Acrivon Therapeutics

ACRIVON PREDICTIVE PRECISION PROTEOMICS (AP3)

OVERCOMING LIMITATIONS OF GENETICS-BASED PRECISION MEDICINE

CORPORATE R&D EVENT

APRIL 24, 2024

FORWARD-LOOKING STATEMENTS

Certain information contained in this presentation includes forward-looking statements within the meaning of the Private Securities Litigation Reform Act of 1995 regarding our future results of operations or financial condition, business strategy and plans and objectives of management for future operations. In some cases, you can identify forward-looking statements because they contain words such as "anticipate," "believe," "contemplate," "continue," "could," "estimate," "expect," "intend," "may," "plan," "potential," "predict," "project," "should," "target," "will," or "would" or the negative of these words or other similar terms or expressions. Our forward-looking statements are based primarily on our current expectations and projections about future events and trends that we believe may affect our business, financial condition and results of operations. The outcome of the events described in the forward-looking statements is subject to risks and uncertainties, including the factors described in our filings with the U.S. Securities and Exchange Commission. New risks and uncertainties emerge from time to time, and it is not possible for us to predict all risks and uncertainties that could have an impact on the forward-looking statements contained in this presentation. The results, events, and circumstances reflected in the forward-looking statements may not be achieved or occur, and actual results, events, or circumstances could differ materially from those described in the forward-looking statements.

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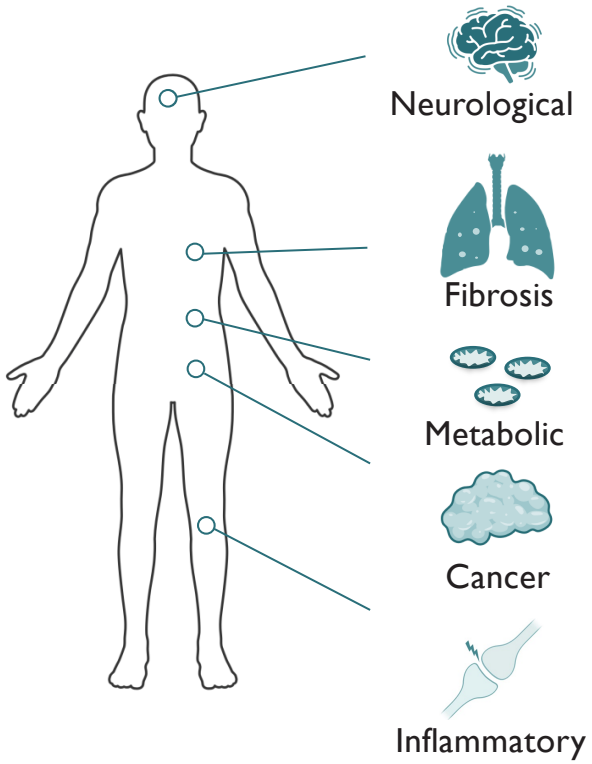
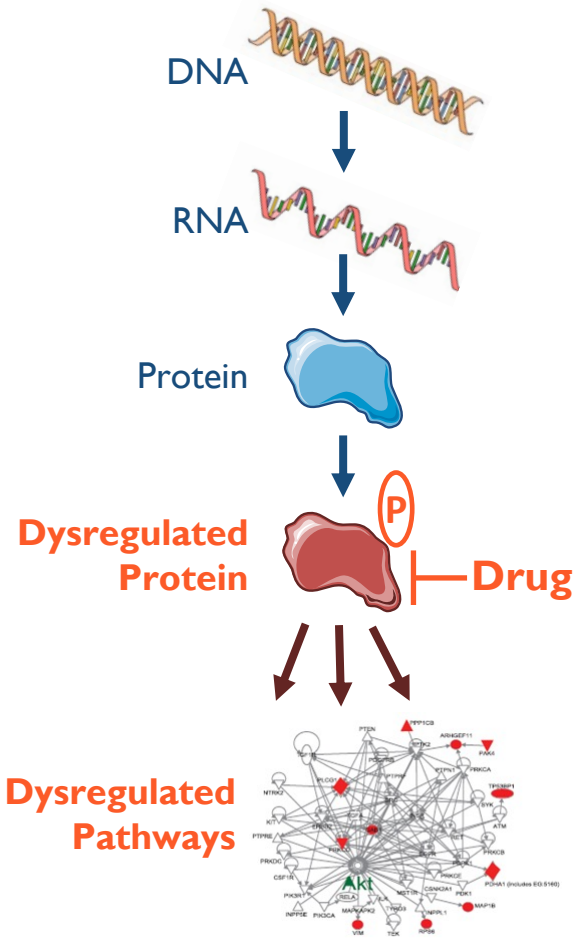
OUTLINE

• Acrivon Therapeutics and AP3 overview	5 minutes
• Initial ACR-368 Phase 2 trial data	25 minutes
• AP3-based drug design: Dual WEE1/PKMYT1 inhibitor ACR-2316 and pipeline	20 minutes
• AP3 Interactome	5 minutes
• Live Q&A	20 minutes

For a comprehensive corporate deck, please visit: https://Acrivon.com

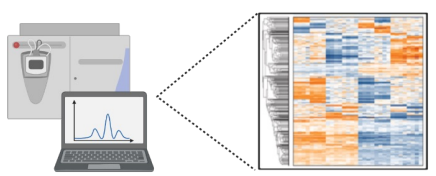
ACRIVON THERAPEUTICS – A NEXT-GENERATION PRECISION MEDICINE COMPANY

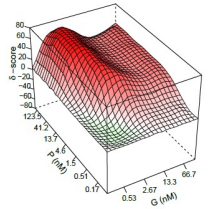


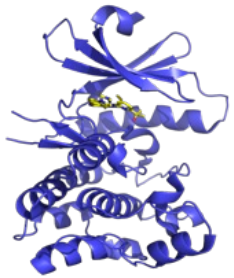


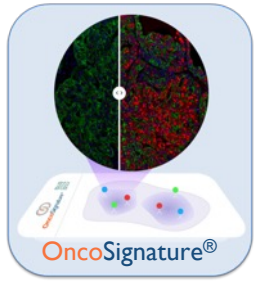
Acrivon Predictive Precision Proteomics (AP3)

- Enables an exact match between the disease-driving, dysregulated pathways with a drug's mechanism of action (Acrivon meaning ≈ exact, accurate)
- Broadly applicable in R&D (biological SAR, resistance, patient responders); leveraged for internal pipeline

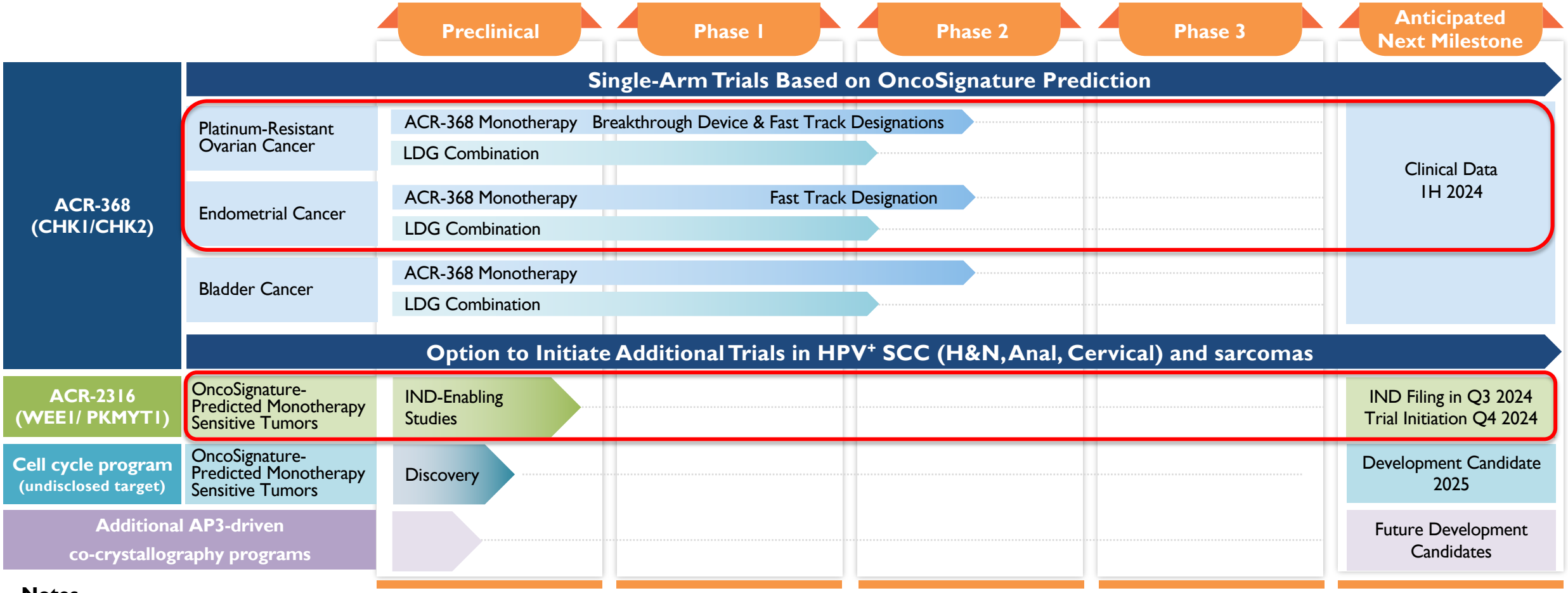








ACRIVON PIPELINE



Notes

ACR-368 Monotherapy	Registrational intent Phase 2 single arm trials based on predicted sensitivity to ACR-368 monotherapy in OncoSignature-positive patients
LDG Combination	Exploratory Phase 1b/2 single arm trials of ACR-368 in combination with low dose gemcitabine, or LDG, in OncoSignature-negative patients

ACRIVON THERAPEUTICS – OVERVIEW

- Next-generation precision medicine; Foundational team pioneers in phosphoproteomics-based R&D

- AP3-based prospective patient responder identification
  - Acrivon in-licensed ACR-368 (Prexasertib) a Lilly flagship program (durable single agent activity across solid cancers, genomics insufficient for patient selection) with 3 key objectives
    1. Increase ORR in ovarian cancer using ACR-368 OncoSignature
    2. Identify and verify robust clinical activity in new indications (AP3 profiling)
    3. Validate our AP3 approach on a challenging drug to provide read through to other drugs (internal pipeline or external)
  - Initial data demonstrate POC for these objectives

- AP3 applied for drug discovery - ACR-2316 and early pipeline
  - AP3 has generated a potent, single agent active WEE1/PKMYT1 development candidate (Biological SAR)
  - Specifically designed for clinical monotherapy development aiming for accelerated pathway
  - ACR-2316 OncoSignature being generated for indication finding prior to clinical development
  - AP3 has enabled streamlined preclinical development and accelerated timelines (IND filing now expected in Q3, 2024)
  - New potential first-in-class cell cycle program for an undisclosed target initiated following the same AP3 approach

- AP3 interactome
  - Proprietary, integrated, ML-enabled compound profiling data (~50 million datapoints/2 wk)
  - Version 1.1.1 of AP3 interactome completed to be applied for AI-based drug discovery and development

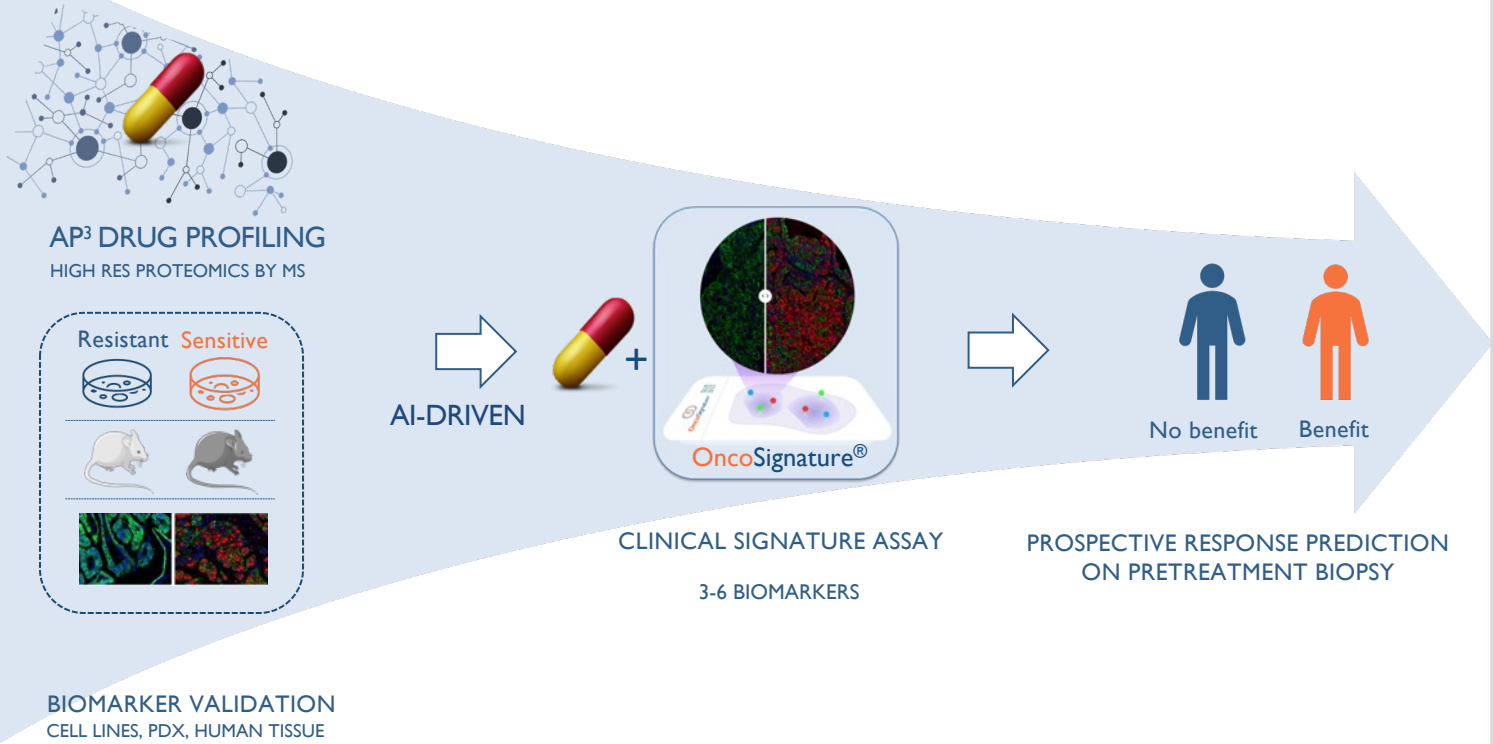
Initial data from the ACR-368 prospective patient selection Phase 2 clinical trial in gynecological cancers

Data cut as of April 1, 2024

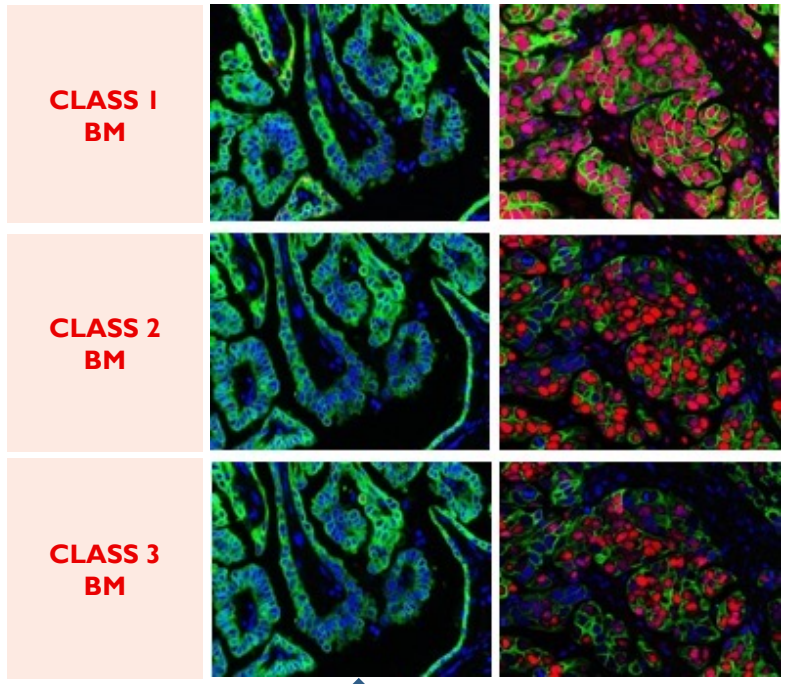
AP3 PLATFORM: DRUG RESPONSE PREDICTION IN INDIVIDUAL PATIENTS

PROPRIETARY WORKFLOW INTEGRATING PROVEN TECHNOLOGIES

STEP 1	STEP 2	STEP 3
BIOMARKER IDENTIFICATION	THERAPEUTIC PACKAGE Acrivon drug with predictive BM assay	PATIENT RESPONDERS Optimal indications and combinations



