
Exscientia
Precision
Designed.
Personalised
Medicine.
January 2023

Forward Looking Statements

This presentation and accompanying oral presentation (referred to herein collectively as the "presentation") contain express and implied forward-looking statements that involve substantial risks and uncertainties. All statements contained in this presentation, other than statements of historical facts, including statements regarding expectations of Exscientia plc ("we," "us", "our," or "Exscientia"), our strategy, future operations, future financial position, future revenues, projected costs, prospects, plans, potential market and growth opportunities, competitive position, market trends, addressable market opportunity and objectives of management, are forward-looking statements. The words "anticipate," "believe," "estimate," "expect," "intend," "may," "might," "plan," "project," "target," "potential," "will," "would," "could," "should," and similar expressions are intended to identify forward-looking statements, although not all forward-looking statements contain these identifying words. Forward-looking statements are based on our management's beliefs and assumptions and on information currently available to our management. Such statements are subject to risks and uncertainties, and actual results may differ materially from those expressed or implied in the forward-looking statements due to various important factors. We may not actually achieve the plans, intentions or expectations disclosed in our forward-looking statements, and you should not place undue reliance on our forward-looking statements.

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This presentation contains estimates, projections and other information concerning our industry, our business and the markets for our products. Information that is based on estimates, forecasts, projections, market research or similar methodologies is inherently subject to uncertainties, and actual events or circumstances may differ materially from events and circumstances that are assumed in this information. Unless otherwise expressly stated, we obtained this industry, business, market and other data from our own internal estimates and research as well as from reports, research surveys, studies and similar data prepared by market research firms and other third parties, industry, medical and general publications, government data and similar sources. While we are responsible for the accuracy of such information and believe our internal company research as to such matters is reliable and the market definitions are appropriate, neither such research nor these definitions have been verified by any independent source.

Patient-first AI: Integrated technologies to discover, design and develop precision medicines.

30+
Programmes consisting of wholly owned, co-owned and partnered

4
Clinical stage compounds*

>$6.5b
Potential partnership milestones (over $3.5b of which are pre-commercial)

10%
Average royalty rate without co-investment

$625m
3Q22 cash with F9M 2022 net cash burn of $15m**

21%
Top-end royalty rate with co-investment in Sanofi collaboration

*Includes out-licensed programmes
**On a constant currency basis as of September 30, 2022

Multiple near-term milestones

Upcoming Pipeline Progress

- Enroll first patient in Phase 1/2 trial for ‘617 (CDK7i) in 1H 2023
- Enroll first patient in IGNITE Phase 1/2 trial for ‘546 ($A_{2A}R$) in 1H 2023
- Kinase X programme continues to advance into clinic; update in 1H 2023
- New patient selection biomarker data on multiple programmes throughout the year
- At least 3 new targets disclosed by YE 2023

Upcoming Platform Advancements

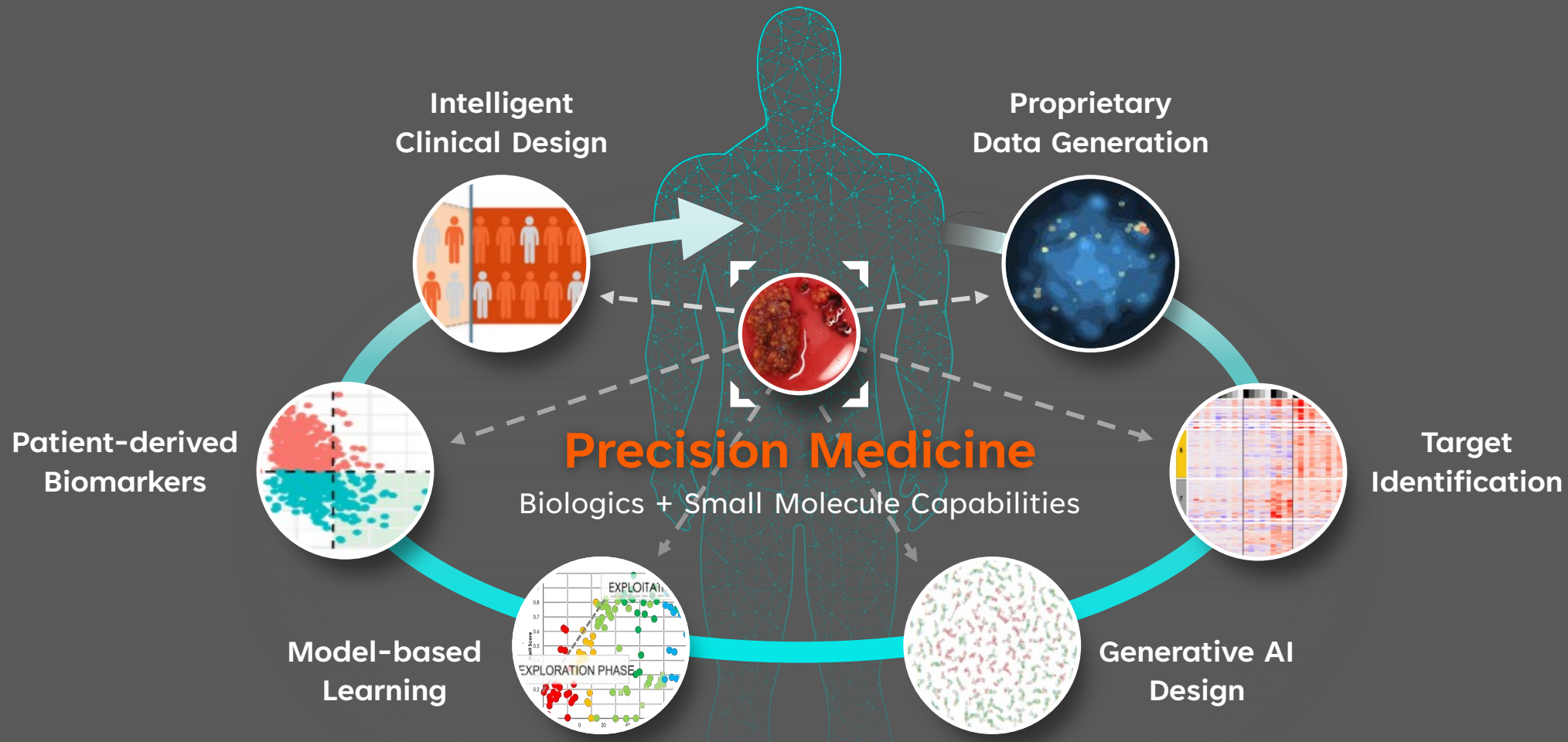
- At least two new partnerships during 2023
- Advancement of first antibody programme from biologics design platform
- Open 50,000 sq ft precision medicine centre of excellence in 1H 2023
- Open 46,000 sq ft of new biologics lab and automation facility by mid-2023
- Additional clinical trials utilising precision medicine platform

Cash balance and expected partner milestones provide foundation to execute on business plan

4 major reasons clinical trials fail

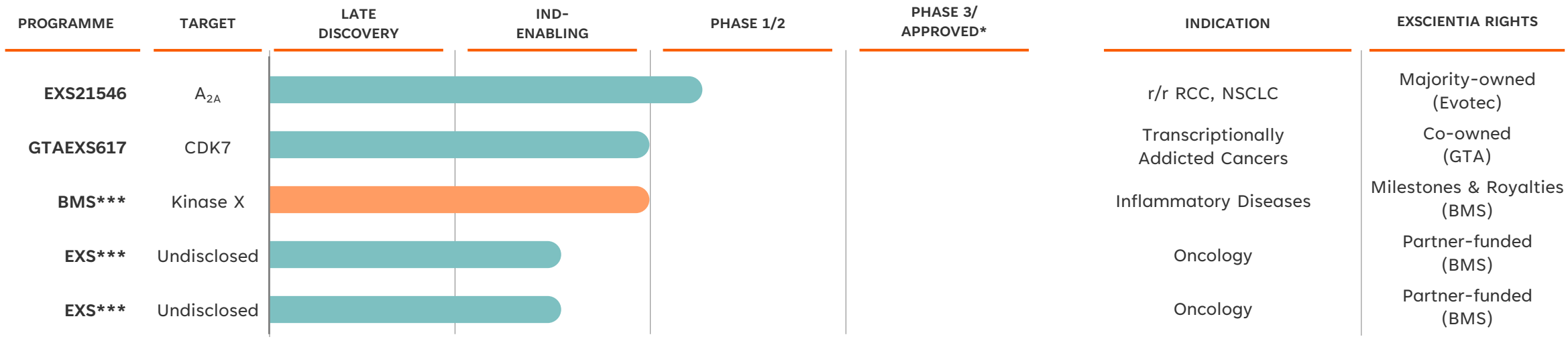
1. **Drug design:** Inadequate safety, potency or bioavailability
2. **Target biology:** Weak target-disease correlation
3. **Patient selection:** Not enrolling patients that are most likely to benefit from the therapy
4. **Clinical trial design:** Biological efficacy was obscured due to protocol issues

Exscientia's solution: Integrating knowledge of the patient and drug



Pipeline advancing to the clinic

Two targets disclosed, three more expected to be disclosed in 1H23



>10 programmes with 50-100% ownership

>20 partnered programmes with substantial economics

Internal focus on precision oncology

Additional clinical programme through DSP collaboration

*Phase 3 may not be required if Phase 2 is registrational

Why are our clinical candidates different?

Differentiated through design and personalised medicine

Target	Phase	Target Market	Key Differentiation
A_{2A}	Phase 1/2	~20-50% of r/r RCC and NSCLC patients estimated to be ABS* high	Novel approach to patient selection allows identification of potential responders
CDK7	Entering Phase 1/2	Multiple relapsed/refractory solid tumour indications	Precision designed PK/PD specific for mechanism. Biomarker identification of high-grade responders
Kinase X	IND-enabling	Multiple immunology indications	Better selectivity, improvements in whole blood potency and predicted human dose <200mg/day
Target 4	IND-enabling	Solid tumour and haematology patients	Uniquely combines reversibility with brain penetration, together with PK to optimise therapeutic index
Target 5	IND-enabling	Multiple haematology indications	Solved potential dose-limiting toxicity issue present in competitor compounds. Precision medicine platform to factor in likely responding groups

Does not include Exscientia-designed programmes that entered clinical trials through DSP collaboration, *Exscientia's biomarker (adenosine burden score)

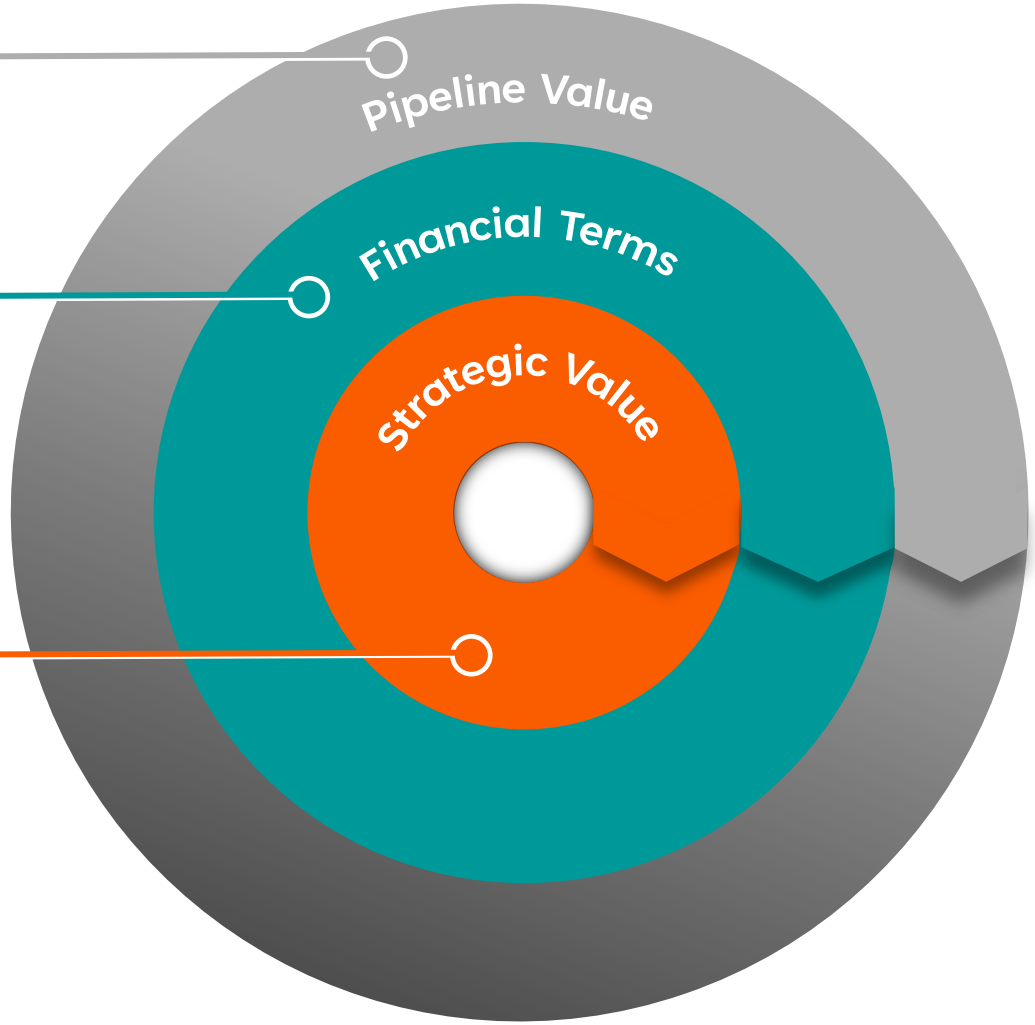
High value partnerships also create strong foundation

Potential for partnership structure to fund majority of Exscientia's operations

- >20 programmes between BMS & Sanofi
- NPV of a Sanofi programme estimated at ~50% of a wholly owned programme

