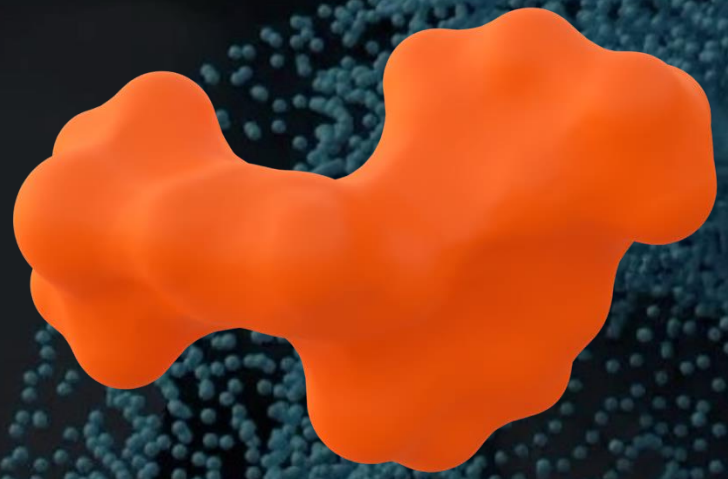
Exscientia



‘617 (CDK7 inhibitor)

ELUCIDATE Phase 1/2 ongoing

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ELUCIDATE: ‘617 Phase 1/2 ongoing in advanced solid tumours

Preclinically identified PD biomarkers to be further assessed in the trial

Two-part trial assessing safety, PK/PD and efficacy of GTAEXS617 in patients with advanced solid tumours*

GTAEXS617 (CDK7 inhibitor)

Phase 1: Dose Escalation

Part A: Monotherapy

Part B: Combination with SoC

- n=up to 30 patients to be enrolled in each part across up to 6 dose schedules to establish the RP2D and PK/PD
- HR+/HER2- CDK4/6i relapsed breast cancer initiating in 2H24/1H25
- Exploring other possible indications

RP2D

Phase 2: Dose Expansion

n=30-60 patients with combination

n will depend on number of disease specific cohorts

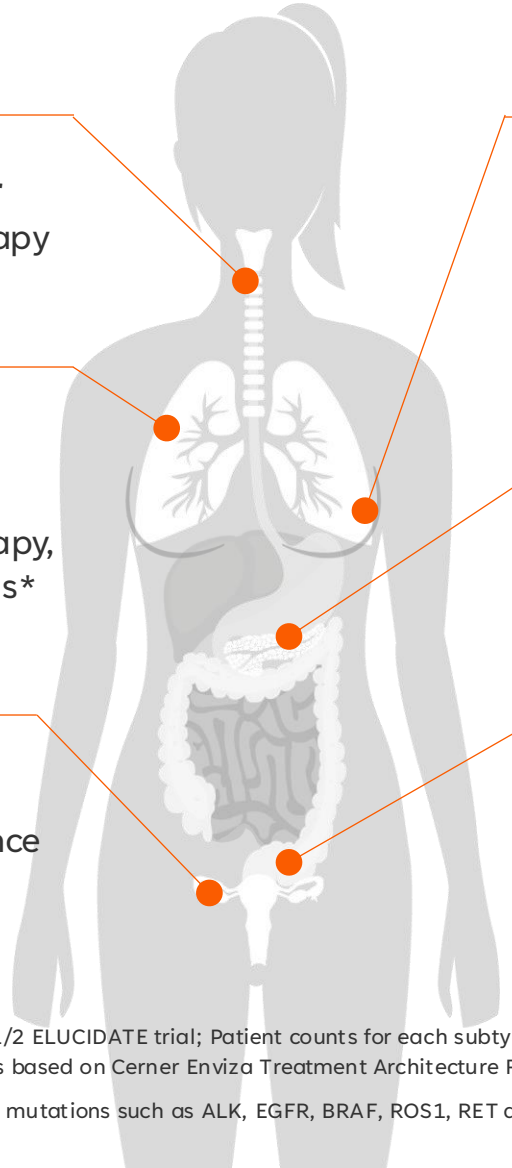
Primary efficacy endpoint: ORR

ELUCIDATE (protocol number GTAEXS617-001) is a Phase 1/2 open-label multi-centre study to assess the safety, pharmacokinetics and anti-tumour activity of GTAEXS617 in patients with advanced solid tumours (who have failed on, refused or are ineligible for the standard of care (SoC))