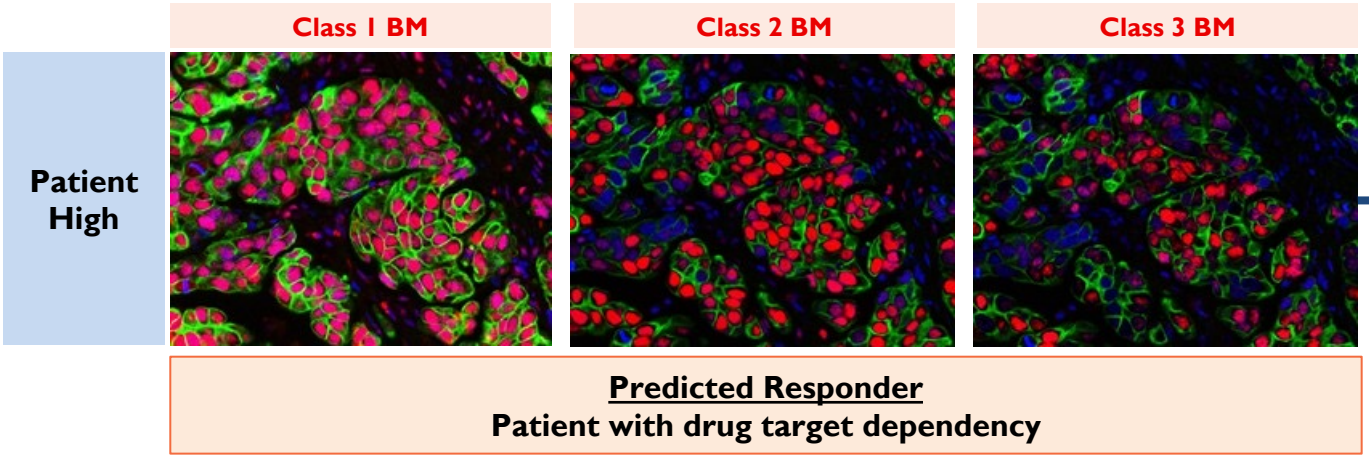
OncoSignature®



Patients without biomarkers critical for drug sensitivity efficiently excluded

"Disease Pathway-Based Method to Generate Biomarker Panels Tailored to Specific Therapeutics for Individualized Treatments": EP 2 229 589, issued June 10, 2015; US2017/0067877A9, pending. OncoSignature® *is a Registered Trademark: US Reg. No. 5,718,472; Int. Cl. 5, 42. Intl. Reg. 1382289*

ACR-368 ONCOSIGNATURE PREDICTION OF DRUG SENSITIVITY: BIOMARKER QUANTITATION ACROSS HUMAN CANCER PATIENT SAMPLES



Tissue ID.	BM1	BM2	BM3
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Fov050855	0.94	0.71	0.67
Fov041300	0.89	0.23	0.55
Fov060380	0.95	0.62	0.51
Fov060050	0.85	0.23	0.51
Fov041285	0.69	0.19	0.5
Fov100142	0.47	0.76	0.47
Fov050764	0.82	0.22	0.47
Fov041267	0.4	0.36	0.36
Fov100146	0.84	0.57	0.26
Fov060382	0.44	0.45	0.22
Fov041269	0.37	0.21	0.2
Fov060302	0.29	0.22	0.15
Fov020067	0.89	0.38	0.09
Fov100003	0.9	0.89	0.08
Fov050700	0.17	0.17	0.07
Fov050139	0.63	0.66	0.05
Fov041138	0.36	0.11	0.05
Fov060152	0.24	0.3	0.03
Fov030062	0.57	0.14	0.02
Fov060133	0.94	0.43	0.01
Fov050666	0.93	0.38	0.01
Fov060371	0.88	0.93	0
Fov020497	0.61	0.43	0
Fov010706	0.54	0.14	0
Fov020386	0.11	0.09	0.12
Fov050734	0.11	0.06	0.08
Fov040872	0.23	0.04	0.36
Fov050659	0.29	0.01	0.02
Fov060293	0.42	0	0
Fov060210	0.1	0.1	0
Fov050840	0.07	0.29	0.01
Fov040855	0.05	0	0.22
Fov050816	0.04	0.33	0.12
Fov060355	0.04	0.27	0
Fov010252	0.03	0.17	0.02
Fov010682	0.03	0.03	0
Fov060553	0.03	0.02	0.03
71768C3	0.02	0	0
Fov150230	0	0.36	0.12
Fov060308	0	0	0

THERAPEUTIC BAR FOR HIGH GRADE PLATINUM-RESISTANT OVARIAN AND ENDOMETRIAL CANCER NEW APPROVALS

- Platinum-resistant ovarian cancer: ≥2nd line SOC* ~12% ORR, mDoR 3.7 – 5.7 months
  - Mirvetuximab: Post 1-3 prior lines, FRa-high PROC (~35% of patients; ORR ~35%, PFS = 5.6 months)
  - ~85% of patients with PROC do not benefit from mirvetuximab
- High grade endometrial cancer: ≥3rd line SOC** ~9% ORR, mDoR 3.1 months
- ACR-368 clinical activity (without patient selection) in past platinum-resistant ovarian trials: ~12% ORR, mDoR >5.6 months
  - (BRCA-mutant and BRCA wild type patients regardless # lines of prior therapy; Lilly-sponsored 46-center, 8-country, N=169 patient study)^

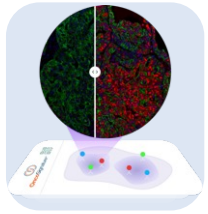
- TPP - high grade PROC: ≥25% ORR with CI lower bound >15%
- TPP - high grade endometrial cancer: ≥20-25% ORR with CI lower bound >15%

* Aurelia trial: Pujade-Lauraine E et al, JCO (2014); Corail trial: Gaillard S et al, JCO (2016)
** Ray-Coquard I et al, BJC (2013)
^ Konstantinopoulos P et al, Gyn Oncol. (2022)

ACR-368-201 STATUS – OVARIAN AND ENDOMETRIAL (LOCKED ONCOSIGNATURE THRESHOLDS, PROSPECTIVE TRIAL)

OncoSignature-positive
ACR-368 RP2D (105 mg/m^2)

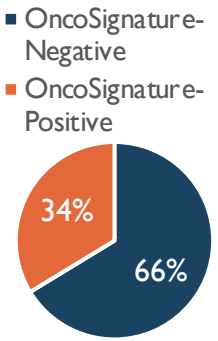
Registrational intent Phase 2



OncoSignature-negative
ACR-368 RP2D (105 mg/m^2) + ULDG RP2D (10 mg/m^2)

Exploratory Phase 1B/2

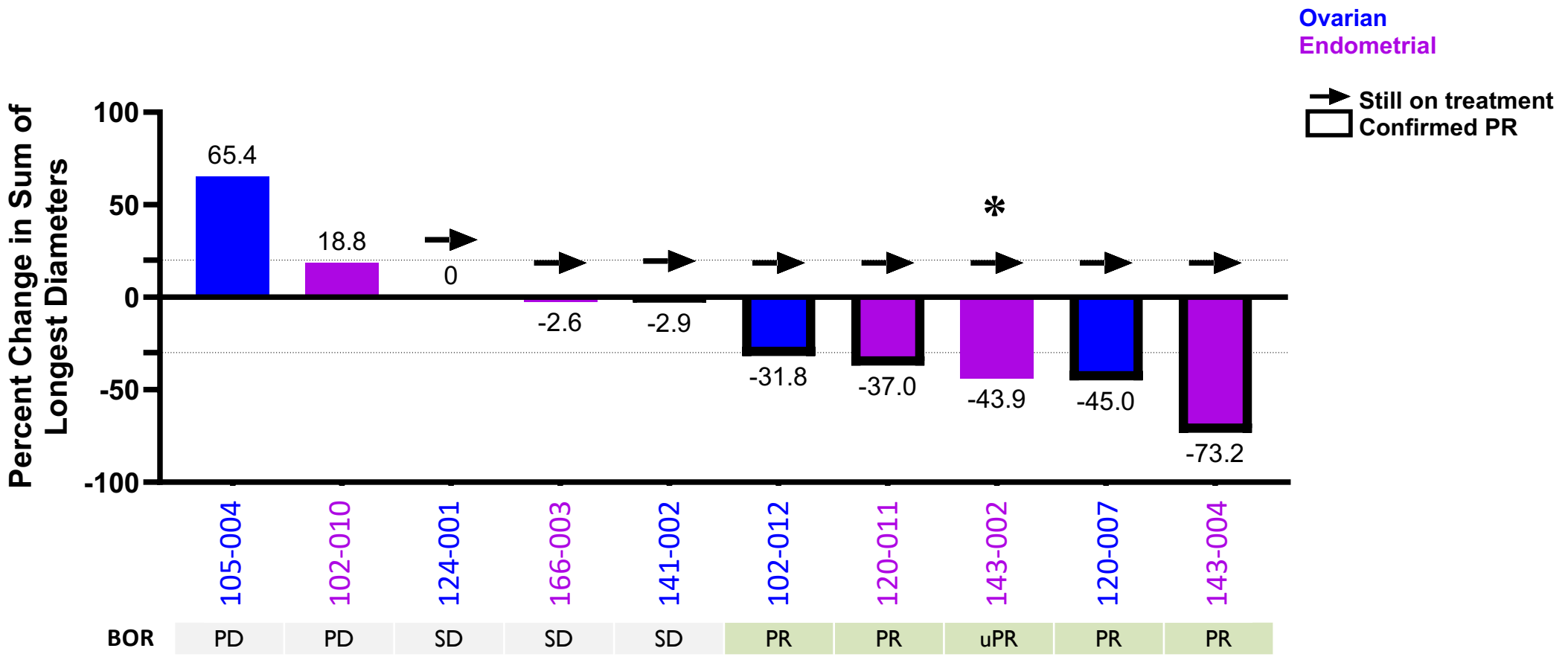
OncoSignature-positive	OncoSignature-negative
Patients enrolled (N = 31)	Patients enrolled (N = 61; incl. 7 to be dosed)
Patients who received at least one dose (N = 26)	Patients who received at least one dose (N = 41)
13 patients awaiting 8-week scan	21 patients awaiting 8-week scan
Patients with at least the 8-wk scan (N = 10)	Patients with at least the 8-wk scan (N = 16)
Patients currently on study (N = 21)	Patients currently on study (N = 28)



OncoSignature-positive rate =34%, consistent with predicted ~35% based on patient tumor sample OncoSignature screening prior to clinical trial

Data cut as of April 1, 2024

ONCOSIGNATURE+ GYN PATIENTS – TUMOR SHRINKAGE (LOCKED THRESHOLDS, PROSPECTIVE EVALUATION PER PROTOCOL)



BOR = Best overall response

*Since data cut off, the one unconfirmed PR has been confirmed bringing the total confirmed PRs to 5

Data cut as of April 1, 2024

ONCOSIGNATURE-POSITIVE PHASE 2 MONOTHERAPY GYN SUMMARY– PROSPECTIVE DATA WITH LOCKED THRESHOLDS

		Ovarian	Endometrial	Total
OncoSignature Positive (Arm 1)	PR (confirmed)	2	3	5
	SD	2	1	3
	PD	1	1	2
	Total	5	5	10
	ORR	40%	60%	50%

-Ovarian: The 95% CI^ for ORR = (12%, 77%). For reference, ovarian SOC ~12%.

-Endometrial: The 95% CI^ for ORR = (23%, 88%). For reference, endometrial SOC ~ 9%

All 5 confirmed responders on treatment; median DoR not reached

^ Agresti-Coull

Data cut as of April 1, 2024

ACR-368 ONCOSIGNATURE PROSPECTIVELY PREDICTS SENSITIVITY TO MONOTHERAPY IN ONGOING PHASE 2 TRIAL

ORR	BM+ (ARM 1) ACR-368 monotherapy	BM- (ARM 2) ACR-368 + ULDG	Patients (N)
Ovarian	40% (2/5)	0% (0/11)	16
Endometrial	60% (3/5)	0% (0/5)	10
Combined	50% (5/10)	0% (0/16)	26

P value (confirmed PRs) = 0.0038

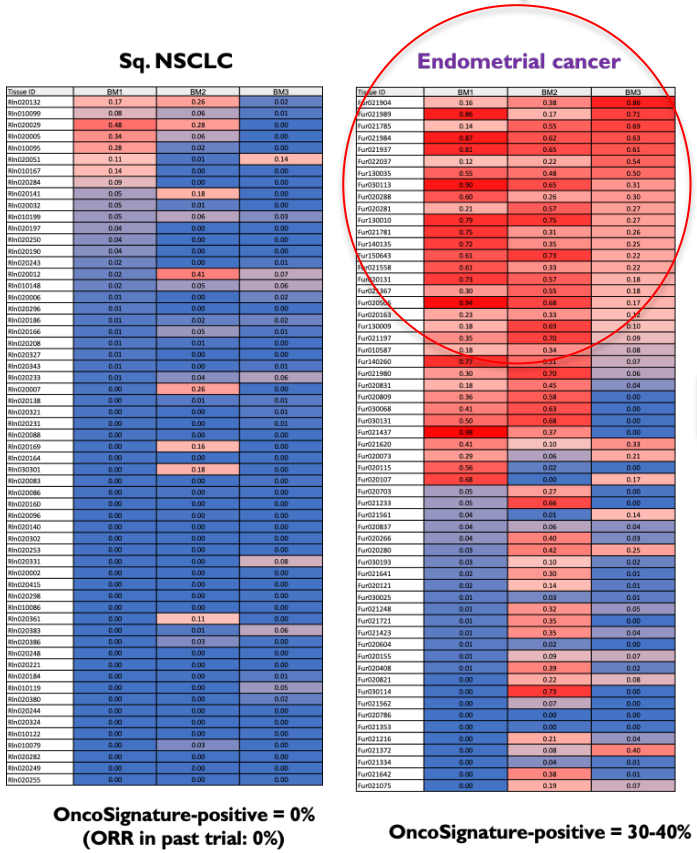
Notes:

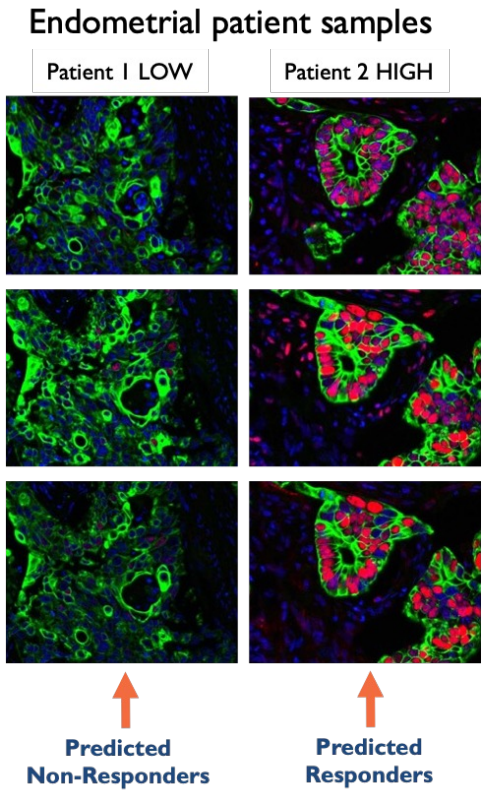
- (non-parametric bootstrap simulation and Fisher test BM+ vs BM-)
- ORR in ovarian all-comer = 12.5%, which is consistent with JTJN study (12%)