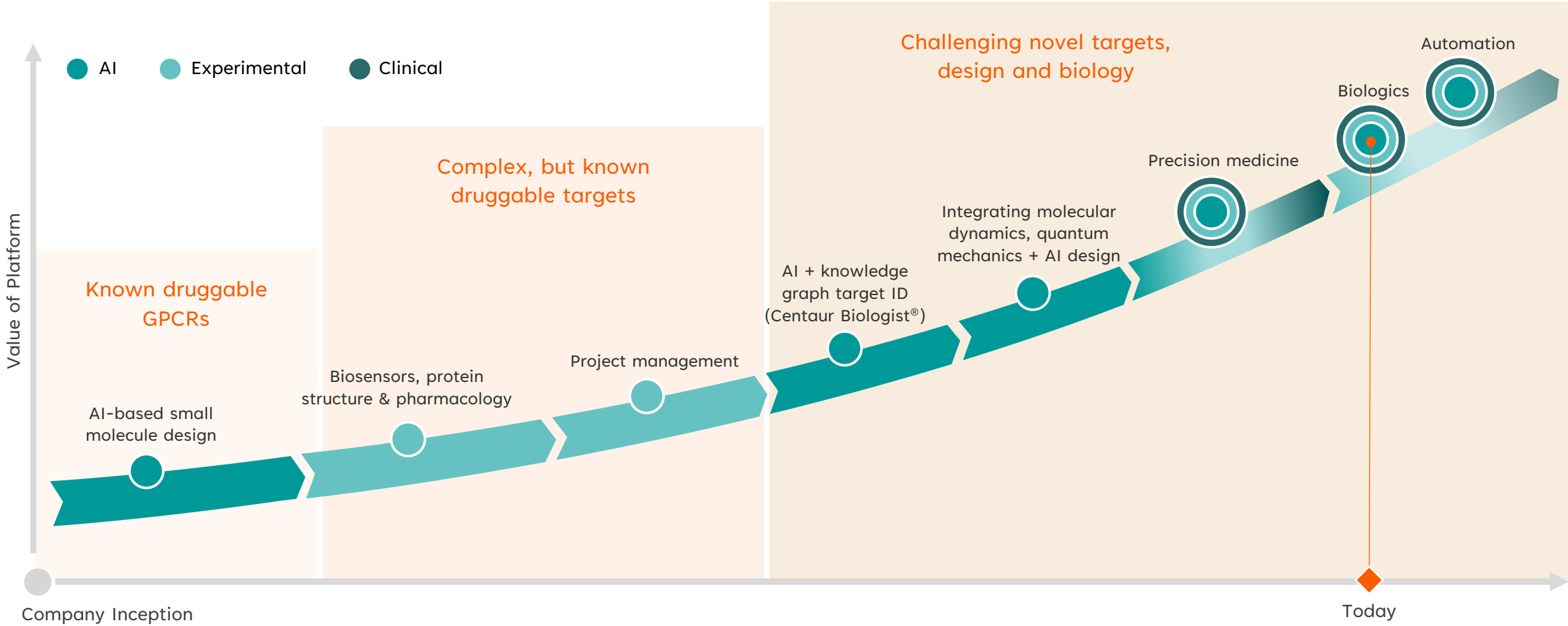
- Upfront payments typically cover operating costs
- >$3.5 billion of potential pre-commercial milestones
- $3.0 billion of potential commercial milestones
- Average royalty rate of 10%
- Co-investment option can take royalties up to 21%

- Strong partners for clinical development and marketing
- Provide therapeutic area expertise for programmes
- Learnings are integrated into broader platform



Expanding technologies enhance value creation

Learning faster by doing more



Timeline for illustrative purposes

Delivering better pipeline candidates, faster

8

Precision designed development candidates

6

Projects in-licensed by partners

1st

Prospective clinical trial showing improvement in cancer treatment outcomes through AI*

70%

Reduction in discovery time from target ID to candidate

80%

Improved capital efficiency in drug discovery

>$3.5b

In pre-commercial milestone potential

*Kornauth et al. Cancer Discovery 2021

Our strategy maintains balance sheet strength

First Nine Months (F9M) 2022 financial performance

($m)	F9M22	F9M21	Comments
Cash inflows from collaborations	$117.3	$67.5	Expect to remain lumpy around development milestones and business development
Net operating cash (outflows)/ inflows	($15.0)	$8.3	Continue to make measured investments into pipeline and platform growth
Capital expenditures	$18.5	$4.5	2022 CapEx expected to be higher YoY with automation and precision medicine expansion
Cash balance*	$624.7	$253.4	Project several years of cash runway

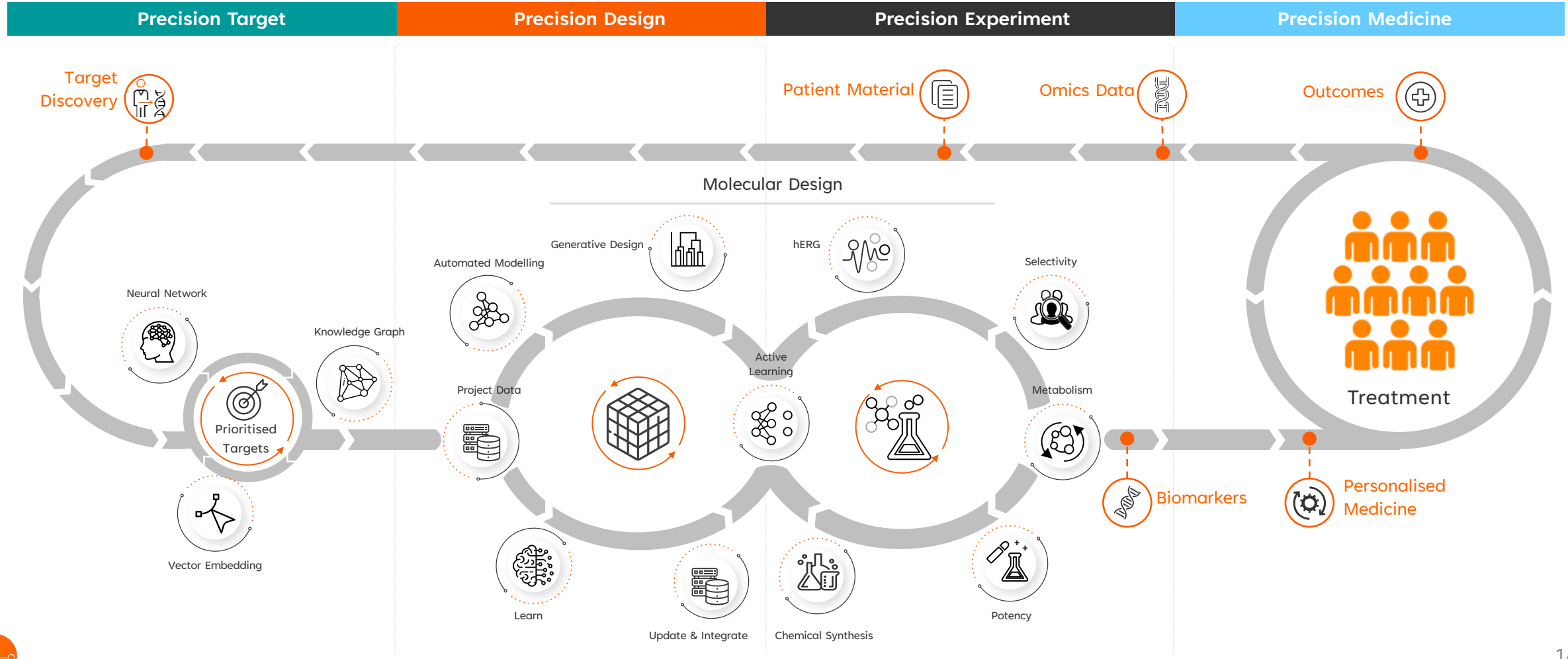
Expect partnerships to continue to moderate cash burn

*Includes fixed term bank deposit, based on constant currency as of September 30, 2022
Shares outstanding: 122.8 million as of September 30, 2022

Technologies

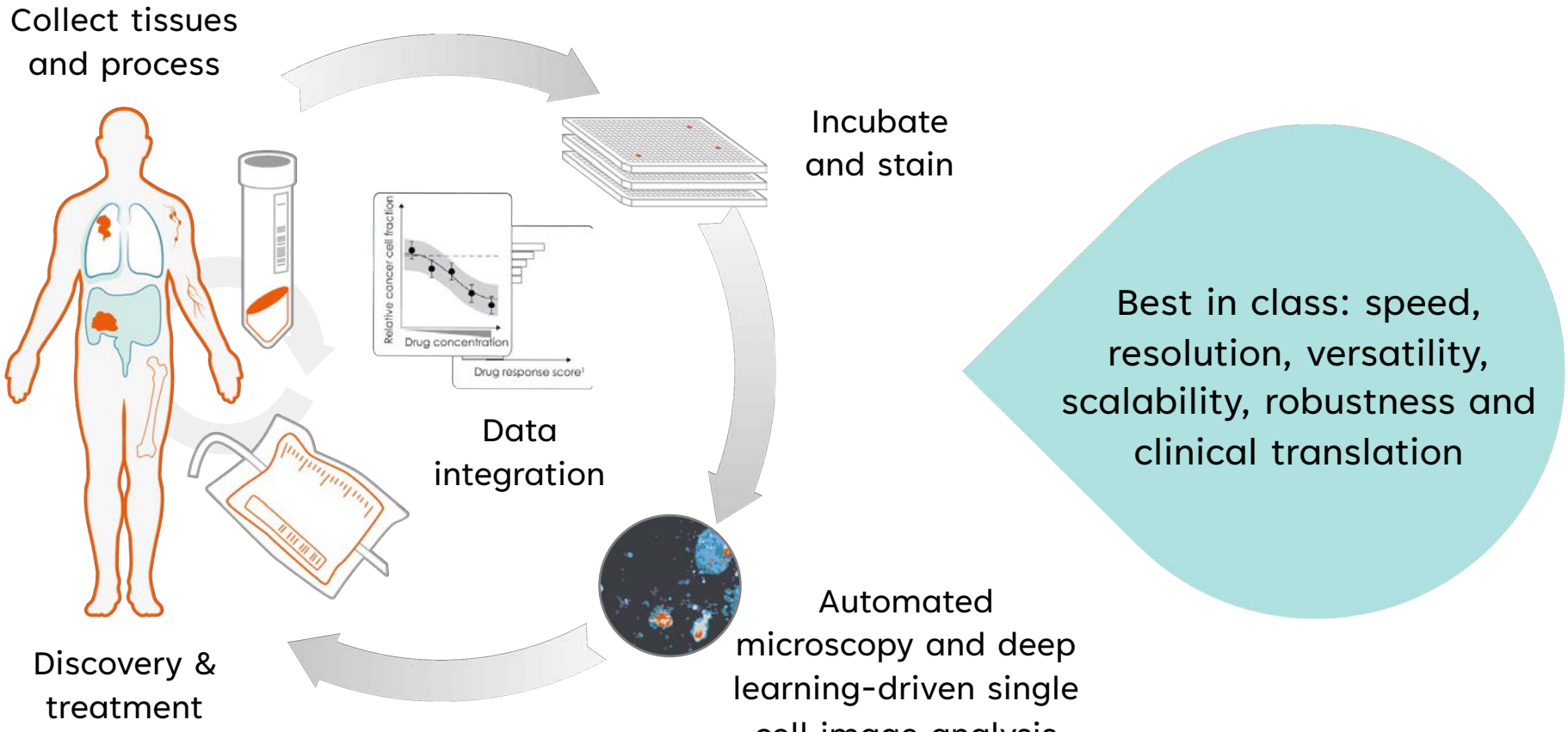
Patient-first AI is a learning process

Our end-to-end architecture brings the patient into every stage of drug creation



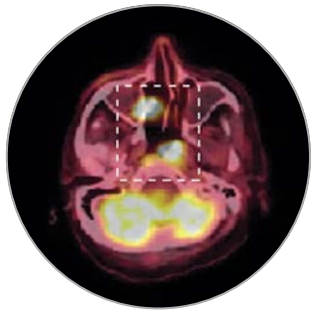
Our differentiated process

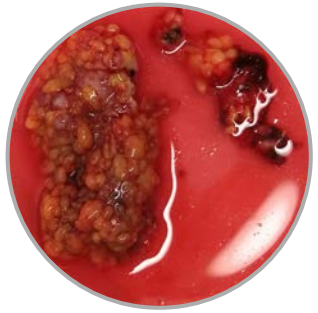
Interrogating drug action in complex primary tissues at the single cell level

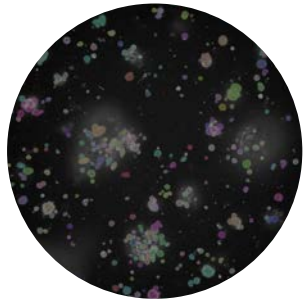


Vladimer et al. *Lancet Hematol*. 2017; Vladimer, Snijder et al. *NCB* 2017; Schmidl, Vladimer et al. *NCB* 2019; Kornauth et al. *Cancer Discovery* 2021

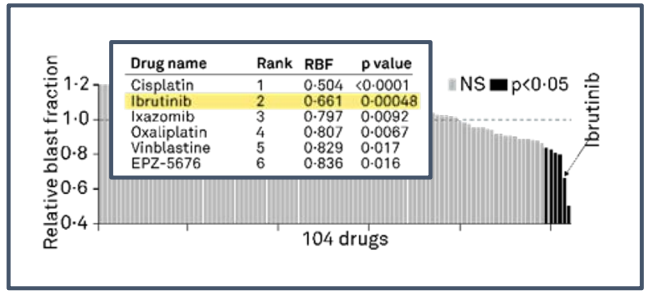
EXALT-1 study: First AI-supported functional precision medicine platform to directly improve cancer treatment & patient outcomes

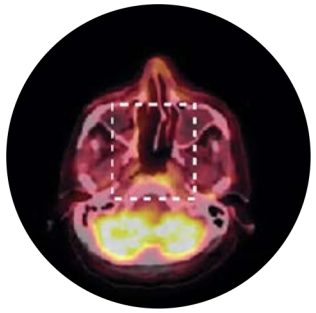






Select the best tolerable treatment in the tumour board





82-year old DLBCL patient intolerant to chemotherapy

Collection of viable tumour tissue - not organoids

Expose to >100 clinically usable drugs in the lab - automate microscopy & single-cell image analysis