*Solid tumours under consideration include ovarian cancer, pancreatic cancer, HR+ HER2- breast cancer, head & neck cancer, NSCLC and colorectal cancer – SoC will vary for each disease cohort

ELUCIDATE: ‘617 Phase 1/2 ongoing in advanced solid tumours

2022 United States incidence for patient sub-groups included in ELUCIDATE trial



5K

Head & Neck Cancer
2nd or later line of therapy

29K

Non-small Cell Lung Cancer
2nd or later line of therapy, without driver mutations*

6K

Ovarian Cancer
Post relapse on/resistance to platinum therapy

12K

Breast Cancer
HR+ and HER2- disease, post relapse on CDK4/6i

8K

Pancreatic Cancer
2nd or later line of therapy

15K

Colorectal Cancer
2nd or later line of therapy

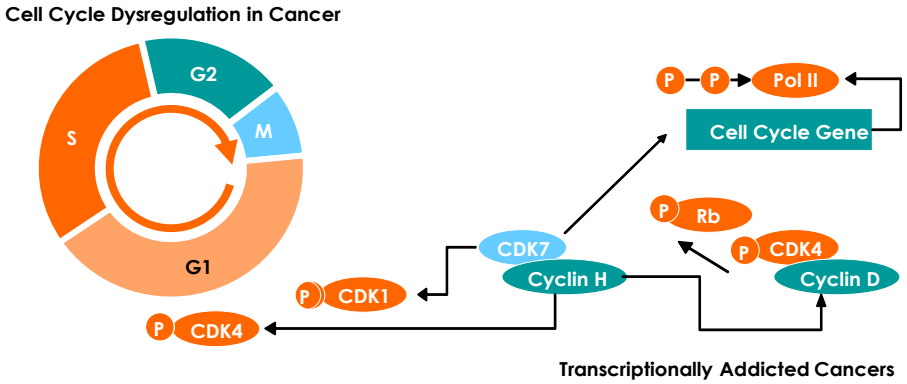
~75K U.S. Patients Lives Annually

Covers all indications included in Ph1/2 ELUCIDATE trial; Patient counts for each subtype based on EvaluatePharma Epidemiology 2022 and primary inclusion criteria for ELUCIDATE Ph 1/2 trial. Treatment rate and progression rates based on Cerner Enviza Treatment Architecture Reports 2022; Numbers have been rounded to the nearest 1000 patients per year;

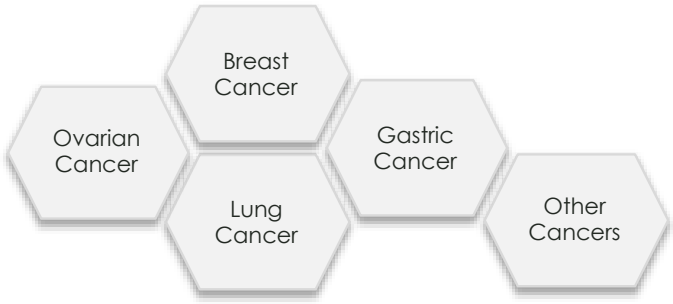
*Excludes currently actionable driver mutations such as ALK, EGFR, BRAF, ROS1, RET and MET

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
  - CDK4/6 inhibitors generated $8.9b in sales in 2022
  - 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 targeted higher 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 8-10 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 novel experimental compounds