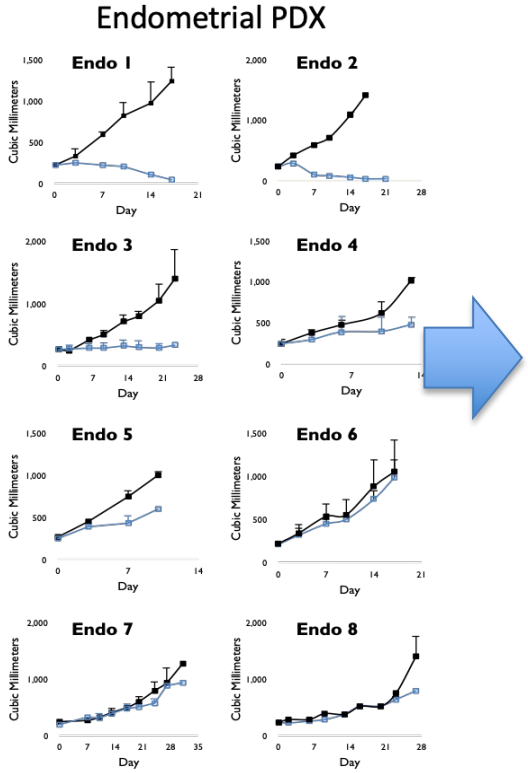
Data cut as of April 1, 2024

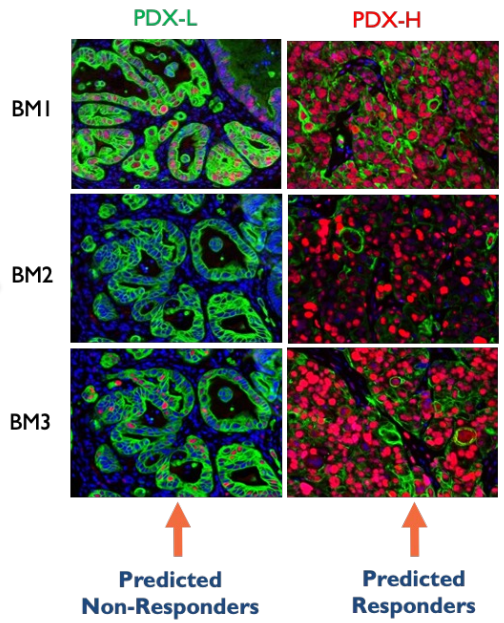
ENDOMETRIAL CANCER IS AN AP3-PREDICTED TUMOR TYPE

ACR-368 OncoSignature-based indication finding prior to trial entry → ACR-368 OncoSignature imaging demonstrates addiction to CHK1/2 DDR axis → Confirmation of predicted sensitivity in genetically non-modified PDX models → Confirmation of ACR-368 OncoSignature prediction in PDX tumor tissues pretreatment





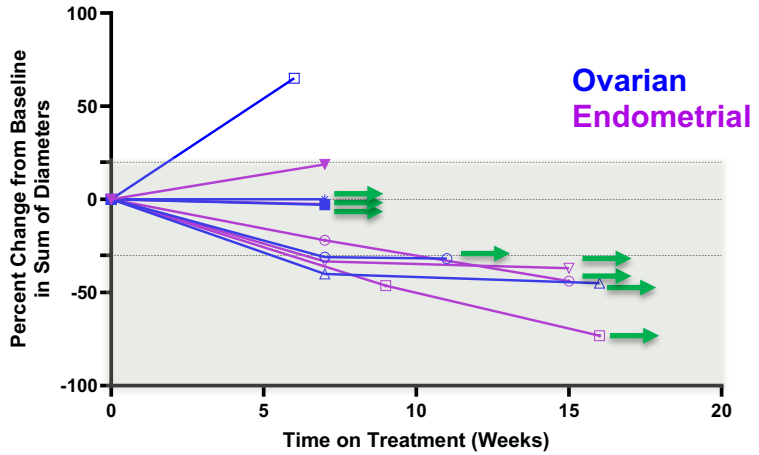




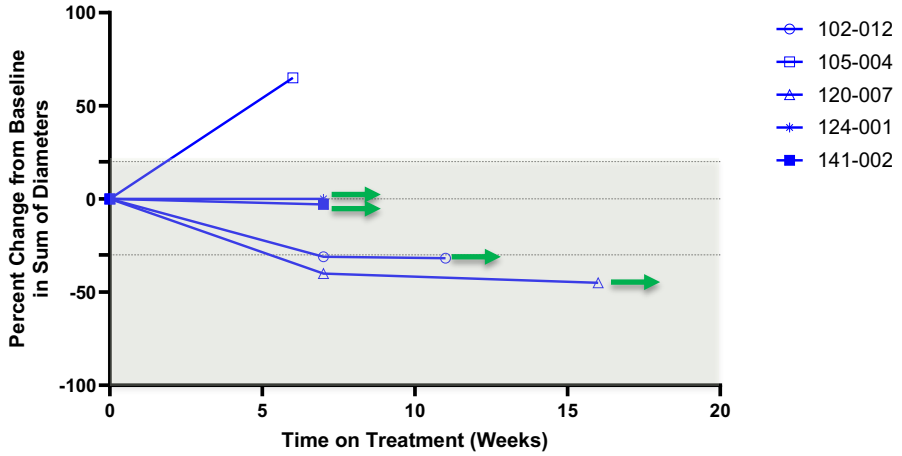
in >1,000 cancer patients treated with ACR-368 in Lilly-sponsored trials, endometrial cancer was not tested

ONCOSIGNATURE+ (ARM 1) SPIDER PLOTS – LOCKED THRESHOLDS

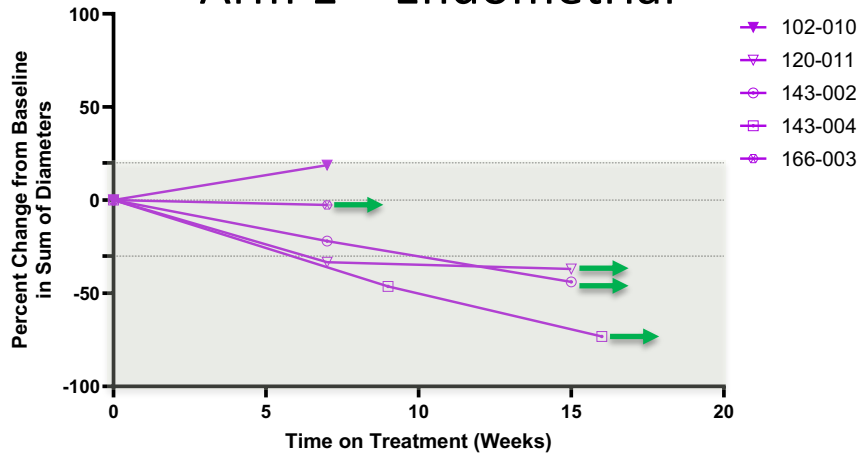
Arm 1 – All GYN Subjects



Arm 1 – Ovarian



Arm 1 – Endometrial



→ Still on treatment

Note: mDoR established in past Phase 2 ACR-368 monotherapy RP2D trials (>200 patients) is between 5.6 months to >10 months (Konstantinopoulos et al, Gyn Oncol (2022); Lee et al, Lancet Oncology (2018))

Data cut as of April 1, 2024

ONCOSIGNATURE-NEGATIVE SUMMARY (GYN INDICATIONS)

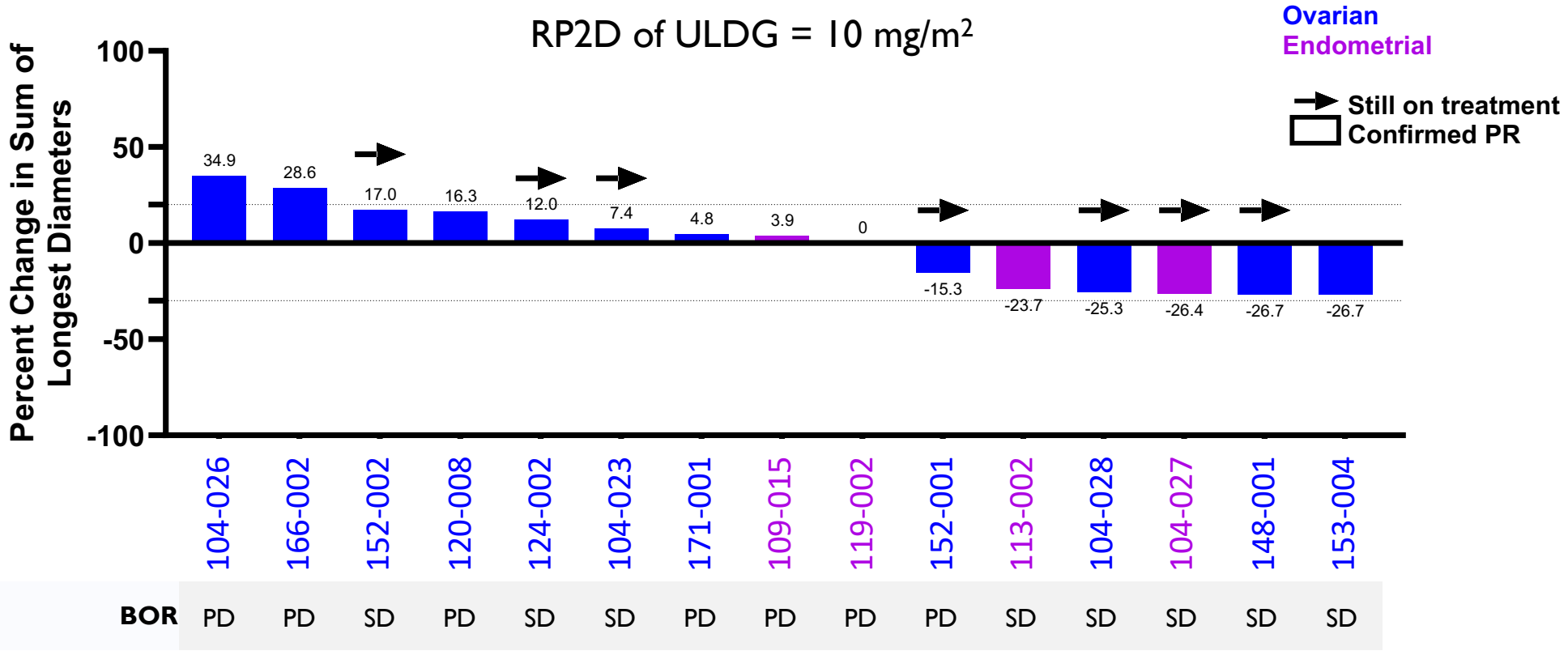
– *PROSPECTIVE DATA WITH LOCKED THRESHOLDS*

Clear ULDG sensitization and activity (see waterfall plot), but no PRs to date with locked thresholds

		Ovarian	Endometrial	Total
OncoSignature Negative (Arm 2)	**PR**			
	SD	6	2	8
	PD	5	3	8
	Total	11	5	**16**
	ORR	0%	0%	0%

Data cut as of April 1, 2024

ONCOSIGNATURE-NEGATIVE (ARM 2)- BEST OVERALL RESPONSE (LOCKED THRESHOLDS, PROSPECTIVE EVALUATION)



BOR = Best overall response; One PD subject censored for lack of target lesion data points available in EDC

Data cut as of April 1, 2024

TREATMENT-RELATED ADVERSE EVENTS BY INDICATION – ONCOSIGNATURE-POSITIVE (ARM 1)

Treatment-related AEs in >15% of subjects, locked thresholds; RP2D (ACR-368 105 mg/m²)

ARM 1	Endometrial		Ovarian		Urothelial		ALL (Arm 1)	
	N = 7		N = 14		N = 2		N = 23	
	All	Gr 3/4	All	Gr 3/4	All	Gr 3/4	All	Gr 3/4
Anemia	2 (29)	2 (29)	8 (57)	2 (14)	1 (50)	0 (0)	11 (48)	4 (17)
Fatigue	2 (29)	1 (14)	3 (21)	0 (0)	1 (50)	0 (0)	6 (26)	1 (4)
Nausea	3 (43)	0 (0)	2 (14)	0 (0)	2 (100)	0 (0)	7 (30)	0 (0)
Thrombocytopenia	0 (0)	0 (0)	2 (14)	2 (14)	0 (0)	0 (0)	2 (9)	2 (9)
Neutropenia	0 (0)	0 (0)	1 (7)	1 (7)	0 (0)	0 (0)	1 (4)	1 (4)
Febrile Neutropenia	0 (0)	0 (0)	2 (14)	2 (14)	0 (0)	0 (0)	2 (9)	2 (9)

Data represented as number of subjects (% of subjects); Non-QC'd data, as of 8 March 2024

AE profile predominantly heme, consistent with previous monotherapy trials at RP2D

TREATMENT-RELATED ADVERSE EVENTS BY INDICATION – ONCOSIGNATURE-NEGATIVE (ARM 2)

Treatment-related AEs in >15% of subjects, locked thresholds, RP2D (ACR-368 105 mg/m²; ULDG = 10 mg/m²)

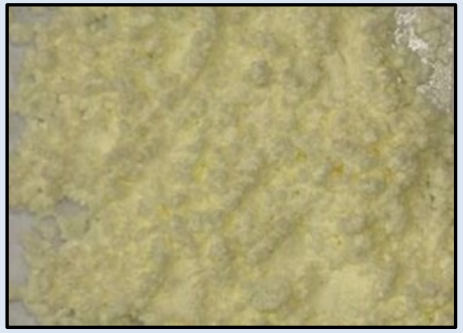
ARM 2	Endometrial		Ovarian		Urothelial		ALL (Arm 2, 10 mg/m² Gem)	
	N = 11		N = 22		N = 5		N = 38	
	All	Gr 3/4	All	Gr 3/4	All	Gr 3/4	All	Gr 3/4
Anemia	2 (18)	2 (18)	10 (45)	3 (14)	3 (60)	1 (20)	15 (39)	6 (16)
Fatigue	1 (9)	0 (0)	9 (41)	2 (9)	1 (20)	0 (0)	11 (29)	2 (5)
Nausea	1 (9)	0 (0)	6 (27)	0 (0)	1 (20)	0 (0)	8 (21)	0 (0)
Thrombocytopenia	2 (18)	1 (9)	3 (14)	2 (9)	4 (60)	2 (40)	9 (24)	5 (13)
Neutropenia	0 (0)	0 (0)	5 (23)	5 (23)	3 (60)	3 (60)	8 (21)	8 (21)
Febrile Neutropenia	1 (9)	1 (9)	4 (18)	4 (18)	1 (20)	1 (20)	6 (16)	6 (16)

Data represented as number of subjects (% of subjects); Non-QC'd data, as of 8 March 2024

AE profile predominantly heme, consistent with previous monotherapy trials at RP2D