Measure drug response using image-based screening

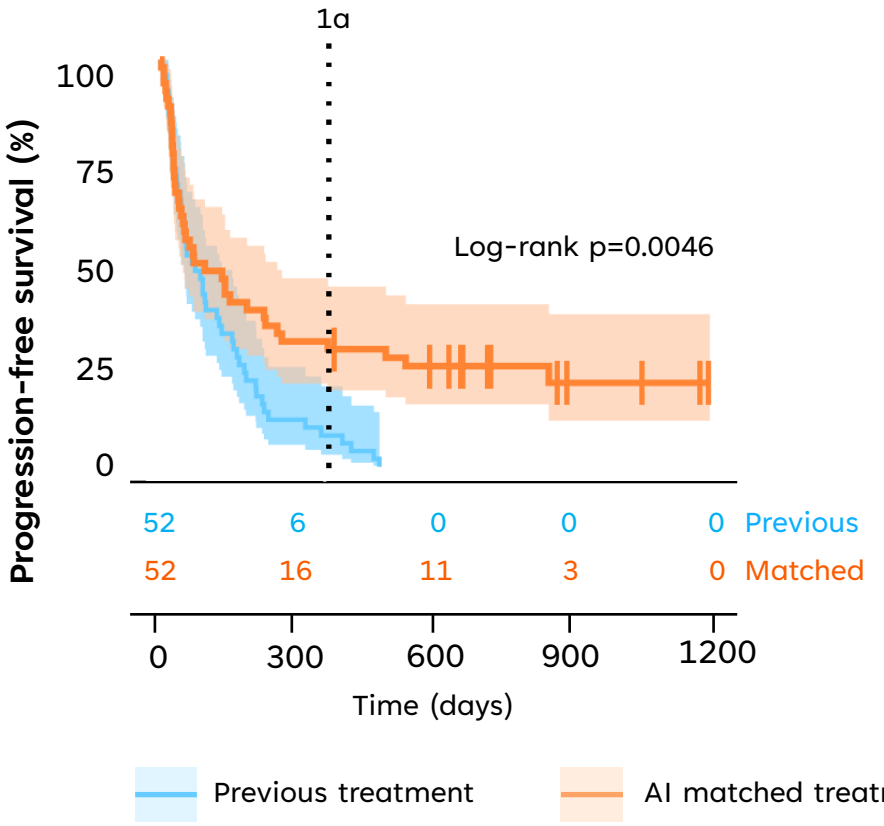
Complete remission 2-years survival at fraction of cost of CAR-T

DLBCL = diffuse large B-cell lymphoma

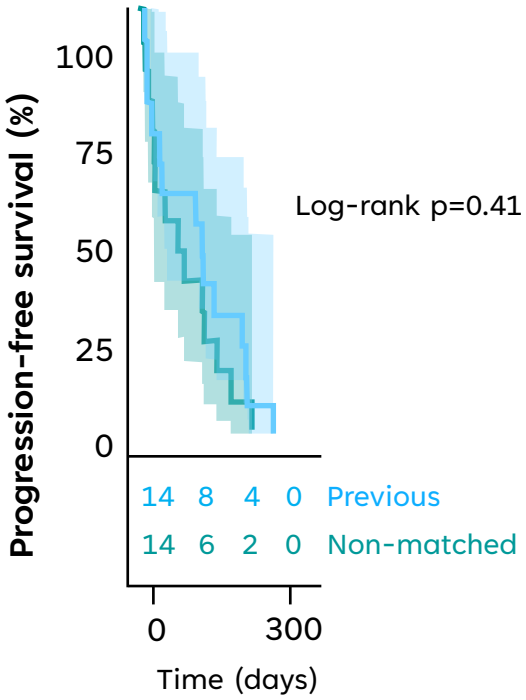
EXALT-1 study results

Patients receiving drugs prioritised by platform had significantly better outcomes

Patients receiving treatment prioritised by platform had improved PFS

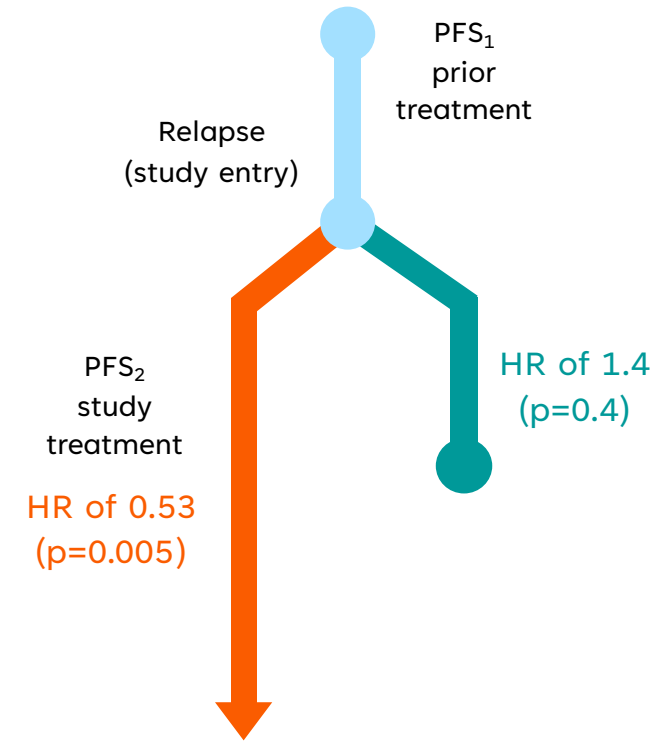


Patients receiving treatment not prioritised by platform did not have improved PFS



Previous treatment | AI matched treatment | Non-matched treatment

CANCER DISCOVERY

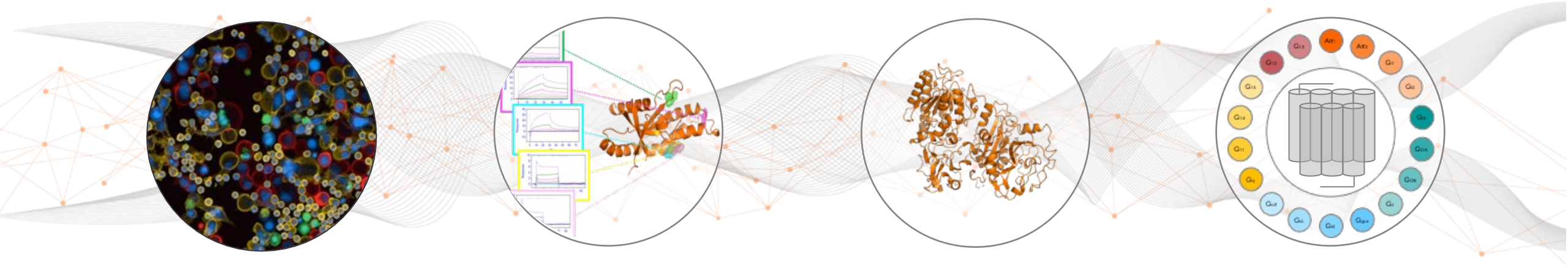


54% of patients demonstrated clinical benefit of >1.3-fold enhanced PFS compared to prior therapy

Kornauth et al. *Cancer Discovery* 2021, 1a: Kaplan–Meier plot comparing scFPM-matched treatment with previous treatment. Dotted line denotes 1-year follow-up

Extensive proprietary data generation capabilities

Over 45,000 sq ft of laboratories producing assays, seed data and structures



Primary tissue disease models

Live patient tissues

Single cell resolution

Deep learning AI

Biobanked samples

World-class biosensors

Proprietary seed data

GPCRs in native state

Label free and automated

High throughput crystallography

Proprietary seed data

Automated Hotspot binding site analysis

Extensive pharmacology

Transducerome mapping

Automated assay development

Polypharmacological profiling

Creating a consistent flow of high-quality targets

Integrated capabilities drive new discoveries

>35%

of pipeline generated using Exscientia target ID platforms

AI

CENTAUR BIOLOGIST®

Applies deep learning to genome-scale datasets to identify connections and predict target-disease associations

Global knowledge graphs

Experimental

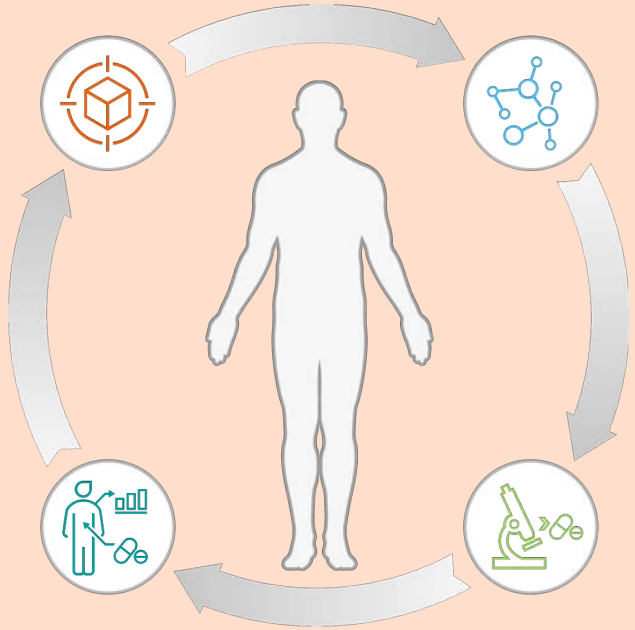
CELLOMICS™

Launched 2022, open-source programme in collaboration with the University of Oxford

Focus on phenotypic assays

Experimental + AI

PRECISION MEDICINE



Proprietary human tissue platform

Single cell phenotypic screening to ID novel targets

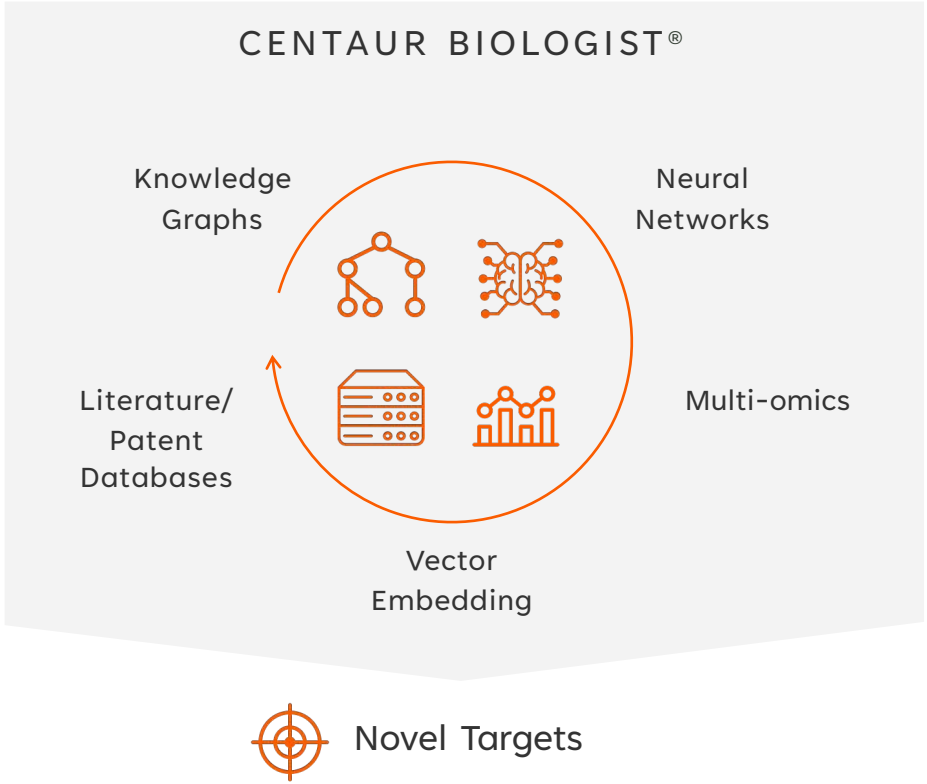
70%

of oncology targets tested on platform

Percentages as of YE 2022

Prioritising and validating targets with Centaur Biologist

AI-driven target identification through deep learning algorithms

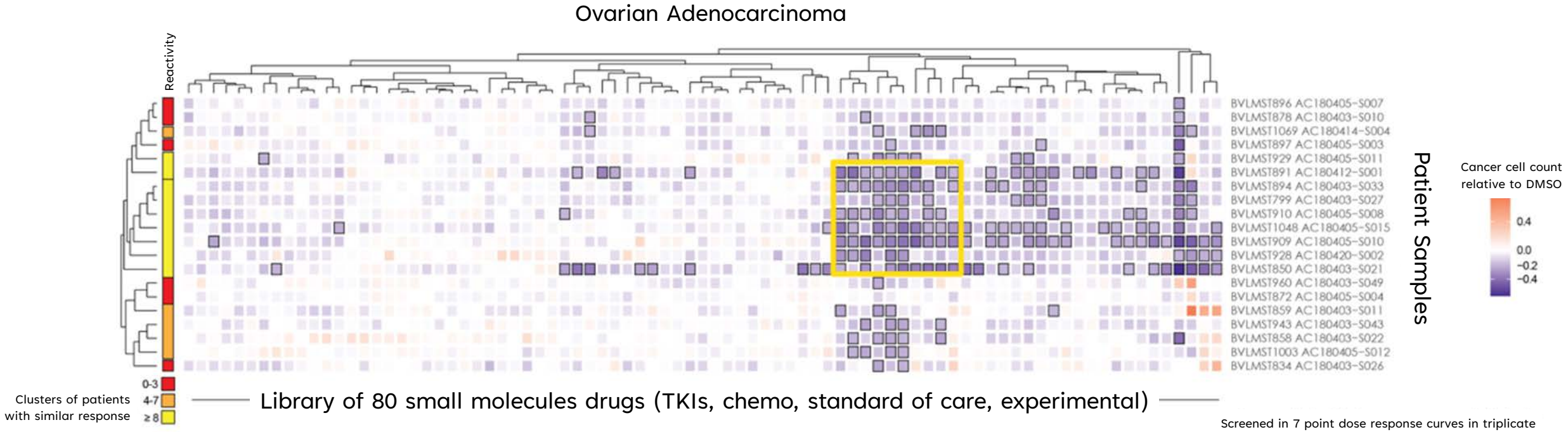


- Applies deep learning to genome-scale datasets to identify connections and predict target-disease associations
- Builds insights from constructing global knowledge graphs
- TrendyGenes algorithm generates graphic representation of literature identifying trends from over 30 million publications
- Disease area agnostic with application to date across oncology, immuno-oncology, immunology and rare disease

