treating lung cancer patients early with *Imfinzi* before and after surgery which reinforces the importance of diagnosing lung cancer early."

Dr Susan Galbraith
Executive Vice President,
Oncology Research & Development

Contents

Attendance at the American Association for Cancer Research (AACR) Annual Meeting

AstraZeneca presented new data across its diverse Oncology pipeline and industry-leading portfolio at the AACR Annual Meeting, 14–19 April 2023.

Data from 70 presentations were featured, including eight oral presentations, a plenary presentation of the AEGEAN Phase III trial of *Imfinzi* (durvalumab) in resectable non-small cell lung cancer (NSCLC), and the first disclosures of preclinical data for five novel molecules across the Company's Antibody Drug Conjugate (ADC), Cell Therapy and Epigenetics scientific platforms.

For any questions or requests for follow-up information, please contact us at IRDirectors@astrazeneca.com

AstraZeneca's ambition in Oncology

Our Oncology strategy is built with one goal in mind – to push the boundaries of science to change the practice of medicine and transform the lives of patients living with cancer. Our broad pipeline of next-generation medicines, together with our focus on excellence in execution, is aimed at expanding treatment options and improving outcomes for patients with solid tumours and haematological cancers.

We focus on four strategic priorities:

1. Pioneering research across multiple scientific platforms and modalities.
2. Advancing innovative clinical strategies to treat patients with early stages of disease and relapsed or refractory patients.
3. Building expertise and leadership in the most prevalent and highest mortality rate tumour types.
4. Delivering across our global footprint.

Latest advances being showcased at AACR

Improving outcomes for patients with resectable lung cancer with *Imfinzi*

- AEGEAN *(Imfinzi)*

Delivering the next wave of ADCs with a proprietary platform

- AZD9592 (EGFR cMET TOP1i)
- AZD5335 (FRα TOP1i)

Building the next generation of cell therapies in solid tumours

- C-CAR031 (armoured GPC3 CAR-T)
- AZD0754 (armoured STEAP2 CAR-T)

First disclosure and preclinical data for an epigenetics molecule targeting PRMT5

- AZ-PRMT5i-1 (MTAP-selective inhibitor)

Harnessing transformational technologies

- Circulating tumour DNA (ctDNA)
- Computational pathology
- Artificial intelligence and data science

Improving outcomes for patients with resectable lung cancer with *Imfinzi*

Overview and approach

What is immuno-oncology (IO)?

The immune system comprises the body's natural defence mechanisms, responsible for responding to external pathogens, such as bacteria and viruses, and protecting us from abnormal internal disease processes like cancer. T-cells and myeloid cells play a major role in this anti-cancer response by recognising and eliminating tumour cells while leaving healthy cells unharmed. Immune checkpoints are a key part of the decision-making process that determines whether T-cells will attack, and whether they can be manipulated by cancer cells in order to evade the immune response.

IO medicines: Unleashing the power of the immune system on cancer

Systemic
Travels through the body to attack cancer cells wherever they may be.

Targeted
Activates and directs the body's own immune system to fight cancer.

Memory
The immune system remembers cancer cells resulting in longer lasting remissions.

Versatile
Used to treat several different types of cancer.

What are we doing in this space?

Checkpoint inhibitors

The first wave of IO therapies, aimed at overcoming immune checkpoints and unleashing the power of the immune system on cancer, have become the backbone of many treatment regimens. Antibody-based checkpoint inhibitors work by engaging a specific receptor (or its ligand) on the surface of immune cells, such as PD-1 or CTLA-4.

But, while these medicines have opened up the field, not all patients respond to checkpoint inhibition and responses are not always as deep or durable as we might hope. Our broad pipeline features a range of potential first-in-class IO therapies across multiple tumour types, as well as novel combinations of IO therapies with the potential to induce deeper, more durable responses.

Bispecific antibodies

We are drawing on our long history of protein engineering to design bispecific antibodies that simultaneously target different immune checkpoints on the same cell. These dual-purpose antibodies aim to improve the specificity and efficacy of treatment by combining both medicines in one and could help to drive more durable responses in the clinic or overcome evolved resistance to blockade of the PD-1/PD-L1 axis.

T-cell engagers

Looking to the future, we are exploring ways to redirect T-cells that do not recognise cancer, as they are more abundant and more potent than those that do. To this end, T-cell engagers (which direct T-cells to the tumour and amplify a patient's own anti-cancer immune response) are a growing area of interest in IO.

Other immune cells

We are exploring different facets of immunity, including the potential of modulating other immune cells, such as myeloid cells, as a way to target cancer. We are also looking at ways of manipulating the tumour microenvironment to make it more amenable for T-cells to function, either through blocking cancer-promoting molecules (e.g. LIF or CD73) or by adding cytokines (e.g. IL-12) to encourage anti-tumour immune responses.

Combinations

Combining IO therapies with drugs designed to kill cancer cells, such as antibody drug conjugates (ADCs), could lead to additive or synergistic responses as dying cells attract the attention of the immune system to further enhance the effect. We are also exploring the potential of combining drugs from our early pipeline with PD-1/PD-L1 checkpoint inhibition to induce deeper and more durable anti-tumour responses.

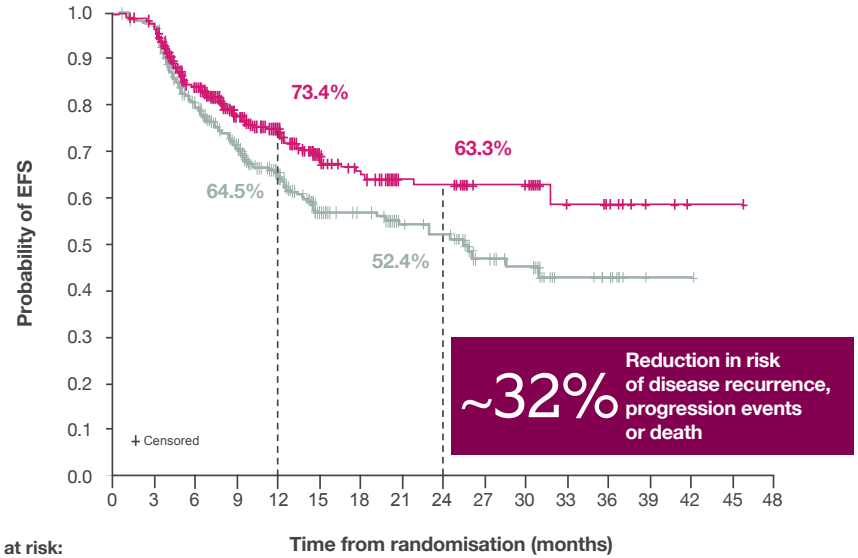
New at #AACR23: Novel *Imfinzi*-based regimen significantly improved patient outcomes in resectable non-small cell lung cancer in AEGEAN Phase III trial

While metastatic disease has long been an area of focus in cancer research, we have been continuously looking for opportunities to treat cancer in earlier stages in which there is the greatest potential for cure.

