CORPORATE R&D EVENT

MARCH 25, 2025

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.

You are cautioned not to place undue reliance on these forward-looking statements, which are made only as of the date of this presentation. We undertake no obligation to update any forward-looking statements or to reflect new information or the occurrence of unanticipated events, except as required by law.

ACRIVON TEAM PARTICIPANTS

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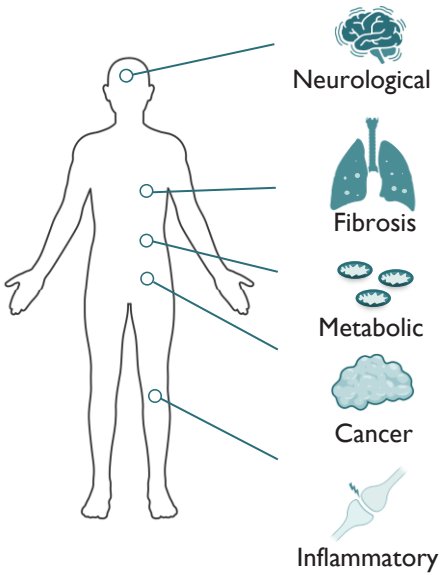
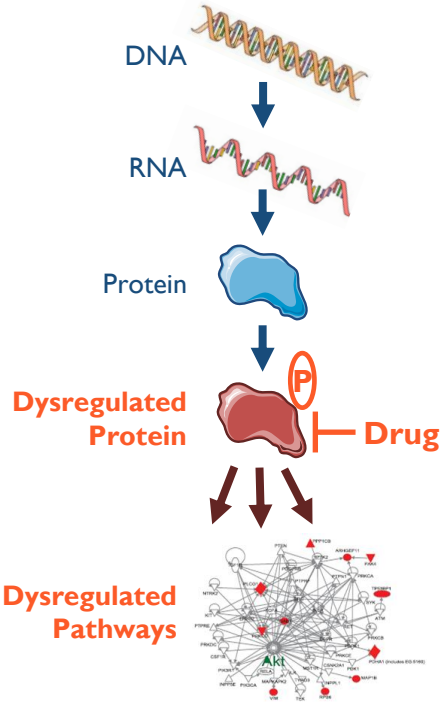
Professor at the University of Copenhagen; Executive Director at the Novo Nordisk Foundation Center for Protein Research; Academic Co-founder of Acrivon

AGENDA

- AP3 Generative Phosphoproteomics: Transforming drug discovery with proteome-wide SAR
- ACR-368 Phase 2 program update
- ACR-2316 Phase 1 program update
- Brief update on preclinical cell cycle program
- Extended runway guidance
- Q&A

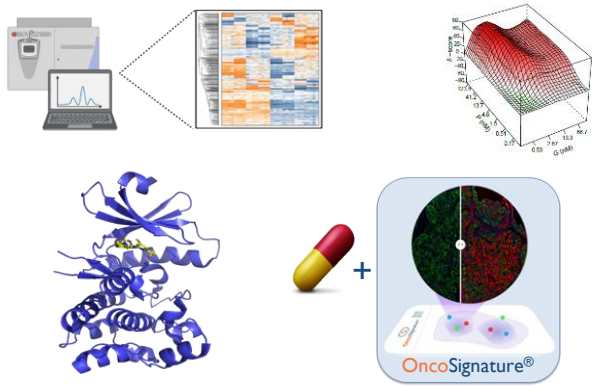
ACRIVON THERAPEUTICS – A NEXT-GENERATION PRECISION MEDICINE COMPANY





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



Blume-Jensen, P & Hunter, T: Oncogenic kinase signaling *Nature* (2001)
Olsen, JV et al: Global, in vivo, and site-specific phosphorylation dynamics in signaling networks *Cell* (2006);
Andersen, JN et al: Pathway-based identification of biomarkers for targeted therapeutics: personalized oncology with PI3K pathway inhibitors *Sci Transl Med* (2010)

STREAMLINED AP3-BASED DRUG DESIGN: INTEGRATED AI/ML-DRIVEN PROTEOME-WIDE SAR

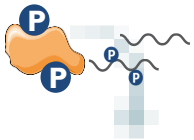
Cellular material
(~0.3 ng / human cell)

↓

Drug treatment

↓

Proteolysis
P-peptide enrichment



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Peptide clean up

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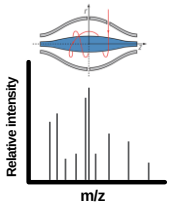
Ultra high resolution
P-proteomic profiling



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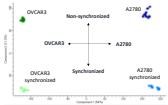
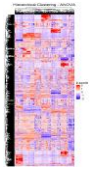
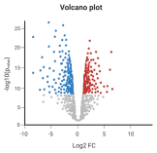
Direct DIA analysis

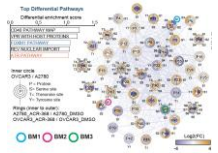
Quantitative mapping (>130,000 phospho-sites covering >9,000 proteins)



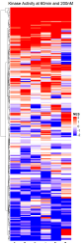
Drug-regulated phospho-sites and biological process enrichment