Enables Exscientia to identify targets with a higher probability of translating into the clinic

Cancer is a heterogenous disease

Our platform is designed to better understand differential response



Evaluating an array of drugs and primary tissues at single cell resolution to quantify cancer cell cytotoxicity uncovers **potential novel target space in ovarian cancer**

Gutierrez-Perez et al, AACR (2022)

Precision objectives for precision design

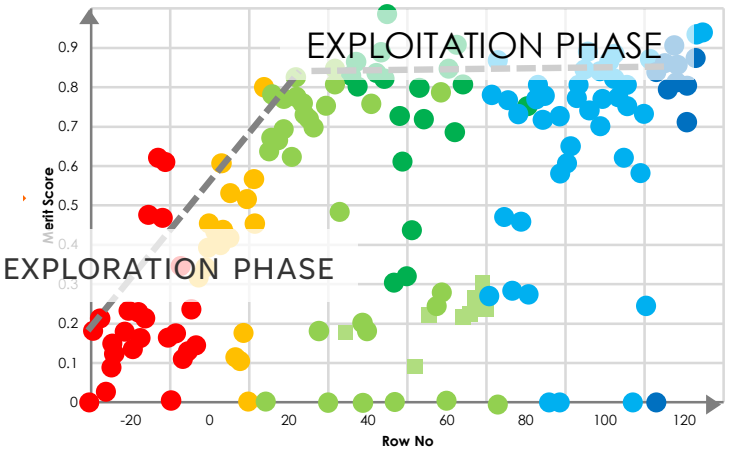
Dozens of endpoints can be optimised in parallel

Target Product Profile

Candidate Criteria	Design Goal
CDK7 IC_{50} (nM)	<10
CDK family selectivity	>100 fold
HCC70 (breast cancer) IC_{50} (nM)	<100
OVCAR-3 (ovarian cancer) IC_{50} (nM)	<100
hERG IC_{50} (μM)	>5
Human microsome Clint μL/min/mg	<15
Human hep Clint μL/min/10^6 cells	<15
Caco-2 A2B (efflux) 10^{-6} cm/s	>3 (<5)

MPO: Multiparameter Optimisation

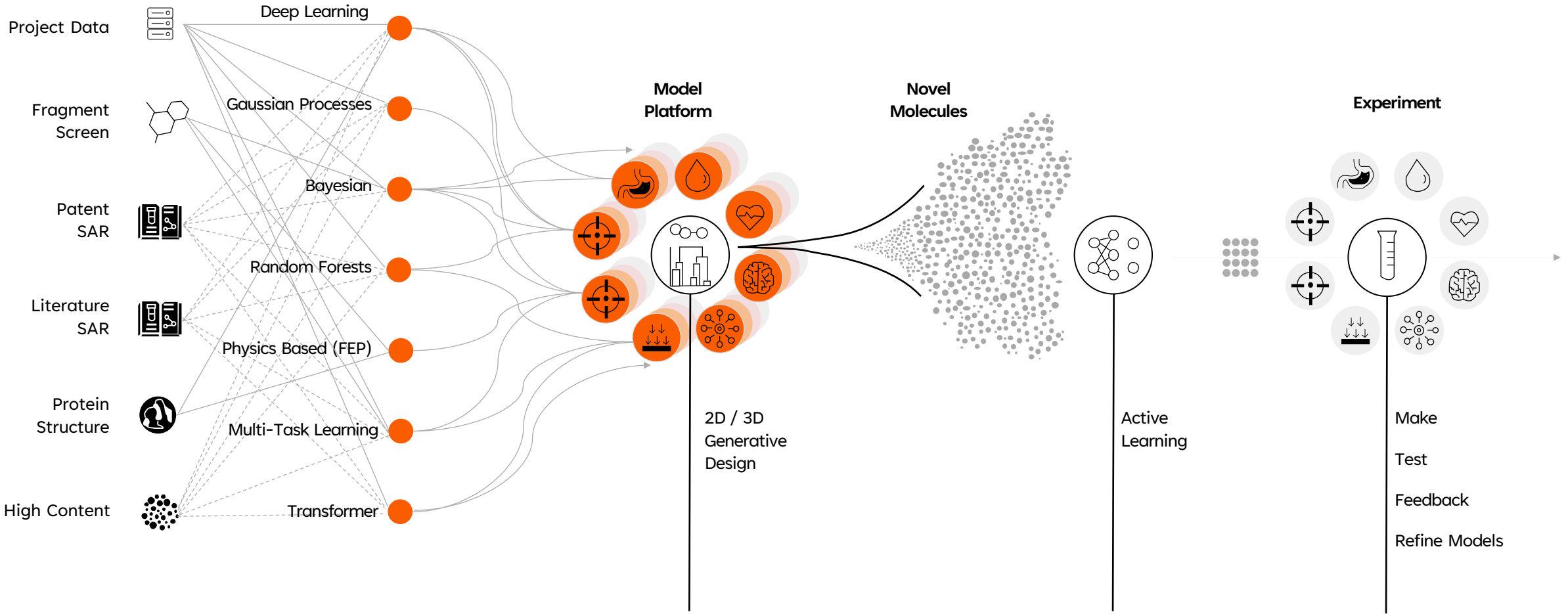
Merit: Project Telemetry

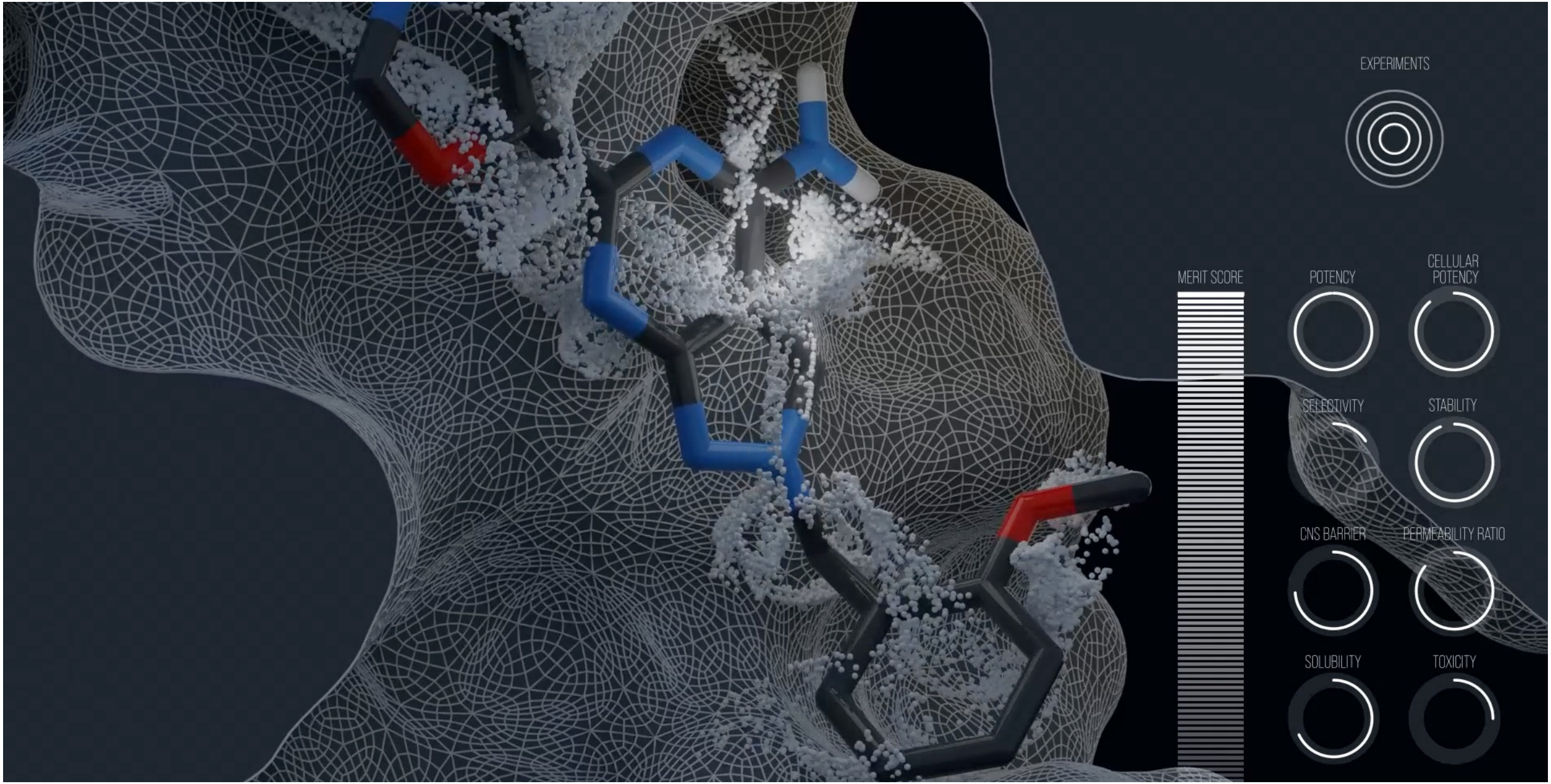


For illustrative purposes

Data and model agnostic

Our AI design platform can optimise complex drugs from diverse starting data



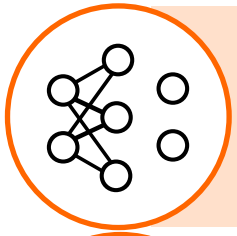


Watch video at: https://bit.ly/EXAIvideo

Active learning AI leads to creative breakthroughs

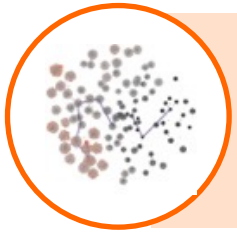
Counterintuitive selection goes against preconceptions and breaks dogma

AI system to maximise information gain



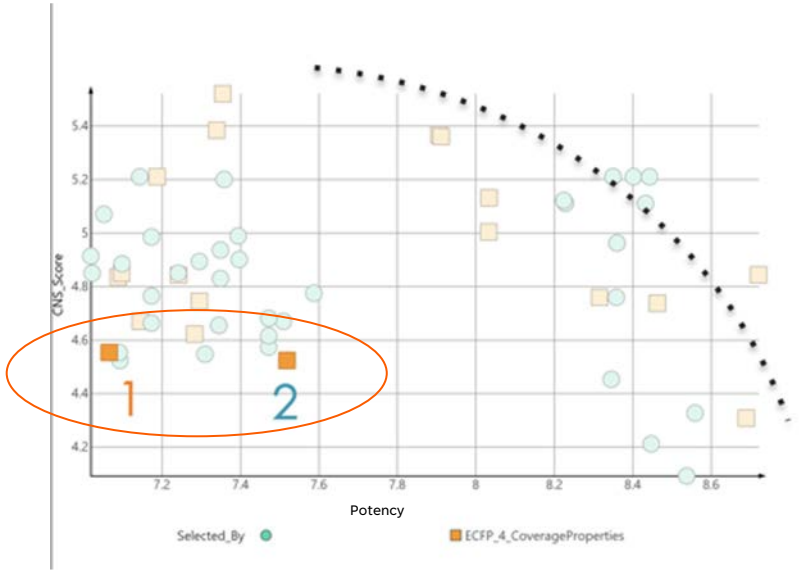
Chooses which compounds to synthesise from output of generative design and predictive models

Mathematically evaluates how much can be learned from each compound



Efficiently explores the available structural and property space

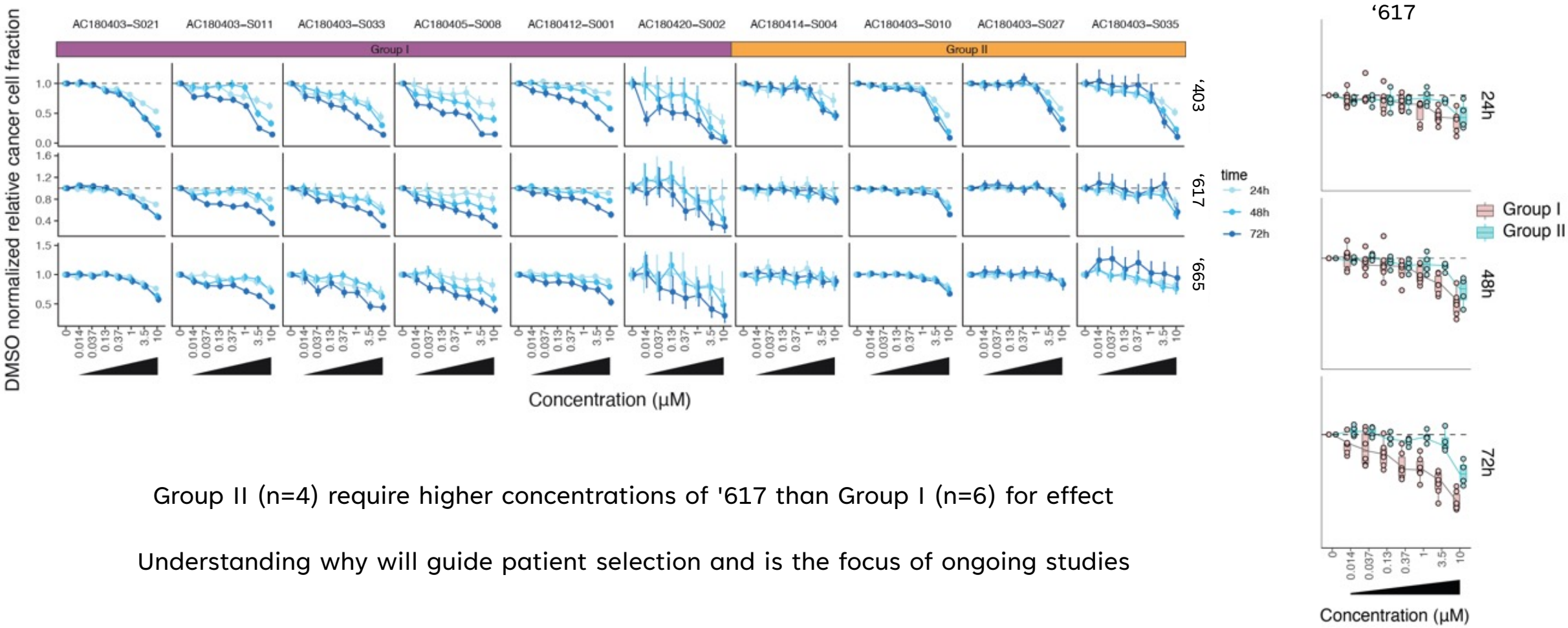
Example of our AI choosing unexpected candidates that led to a design breakthrough and development candidate