In lung cancer, the majority of patients with resectable disease eventually develop recurrence despite complete tumour resection and adjuvant chemotherapy. Therefore, by moving immuno-oncology treatment into the neoadjuvant (before surgery) and adjuvant (after surgery) settings, we can maximise the potential of our medicines, and optimise treatments for patients.

The AEGEAN results showed that adding *Imfinzi* both before and after surgery is an important new approach that has the potential to become a backbone combination approach for the treatment of resectable non-small cell lung cancer (NSCLC). These data further validate our scientific leadership in moving lung cancer treatment to earlier stages of disease in which patients have the highest potential for cure, as we have already seen in other areas of our clinical development programme, as seen with *Tagrisso* in the ADAURA trial.

First planned interim analysis of event-free survival (EFS)*

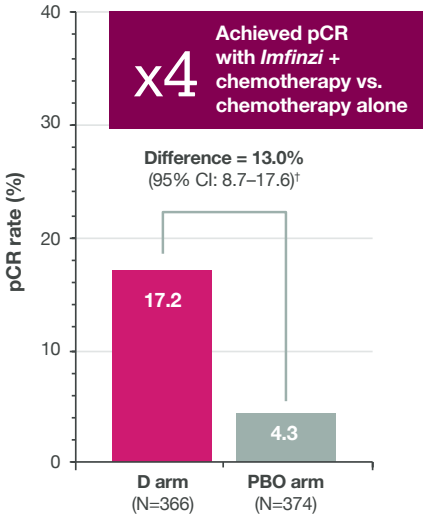


No. at risk:																
D arm	366	336	271	194	140	90	78	50	49	31	30	14	11	3	1	1	0
PBO arm	374	339	257	184	136	82	74	53	50	30	25	16	13	1	1	0	0

	D arm	PBO arm
No. events / no. patients (%)	98/366 (26.8)	138/374 (36.9)
mEFS, months (95% CI)	NR (31.9–NR)	25.9 (18.9–NR)
Stratified HR (95% CI)	0.68 (0.53–0.88)	
Stratified log-rank P-value	0.003902	

EFS is at 32% data maturity

Final analysis of pathologic complete response (pCR)†



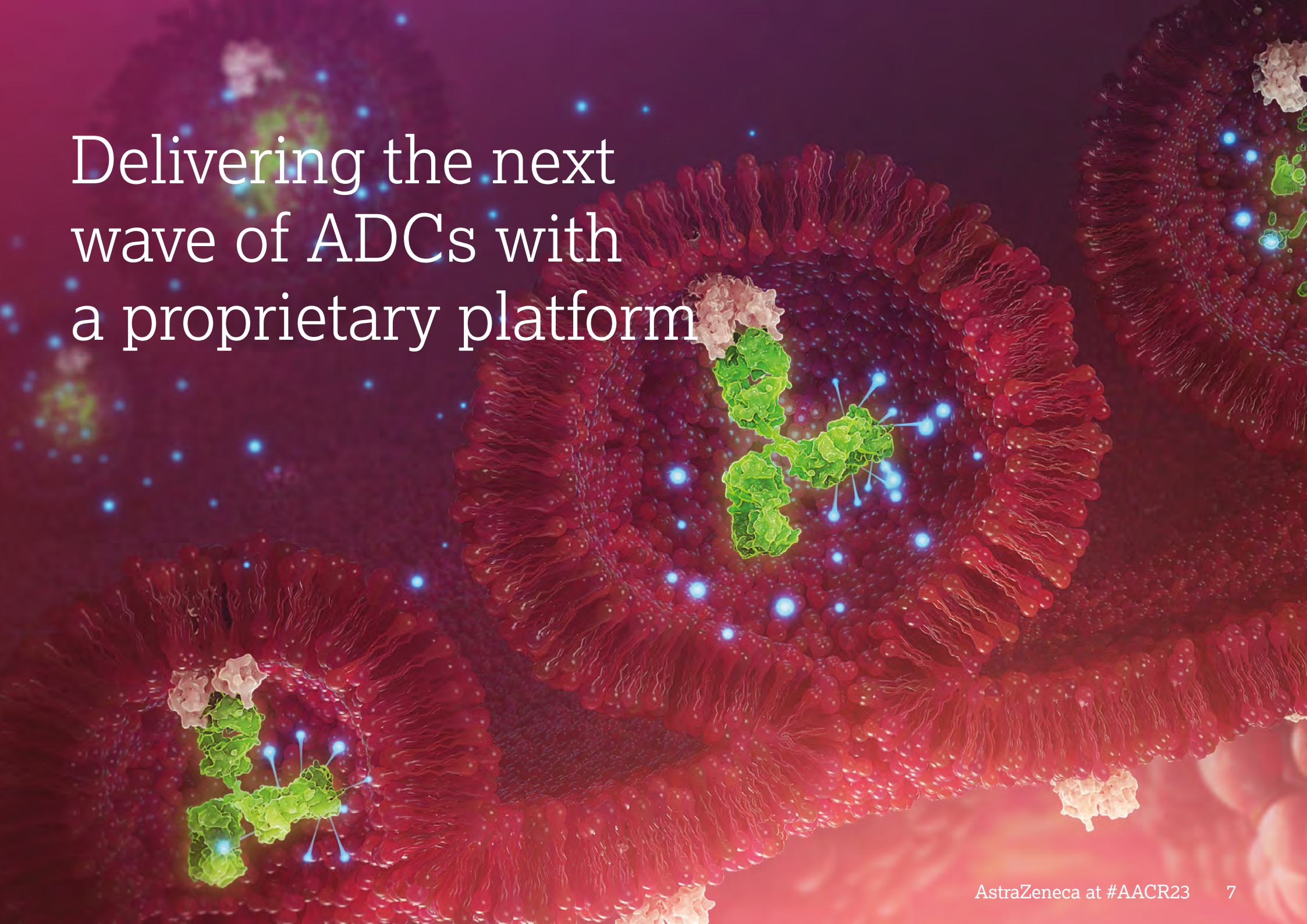
Heymach, JV. CT005 - AEGEAN: A phase 3 trial of neoadjuvant durvalumab + chemotherapy followed by adjuvant durvalumab in patients with resectable NSCLC. AACR 2023. Abstract #CT005.

EFS is defined as time from randomisation to the earliest of: (A) progressive disease (PD) that precludes surgery; (B) PD discovered and reported by the investigator upon attempting surgery that prevents completion of surgery; (C) local/distant recurrence using BICR per RECIST v1.1; or (D) death from any cause.

BICR, blinded independent central review; CI, confidence interval; D, durvalumab; (m)EFS, (median) event-free survival; HR, hazard ratio; IASLC, International Association for the Study of Lung Cancer; NR, not reached; PBO, placebo; pCR, pathologic complete response; RECIST, Response Evaluation Criteria in Solid Tumours.