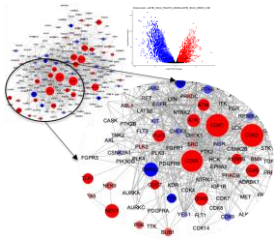


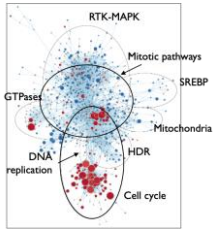




Drug-regulated pathway activity mapping and reconstitution







Week 0

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Turn-around
<2 weeks

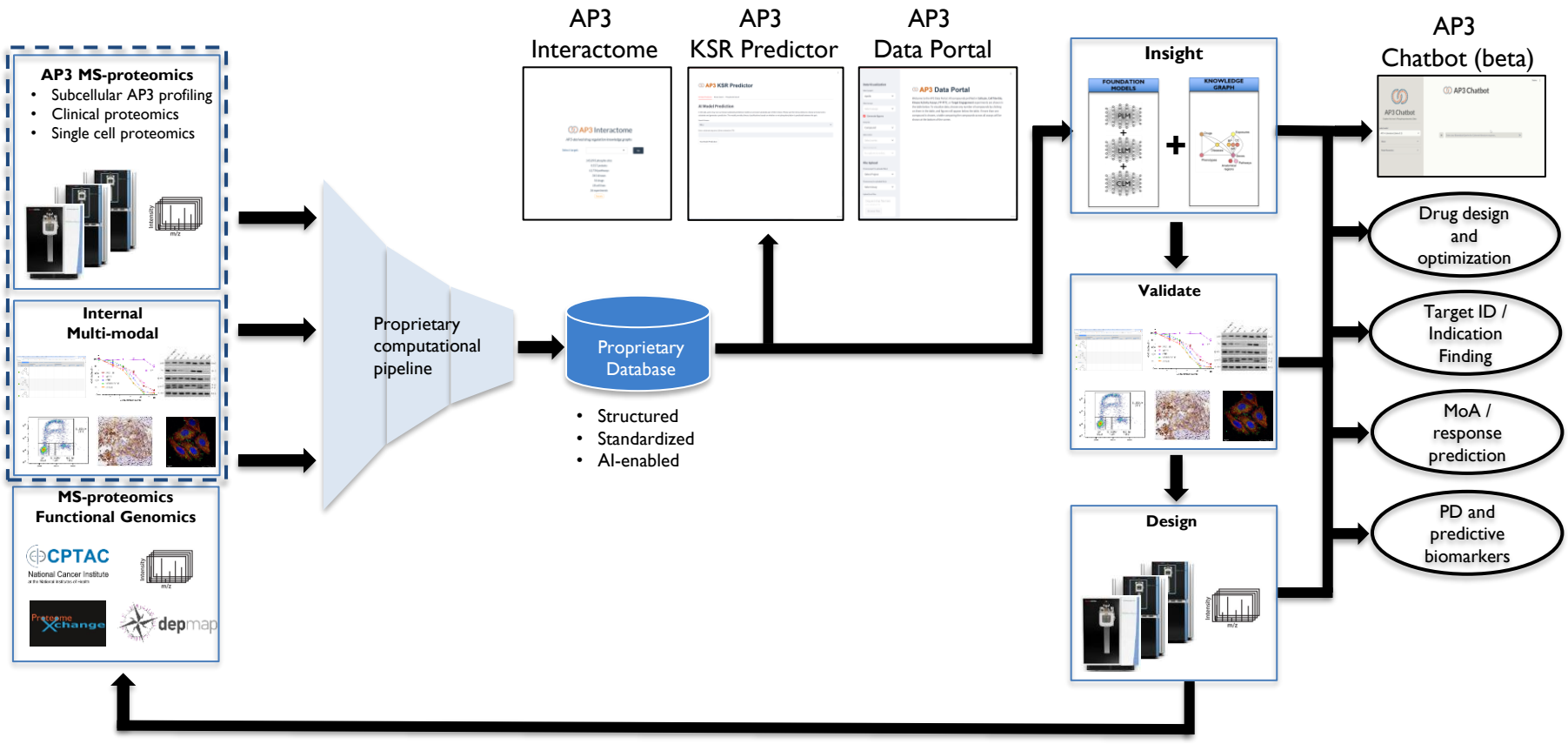
↓

Week2

High resolution and throughput MS-based P-proteomics

Proprietary generative phosphoproteomic AP3 analyses with actionable results

AP3 GENERATIVE PHOSPHOPROTEOMICS PLATFORM



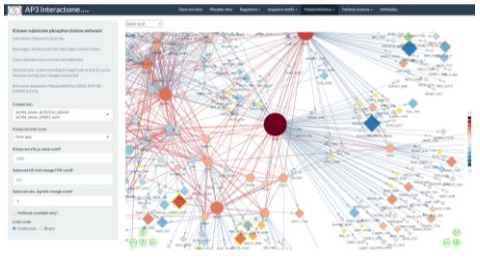
AP3 INTERACTOME: PROPRIETARY INTERACTIVE DATA ANALYSIS INFRASTRUCTURE FOR ALL ACRIVON DATA AND EXPERIMENTS

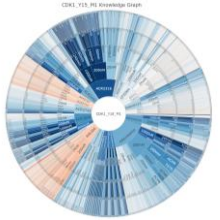
Actionable data across all AP3 experiments accessible for all Acrivon scientists
Fully scripted, algorithm-based machine learning-enabled pathway and biomarker analyses

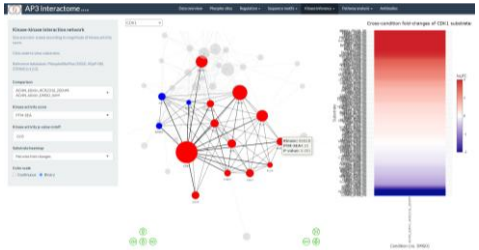
~150,000 phosphosites

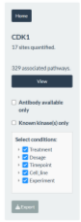
~50,000 phosphosites

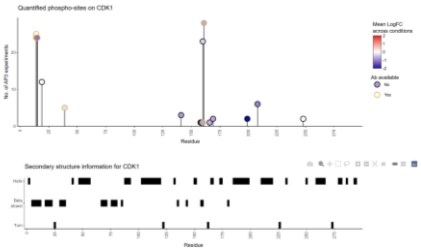
- Data completeness Peptide IDs CV
- Pearson Correlation
- PCA Hierarchical Clustering
- Filtering Batch effects Normalization Imputation
- ANOVA Hierarchical clustering
- DEPs Volcano Plots
- Consensus Sequence Motif
- Pathway Enrichment
- Kinase Inference
- Network Mapping
- Biomarkers Machine-Learning



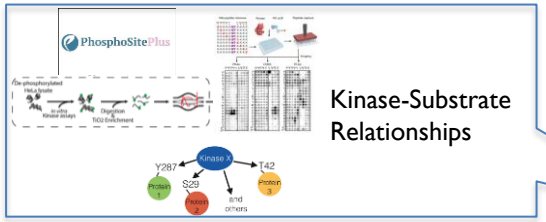








PREDICTION BEYOND TRAINING - GENERATIVE PHOSPHOPROTEOMICS



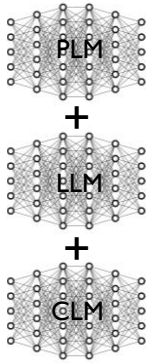
Kinase-Substrate Relationships

- Proprietary AP3 data
- External data

- Protein-Ligand Interactions
- Properties

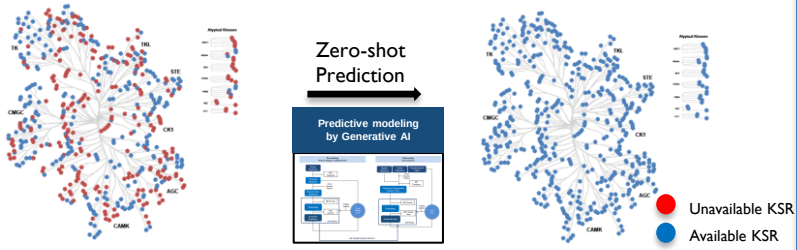
Fine-Tuning

FOUNDATION MODELS

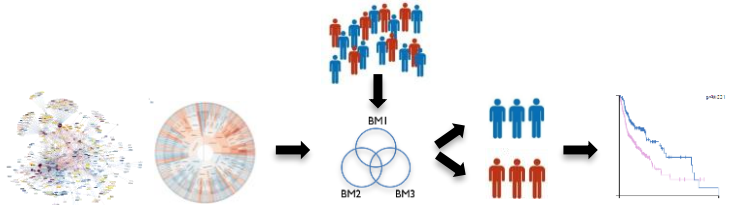


PLM = Protein Language Model
LLM = Large Language Model
CLM = Chemical Language Model

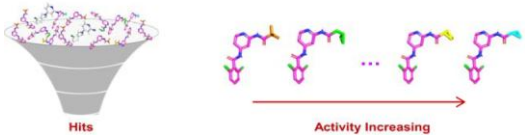
AP3 KSR Predictor:
Generative AI uncovers kinome-wide substrate relationships



MoA and Biomarkers

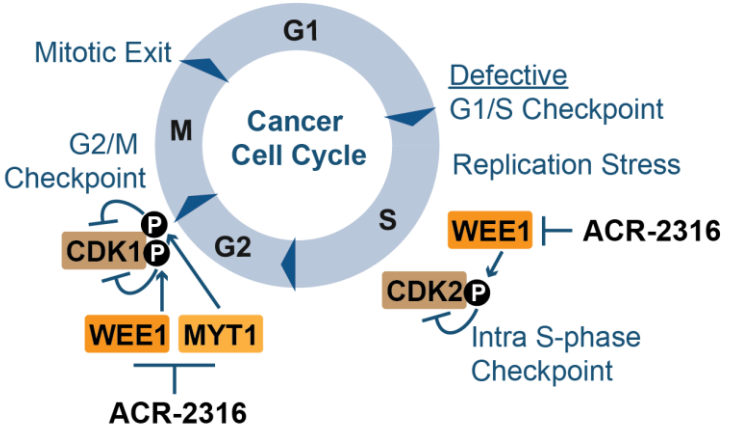


Hit Identification and Optimization

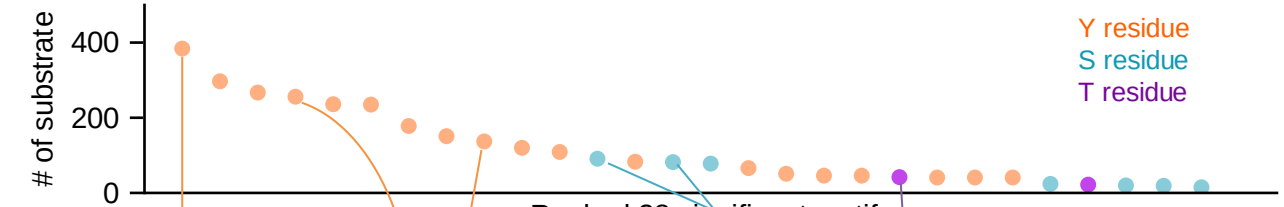


WEE1 AND PKMYT1 - CRITICAL CELL CYCLE CHECKPOINTS IN HUMAN CANCER

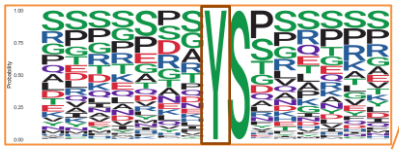
- WEE1 and PKMYT1 regulate S and G2-M cell cycle checkpoints to ensure proper DNA replication and mitotic completion
- Defective DNA repair is highly prevalent in cancers, creating a dependency on checkpoint proteins
- Only known substrate for WEE1 is CDK-Y15 and for PKMYT1 is CDK-T14, both of which inhibit CDK activity
- Despite demonstrated clinical activity, the Therapeutic Index has been challenging
- AP3 was applied to design a molecule with potent single agent activity and exquisite selectivity to achieve an expanded Therapeutic Index