20 compounds (square) are selected by active learning

Defining patient selection during drug design

'617: Ovarian cancer patient samples stratify into two groups



Group II (n=4) require higher concentrations of '617 than Group I (n=6) for effect

Understanding why will guide patient selection and is the focus of ongoing studies

Besnard et al, AACR (2022)

Advancing to the clinic

Design *patient-centric* drugs with an improved probability of success

- Use precision medicine platform with patient samples to profile high response populations for that specific drug prior to initiating clinical trials
- Validate signatures early in the clinic:
  - Initial clinical trials to occur with concurrent prospective biomarker testing
  - Positive and negative controls provide validation of biomarker/signature
- Use validated biomarker/signature to enrich later clinical trials with patients expected to have the highest response
- Leverage adaptive trial design to build efficiency into clinical programmes
- Platform supports analysis of mono or combination therapies

‘546
IGNITE Phase 1/2 initiated
FPI expected in 1H 2023

IGNITE: ‘546 Phase 1/2 initiated in RCC & NSCLC

Exscientia’s biomarker signature for patient selection to be tested during trial

Two-part trial assessing safety, PK, PD and efficacy of EXS21546:

EXS21456 + PD-1 inhibitor

Part 1: Dose Escalation	Part 2: Dose Expansion
n=up to 30 relapsed/refractory RCC and NSCLC patients Across up to 7 dose levels to establish MTD	n=up to 80 relapsed/refractory RCC and NSCLC patients Primary efficacy endpoint: ORR

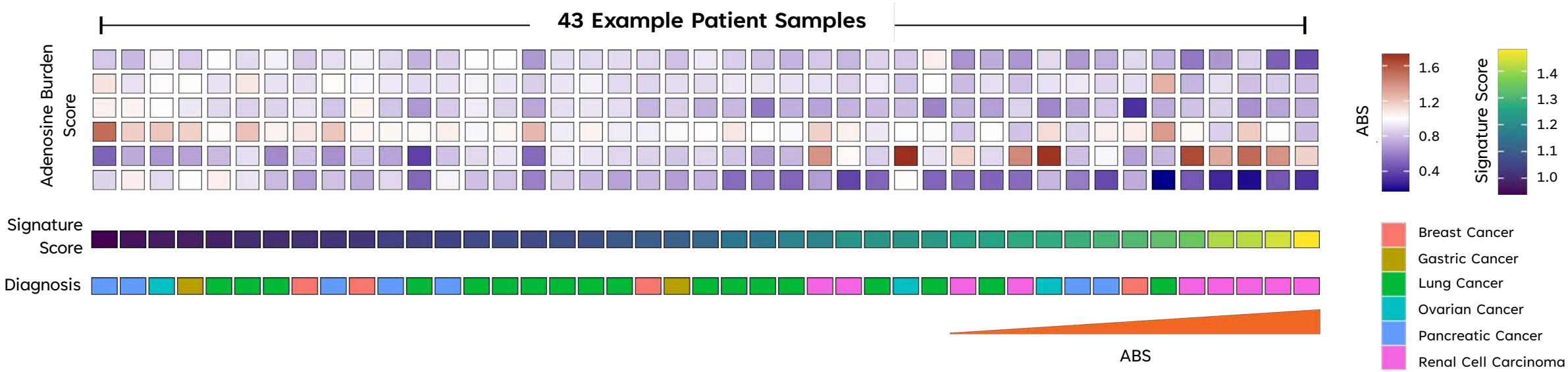
MTD

Biomarker signature, adenosine burden score (ABS) to be evaluated

IGNITE is multicentre, open-label, two-stage clinical trial to evaluate safety, pharmacokinetics, pharmacodynamics and efficacy of EXS21546 in combination with PD-1 inhibition. Study to enroll patients who have progressed after checkpoint inhibition

Enriching for patients that will benefit most from ‘546

Developed novel adenosine-pathway activity signature



Exscientia's ‘546 response signature, the adenosine burden score (ABS), was developed using single cell transcriptomics of primary samples after *ex vivo* perturbation with stabilised adenosine

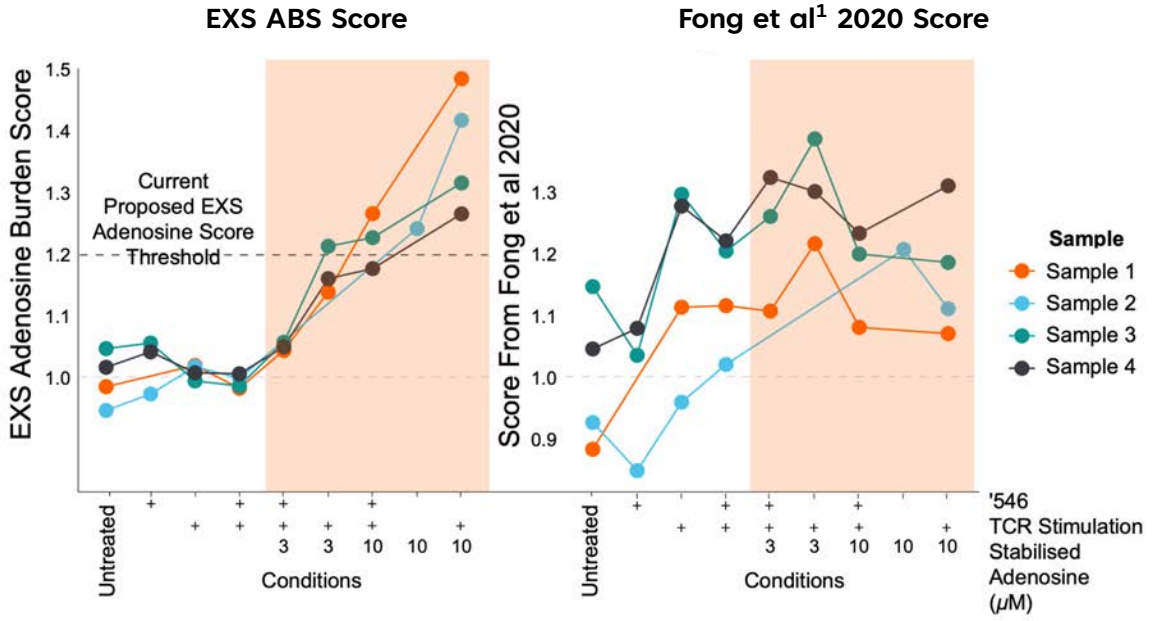
- Expected to enrich patients more likely to respond to adenosine-pathway inhibition
- Supported with biological validation including soluble factor data
- Exscientia’s ABS is differentiated from other published “adenosine signatures”

Vladimer et al, ESMO I-O (2022)

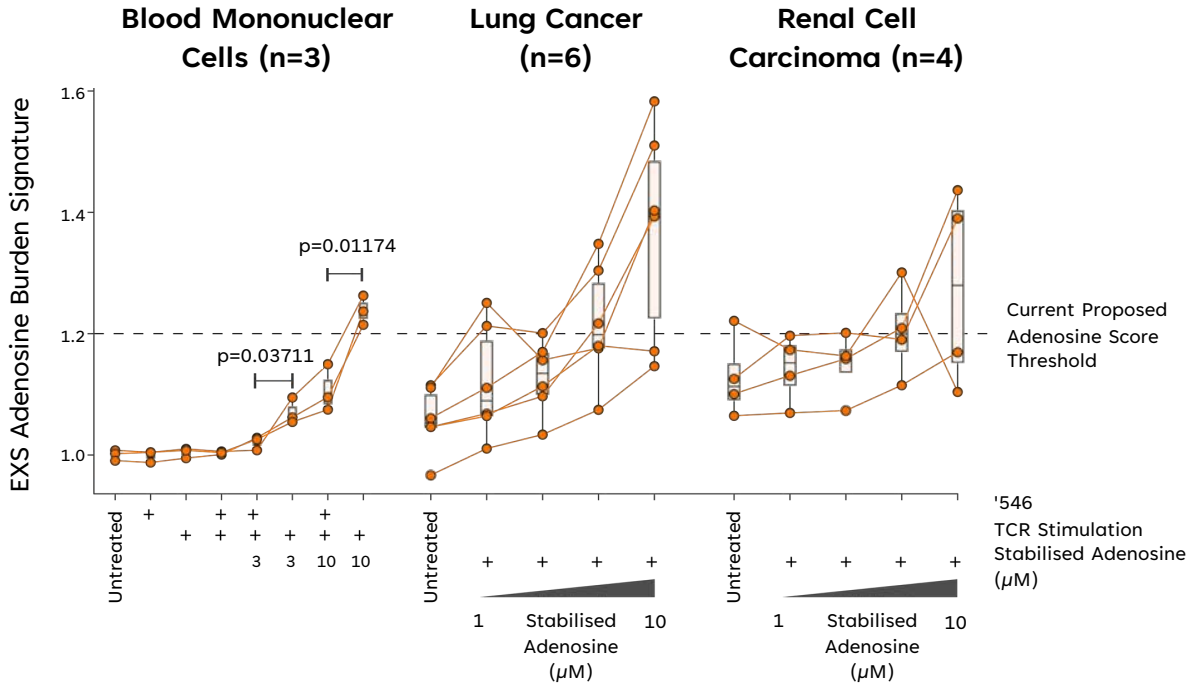
Biological validation of the ABS in various models

Ensuring the signature performs as expected *ex vivo* in the presence of adenosine

Exscientia's ABS outperforms published signature in the presence of stabilised adenosine



ABS also behaves well in cancer patient samples exposed to stabilised adenosine *ex vivo*



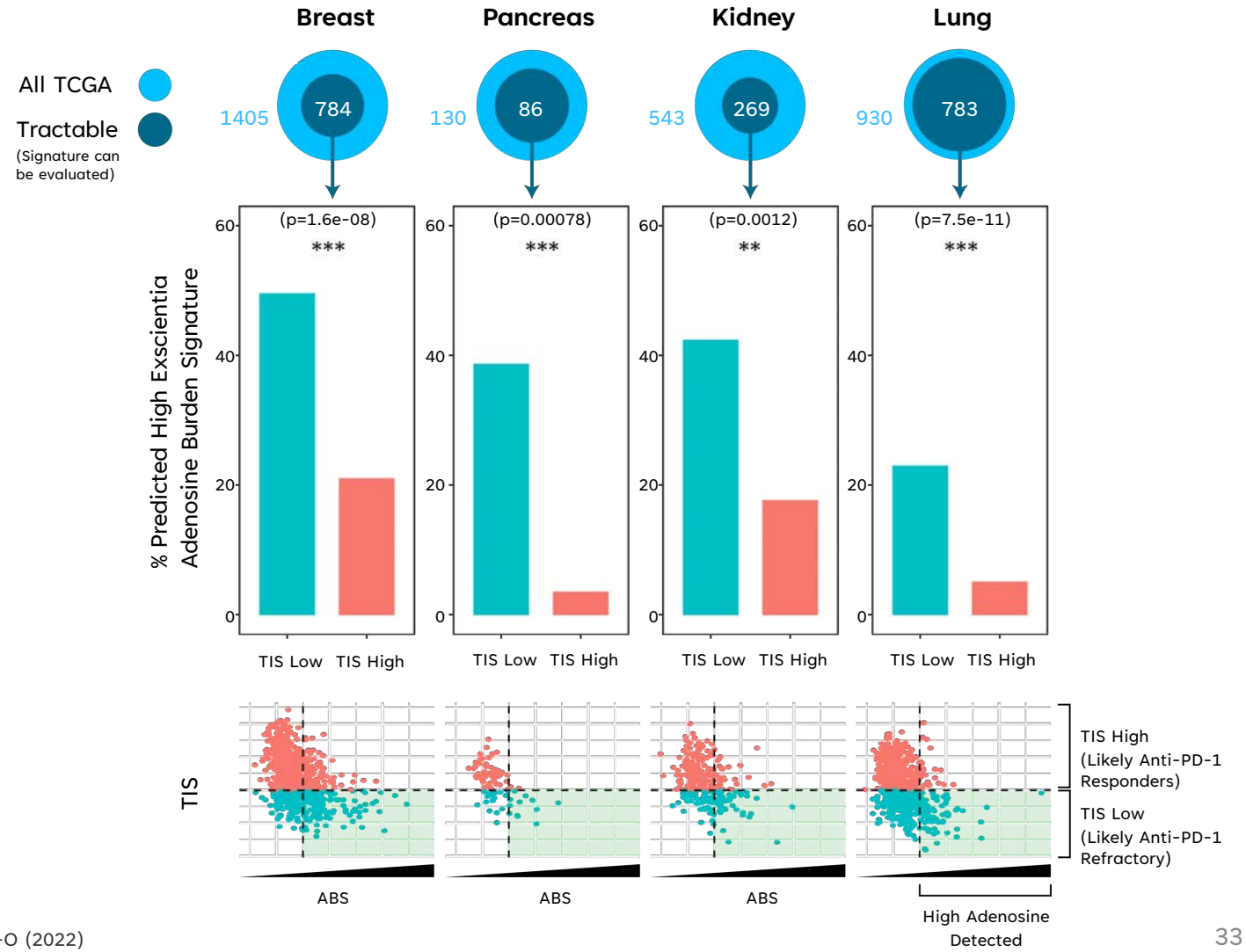
1. Fong et al, Cancer Discovery (2020); ABS = adenosine burden score, stabilised adenosine = 2-chloroadenosine (CADO); Vladimer et al, ESMO I-O (2022)

Connection between high adenosine and inflammation

High ABS correlates with weakened local immune response in microenvironment

Biological validation of ABS in a larger sample cohort (TCGA[1]):