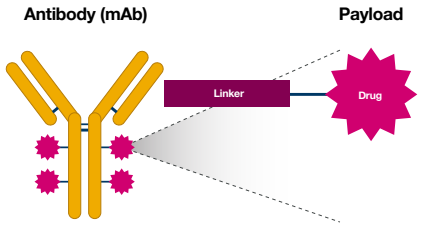
*Using RECIST v1.1 (BICR)
†Per IASLC 2020 methodology

Delivering the next wave of ADCs with a proprietary platform



Overview and approach

What are antibody drug conjugates (ADCs)?

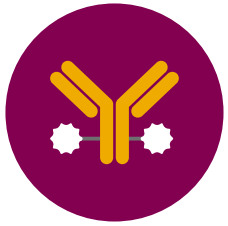


ADCs are targeted medicines that combine a monoclonal antibody (mAb), which binds to target proteins on the surface of cancer cells, with a drug, typically a cytotoxic chemotherapy agent, via a chemical linker.

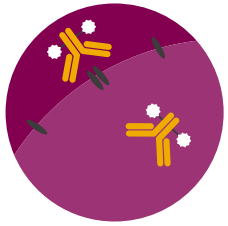
Unlike conventional chemotherapy treatments that can damage healthy cells, ADCs are targeted medicines that deliver chemotherapy agents directly to cancer cells. After binding to the target (protein or receptor), the ADC releases the cytotoxic drug into the cancer cell.

'Fully human' monoclonal antibodies (which have been engineered to carry human antibody genes) are an ideal delivery platform for ADCs. They are highly targeted and cell-specific, binding to specific proteins on the cell surface of cancer cells. They have a long circulating half-life and offer minimal immunogenicity. The stable chemical connections or 'linkers' that join the antibodies and cytotoxic drugs together are highly stable to prevent cleaving (splitting) before the ADC enters the tumour. The anticancer drugs (or 'payloads') penetrate the tumour and cause cell death either by damaging the DNA of cancer cells or by preventing new cancer cells from forming and spreading.

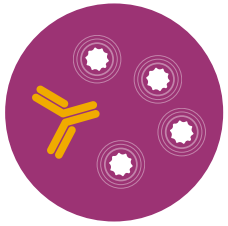
How ADCs work



A cancer-killing drug is attached to the mAb through a stable connection (linker) creating the ADC.



The mAb component binds to the target on the tumour cell surface and the ADC is internalised.



Once inside the cancer cell, the linker is selectively cleaved and the cytotoxic payload is released into the cell.

What are we doing in this space?

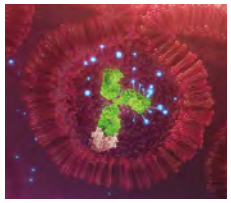
- Our vision is for ADCs to replace chemotherapy and become the backbone of cancer therapy
- We are building on the success of *Enhertu* (trastuzumab deruxtecan) and the emerging promise of datopotamab deruxtecan, both of which are being jointly developed with Daiichi Sankyo
- We are building next-generation ADCs with our robust internal technology and portfolio:
  - Using novel chemistry to design linker warheads to achieve optimal target coverage and efficacy
  - Targeting solid tumours and haematological malignancies

The strength of our emerging proprietary ADC technology is demonstrated with data across three assets

Leveraging in-house ADC capabilities and technology to accelerate innovation

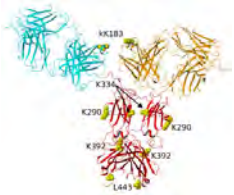
Targets and antibodies

Efficient delivery of antibody drug conjugate (ADC) payload



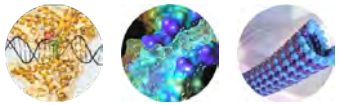
Linkers

Optimise conjugation chemistry and overcome potential off-target toxicity

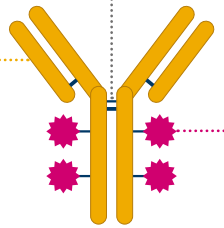


Warhead

Match disease biology and enable biology-driven combinations to overcome resistance



Topoisomerase inhibitors | DNA intercalators crosslinkers | Microtubule inhibitors



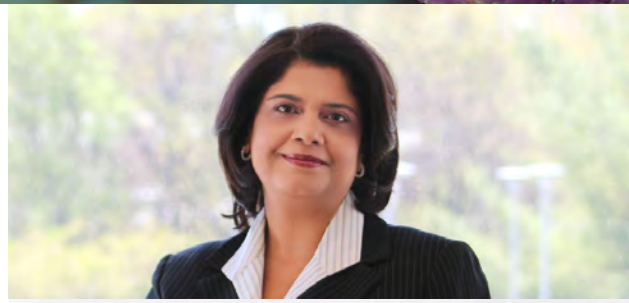
Three areas of future investment for clinical success:

1 **Biomarkers**

2 **Mechanistic and IO combinations**

3 **Understanding resistance**

cMET, mesenchymal-epithelial transition factor; EGFR, epidermal growth factor receptor; FRα, folate receptor alpha; IO, immuno-oncology; MMAE, monomethyl auristatin E; TOP1i, topoisomerase 1 inhibitor.



"It's been an incredible few years as we have established our in-house ADC capabilities, delivered our proprietary discovery platform and advanced our pipeline into the clinic. I'm especially excited to see how our programs deliver in the clinic and our continued innovation in this space."

Puja Sapra
Senior Vice President, Biologics Engineering & Targeted Delivery, Oncology Research & Development

AstraZeneca in-house ADC pipeline

Phase I:
AZD8205 (B7H4 TOP1i)
AZD9592 (EGFR cMET TOP1i)
CMG901 (Claudin 18.2 MMAE)

Preclinical:
AZD5335 (FRα TOP1i)

New at #AACR23: AstraZeneca's proprietary ADC technology

AZD9592 (EGFR cMET TOP1i)

Two oral presentations featured the first preclinical and translational results for AZD9592, a bispecific antibody drug conjugate (ADC) designed to deliver targeted chemotherapy to cancer cells with a topoisomerase 1 inhibitor (TOP1i) warhead using the Company's proprietary linker technology.

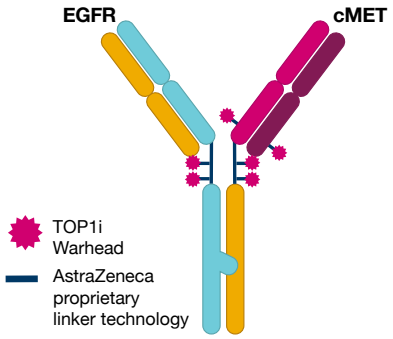
AZD9592 binds to two known oncogenic drivers: epidermal growth factor receptor (EGFR) and mesenchymal-epithelial transition factor (cMET). These two drivers are often co-expressed in solid tumours including in non-small cell lung cancer (NSCLC) and head and neck squamous cell carcinoma (HNSCC).

Proprietary ADC technology with unique cleavable linker and novel topoisomerase warhead.