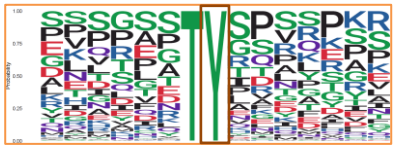


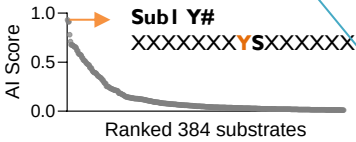
AP3-DERIVED WEE1 SUBSTRATE ENSEMBLE ENRICHED FOR NOVEL CONSENSUS MOTIFS ENABLING PROTEOME-BASED DRUG DESIGN

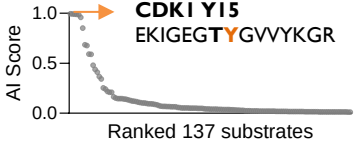


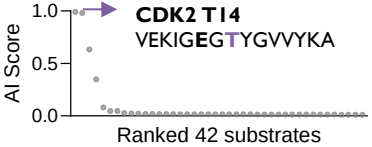
- Tyrosine centric motifs dominate







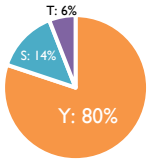




AP3-PREDICTED WEE1 SUBSTRATES AND PATHWAYS FOR PROTEOME-WIDE SAR COMPOUND DESIGN

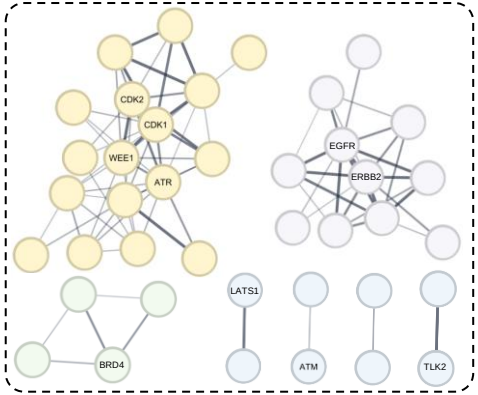
Pathway clusters of selected WEE1 substrates