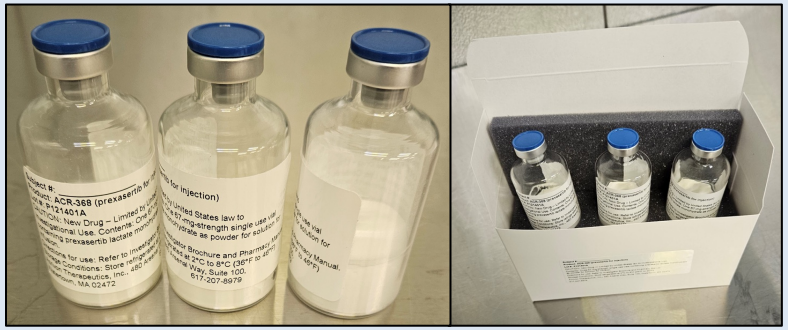
ACR-368 MANUFACTURING STATUS

Active Pharmaceutical Ingredient



- Registration and validation campaigns complete with release of all batches
- 21.9 kg of GMP API in stock (enough for >100k doses)

Drug Product

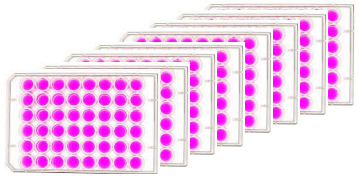


- Registration campaign complete with all three batches released and yield ~95%

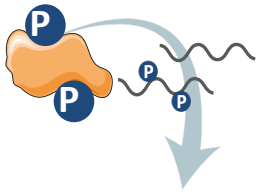
AP3-based drug design: ACR-2316, a potential first-in-class, dual WEE1/PKMYT1 inhibitor and pipeline

STREAMLINED AP3-BASED BIOLOGICAL SAR OPTIMIZATION FOR SINGLE AGENT ACTIVITY OF PRECLINICAL PROGRAMS

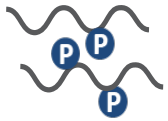
Drug treatment



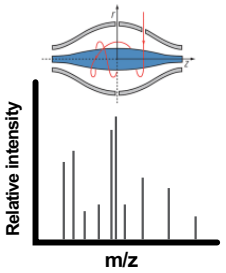
Proteolysis
P-peptide enrichment



Peptide clean up



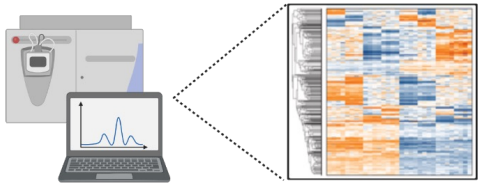
Ultra high resolution
P-proteomic profiling

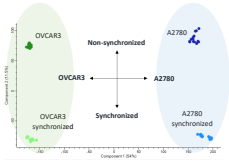


Direct DIA analysis

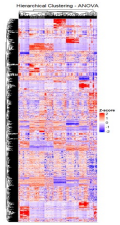
High resolution and throughput MS-based P-proteomics

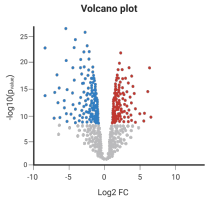
Quantitative mapping (>35,000 phospho-sites covering >4,500 proteins)

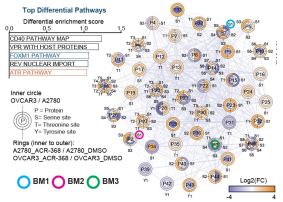




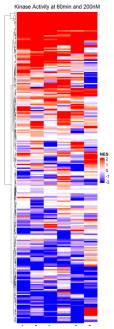
Drug-regulated phospho-sites and biological process enrichment

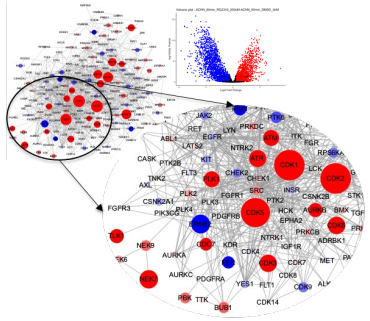


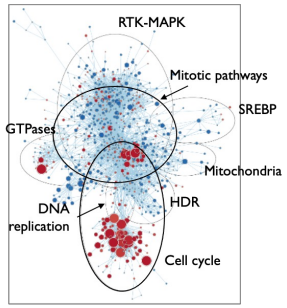




Drug-regulated pathway activity mapping and reconstitution







Proprietary pipe for automated AP3 analyses with actionable results

Week 0

Turnaround <2 weeks

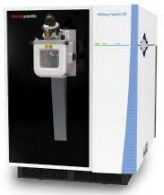
Week 2

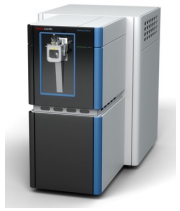
AP3: TIGHT, HIGH-RESOLUTION DATA WITH DEEP COVERAGE

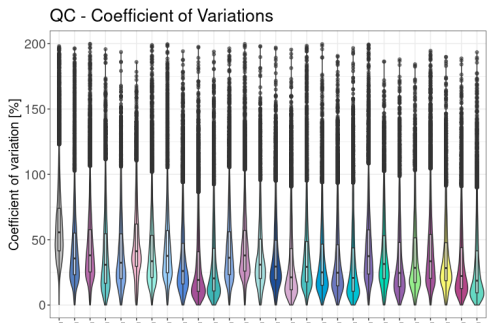
35388 p-sites

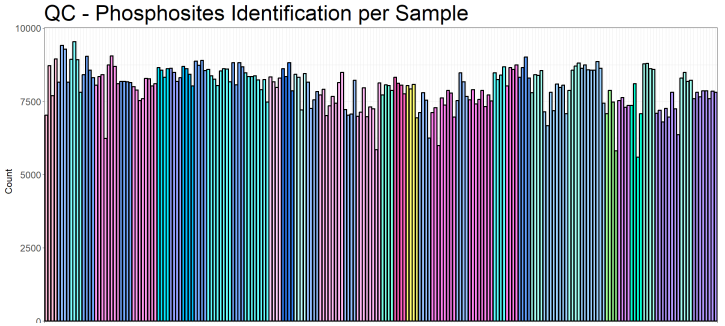
15733 p-sites

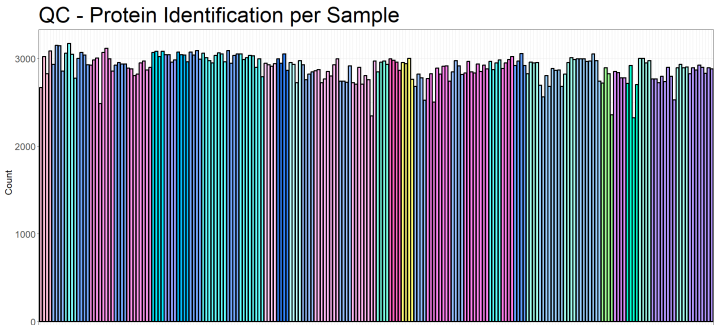
QC MS Data → Data Clean Up → QC Processed Data → Volcano Plots → Hierarchical Clustering → Consensus Sequence Motif → Kinase Inference → Pathway Enrichment → Functional Annotation → Network Mapping → Biomarkers

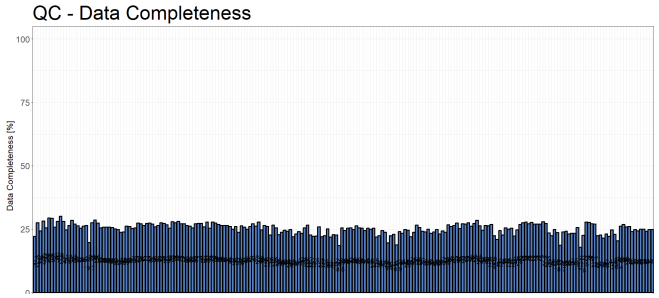


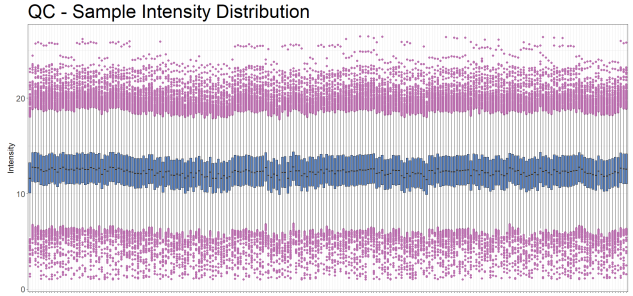


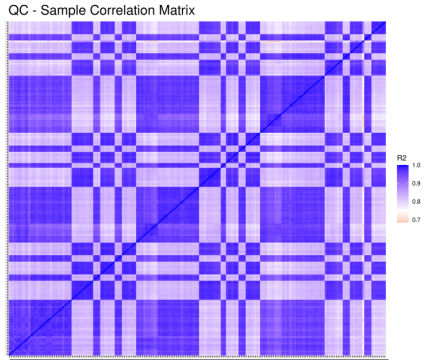












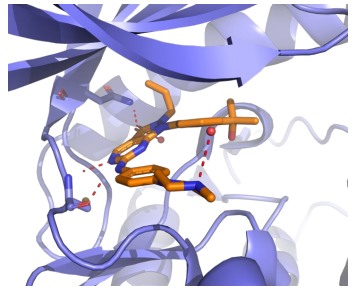
- ✓ Acrivon proprietary compound data (~50 million data points per experiment); dozens of compounds profiled
- ✓ Miniaturized, high throughput, scalable: <2 weeks turn-around, automated AI computational analyses in 1 day
- ✓ Actionable results: Resistance mechanisms, rational combinations, drug-tailored OncoSignature patient selection

ACR-2316 –UNIQUELY ENABLED BY AP3 TO OVERCOME LIMITATIONS OF CURRENT WEE1 AND PKMYT1 INHIBITORS

Program goals:

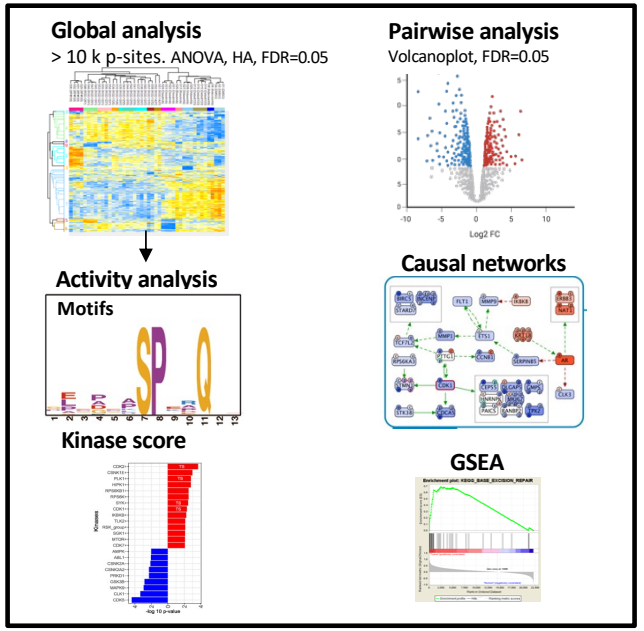
- **Superior single agent activity (AP3)**
  - AP3-guided design to overcome WEE1 and PKMYT1 single inhibitor resistance through balanced dual inhibition
- **High selectivity and potency (co-crystallography)**
  - Structure-guided design to limit adverse events (AEs) to be on-target, transient, mechanism-based
- **Streamlined clinical development (ACR-2316 OncoSignature)**
  - To identify/prioritize sensitive indications prior to clinical start and for drug target engagement-based dose optimization

Co-crystallography for drug design and selectivity



Zhu et al, J. Med. Chem. (2017)

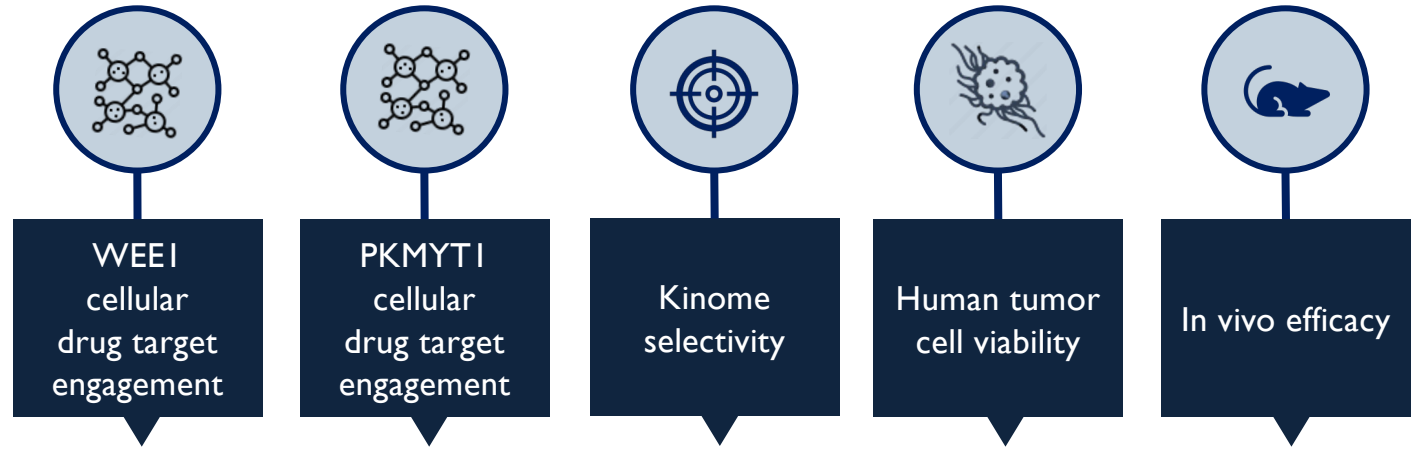
AP3 used for biologically optimal SAR



ACR-2316: Rationally designed WEE1/PKMYT1 development candidate

- ✓ AP3-based SAR from >40 co-crystals (1.5-3.1 Å) of novel WEE1/PKMYT1-selective series
- ✓ 5-20-fold more potent in preclinical models than clinical benchmarks
- ✓ Superior anti-tumor efficacy with complete tumor regression across models
- ✓ High selectivity ensures transient, short-lived, mild AEs
- ✓ Potent WEE1 inhibition, balanced PKMYT1 inhibition, overcomes resistance

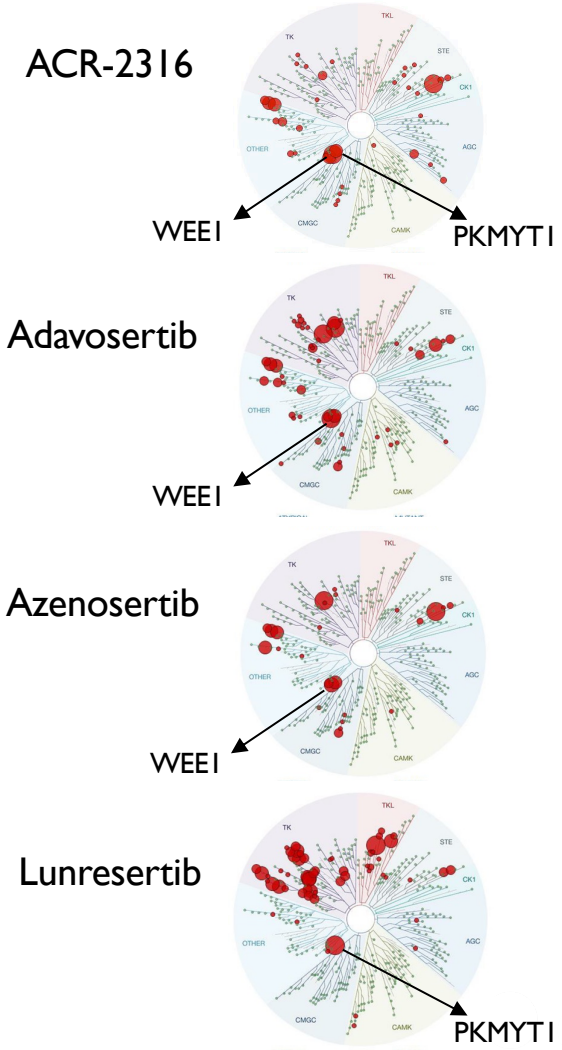
ACR-2316 SHOWS ATTRACTIVE PROFILE IN ONGOING PRECLINICAL STUDIES



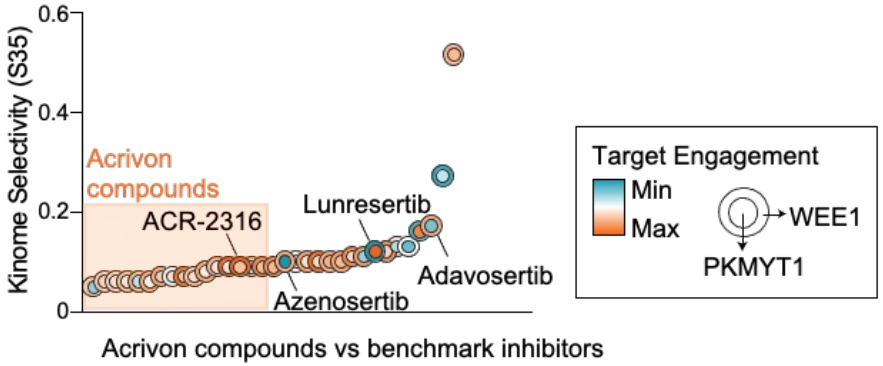
Relative performance	WEE1 cellular drug target engagement	PKMYT1 cellular drug target engagement	Kinome selectivity	Human tumor cell viability	In vivo efficacy
ACR-2316	**++++**	**++**	**++++**	**++++**	**++++**
Adavosertib	++	-	++	+++	++
Azenosertib	++	-	+++	++	++
Debio0123	+	-	++++	+	+
Lunresertib	-	+++	+	+	+