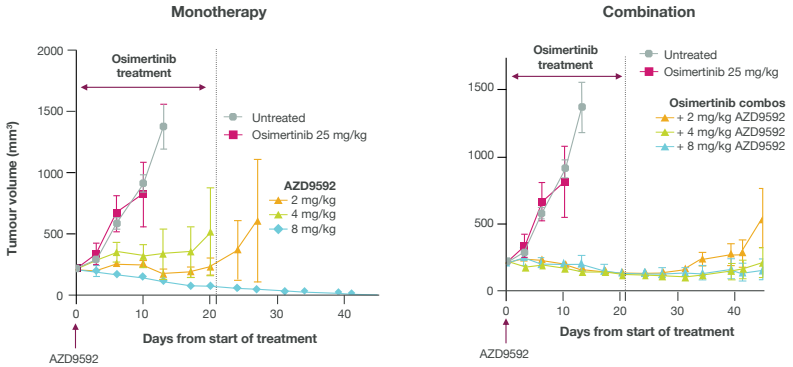
Preclinical findings

This is the Company's first bispecific ADC to enter the clinic and showed a promising efficacy and safety profile in preclinical models, with evidence for DNA damage dependent tumour cell death as the mechanism of action.

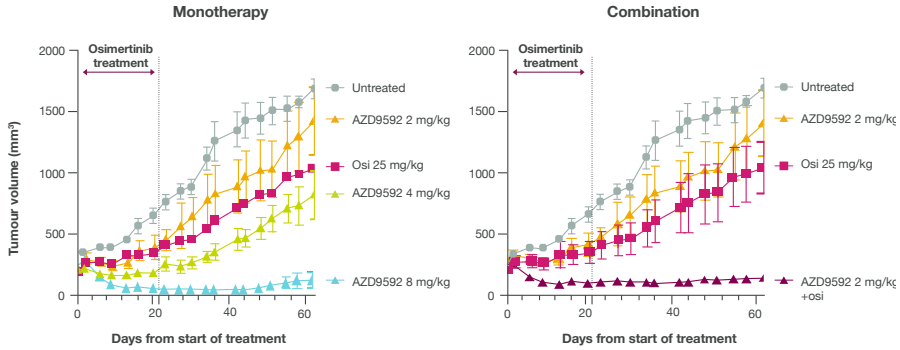
AZD9592



NSCLC: Acquired resistance



NSCLC: Primary resistance



Comer F et al. AZD9592: an EGFR-cMET bispecific antibody-drug conjugate (ADC) targeting key oncogenic drivers in non-small-cell lung cancer (NSCLC) and beyond. AACR 2023. Abstract #5736; McGrath L et al. Evaluation of the relationship between target expression and in vivo anti-tumour efficacy of AZD9592, an EGFR/c-MET targeted bispecific antibody drug conjugate. AACR 2023. Abstract #5737.

New at #AACR23: AstraZeneca's proprietary ADC technology

AZD5335 (FRα TOP1i)

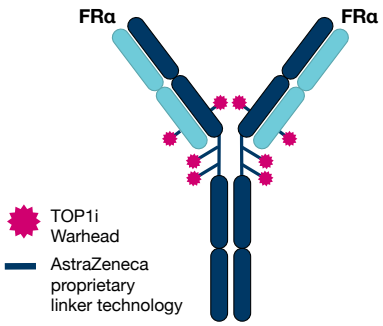
The first preclinical results were presented for another antibody drug conjugate (ADC), AZD5335, a promising therapeutic candidate for the treatment of certain ovarian cancers. This ADC has a folate receptor alpha (FRα) targeting antibody linked to a proprietary topoisomerase 1 inhibitor (TOP1i) warhead.

In-house TOP1i warhead **enables accelerated** ADC pipeline development.

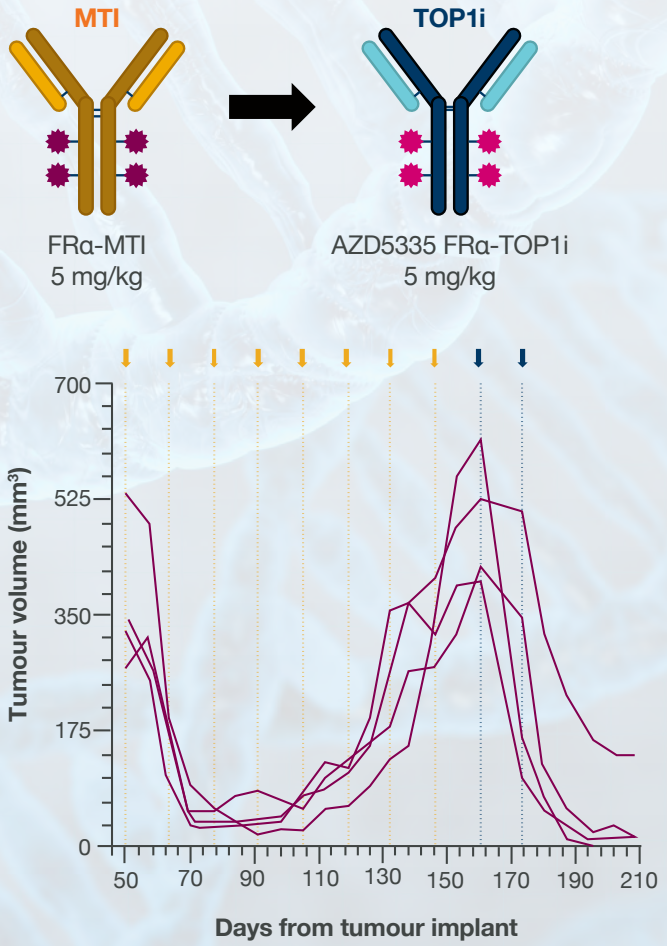
Preclinical findings

A robust anti-tumour response was reported in FRα-expressing preclinical models that are resistant to another FRα ADC with a microtubule inhibitor warhead. AZD5335 was active in models with either high or low levels of target expression as detected by computational pathology.