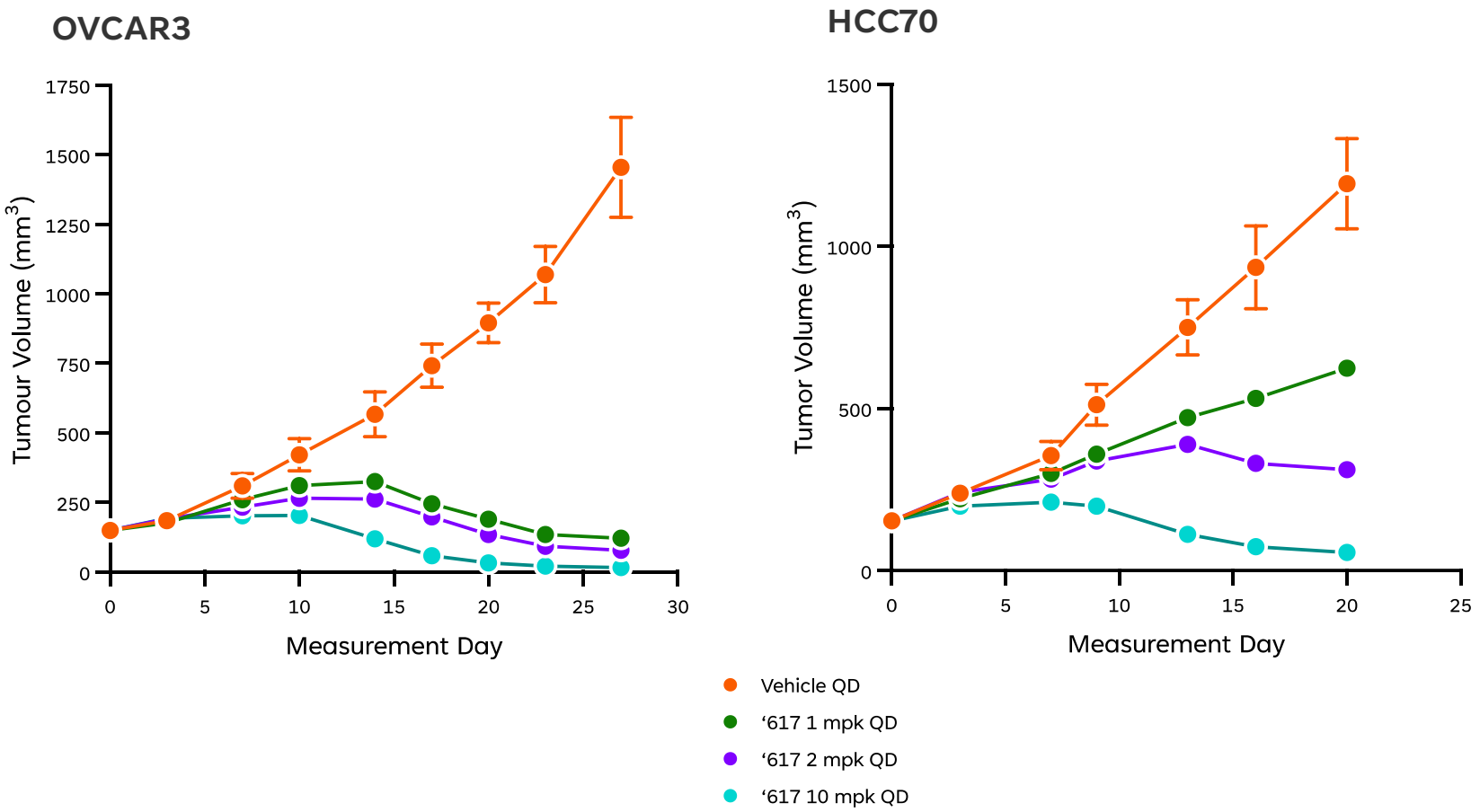
	Assay	Candidate Criteria	Competing Phase 1 Candidate	Competing Phase 1/2 Candidate	Exscientia Candidate ‘617
Target affinity and selectivity	CDK7 IC_{50} (nM)	<10			
	CDK family selectivity	>100 fold			
Cell potency	HCC70 (breast cancer) IC_{50} (nM)	<100			
	OVCAR-3 (ovarian cancer) IC_{50} (nM)	<100			
Safety and metabolism	hERG IC_{50} (µM)	>5			
	Human microsome Clint µL/min/mg	<15			
	Human hep Clint µL/min/10^6 cells	<15			
	Predicted human half-life (hrs)	<15			
Permeability/ transporter liability	Caco-2 A2B (efflux) 10^{-6} cm/s	>3 (<5)			
General properties	Solubility pH 7.4 µg/ml	>50			
	F % (p.o.)	>30%			