WEE1 is primarily a protein Tyrosine kinase

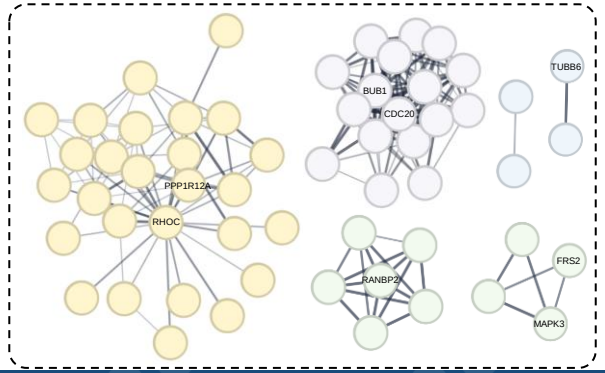


Phosphorylation residues
Total: 2192

GO:0004672, **Protein kinase activity**, FDR: 6.94E-17

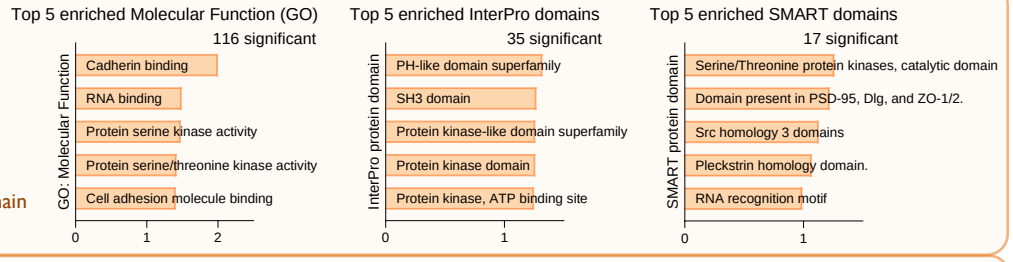


Reactome:HSA-194315, Signaling by **Rho GTPases**, FDR: 1.73E-29

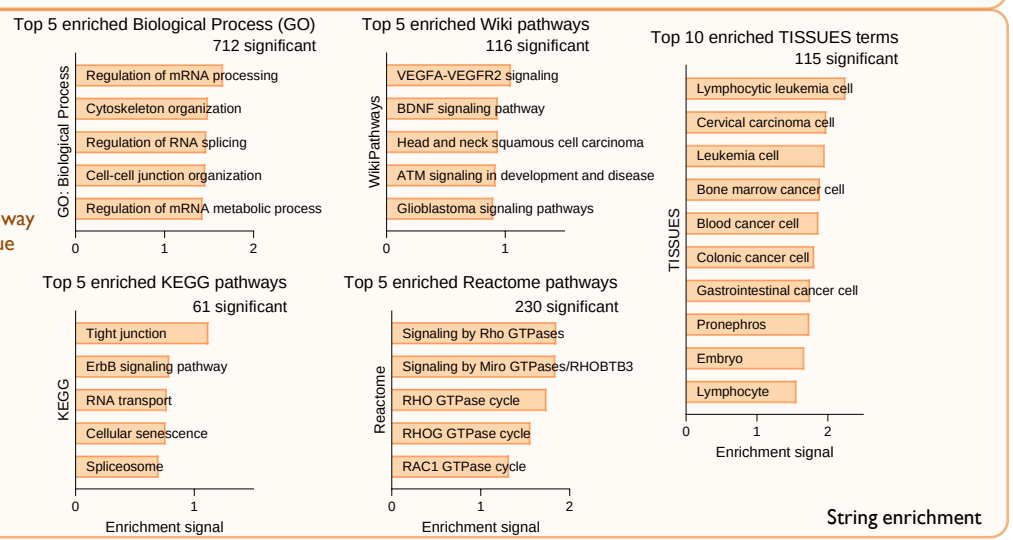


Pathway enrichment based on key substrates

Protein domain and function



Protein pathway and cell/tissue

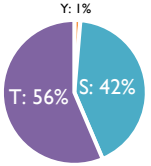


String enrichment

AP3-PREDICTED PKMYT1 SUBSTRATES AND PATHWAYS FOR PROTEOME-WIDE SAR COMPOUND DESIGN

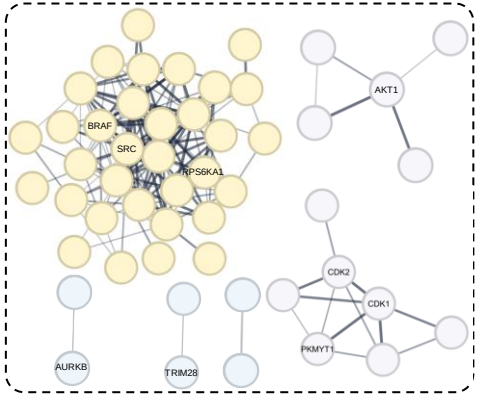
Clusters of selected PKMYT1 substrates

PKMYT1 is a protein Ser/Thr kinase

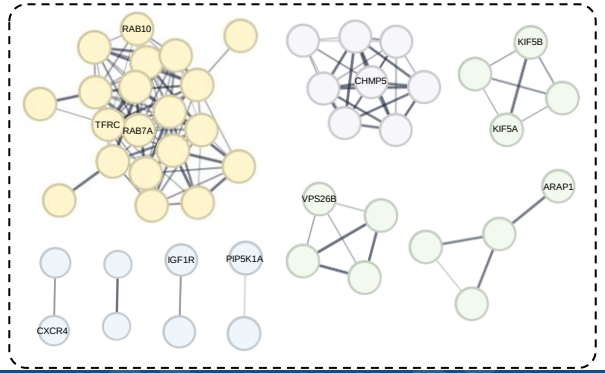


Phosphorylation residues
Total: 3344

GO:0004672, **Protein kinase activity**, FDR: 3.36E-13

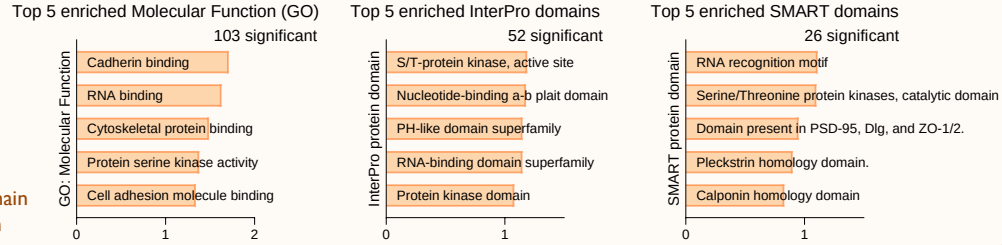


KEGG:hsa04144, **Endocytosis**, FDR: 3.74E-06

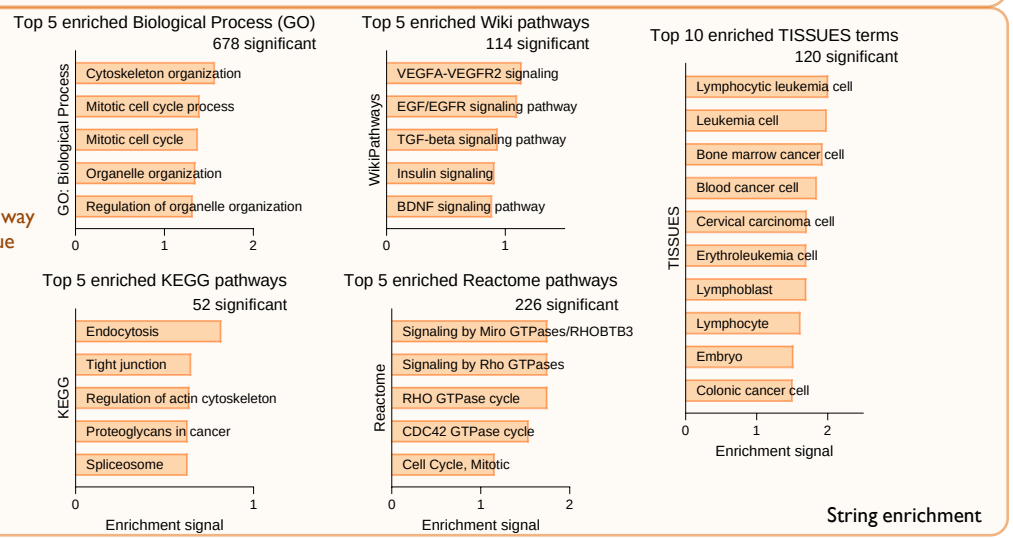


Top enrichment from PKMYT1 substrates

Protein domain and function



Protein pathway and cell/tissue



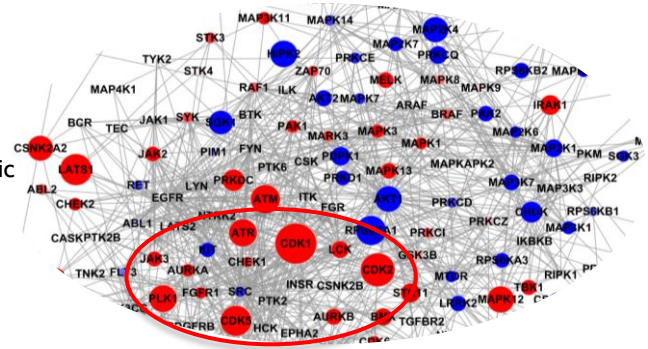
String enrichment

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

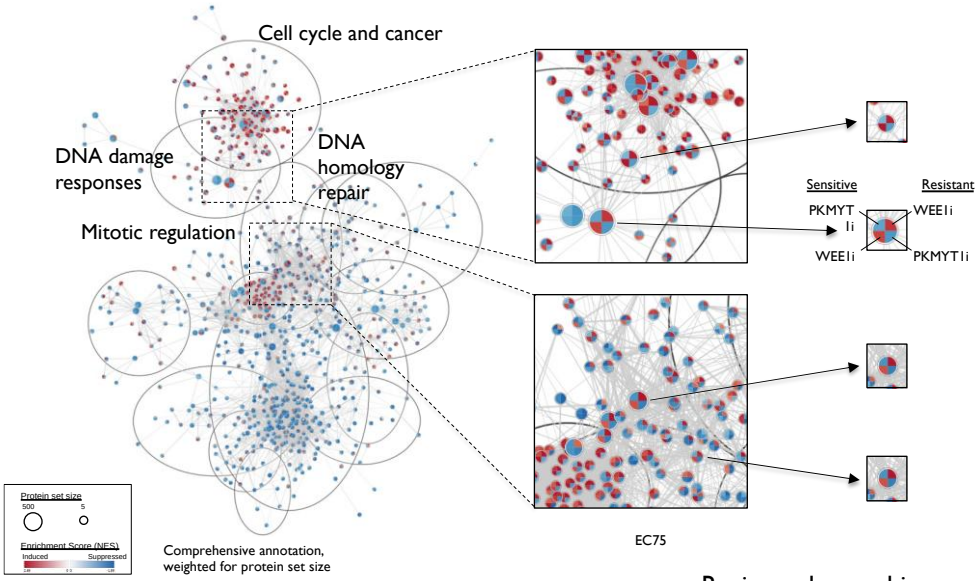
ACR-2316: Rationally designed, differentiated WEE1/PKMYT1 inhibitor for superior Therapeutic Index

- ✓ Strong anti-tumor efficacy with complete tumor regression across models
- ✓ High selectivity (co-crystallography) and short elimination T1/2 to ensure transient, mild AEs
- ✓ Potent WEE1 inhibition, balanced PKMYT1 inhibition, overcomes resistance and enables robust activation of CDK1, CDK2, and PLK1 for mitotic catastrophe
- ✓ Development in high unmet need solid tumor types predicted sensitive by AP3

Potent mitotic catastrophe

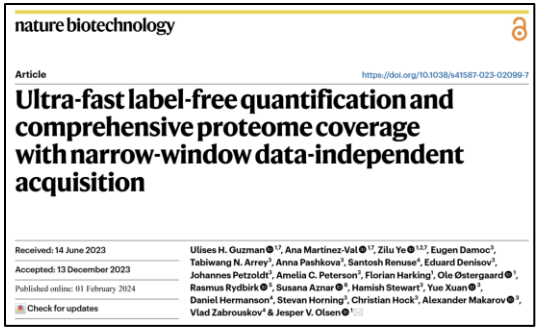


AP3-based proteome-wide SAR for desirable pathway effects and suppressing resistance mechanisms



Reciprocal quenching

SINGLE CELL PHOSPHOPROTEOMICS PIONEERED IN OLSEN LAB



nature biotechnology

Article

https://doi.org/10.1038/s41587-023-02099-7

Ultra-fast label-free quantification and comprehensive proteome coverage with narrow-window data-independent acquisition

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Check for updates

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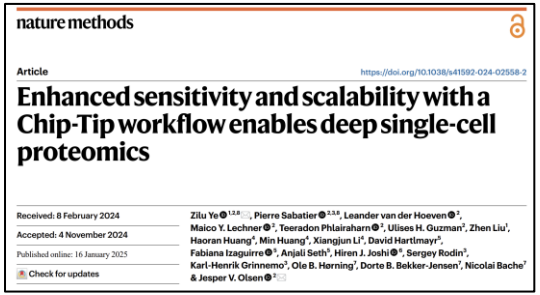
- Seminal article on revolutionizing and SCP-enabling Astral MS technology



nature methods

Article

https://doi.org/10.1038/s41592-024-02558-2

Enhanced sensitivity and scalability with a Chip-Tip workflow enables deep single-cell proteomics