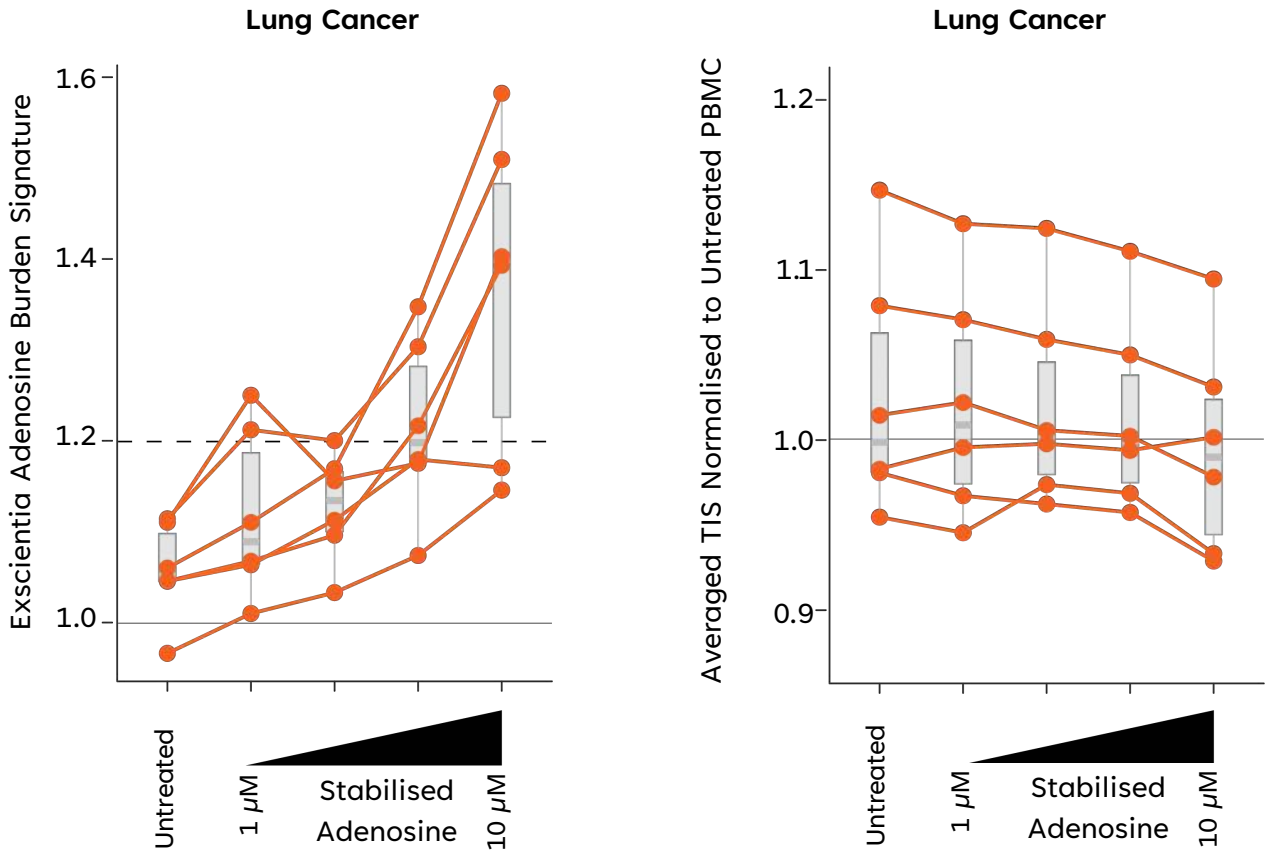
- Higher adenosine as determined by ABS correlates to lower inflammation in the tumour as determined by the tumour inflammation signature (TIS)
- Patients who have high ABS have a low TIS score
- TIS is a predictive signature for anti-PD-1 response[2]



1. The Cancer Genome Atlas Project (TCGA) dataset, NCI
2. Damotte et al, Journal of Translational Medicine (2019); Vladimer et al, ESMO I-O (2022)

Findings from ABS inform ‘546 clinical strategy

Correlation between higher ABS and lower TIS[1] *ex vivo* with stabilised adenosine



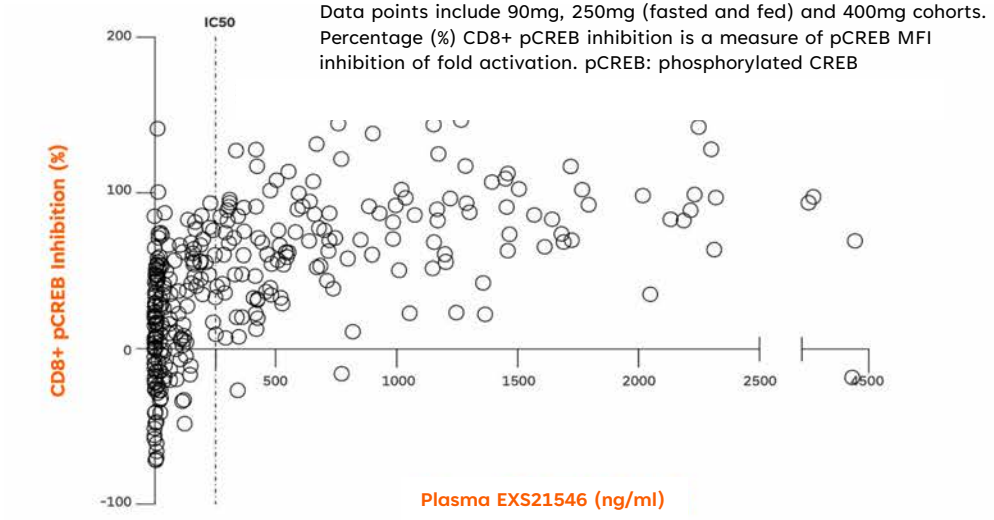
- ‘546 may be able to reverse immuno-suppression
- Potential to increase the likelihood of responding to checkpoint inhibitors in patients who relapse or did not respond originally

Damotte et al, Journal of Translational Medicine (2019) Vladimer et al, ESMO I-O (2022)

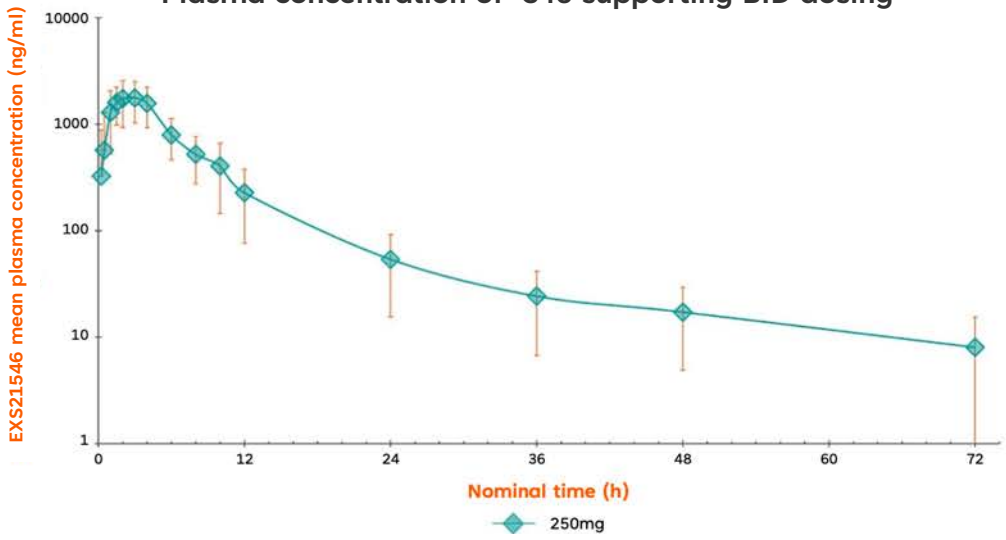
‘546 achieved targeted objectives in Phase 1a study

Potency, selectivity, PK, low expected brain exposure achieved

Correlation of ‘546 plasma exposure to inhibition of $A_{2A}R$ signalling



Plasma concentration of ‘546 supporting BID dosing

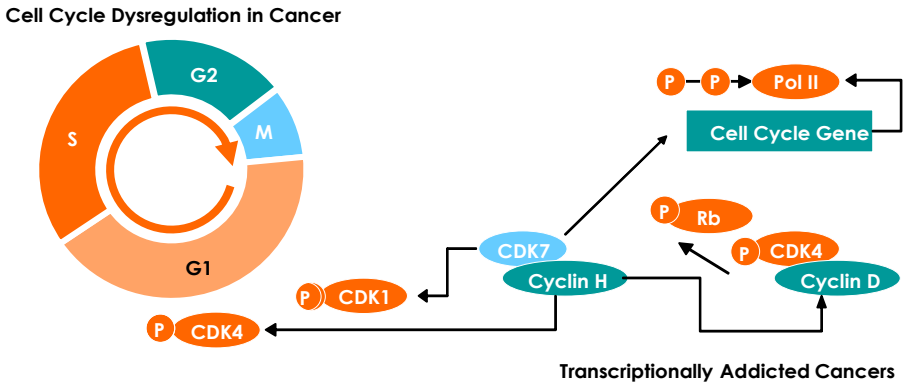


- Observed human PK for ‘546 in line with predictions from preclinical modelling
  - Supports BID dose for continuous A_{2A} receptor inhibition over a dosing interval
- ‘546 showed dose-dependent inhibition of CREB phosphorylation in CD8-positive cells
  - PD profile mirrored plasma exposure
- Level of lasting target engagement identified
  - Inhibition of A_{2A} receptor signalling sustained over BID dosing period

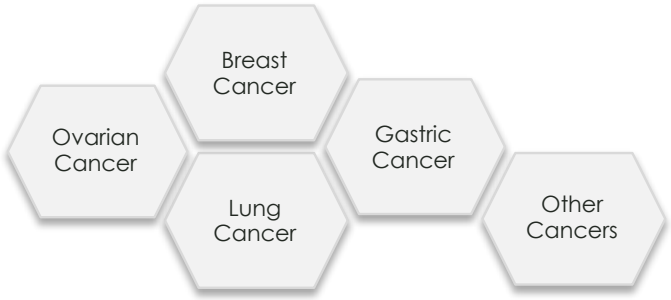
‘617
CTA filed for Phase 1/2
FPI expected in 1H 2023

CDK7: inhibition provides broad oncology opportunity

Dual targeting of cell cycle and transcription mechanisms



CDK7: Potential for multiple cancer indications



Importance of cell cycle inhibition

- CDK4/6 inhibitors have demonstrated the potential for cell cycle inhibitors to impact cancer
  - Ibrance (palbociclib) generated $5.4b sales in 2021
  - 65-75% of patients show response, but acquire resistance

Transcription and cell cycle dysregulation are both hallmarks of cancer

- Inhibiting both may be more effective in controlling growth
- Aberrant CDK7 overexpression is common in multiple indications and associated with poor prognosis
- Majority of cancers are 'transcriptionally addicted' with c-Myc overexpression

Potential for first line therapy or for CDK4/6 refractory patients

ER+: Oestrogen receptor positive. About 80% of all breast cancers are ER+.
Source: Xu et al. 2020 *Nature*; Sava et al. 2020 *Cancer and Metastasis Reviews*

Precision design to maximise effectiveness

Mechanism requires a tightly controlled target product profile

Non-covalent Potency and Selectivity

- Both potency and selectivity are critically important
- Early entrants increased potency and selectivity by covalent bonding
- This dramatically increased off target toxicity, leading to discontinuation

Design needs to achieve potency and selectivity non-covalently

Short Therapeutic Window

- Ideal therapeutic coverage would be 6-8 hours at IC_{80}
- Longer periods would lead to increasing systemic toxicity

Product needs to be highly potent, but with a short half-life

Bioavailable

- CDK7 inhibition will lead to toxicity if it remains at any site other than the tumour
- Absorption variability will cause either supra-doses or sub-therapeutic dosing

Goal is for very rapid absorption at the lowest possible dose

Our ‘617 candidate resolves critical design issues

Designed in <12 months and just 136 experimental compounds

	Assay	Candidate Criteria	Competing Phase 1 Candidate	Competing Phase 1/2 Candidate	‘617
Target affinity and selectivity	CDK7 IC_{50} (nM)	<10	6	30	2
	CDK family selectivity	>100 fold		<20	
Cell potency	HCC70 (breast cancer) IC_{50} (nM)	<100	2.5	500	4.2
	OVCAR-3 (ovarian cancer) IC_{50} (nM)	<100	0.8		0.8
Safety and metabolism	hERG IC_{50} (µM)	>5	5	24	>30
	Human microsome Clint µL/min/mg	<15	9	3.6	<3
	Human hep Clint µL/min/10^6 cells	<15	7	<15	2
Permeability / transporter liability	Caco-2 A2B (efflux) 10^{-6} cm/s	>3 (<5)	0.55 (51)	0.14 (107)	5.3 (4)
General properties	pH 7.4 µg/ml	>50	132	>100	120
	F % (p.o.)	>30%	100%	30 %	77%

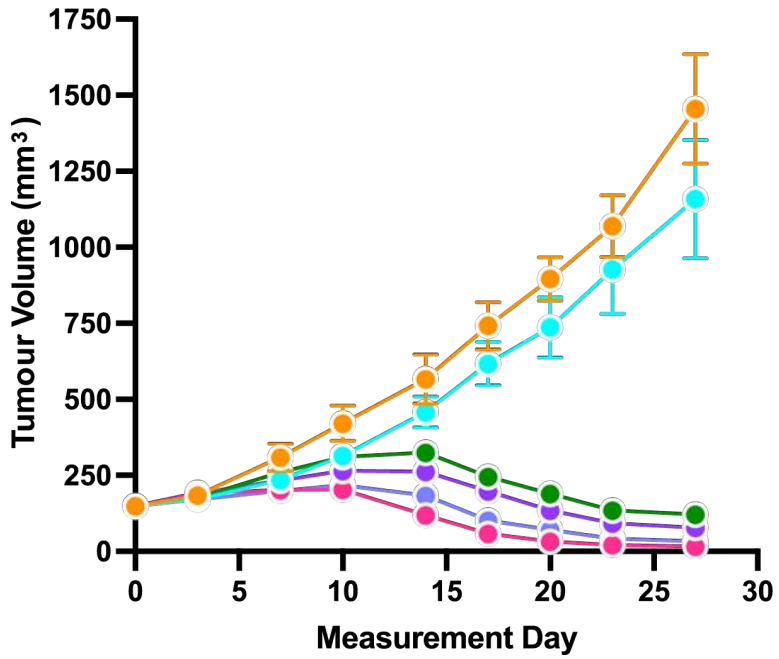
Meets or exceeds criteria Minor deviation Major deviation