AZD5335



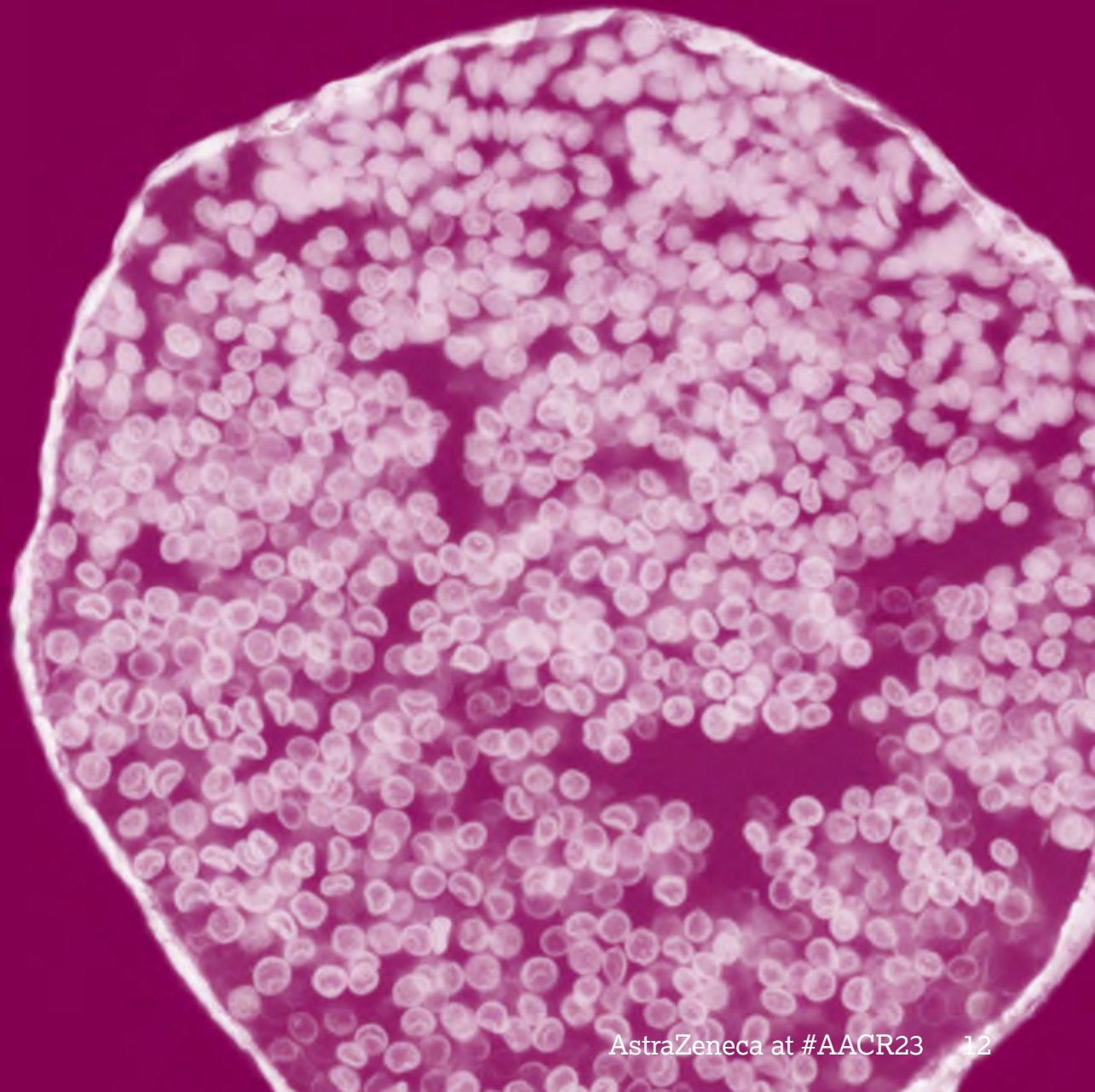
AZD5335 achieved a response in FRα-MTI-resistant models



Gymnopoulos M, et al. First disclosure of AZD5335, a TOP1i-ADC targeting low and high FRΠ-expressing ovarian cancer with superior preclinical activity vs FRΠ-MTI ADC. AACR 2023. Abstract #LB025/17.

FRα, folate receptor alpha; MTI, microtubule inhibitor; TOP1i, topoisomerase 1 inhibitor.

Building the next generation of cell therapies in solid tumours



Building the next generation of cell therapies in solid tumours

What is cell therapy?

Cell therapy is a promising, rapidly advancing field with the potential to transform medicine across disease areas in which significant need exists. In oncology, cell therapy refers to the removal of tumour cells using engineered immune cells.

Current approaches in oncology are largely focused on autologous chimeric antigen receptor-T cell (CAR-T) therapies, in which the immune system's T cells are extracted from a patient and genetically modified to recognise their specific cancer. While this has proved highly effective in some haematological cancers, replicating this success in solid tumours has been challenging. The hostile microenvironment surrounding solid tumours can limit the accessibility and functionality of cell therapies.

What are we doing in this space?

We are advancing a pipeline of next-generation autologous cell therapies that are designed to overcome current barriers to the widespread adoption of cell therapies in oncology. This includes novel ways to engineer or 'armour' CAR-Ts to overcome the immune-suppressive tumour microenvironment and enhance their potential effectiveness in hard-to-treat solid tumours, and off-the-shelf patient-ready cell therapies.

We are also exploring the potential of T-cell receptor therapies (TCR-Ts) following the recent acquisition of Neogene Therapeutics. TCR-Ts are an emerging modality that enables the identification of intracellular targets, unlocking biology that was previously inaccessible by cell therapy.

neogene THERAPEUTICS

"A key aspect of our research is exploring how to engineer a better T cell – we're essentially taking what nature has given us and going a step further to build immunity beyond what our immune response is capable of doing on its own, to detect and eradicate cancer more effectively"

Mark Cobbold
Vice President, Head of Immuno-Oncology, Discovery and Oncology Cell Therapy

AstraZeneca CAR-T pipeline

Phase I:

C-CAR031 (armoured* GPC3 CAR-T) - IIT trial by Cellular Biomedicine group (CBMG)†

Preclinical:

AZD5851 (armoured* GPC3 CAR-T)

AZD0574 (armoured* STEAP2 CAR-T)

Armoured* Claudin 18.2 CAR-T

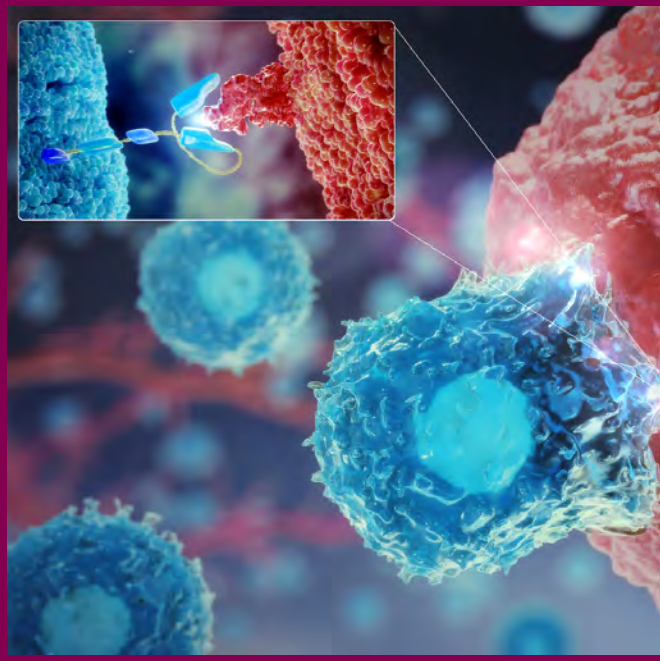
Neogene TCR-T pipeline

Preclinical:

NT-125 (fully individualised, multi-specific TCR-T)‡

NT-175 (armoured P53-specific TCR-T)

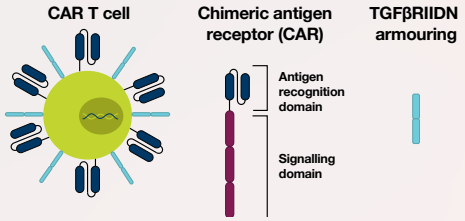
*AstraZeneca's dominant negative transforming growth factor β receptor II (TGFβRIIDN) armouring
†CAR-T designed by AstraZeneca, based on AZD5851
‡An autologous, fully individualised, multi-specific (including up to 5 TCRs) TCR therapy that derives neoantigen-specific TCRs from a patient's tumour infiltrating T cells, identified using Neogene's proprietary neoantigen-specific TCR identification and selection platform



New at #AACR23: Armoured CAR-T cell therapy in solid tumours

C-CAR031 (armoured GPC3 CAR-T) and AZD0754 (armoured STEAP2 CAR-T)

AstraZeneca's dominant negative transforming growth factor β receptor II (TGFβRIIDN) armouring is designed to resist the immuno-suppressive tumour microenvironment and enhance the potential effectiveness of chimeric antigen receptor T cells (CAR-T) in solid tumours.