Received: 8 February 2024

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Check for updates

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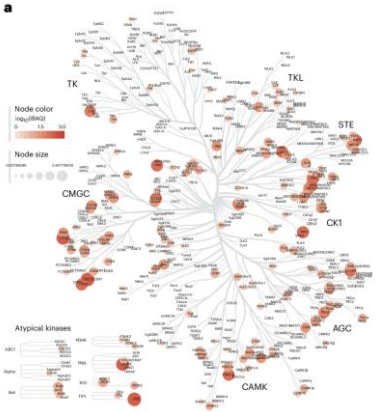
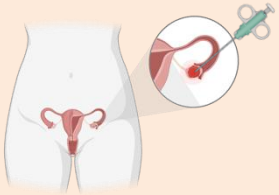
- >5,000 proteins in individual HeLa cells
- The first single cell phospho-proteome



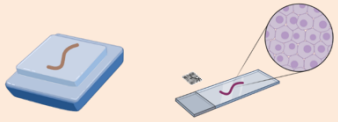
ACR-368 PHASE 2 UPDATE

ACR-368-201 TRIAL DESIGN

Pretreatment tumor biopsy



Routine FFPE processing



Biopsy in FFPE block

Automated tumor Region-of-Interest biomarker scoring

OncoSignature+

BM+

OncoSignature-

BM-

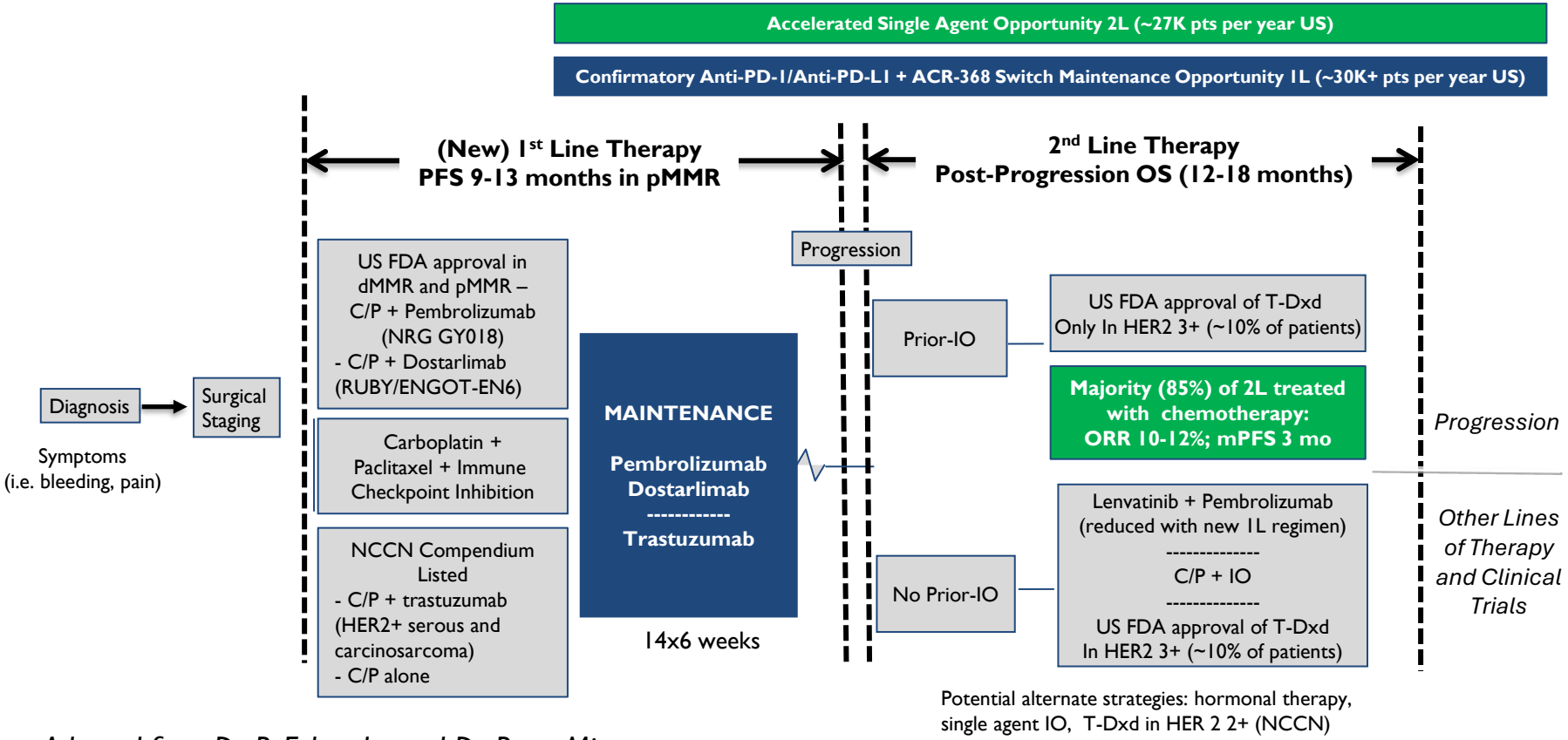
ACR-368 monotherapy

Single Arm Simon 2 Stage Monotherapy Phase 2b with Registrational Intent

ACR-368 + LDG combination

Single Arm Phase 1b/2 Exploratory Combination with Low Dose Gemcitabine

EVOLVING TREATMENT LANDSCAPE FOR THE MANAGEMENT OF ADVANCED STAGE OR RECURRENT ENDOMETRIAL CANCER



Adapted from Dr. R. Eskander and Dr. Raza Mirza

DEMOGRAPHICS AND BASELINE CHARACTERISTICS

Subject Demographics	BM+ N = 20	BM- N = 38
Median Age (range)	65.0 (54, 76)	67.5 (42, 80)
Race, n (%)		
White	14 (70)	22 (58)
Black/African American	4 (20)	9 (24)
Asian	1 (5)	5 (13)
Unknown/Other	1 (5)	2 (5)
Stage, n (%)		
III	4 (20)	14 (37)
IV	**16 (80)**	**22 (58)**
Unknown	0	2 (5)
Histology, n (%)		
Serous	**12 (60)**	**15 (40)**
Clear-cell carcinoma	0	4 (10)
Carcinosarcoma	**3 (15)**	**4 (10)**
Endometroid, G3	4 (20)	11 (29)
Other	1 (5)	4 (10)
ECOG Status at Baseline, n (%)		
0	10 (50)	18 (47)
1	10 (50)	20 (53)

Subject Demographics	BM+ N = 20	BM- N = 38
Median (Mean) Number of Prior Lines	**2 (2.6)**	**3 (3)**
Best Overall Response to Last Prior Line		
Refractory*	**12 (60)**	**19 (50)**
Relapsed disease	6 (30)	14 (37)
Unknown	2 (10)	5 (13)
MMR Status, n (%)		
pMMR	**13 (65)**	**19 (50)**
dMMR	2 (10)	9 (24)
Unknown	5 (25)	10 (26)
TP53 Status, n (%)		
Mutant	**11 (55)**	**12 (32)**
Wildtype	3 (15)	7 (18)
Unknown	6 (30)	19 (50)
Prior exposure to PD-1/PD-L1, n (%)		
Yes	**20 (100)**	**36 (95)**
No	0	2 (5)
Prior exposure to Pembro/Len, n (%)		
Yes	**13 (65)**	**23 (61)**
No	7 (35)	15 (39)

EDC data extract Feb 25, 2025. *Best overall response to last prior line = PD

DISPOSITION

Subject Disposition	BM+	BM-
Received at least 1 dose*	23	44
Efficacy evaluable (EE) population, n (%)**	20 (100)	38 (100)
Efficacy-Evaluable Still on Treatment, n (%)	5 (25)	7 (18)
Reason for Discontinuing Treatment, n (%)		
PD	10 (50)	28 (74)
PI Decision	2 (10)	0
Unacceptable Tox	1 (5)	2 (5)
Subject Decision	2 (10)	1 (3)
Discontinued Study, n (%)	7 (35)	17 (45)
Reason for Discontinuing Study, n (%)		
Death	6 (30)	15 (40)
Unknown/Other	1 (5)	0
Lost to follow-up/subject withdrew consent	0	2 (5)