DUAL WEE1/PKMYT1 INHIBITOR ACR-2316 DEMONSTRATES STRONG POTENCY AND SELECTIVITY

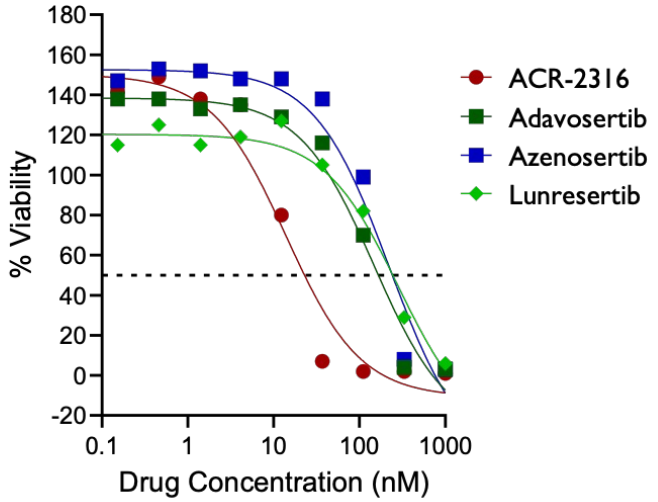
KinomeScan (468 kinases @ 1μM)



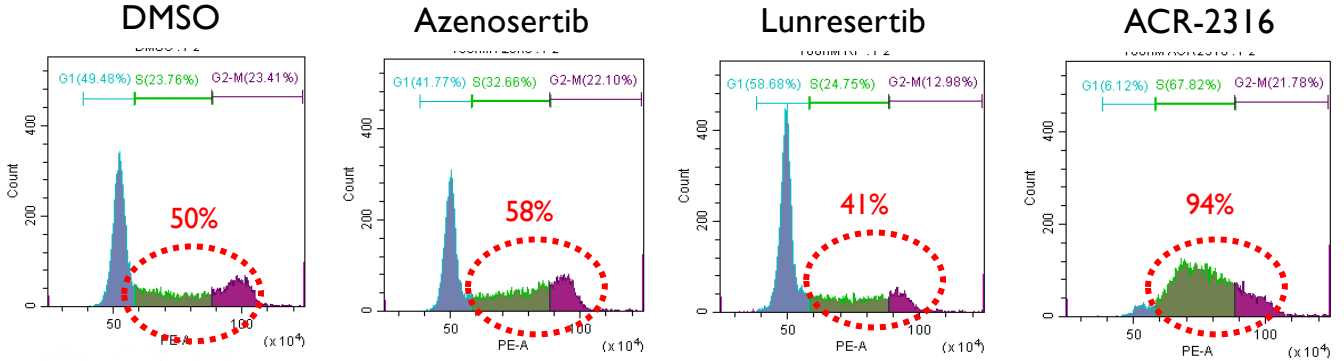
ACR-2316 is highly selective (KinomeScan)



ACR-2316 potently inhibits cancer cell viability



ACR-2316 exerts potent cell cycle effects with pronounced S-G2/M accumulation

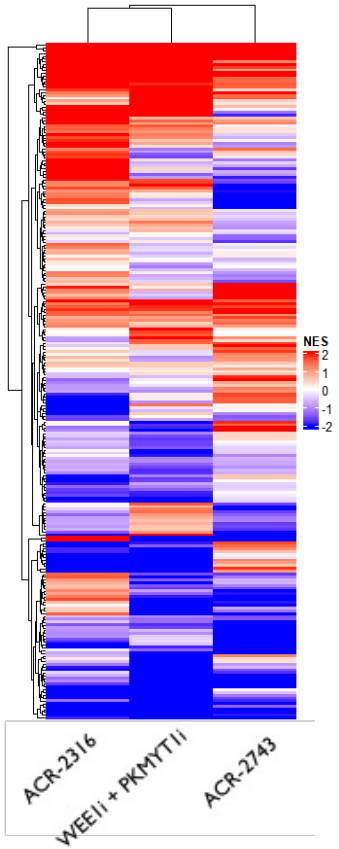


100 nM, 24 hour

OPTIMIZED DUAL INHIBITORS SHOW DESIRABLE PATHWAY EFFECTS

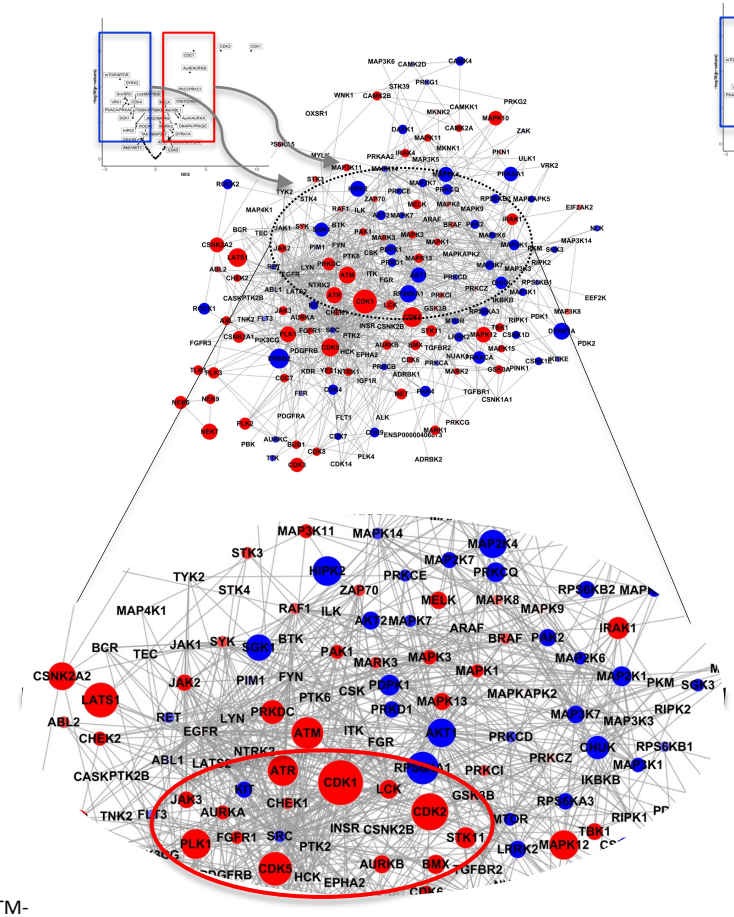
Optimized dual WEE1 and PKMYT1 inhibitors affect cell cycle and canonical MAPK pathways in desirable manner