- Potent biochemical and cellular activity
- High selectivity
- Excellent bioavailability and efflux

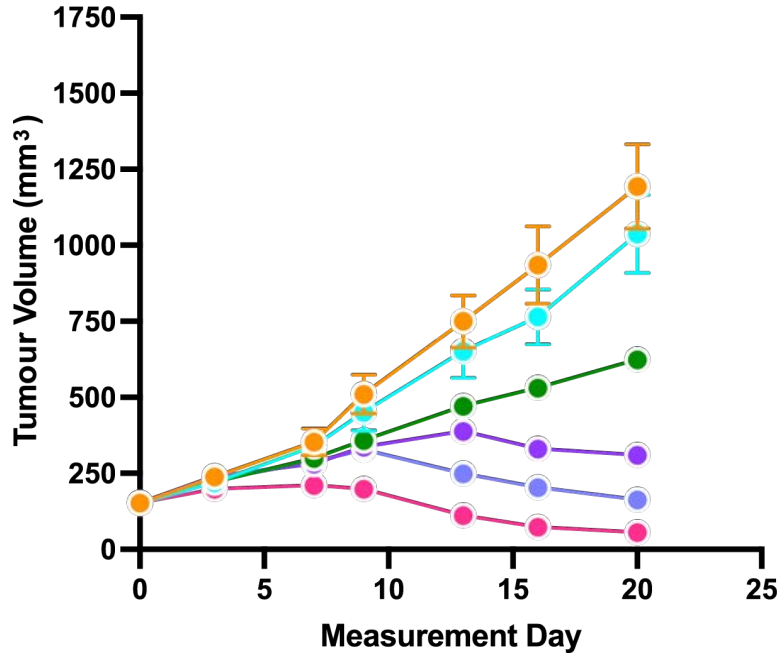
‘617 is highly effective in classical models

Potent anti-tumour activity demonstrated in multiple solid tumour types

OVCAR3



HCC70



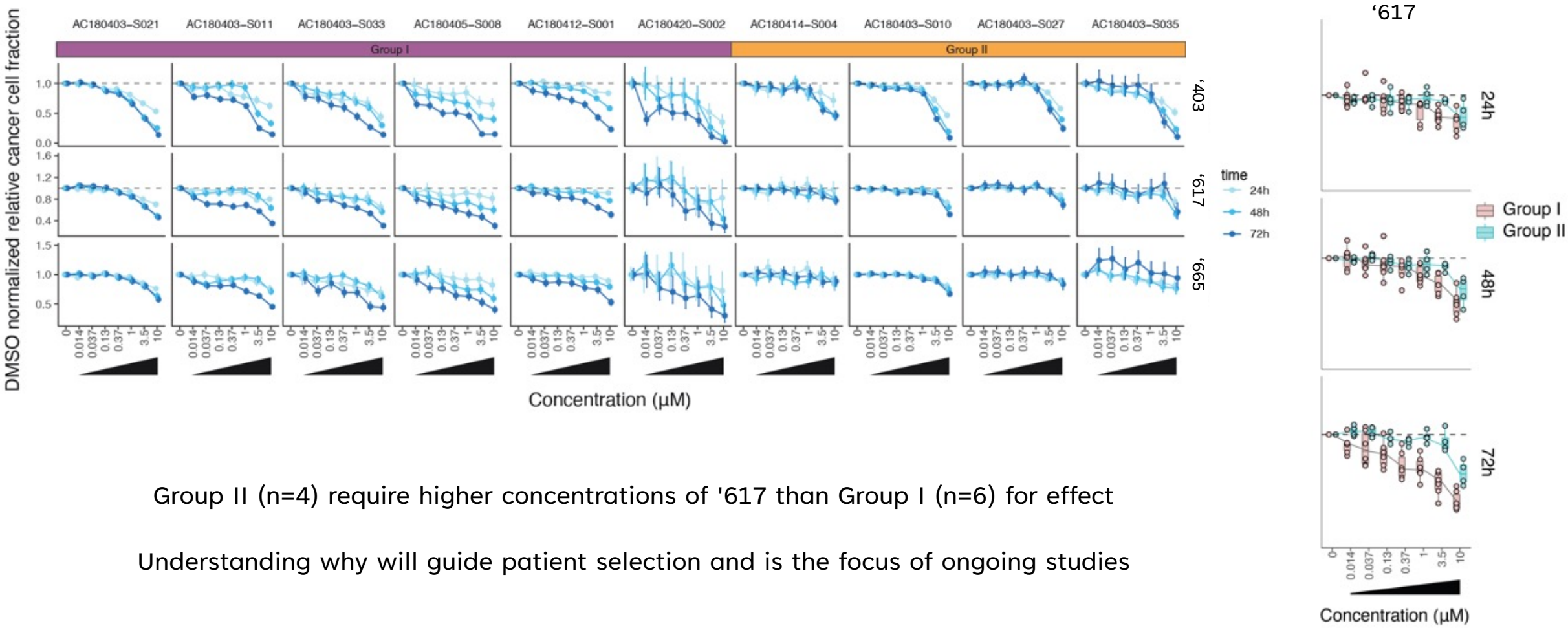
‘617: Differentiated CDK7i

- High on-target potency and selectivity
- Strong *in vivo* anti-tumour profile, as demonstrated in both TNBC and ovarian cancer

Besnard et al, AACR (2022)

Defining patient selection during drug design

'617: Ovarian cancer patient samples stratify into two groups



Group II (n=4) require higher concentrations of '617 than Group I (n=6) for effect

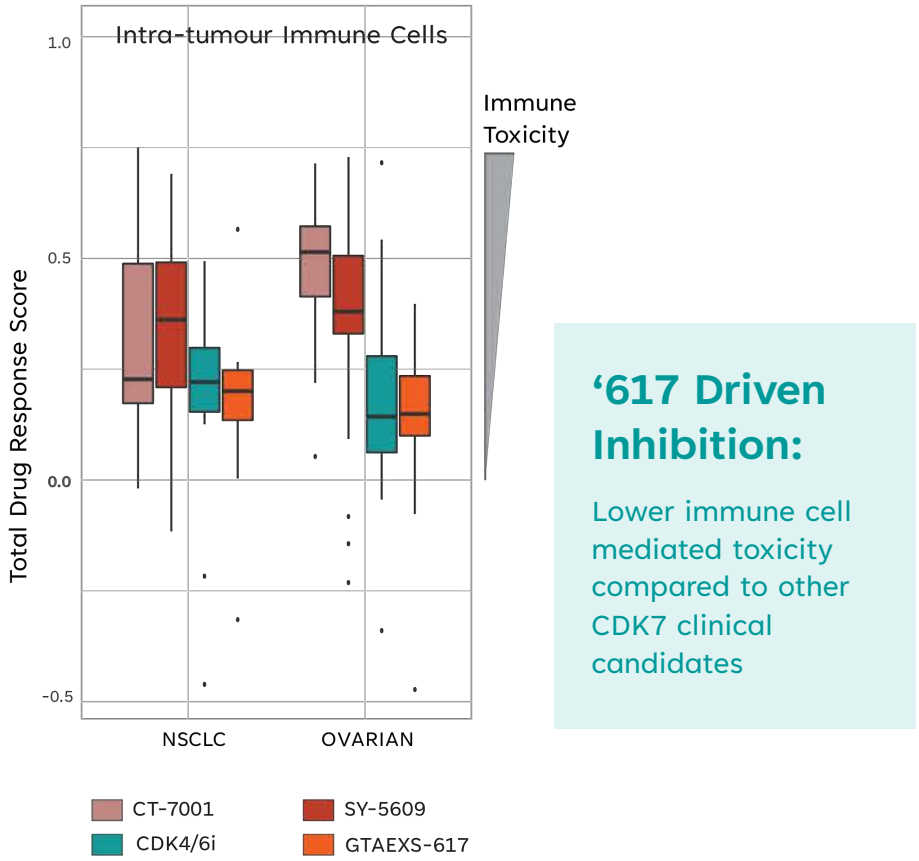
Understanding why will guide patient selection and is the focus of ongoing studies

Besnard et al, AACR (2022)

Identifying MOA-specific patient selection marker for CDK7

Functional drug assessment using a platform with proven translation

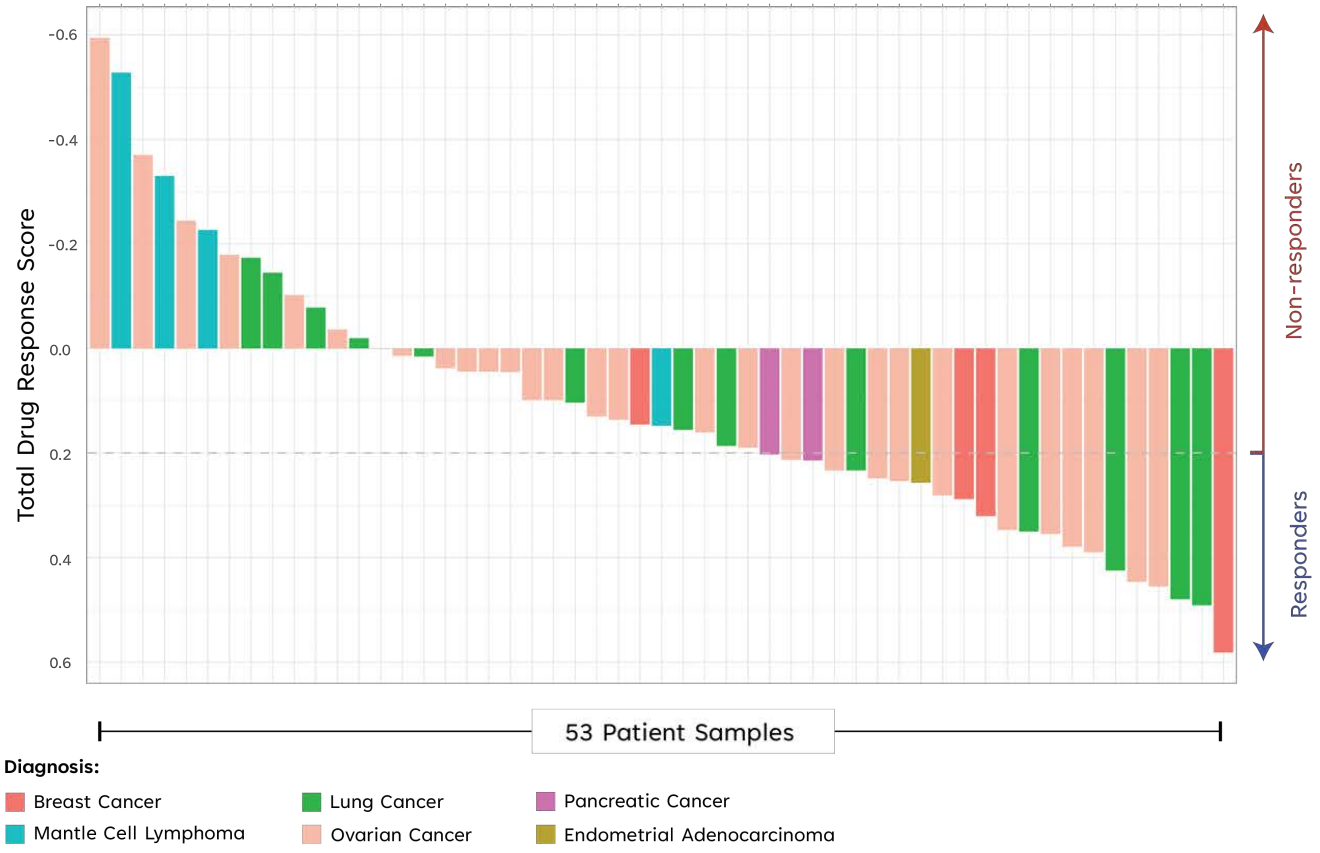
Reduced immune toxicity endpoints



'617 Driven Inhibition:

Lower immune cell mediated toxicity compared to other CDK7 clinical candidates

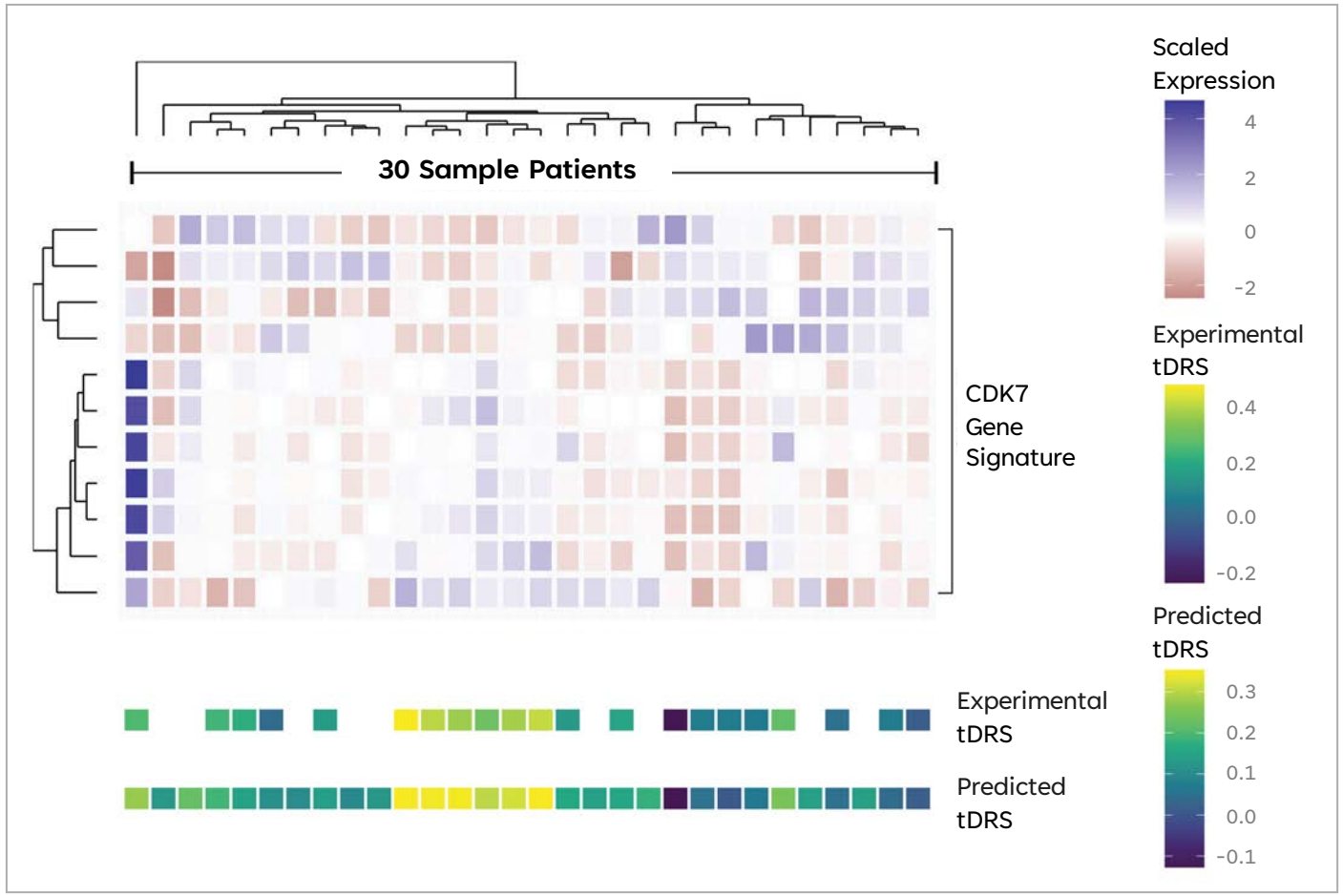
Platform can identify responders and non-responders across tumour types