The first clinical data for **CAR-T cell therapy in solid tumours** utilising AstraZeneca's innovative research and armouring platform were presented at AACR.

Zhang Q et al. First report of preliminary safety, efficacy, and pharmacokinetics of C-CAR031 (GPC3-specific TGFBRIIDN CAR-T) in patients with advanced HCC. AACR 2023. Abstract #CT097/5; Van Dyk D et al. Antitumour activity of AZD0754, a dnTGFbR2 armored STEAP2 targeted CAR-T therapy, in preclinical models of prostate cancer. AACR 2023. Abstract #LB085/1

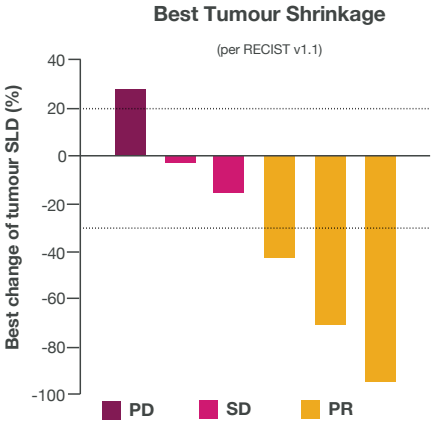
CR, complete response; PD, progressive disease; PR, partial response; RECIST, Response Evaluation Criteria in Solid Tumours; SD, stable disease; SLD, sum of longest diameters.

C-CAR031 (armoured GPC3 CAR-T)

In cell therapy, the first clinical data were presented for C-CAR031, a novel TGFβRIIDN-armoured Glypican 3 (GPC3) targeting CAR-T therapy that is being investigated for liver cancer. C-CAR031 was designed by AstraZeneca and is being manufactured and developed in China by Cellular Biomedicine group (CBMG).

Clinical findings

Early results showed C-CAR031 is well tolerated with promising anti-tumour activity seen with objective responses in several patients to date.

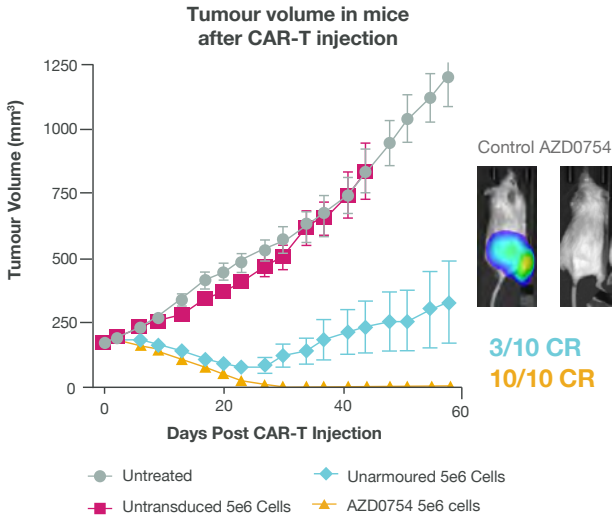


AZD0754 (armoured STEAP2 CAR-T)

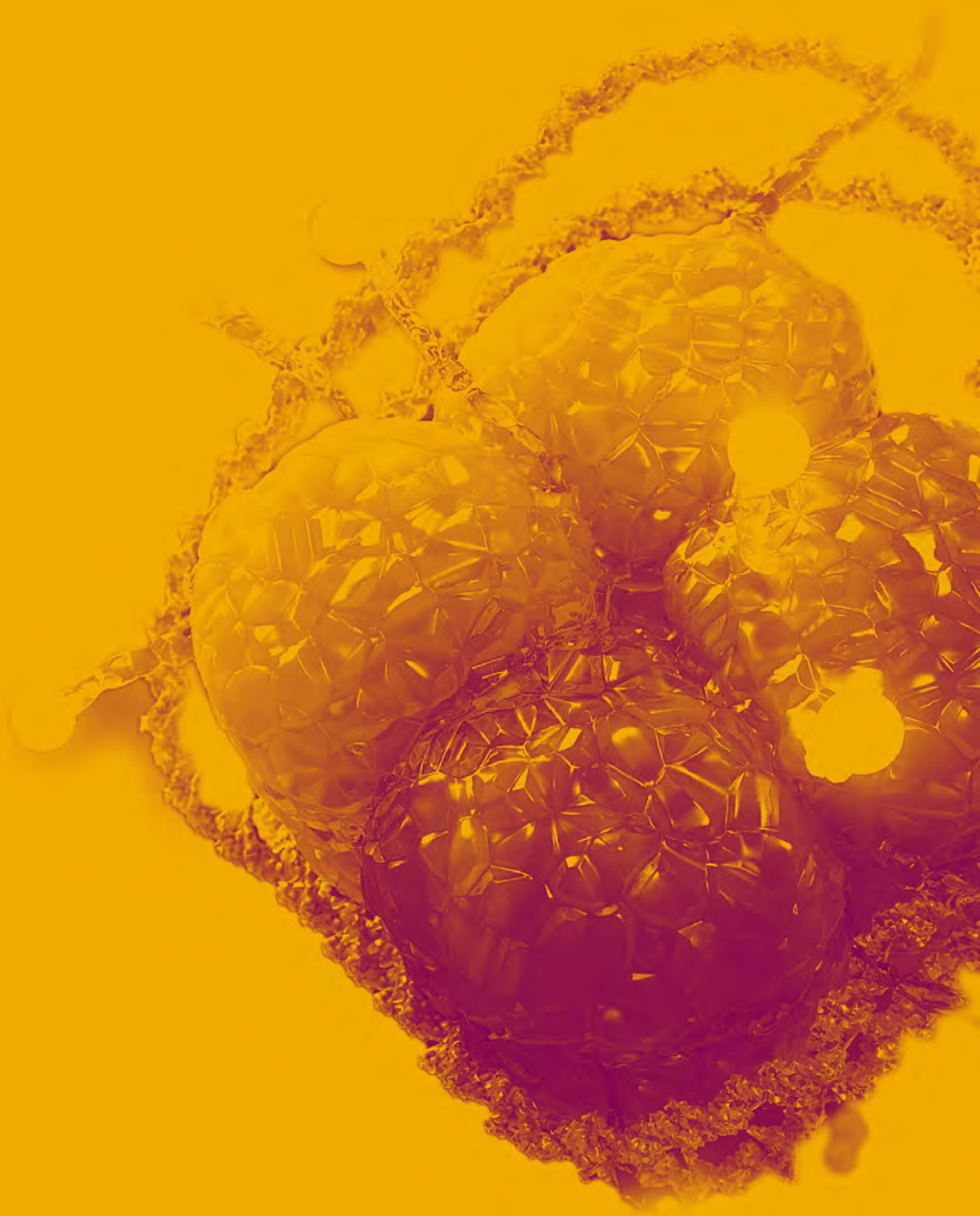
AZD0754 is a novel TGFβRIIDN-armoured CAR-T targeting STEAP2, a protein commonly overexpressed in prostate cancer. This is the first potential cell therapy to be designed, manufactured and developed by AstraZeneca.