EDC data extract Feb 25, 2025. BM- includes all subjects treated with ACR-368 + ultra-low dose gemcitabine (ULDG) at RP2D (105 mg/m^2 and 10 mg/m^2, respectively)
*Patients enrolled with locked OncoSignature; **EE population includes subjects enrolled, that either had a post-baseline scan or discontinued treatment due to progressive disease (PD)

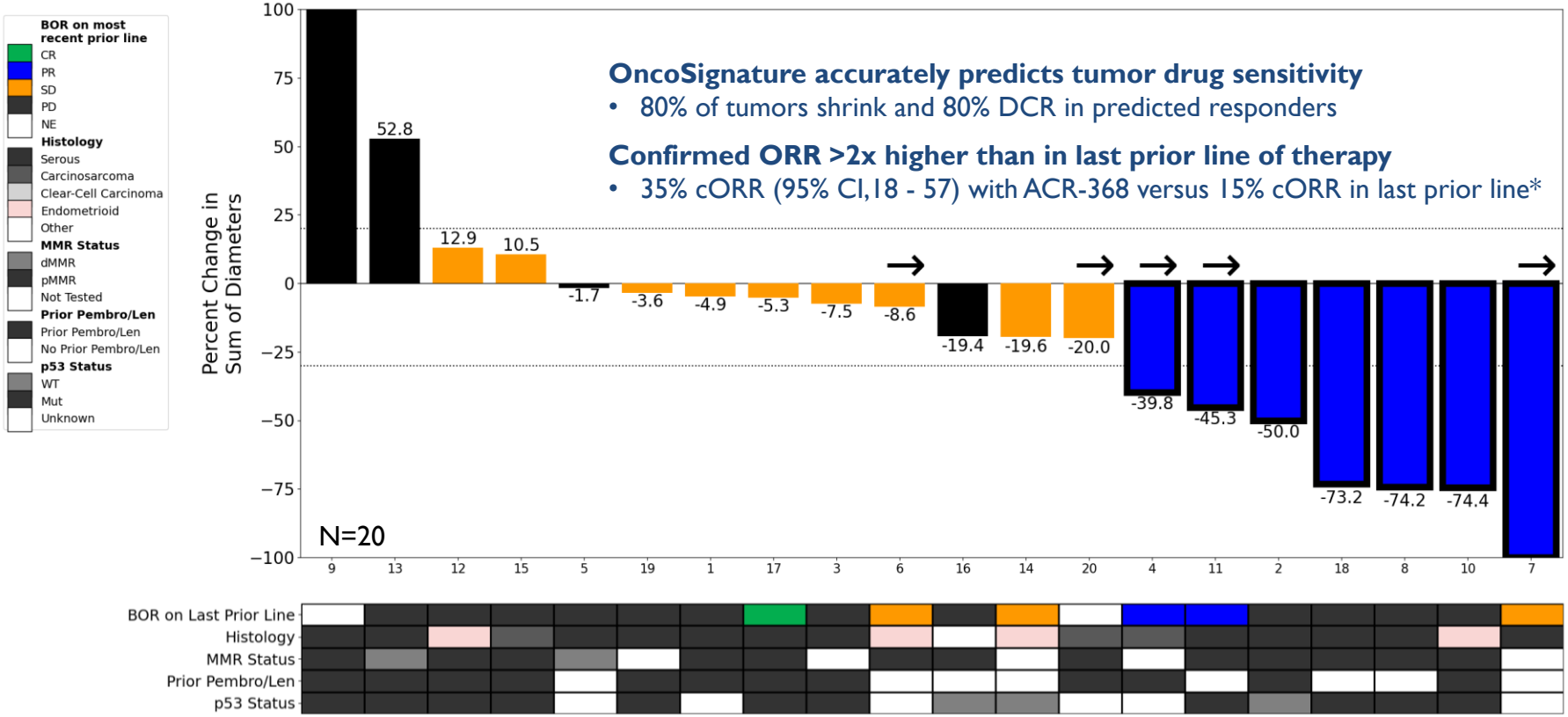
ENCOURAGING SAFETY PROFILE

- **Limited, transient, reversible, mechanism-based hematological AEs, which typically occurred during the first 1-2 cycles of therapy**
- **Notable absence of GI toxicities, long-lasting myelosuppression or the typical more severe non-hematological AEs commonly seen with ADCs and chemotherapy**

Treatment-Related Adverse Events, n (%)	BM+ (N=26)		BM- (N=51)	
	All Grades	Grades 3/4	All Grades	Grades 3/4
Thrombocytopenia	14 (54)	4 (15)	30 (59)	18 (35)
Anemia	13 (50)	7 (27)	29 (57)	23 (45)
Neutropenia	8 (31)	7 (27)	14 (28)	12 (24)
Leukopenia	5 (19)	3 (12)	11 (22)	10 (20)
Lymphopenia	2 (8)	0	5 (10)	3 (6)
Febrile neutropenia	1 (4)	1 (4)	4 (8)	4 (8)
Acute kidney injury	2 (8)	2 (8)	0	0

TRAEs with Grades 3/4 percentages ≥5% for either group are included in this table. No fatal TRAE in either group.
G-CSF is encouraged for ACR-368 monotherapy and mandated for the combination of ACR-368 + LDG

BEST OVERALL RESPONSE* – ENDOMETRIAL BM+ PATIENTS



OncoSignature accurately predicts tumor drug sensitivity

- 80% of tumors shrink and 80% DCR in predicted responders

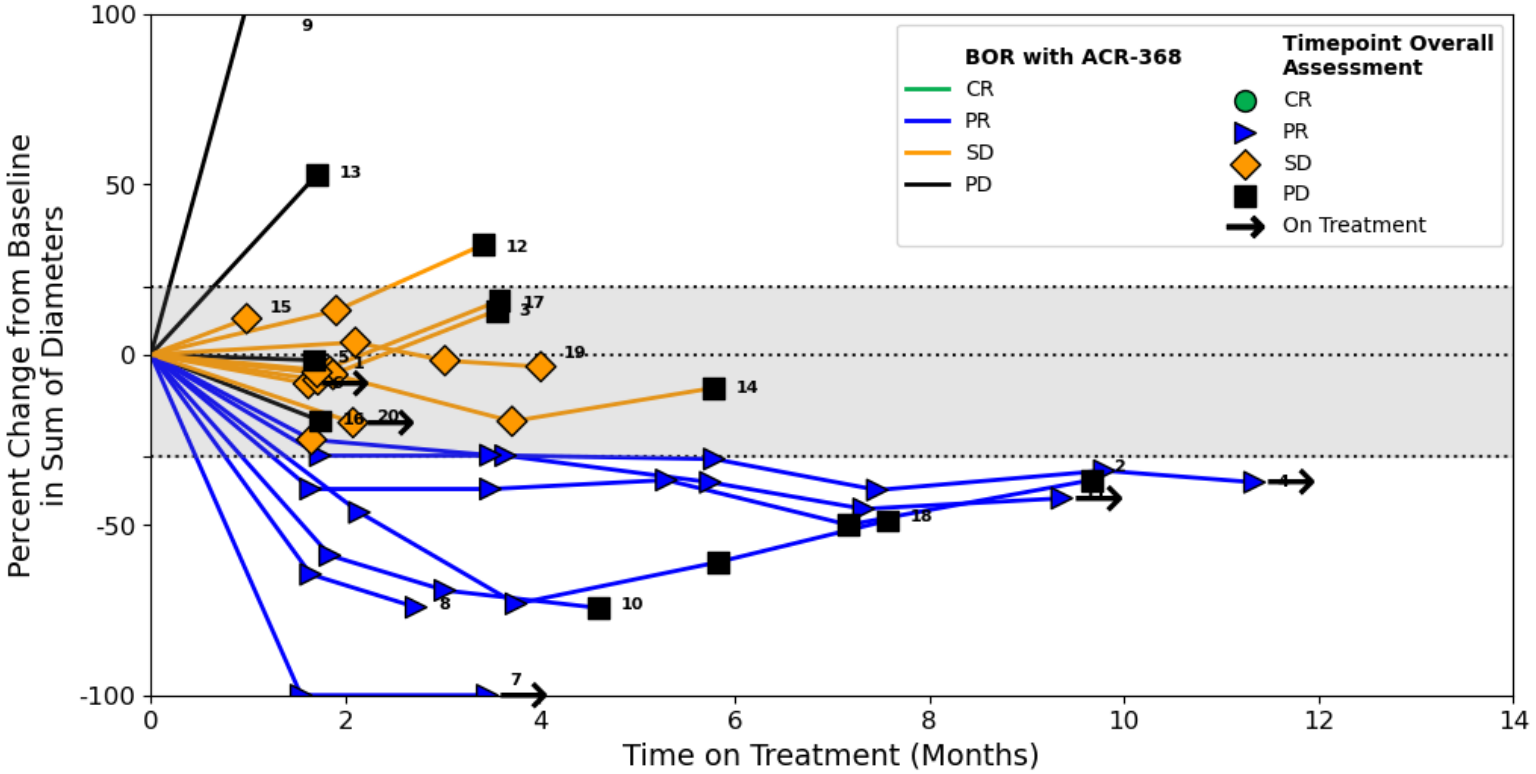
Confirmed ORR >2x higher than in last prior line of therapy