- Potent biochemical and cellular activity
- High selectivity
- Optimised half-life
- Excellent bioavailability and efflux

Meets or exceeds criteria | Minor deviation | Major deviation | Not tested

‘617 is highly effective in classical models

Potent anti-tumour activity demonstrated in multiple solid tumour types



Besnard et al, AACR (2022)

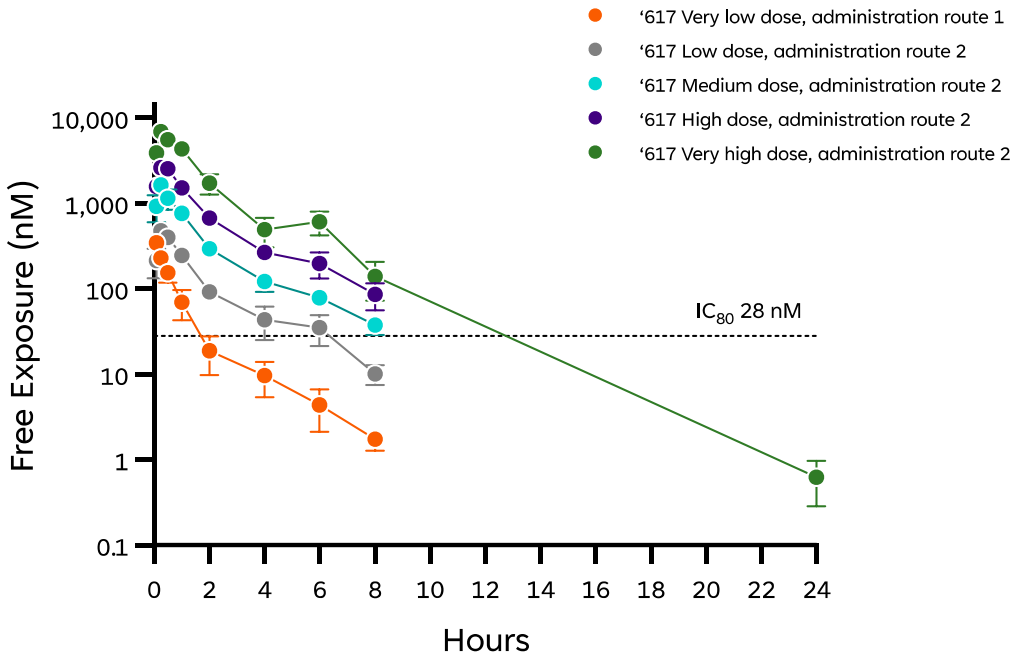
‘617: Differentiated CDK7i

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

‘617 half-life optimises benefit-risk

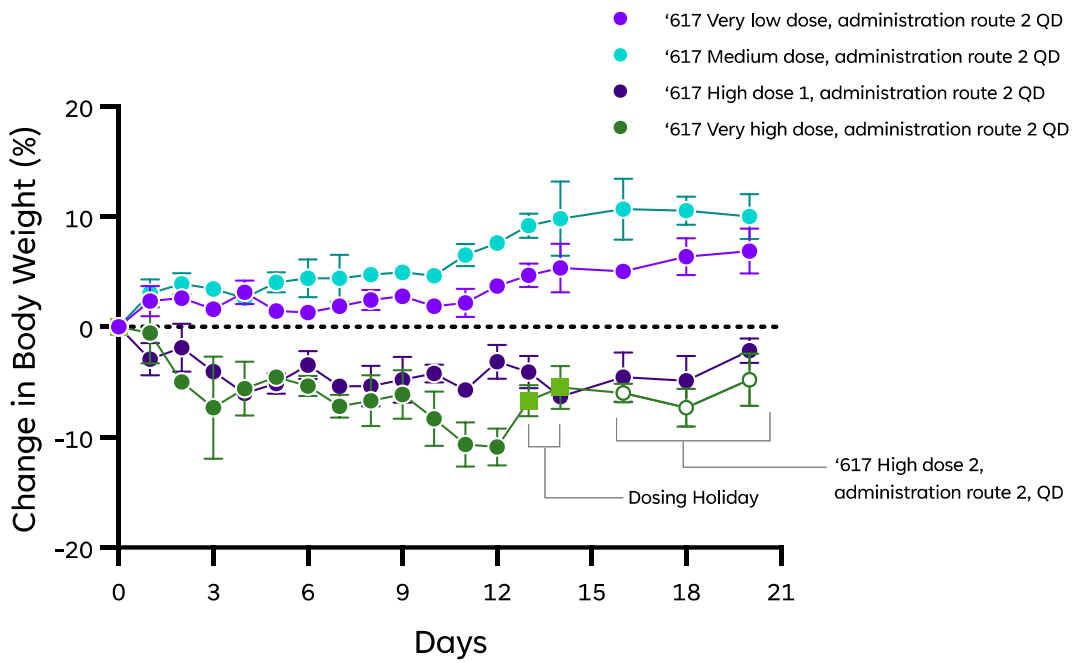
>10 hr of IC_{80} exposure predicted to increase toxicity risk

‘617 Single Dose Pharmacokinetics Data in CD1 Mice



CD1 mouse models predict 8-10 hr of IC_{80} exposure at a medium dose, increasing to >10 hr with a high dose (*Consistent with initial observations in clinic)*