Preclinical findings

The data showed encouraging preclinical safety signals *in vitro* and *in vivo*, and support future clinical development of this potential first-in-class CAR-T therapy. STEAP2 is highly prevalent in prostate cancer cells, with limited normal tissue expression.



First disclosure and preclinical data for an epigenetics molecule targeting PRMT5



Overview and approach

What is epigenetics?

Building on decades of progress describing and targeting genetic changes identified in cancer, new technological advances have enabled a similar initiative for epigenetic biology. These epigenetic changes – not to the sequence of the genome but rather to how it is interpreted and expressed – can lead to cancer and cause resistance to current anticancer therapies. AstraZeneca believe advances in epigenomic capabilities and inhibitors targeting key processes, including chromatin remodelling and RNA modification, will advance the next wave of innovation in cancer treatment.

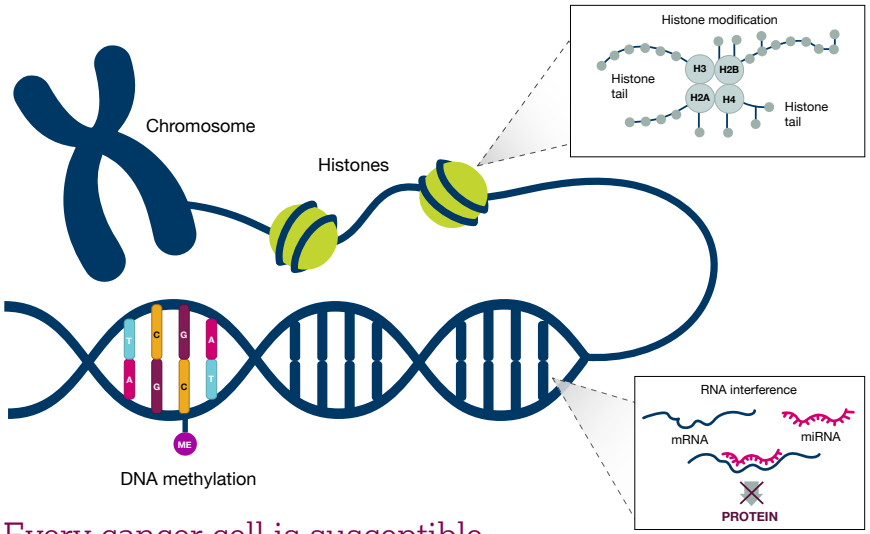
Our areas of focus include:

- Monitoring epigenetic changes to detect cancer earlier
- Understanding epigenetic mechanisms of resistance to anti-cancer therapy
- Developing new ways to attack cancer by modulating gene expression
- Exploiting cancer-specific vulnerabilities, through ‘synthetic lethality’
- Using epigenetics to reinvigorate the anti-tumour immune response

*Including DNA, RNA and histone

“Epigenetics represents a relatively untapped opportunity in cancer biology for researchers, oncologists and tomorrow’s patients living with cancer.”

Stephen Fawell
Vice President, Head of Oncology Discovery, Oncology Research & Development



Every cancer cell is susceptible to epigenetic* modification

Adapted from American Society of Hematology 2021

New at #AACR23: First disclosure and preclinical data for an epigenetics molecule targeting PRMT5

Epigenetic therapy is one of AstraZeneca's six core scientific areas of focus. The modality is the latest addition to the Company's diverse portfolio, which is designed to attack cancer from multiple angles and redefine outcomes for patients with high unmet needs.

At AACR, the first preclinical data were presented for the novel lead epigenetics molecule, AZ-PRMT5i-1, a potent methylthioadenosine phosphorylase (MTAP)-selective PRMT5 inhibitor with anti-tumour activity in MTAP-deleted tumours. Loss of the *MTAP* gene occurs across approximately 15% of all tumours, which provides an opportunity to selectively target PRMT5 in these tumours and spare healthy tissue.

Preclinical data for AZ-PRMT5i-1 showed robust pharmacodynamic and anti-tumour activity

Smith JM et al. Identification of a novel series of MTAP-selective PRMT5 inhibitors, and first disclosure of AZ-PRMT5i-1. AACR 2023. Abstract #3088/1; Lynch J et al. AZ-PRMT5i-1: A potent MTAP-selective PRMT5 inhibitor with pharmacodynamic and monotherapy anti-tumor activity in MTAP-deleted tumours. AACR 2023. Abstract #6272/10.

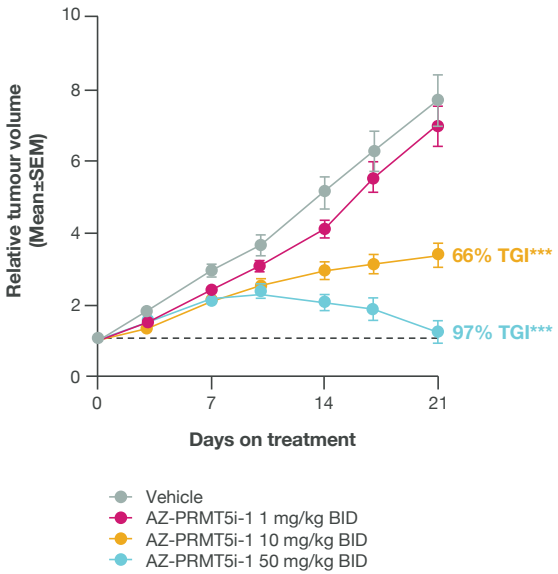
BID, twice a day; NS, not significant; NSCLC, non-small cell lung cancer; PDX, patient derived xenograft; SEM, standard error of the mean; TGI, tumour growth inhibition.