- 35% cORR (95% CI,18 - 57) with ACR-368 versus 15% cORR in last prior line*

*BOR of either BICR or INV

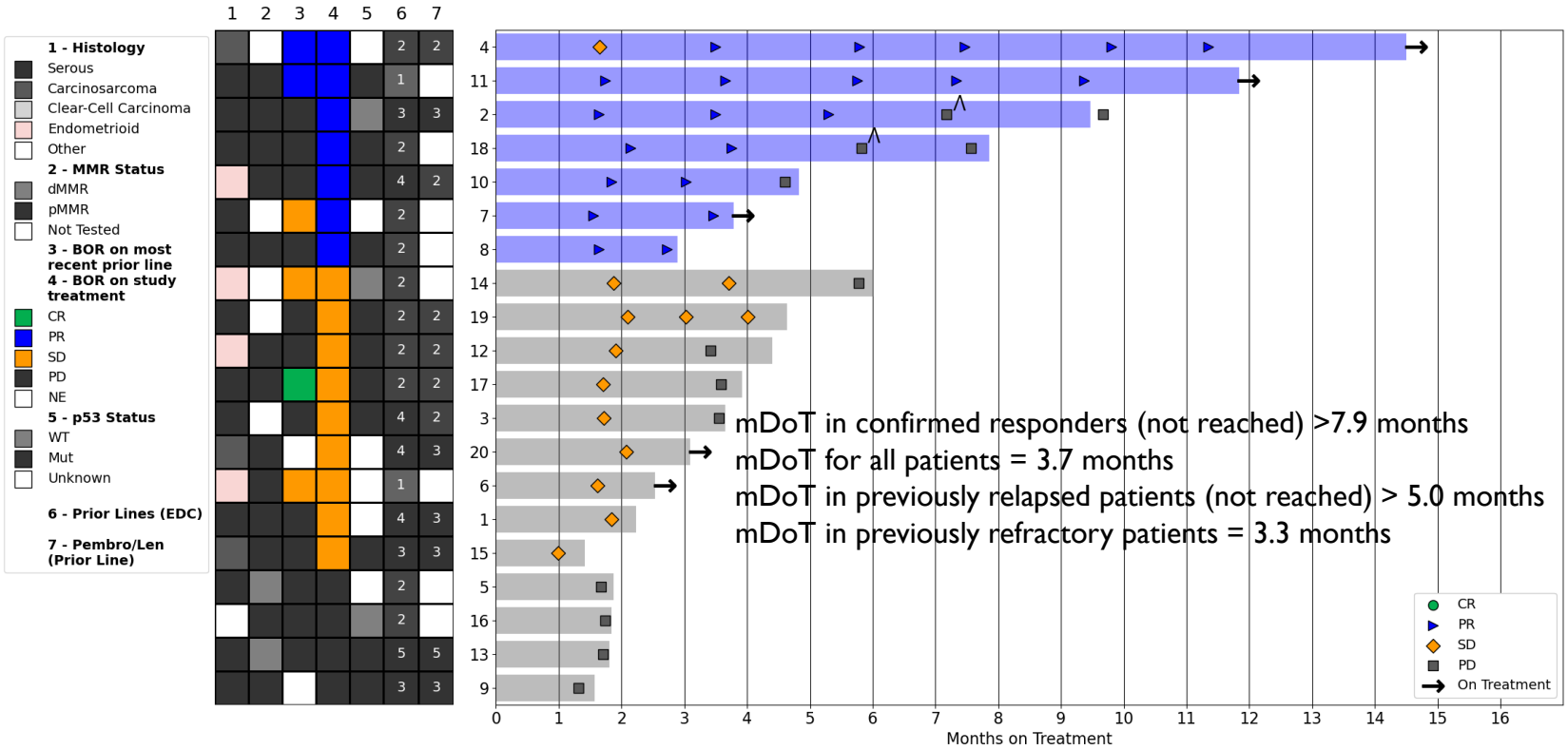
** cORR of ACR-368 is 39% versus 17% in the last prior line for the patients (N =18) with known BOR in last prior line

SPIDER PLOT – BM+ PATIENTS



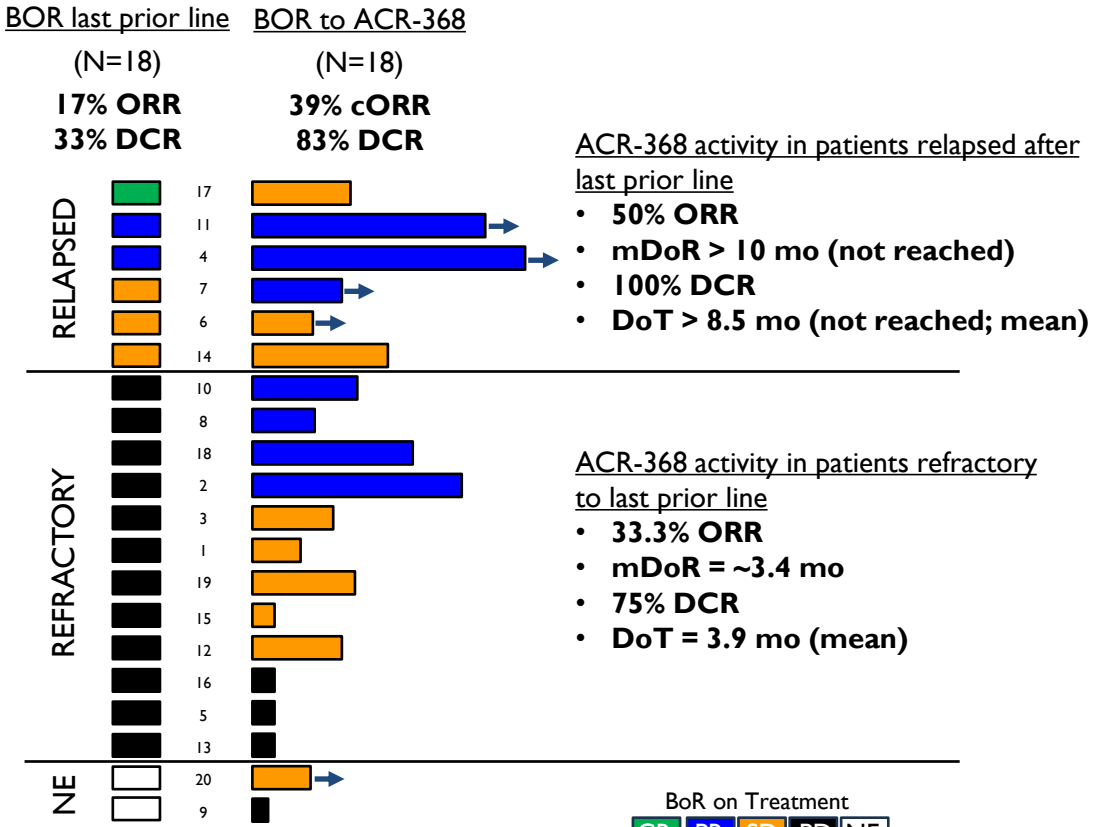
Spider plots based on last RECIST imaging for each patient

SWIMMER PLOT – BM+ ENDOMETRIAL CANCER PATIENTS



^ Patient #2 had PR per INV and Patient #18 had SD per BICR at second to last imaging

ACR-368 ACTIVITY COMPARED TO LAST PRIOR LINE (N=18*)