Substrate motif-inferred kinase activities

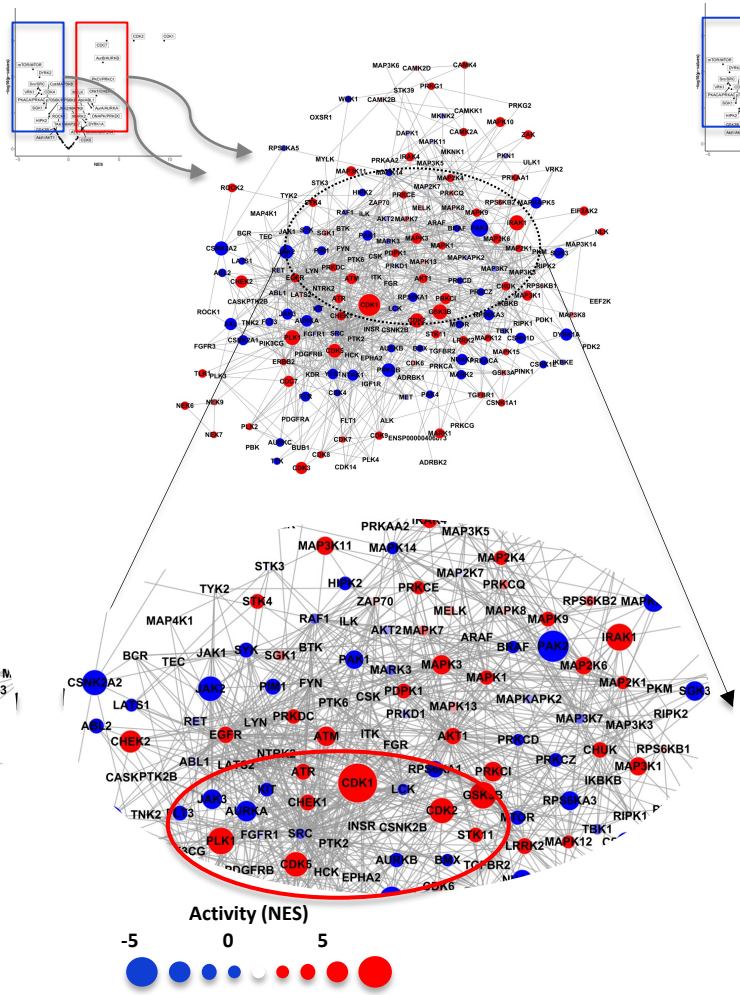


Kinase activity based on proprietary PTM-SEA-based hybrid workflow and analyses

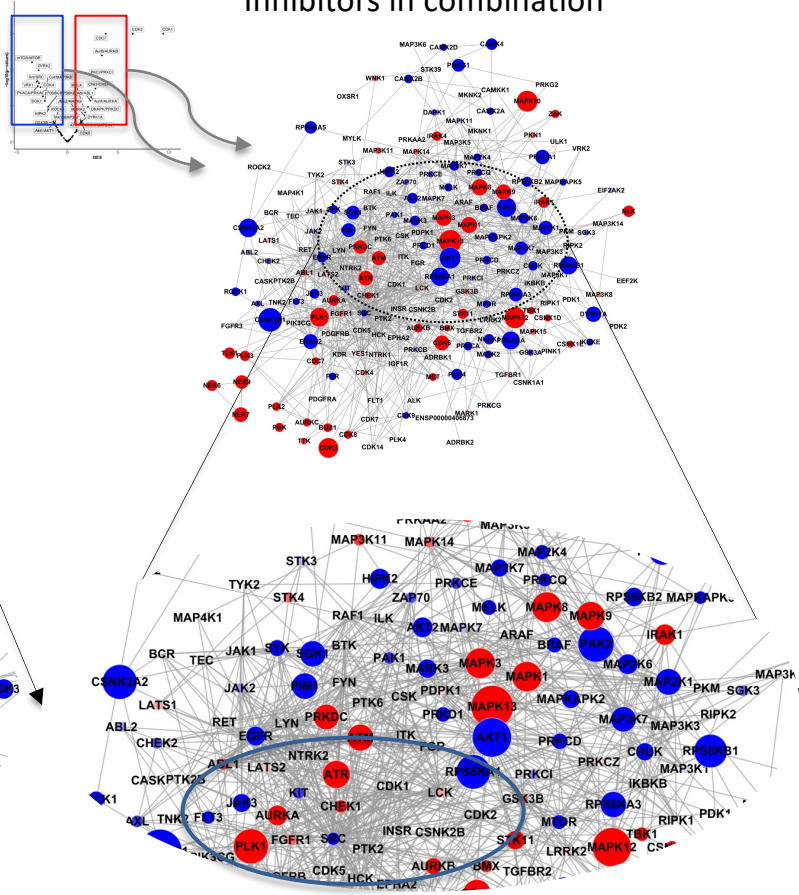
ACR-2316



ACR-2743

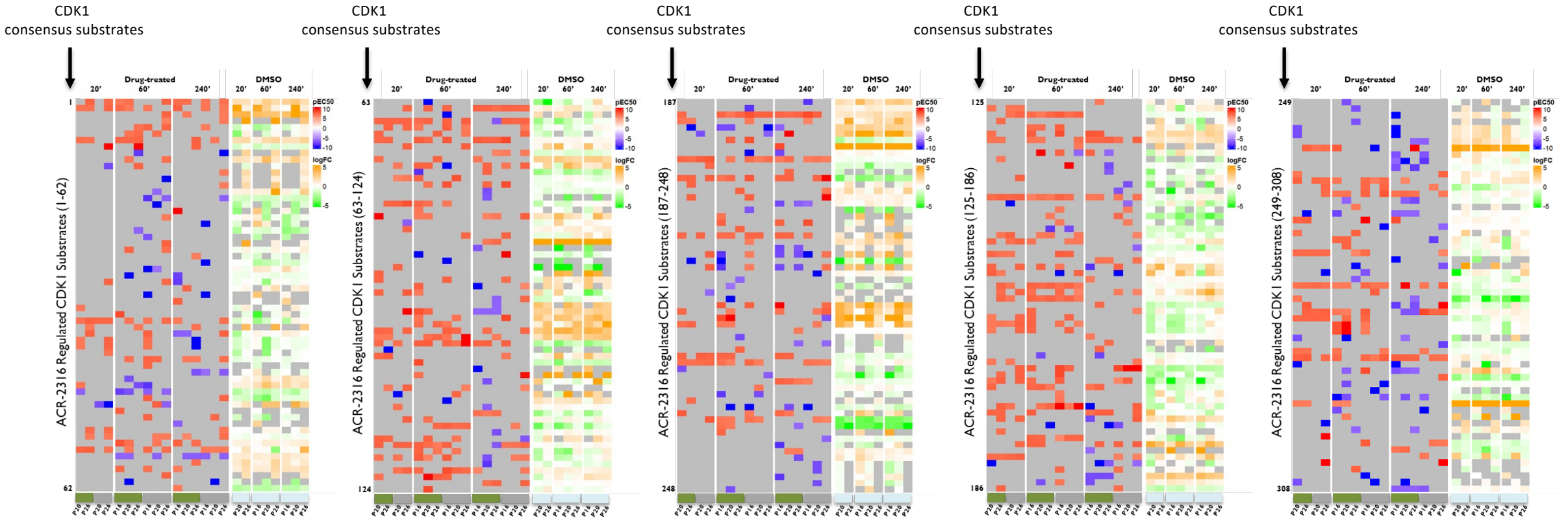


Benchmark* WEE1 + PKMYT1 Inhibitors in combination



*Clinical-stage selective WEE1 and PKMYT1 inhibitors

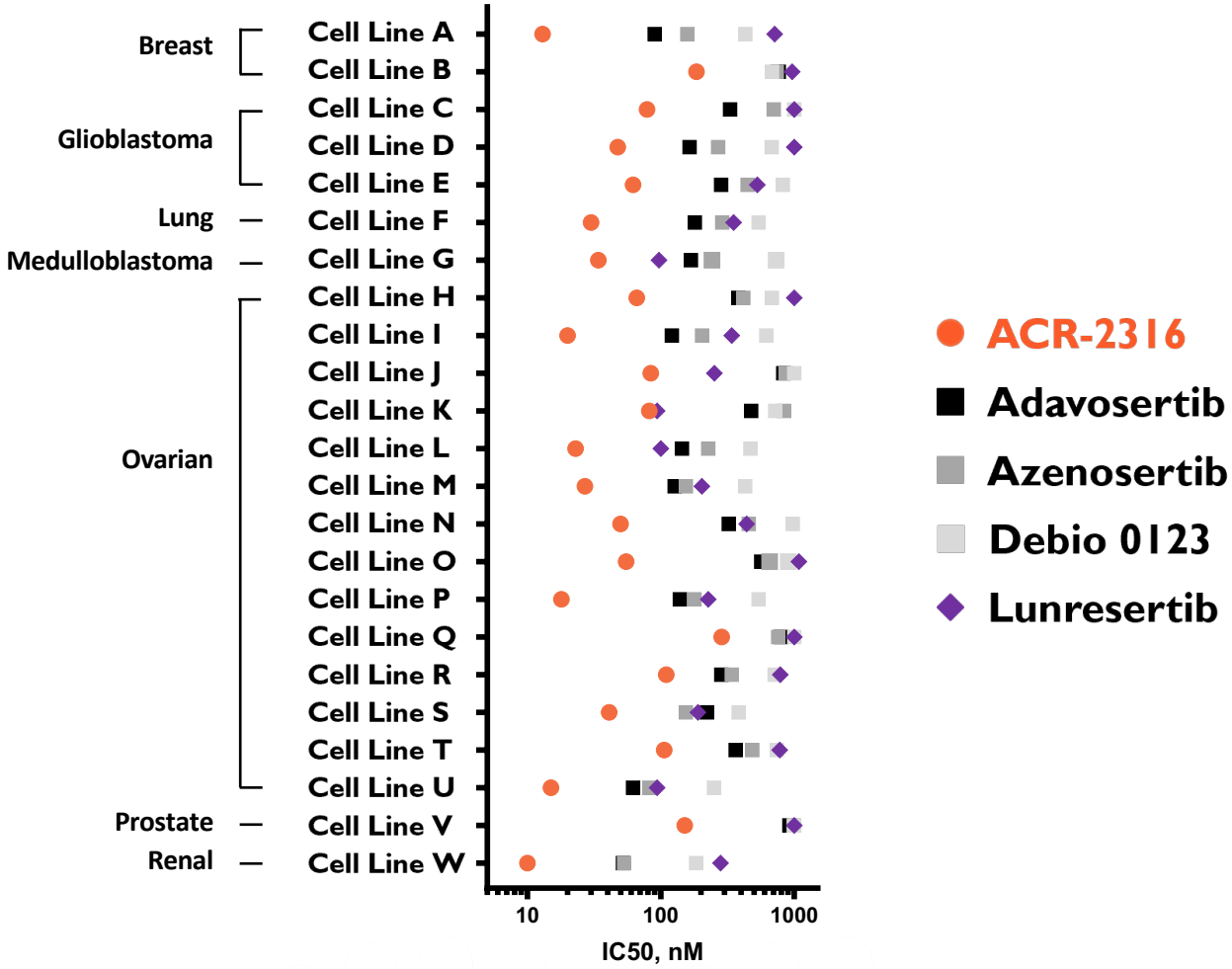
ACR-2316 POTENTLY ACTIVATES CDK1, REGULATING >300 CDK1 CONSENSUS SUBSTRATES AND DRIVING MITOTIC CATASTROPHE



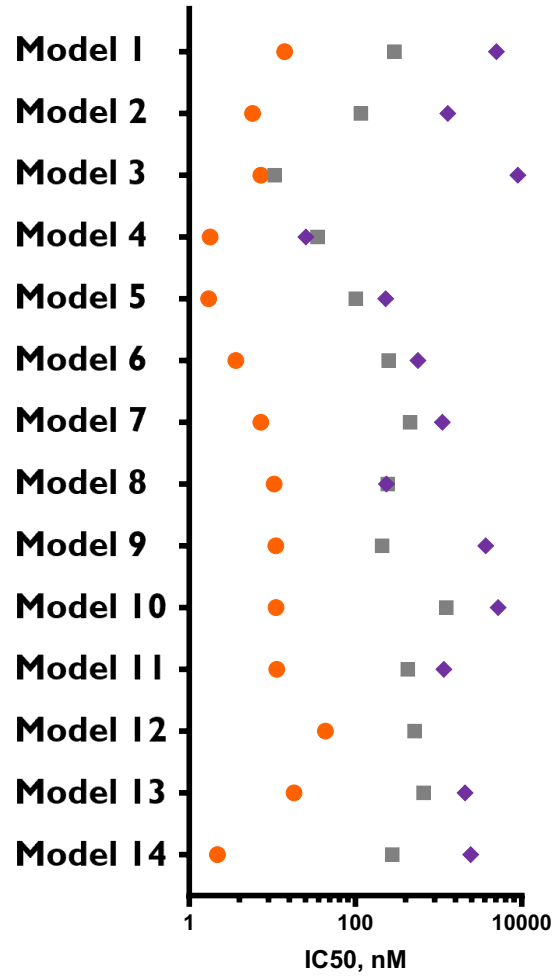
- Unbiased quantitation of ACR-2316-regulated CDK1 substrate p-sites (308) in intact cells based on CDK1 consensus recognition motif (Acrivon proprietary hybrid database approach) across multiple experiments
- Actionable insight into drivers of mitotic catastrophe and on-target CDK1-driven pathways

ACR-2316 IS HIGHLY POTENT ACROSS HUMAN TUMOR CELL LINES AND PATIENT-DERIVED *EX VIVO* TUMOR MODELS

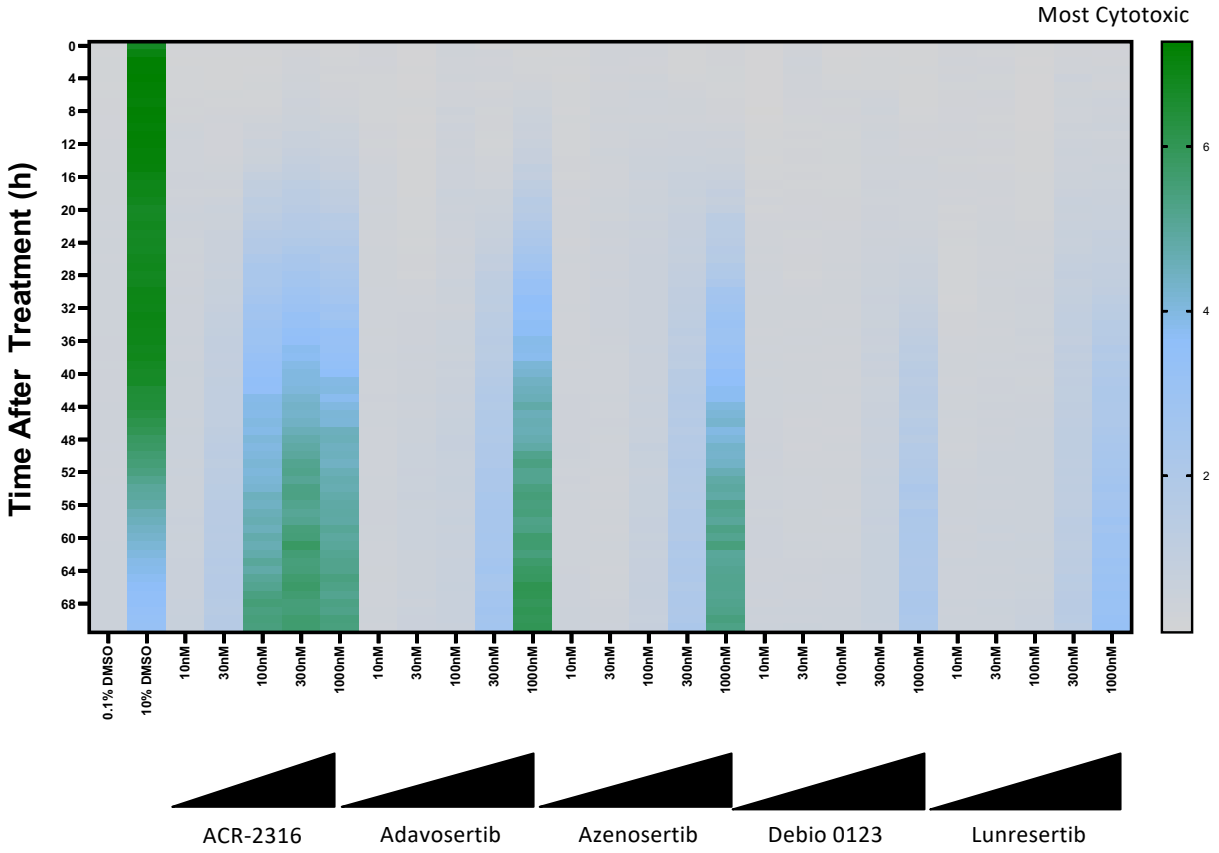
Human tumor cell lines

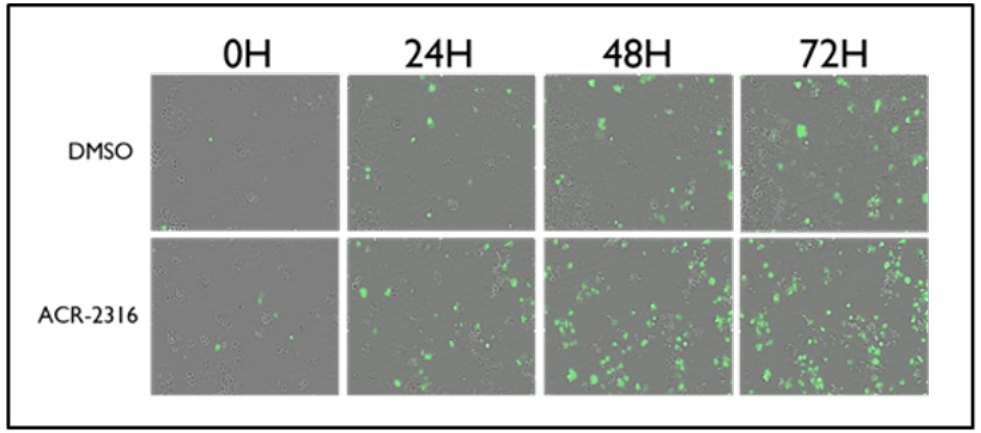


Patient-derived *ex vivo* tumor models



ACR-2316 INDUCES POTENT CELL DEATH COMPARED TO BENCHMARK WEE1 AND PKMYT1 INHIBITORS

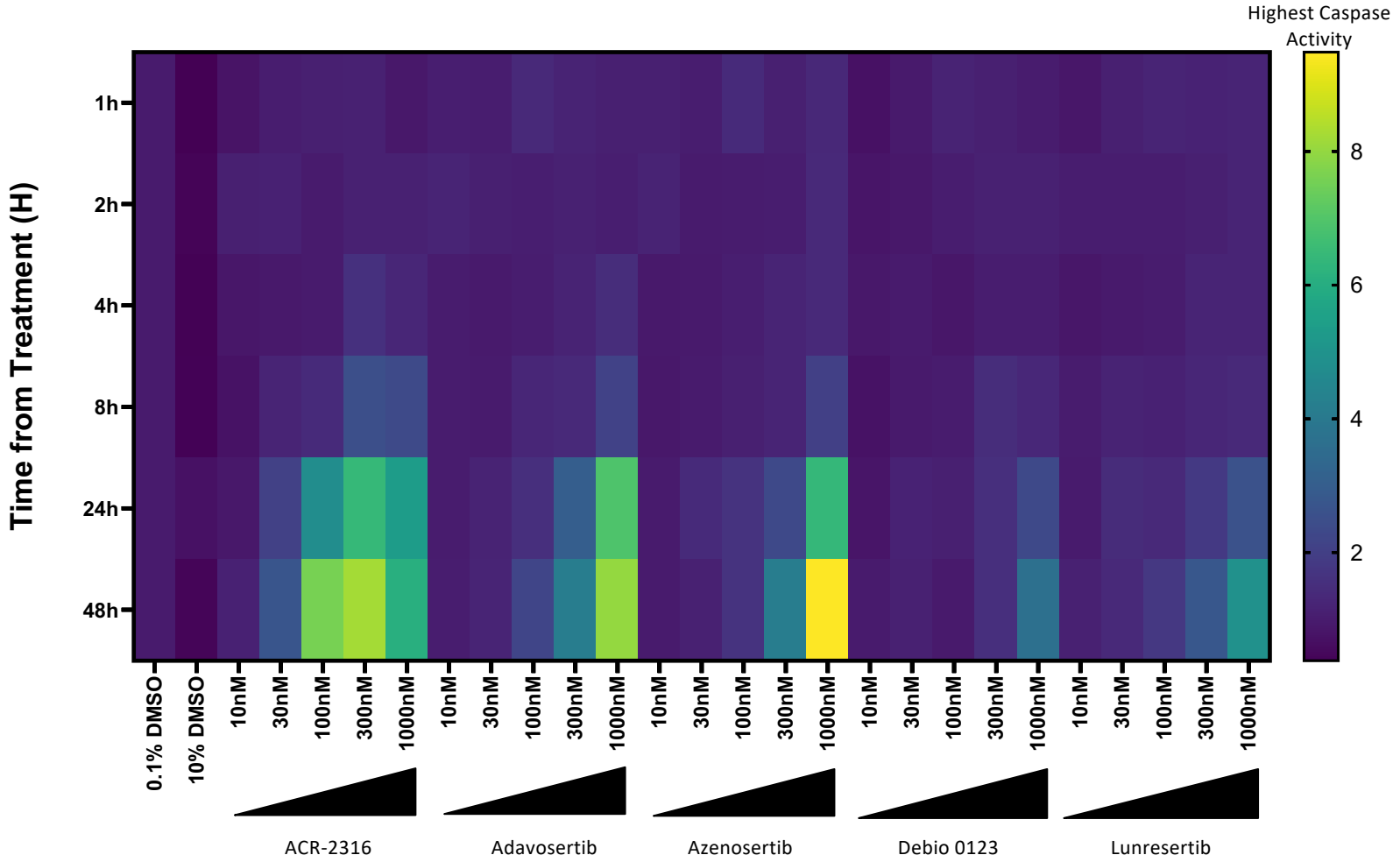




Representative images of human tumor cells treated with 100 nM ACR-2316 vs control vehicle (green fluorescence = dead cells