***p=<0.001

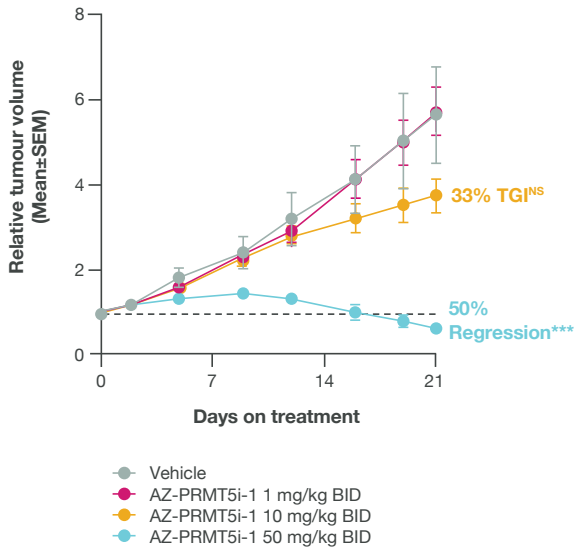
Preclinical findings

The preclinical results demonstrated MTAP selectivity and promising anti-tumour activity.

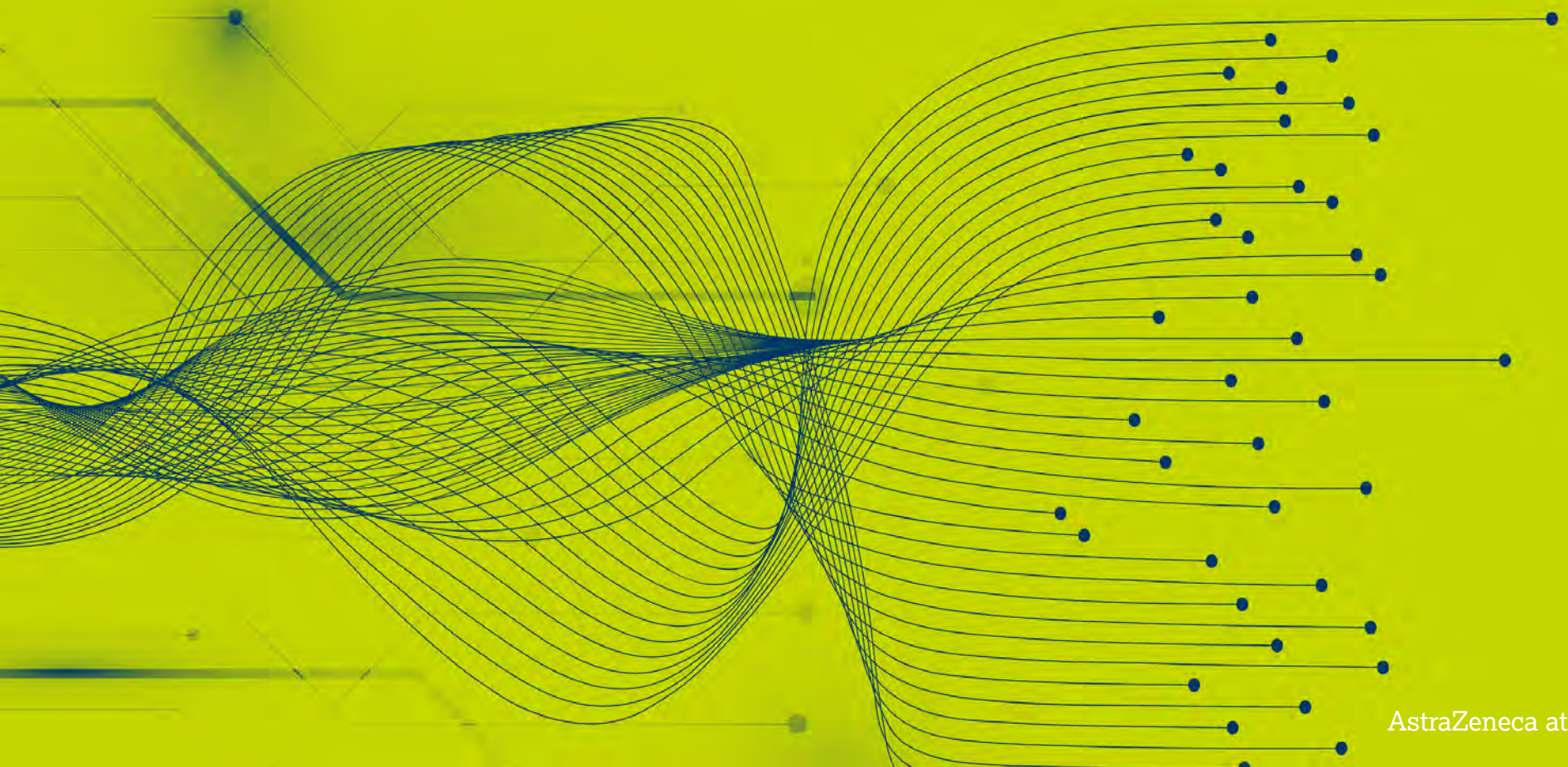
Efficacy in MTAP-null NSCLC PDX



Efficacy in MTAP-null Gastric PDX



Harnessing transformational technologies



Harnessing transformational technologies to revolutionise current treatment paradigms

Transformational technologies, including circulating tumour DNA (ctDNA), computational pathology, and data science and artificial intelligence (AI), underpin the success of progressing AstraZeneca's pipeline. Several presentations at AACR showcase the Company's efforts to harness the power of these technologies to better understand complex cancer biology, identify and select patients for treatment, and increase the probability of success in the clinic.

Computational pathology

We're pioneering new computational pathology approaches, combining digital pathology and big data with cutting-edge AI to enhance patient selection and enable more personalised treatments.

Right now, we're pioneering the use of Quantitative Continuous Scoring within our clinical trial portfolio, with regulatory approval as a first-in-class AI-driven diagnostic as a future goal. We are exploring its use in multiple indications, such as non-small cell lung cancer and hepatocellular carcinoma, and envisage it as a valuable future tool for patient selection in clinical trials and cancer care.

Learn more about computational pathology at AACR:

- A unified computational pathology method to quantify HER2 expression from raw IHC and IF images in breast cancer
- Evaluation of the relationship between target expression and in vivo anti-tumour efficacy of AZD9592, an EGFR/c-MET targeted bispecific antibody drug conjugate

AI and data science

Our AI and data science capabilities are allowing us to build transformer-based models to apply more knowledge, including translational problems (e.g. biomarker discovery and survival analysis) to deep learning models.

By harnessing the 'omics' modalities, including genomic, transcriptomic, epigenomic and proteomic information about patients, we hope to identify key drivers for cancer progression and advance our understanding of risk factors.

Learn more about AI and data science at AACR:

- Enhancing the utilisation of deep learning to predict patient response in small immunotherapy cohorts using real world data

ctDNA

Most cells release DNA into the bloodstream as they are damaged, dying or dead, and cancer cells are no exception. Through the use of technologies, such as next-generation sequencing, these fragments of DNA can reveal a wealth of information about cancer, without the need for invasive surgical biopsies, such as the likely presence and burden of cancer within the body and the presence of particular genetic mutations that may be able to inform the optimal treatment for each patient.

Learn more about ctDNA at AACR:

- Baseline and on-treatment plasma-based genomics as a predictor of outcomes in SAVANNAH: savolitinib + osimertinib in EGFRm MET overexpressed/ amplified NSCLC post-osimertinib

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