‘617 Preclinical Maximum Tolerated Dose (MTD) Study

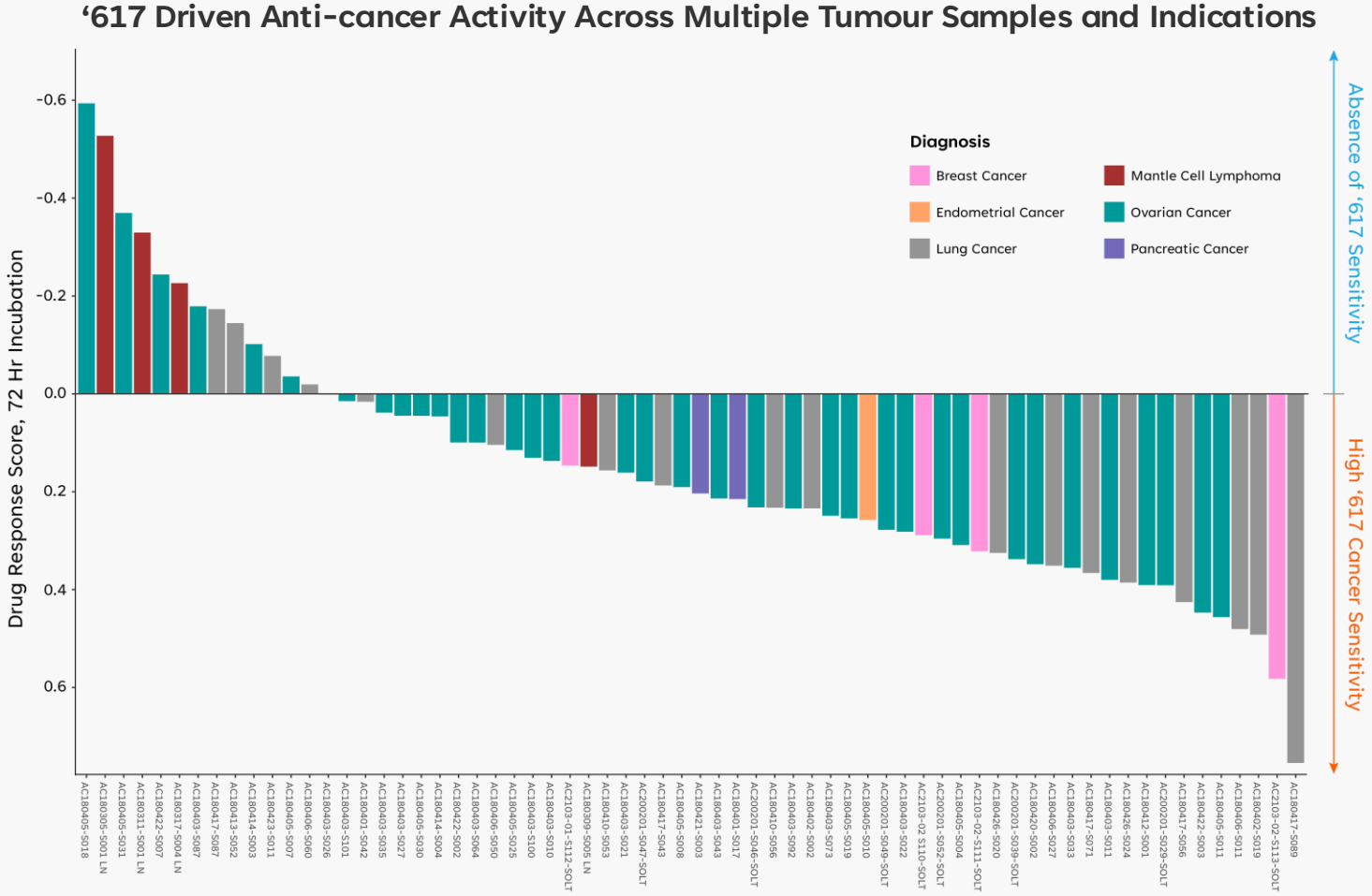


>10 hr IC_{80} exposure with a high dose leads to significant body weight impact, not seen with a medium dose* signalling optimal benefit risk with <10 hr of exposure