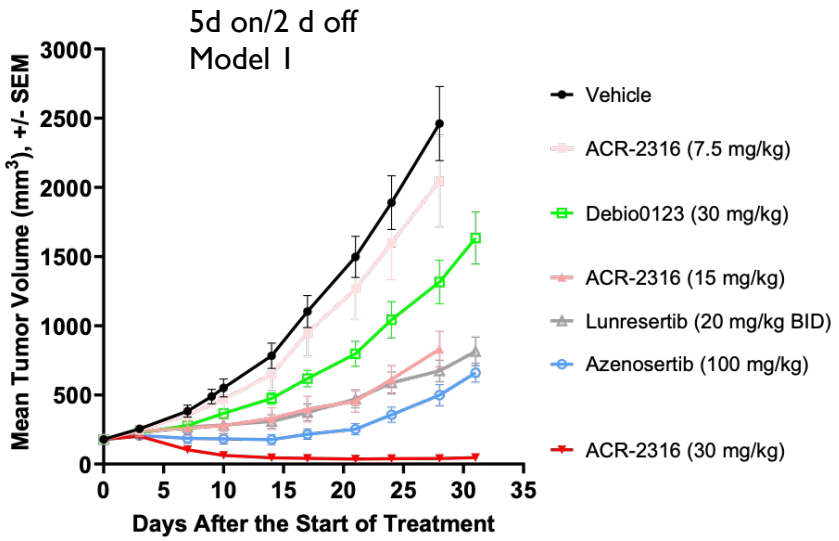
CellTox-Green Assay

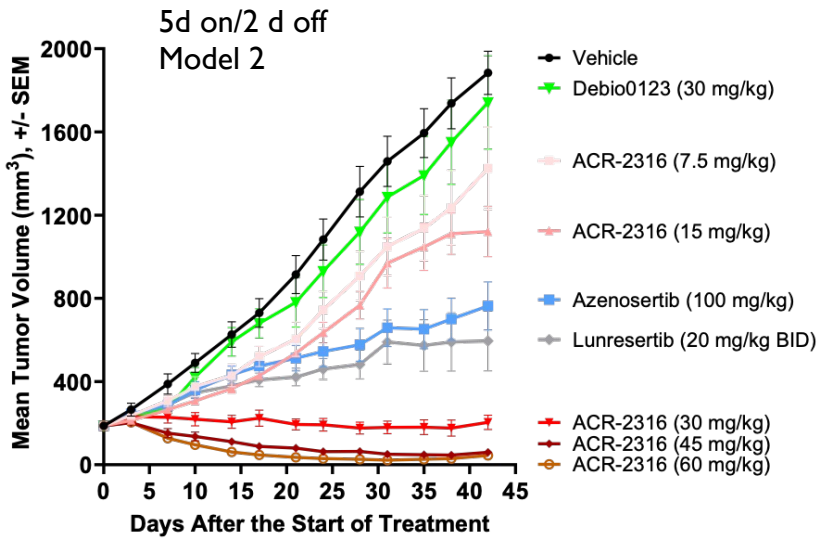
ACR-2316 INDUCES POTENT CASPASE 3/7 CLEAVAGE COMPARED TO BENCHMARK WEE1 OR PKMYT1 INHIBITORS

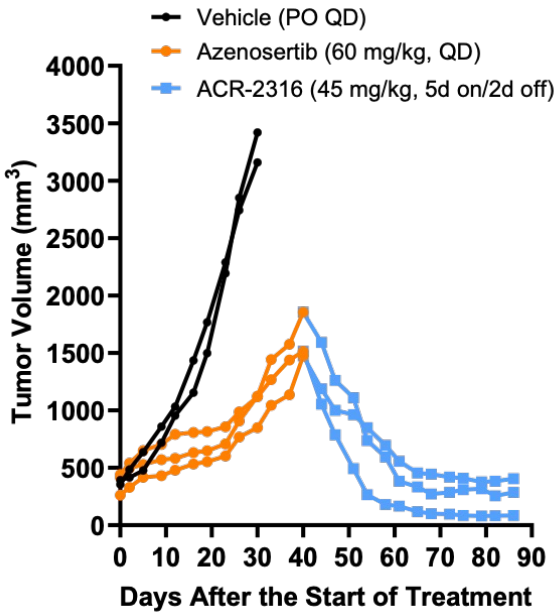


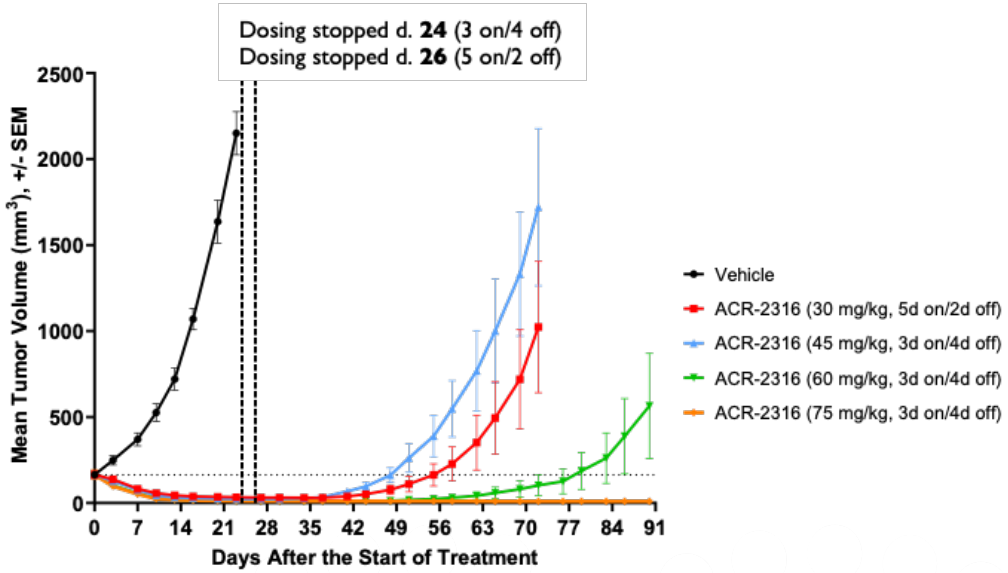
Caspase 3/7-Glo Assay

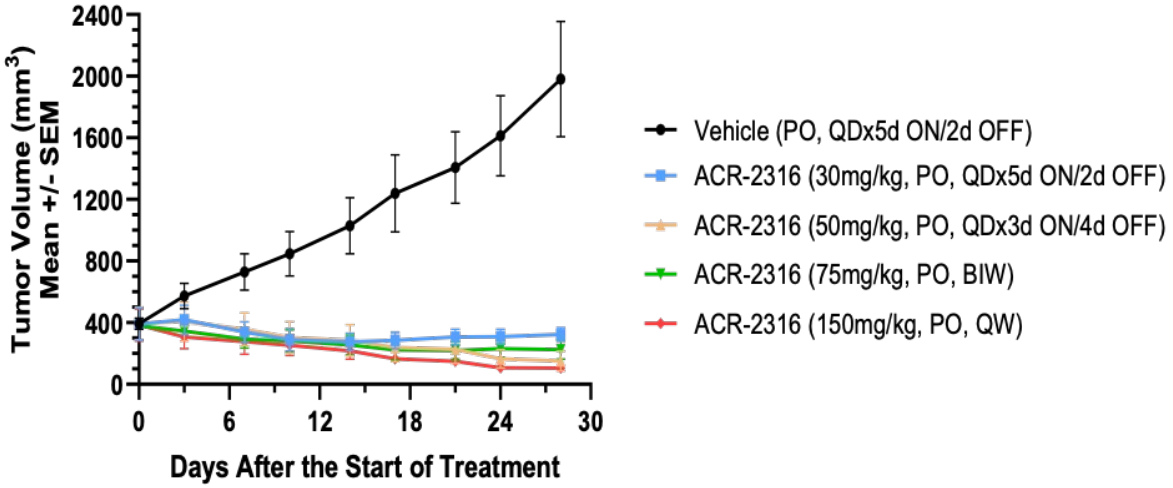
ACR-2316 INDUCES COMPLETE TUMOR REGRESSION ACROSS MODELS AND DOSING REGIMENS







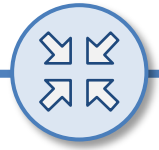




EXPEDITING ACR-2316 TOWARDS CLINICAL MONOTHERAPY DEVELOPMENT

A novel, AP3-enabled, internally discovered dual WEE1 / PKMYT1 inhibitor

Rational Design



- Optimized via AP3
- AP3-enabled design for optimized single agent activity

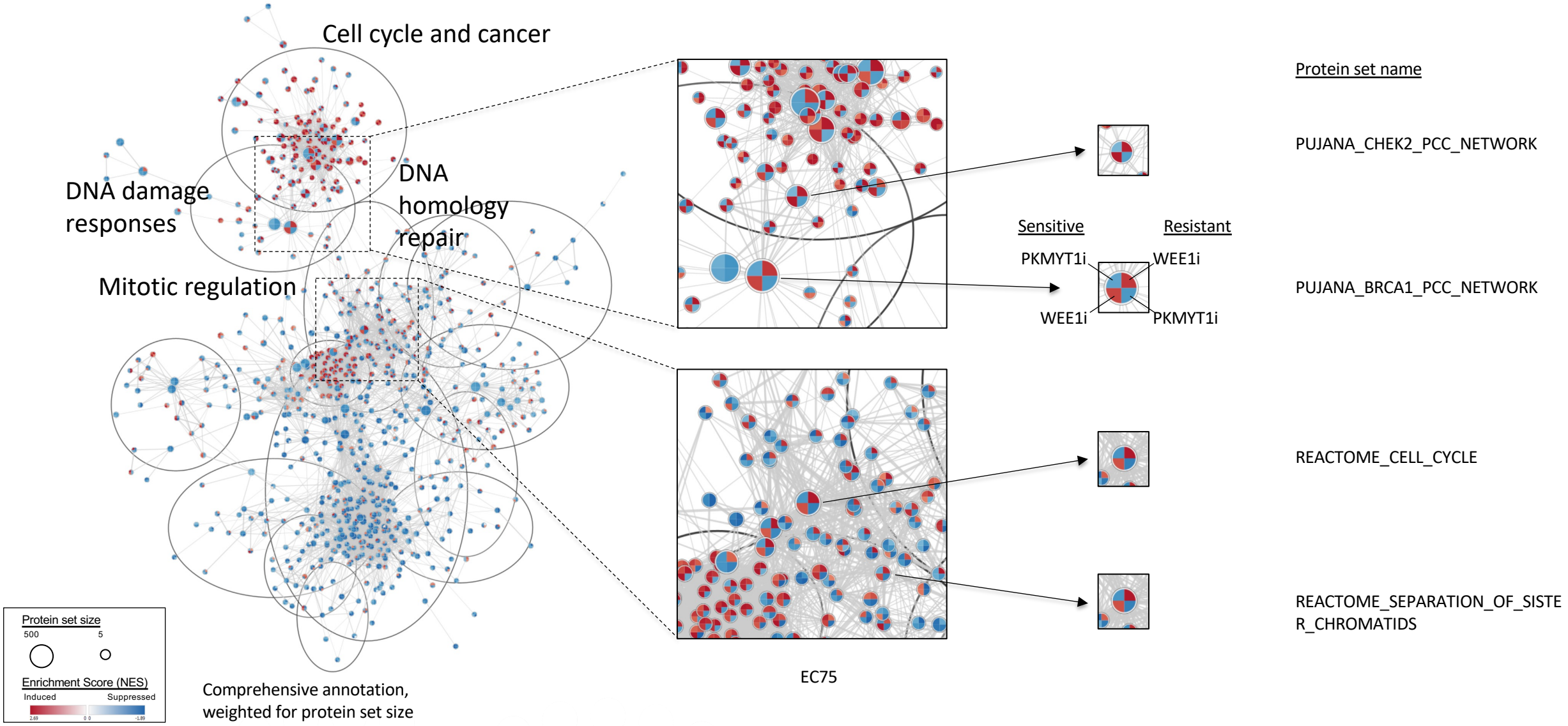
Superior Profile

- Potent anti-tumor activity across human tumor cell lines and in tumor-bearing mice vs benchmarks

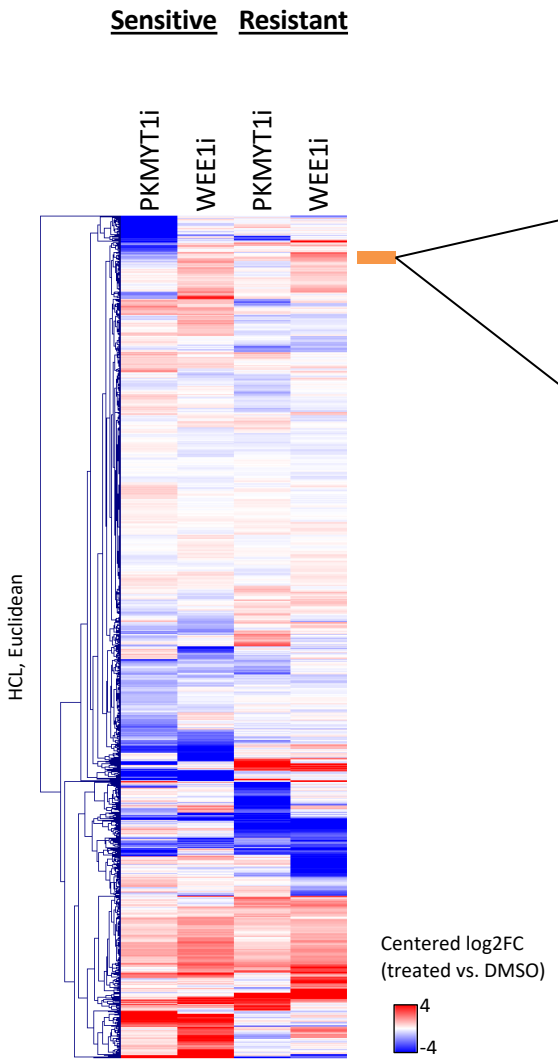
Streamlined Development

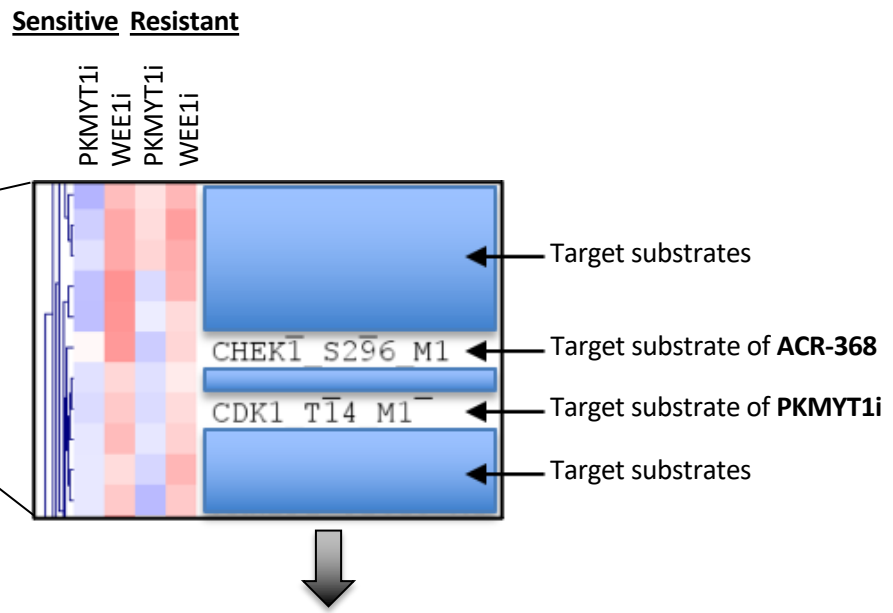
- Aiming for expedited monotherapy development
- Advancing towards IND in Q3, 2024, FIH in Q4, 2024
- OncoSignature test in development for indication finding
- Dose optimization to be guided by drug target engagement (BM2)