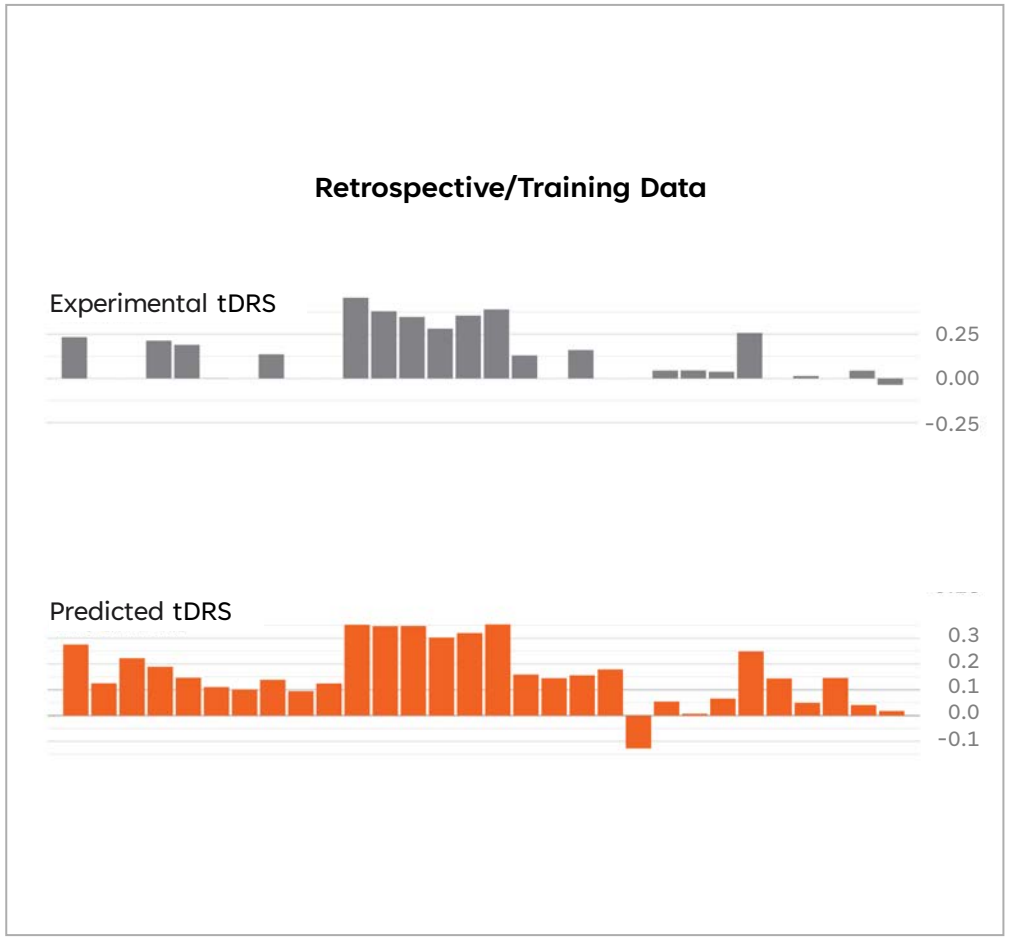


Durinikova et al, ENA (2022)

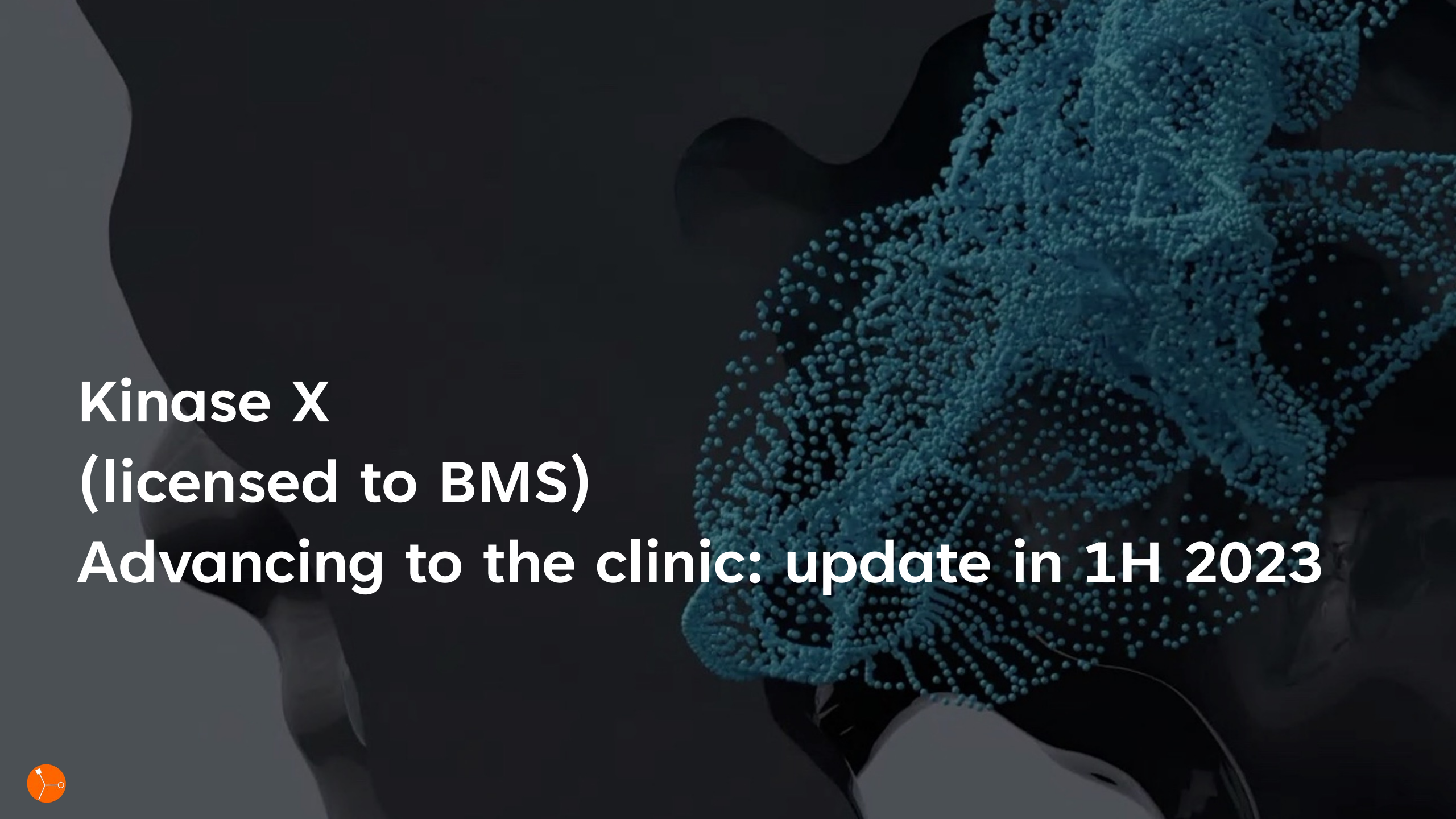
Establishing ‘617 response predictor model

Multimodal analysis of functional and matched transcriptomics data





tDRS = Total Drug Response Score; Durinikova et al, ENA (2022)



Kinase X
(licensed to BMS)
Advancing to the clinic: update in 1H 2023

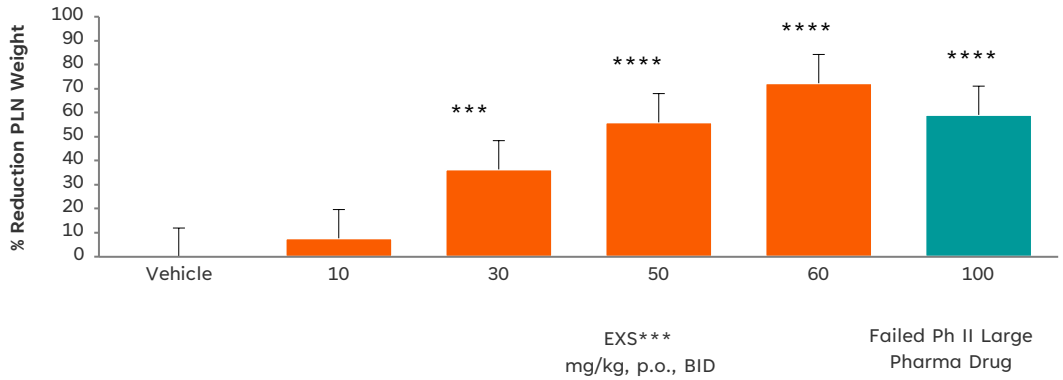
Kinase X: In-licensed to BMS in August 2021

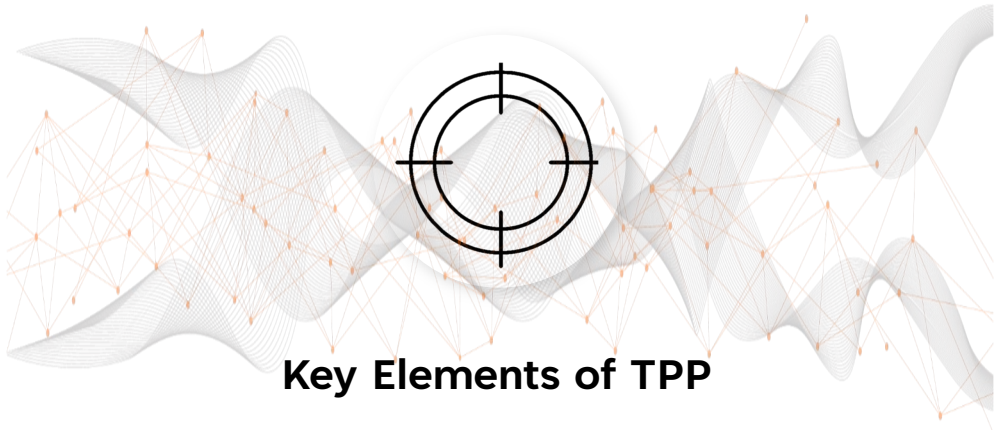
Expert led AI-design to deliver against a high-value target

Potential First-In-Class Immunology Asset

- High-value immunology target that had **eluded many large Biopharmas** due to selectivity challenges
- **Balanced profile** provided improvements in human whole blood potency and predicted human dose <200mg/day
- **Excellent selectivity** versus near neighbours and broad kinome

Better Efficacy at Approximately Half the Dose





Key Elements of TPP

- 24h coverage of IC_{80} required to drive efficacy
- Predicted human dose <200mg/day
- High demands on target potency, selectivity, pharmacokinetics
- Robust translation into cellular and human whole blood assays

Large pharma failures on an attractive target

Potential first-in-class immunology target

Differentiated Kinase X inhibitor Profile

Characteristic	Assay	EXS****	Failed mid-clinical large pharma drug candidate	Failed early clinical large pharma drug candidate
Target potency and selectivity	Biochemical	Meets or exceeds criteria	Meets or exceeds criteria	Major deviation
	Near Neighbours	Meets or exceeds criteria	Major deviation	Meets or exceeds criteria
	Full kinome	Meets or exceeds criteria	Minor deviation	Meets or exceeds criteria
Cellular potency	T-cell	Meets or exceeds criteria	Meets or exceeds criteria	Major deviation
	Human whole blood	Meets or exceeds criteria	Meets or exceeds criteria	Major deviation
Safety and metabolic stability	hERG	Meets or exceeds criteria	Minor deviation	Meets or exceeds criteria
	Microsomal stability	Minor deviation	Major deviation	Meets or exceeds criteria
	Hepatocyte stability	Meets or exceeds criteria	Minor deviation	Minor deviation
General properties	Permeability	Meets or exceeds criteria	Meets or exceeds criteria	Meets or exceeds criteria
	Unbound drug	Meets or exceeds criteria	Minor deviation	Meets or exceeds criteria
	Solubility	Meets or exceeds criteria	Major deviation	Minor deviation

Meets or exceeds criteria Minor deviation Major deviation

Our approach

Fragments. 2D and 3D generative design. Hotspots and multi-task models

Experiment

- Diverse ligand data sources. Proprietary fragment and kinase focussed **SPR screens** provided additional **seed data**
- Established and routinely executed key human whole blood assay

Expert-led AI Solutions

- **Generative design** rapidly explored selectivity-focussed scaffolds. **MERIT** analysis quantified the most promising
- **Hotspot** and **multi-task models** drove local and global kinase selectivity, respectively

Best-in-class Compound

- Nominated candidate designed in **<11 months** and was **150th novel compound** prepared
- Demonstrates close relationship at Exscientia of AI and experiment
- Elegant solution to a challenging problem. Nominated candidate <400 MW

Exscientia

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