*Dose showing maximum tumour growth inhibition in ovarian and TNBC models

Evaluating the potential of ‘617 in primary tissue

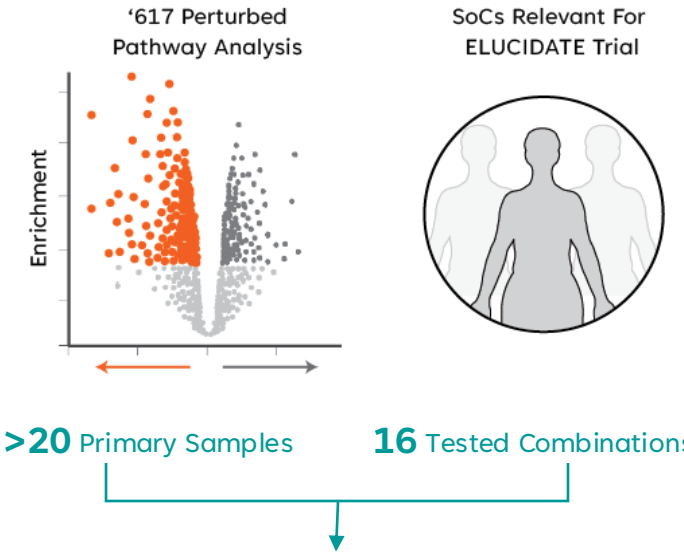
Exploring monotherapy and rational combinations with standard of care