AP3-DERIVED DUAL POTENCY OPTIMIZATION TO OVERCOME WEE1 INHIBITOR RESISTANCE: RECIPROCAL QUENCHING

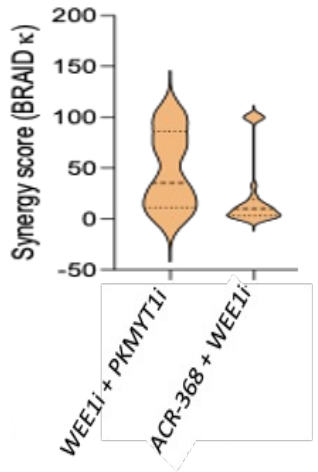


AP3 RECIPROCAL QUENCHING REVEALS OPTIMAL TARGET POTENCY PROFILE FOR DUAL WEE1/PKMYT1 INHIBITOR

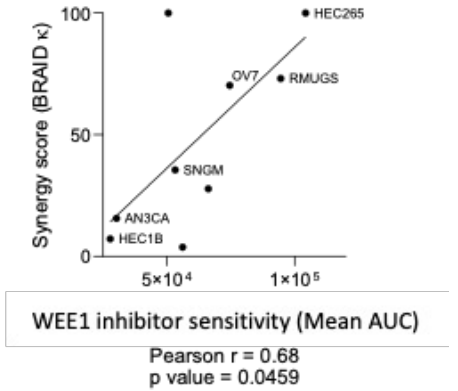




Optimal potency ratio results in significant synergy

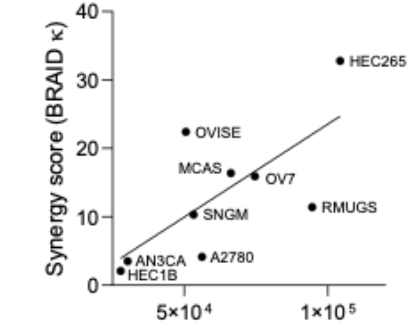


WEE1i and PKMYT1i dual inhibition synergy



Pearson r = 0.68
p value = 0.0459

WEE1i and ACR-368



Pearson r = 0.71
p value = 0.0324

AP3 INTERACTOME V.1: PROPRIETARY INTERACTIVE DATA ANALYSIS INFRASTRUCTURE

Actionable data across all AP3 experiments accessible for all Acrivon scientists
Fully scripted, algorithm-based machine-learning enabled pathway and biomarker analyses

50,000 phosphosites

20,000 phosphosites

Data completeness Peptide IDs CV

Pearson Correlation

PCA Hierarchical Clustering

Filtering Batch effects Normalization Imputation

ANOVA Hierarchical clustering

DEPs Volcano Plots

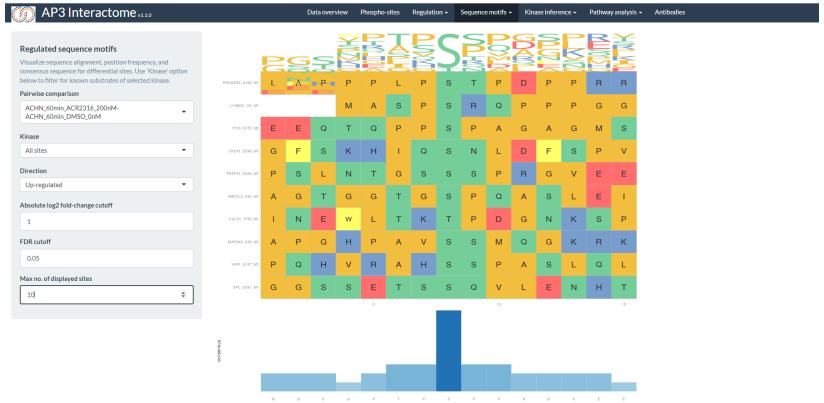
Consensus Sequence Motif

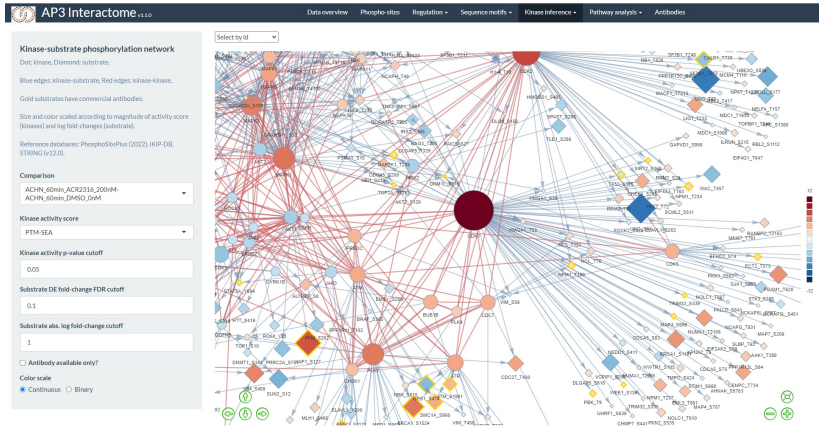
Pathway Enrichment

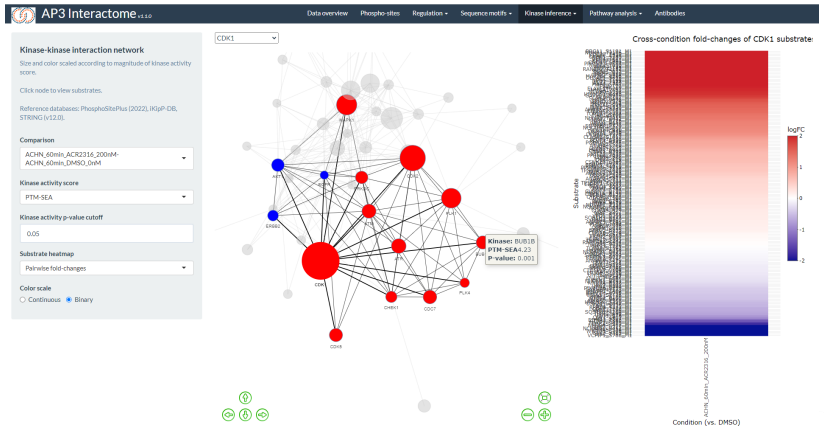
Kinase Inference

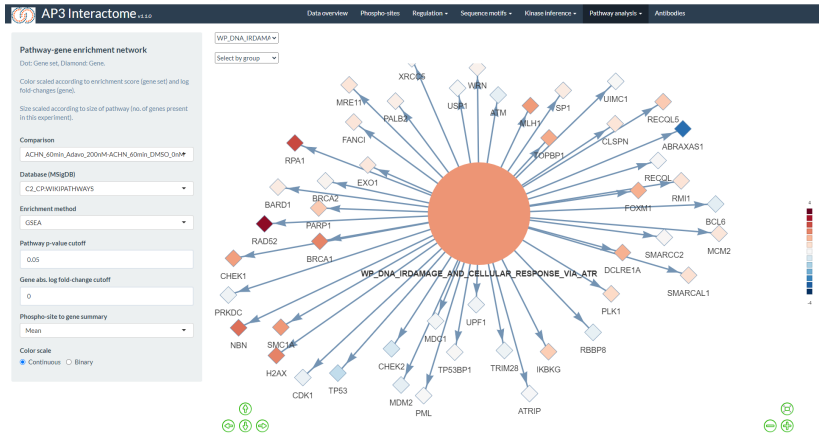
Network Mapping

Biomarkers



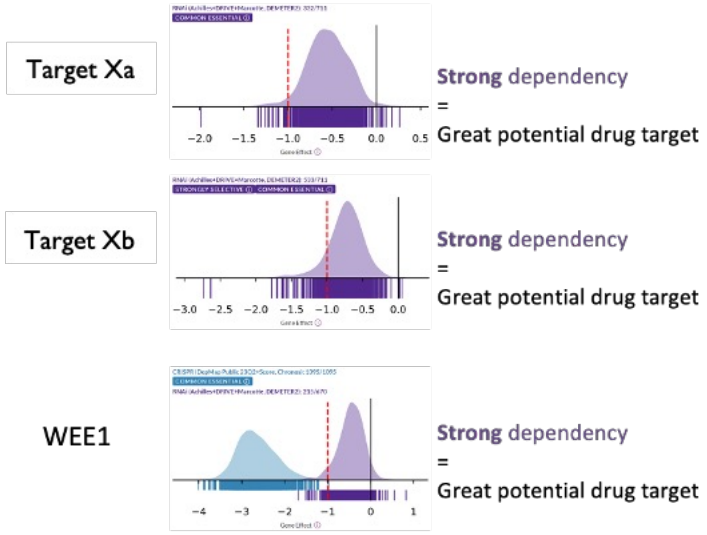




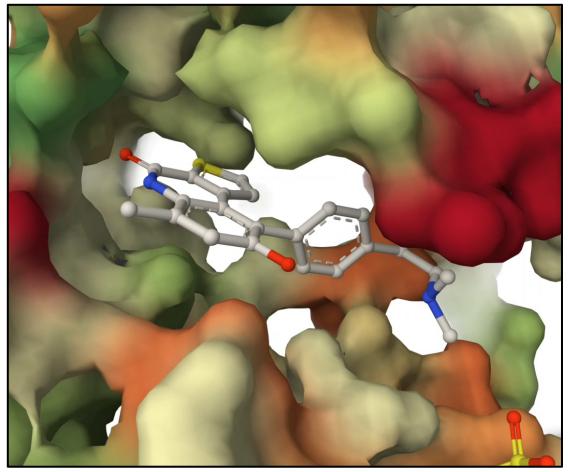


CELL CYCLE REGULATORY PIPELINE PROGRAM (UNDISCLOSED TARGET)

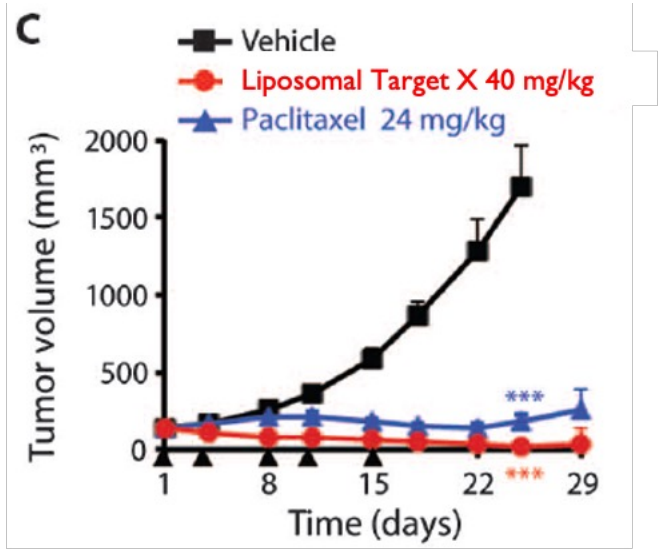
- Target X – an exciting cancer drug target, no/minimal competitor programs, perfectly suited for AP3 platform
- DepMap data suggest suggest target X is an essential gene for cancer cell viability
- Strong mechanistic target rationale for role in oncogenesis
- Highly selective tool compound shows strong anti-tumor efficacy in rodent models
- Tool compound AP3 profiling supports selectivity
- New preclinical program leveraging co-crystallography and AP3 infrastructure successfully built for ACR-2316



Genetic dependency analysis across CCLE (n > 700)



Tool compound is a selective target X inhibitor
(originally believed to be inhibitor for another target)



Development candidate 2025

FINANCIAL HIGHLIGHTS – MARCH 31, 2024 PRO-FORMA

Cash and marketable securities

~$234M

Pro-forma as of Mar-31-2024
Including net proceeds from recent PIPE

Projected runway into

H2'26

Current operating plan, including PIPE
Not including additional financing

Fully Diluted Shares Outstanding

~43.8M

Pro-forma as of Mar-31-2024
Including shares and pre-funded warrants issued with recent PIPE

Note: Unaudited

KEY TAKE-AWAYS

Initial clinical data (cut-off date April 1, 2024) have demonstrated prospective validation of our AP3 platform and our key objectives

- 50% confirmed overall response rate observed in patients with OncoSignature-positive gynecological (ovarian and endometrial) cancers
  - Enrichment of ORR in ovarian cancer (40% confirmed ORR)
  - AP3-based prediction of endometrial cancer to be sensitive to ACR-368 now proven with clinical data and confirmed ORR of 60%
  - All 5 confirmed responders still on treatment, with mDoR not yet reached

- Initial, prospective validation of the AP3-based ACR-368 OncoSignature assay has demonstrated clear segregation of RECIST responders in the OncoSignature-positive versus OncoSignature-negative arms (p-value=0.0038)

Streamlined AP3-based drug design - ACR-2316, our internally-discovered dual WEE1/PKMYT1 inhibitor

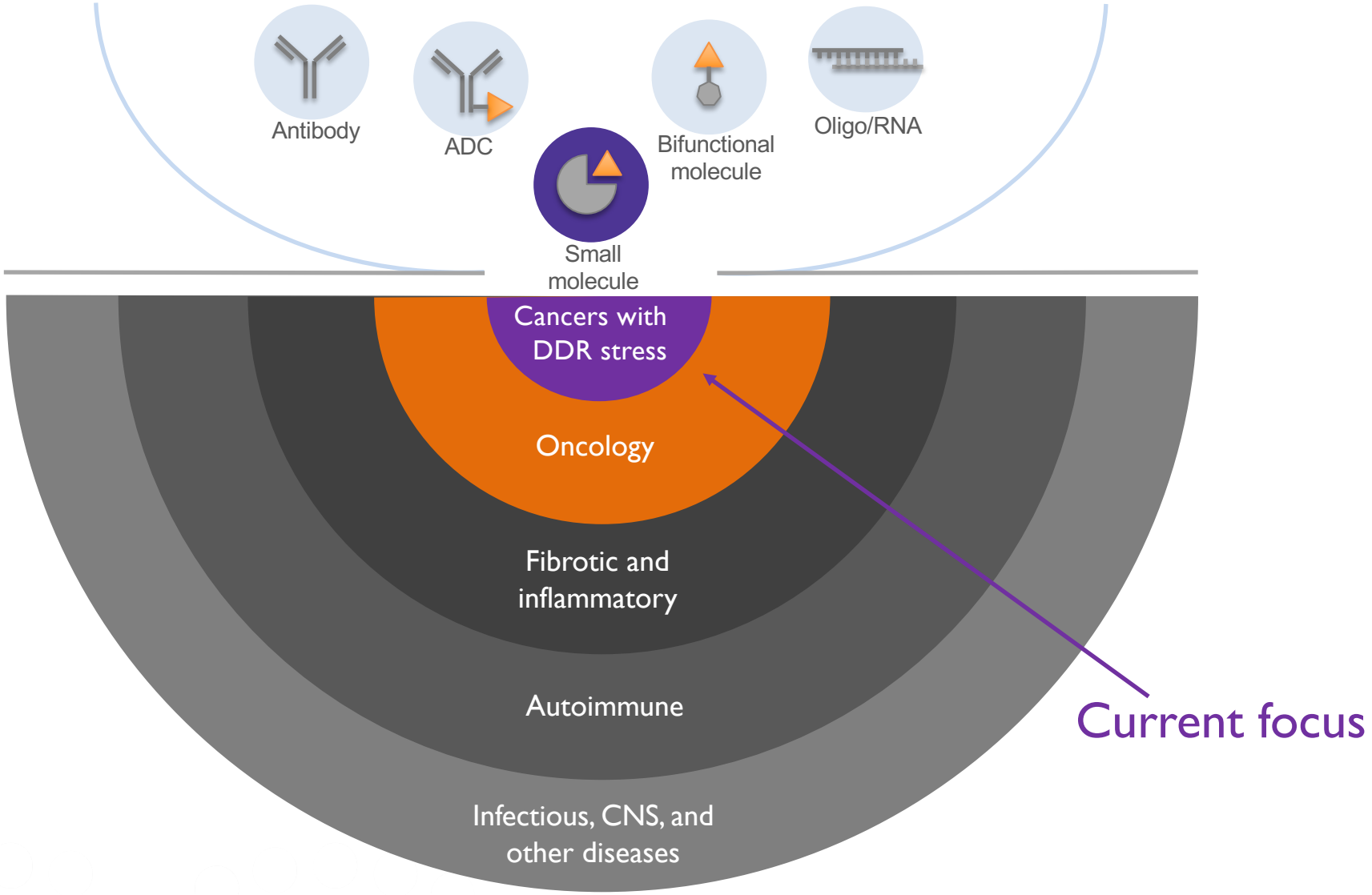
- Demonstrated superior single agent activity in preclinical studies compared to clinical benchmarks
- Accelerated timelines with anticipated IND filing in Q3 and first-in-human in Q4, 2024

Pro-forma cash and marketable securities ~$234M with runway projected to second half of 2026

THE AP3 APPROACH IS MODALITY AND DISEASE AGNOSTIC

Therapeutic modalities

Therapeutic areas



- **Q&A session**