Key Translational Platform Findings

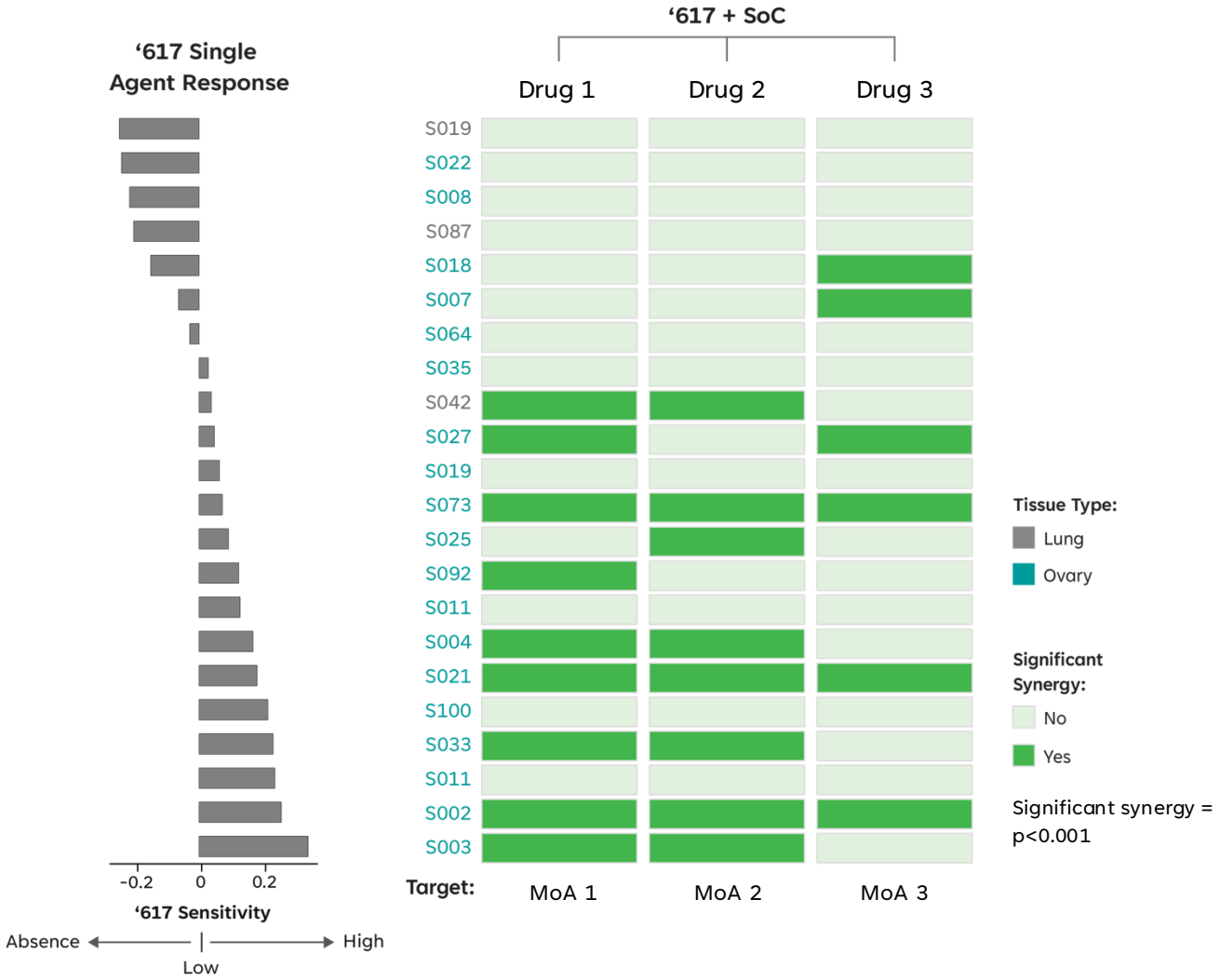
‘617 has demonstrated anti-cancer activity across multiple tumour samples and indications; 4/6 indications are being studied in ELUCIDATE.

Rational Design for Combination Testing



Expanding potential ‘617 efficacy with rational combinations

Synergies for ‘617 in combination preclinically in ovarian and lung cancer models



Drug 1 + ‘617
Related MoA

Synergy in 9/22 patients

Drug 2 + ‘617
Complementary MoA

Synergy in 8/22 patients

Drug 3 + ‘617
Multi-indication SoC

Synergy in 6/22 patients

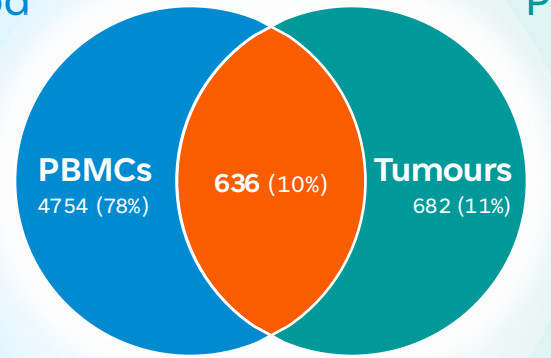
Importantly, some of this synergy is observed for samples with both low ‘617 and Drug 3 sensitivity

We are now integrating functional and omics data to validate the combination biology in sensitive primary patient material.

Proposed PD biomarker of ‘617 exposure

Preclinically identified PD biomarkers to be validated alongside ELUCIDATE

Cancer Patients’ Peripheral Blood

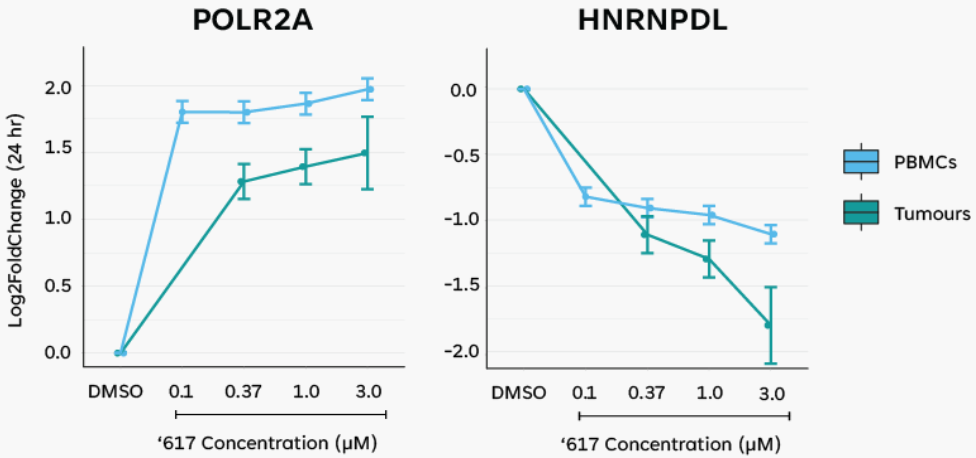


Number of genes modulated upon 24 hr ‘617 exposure

- \+ Provide non-invasive biomarkers
- \+ ‘617 effects on healthy cells
- – Partially informative of tumour effects

Cancer Patients’ Primary Tumours

- \+ ‘617 effect on cancer cells
- \+ Comparison vs healthy cells



Two examples of preclinically identified PD biomarkers to be evaluated via transcriptomic profiling of peripheral blood and FFPE tumour samples from ELUCIDATE

PBMC = peripheral blood mononuclear cells; FFPE = formalin-fixed paraffin-embedded

Exscientia

Exscientia plc

OXFORD HEADQUARTERS
The Schrödinger Building
Oxford Science Park
Oxford OX4 4GE

investors@exscientia.ai

Registered address: The Schrödinger Building,
Oxford Science Park, Oxford, OX4 4GE, United Kingdom

Registered number: 13483814