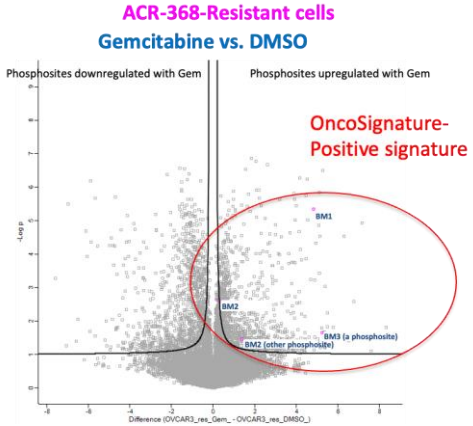
*18 out of 20 annotated for BOR in last prior line

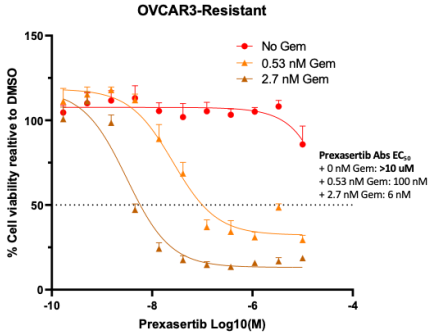
BM+ Key Take Home Messages

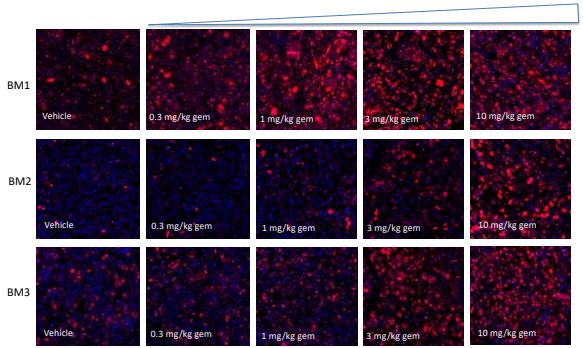
- All patients have progressed on prior platinum-based chemo and prior anti-PD-1
- Median/mean of prior lines = 2/2.6
- **66% of patients refractory to last prior line, 33% of patients relapsed on last prior line**
- **The ORR is >2x higher with ACR-368 in current line than ORR in the previous line**
- **ACR-368 is active even in refractory patients (33% cORR and 75% DCR)**
- Long durability with ACR-368 in relapsed patients (4/6 still on treatment)
- ACR-368 may be optimally positioned in patients who were not refractory to last prior line of therapy

AP3-PREDICTED SENSITIZATION TO ACR-368 BY LDG CORRELATES WITH ONCOSIGNATURE UPREGULATION

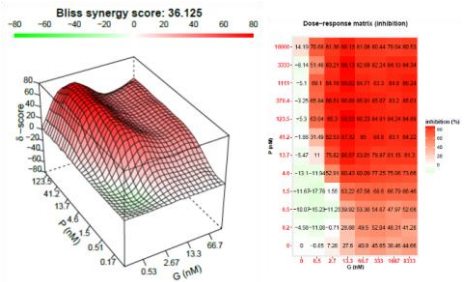
Preclinical data



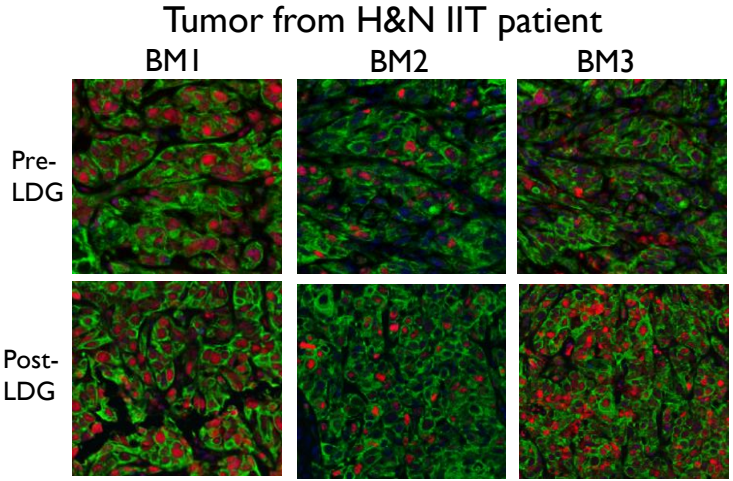


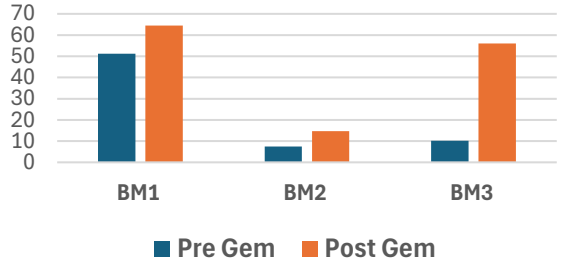


A427 human lung tumor xenograft

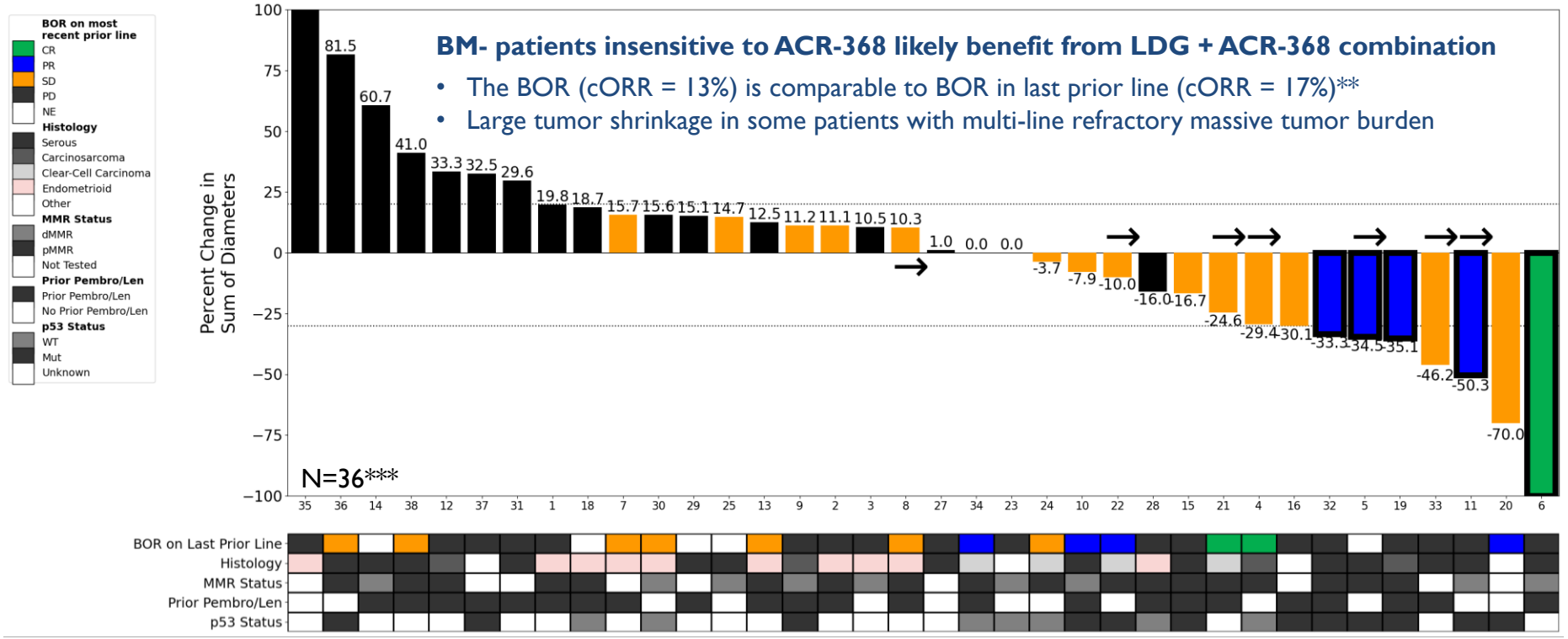


Clinical data





BEST OVERALL RESPONSE* – ENDOMETRIAL BM- PATIENTS



*BOR of either BICR or INV

** cORR of ACR-368 is 16% versus 19% in the last prior line for the patients (N =31) with known BOR in last prior line

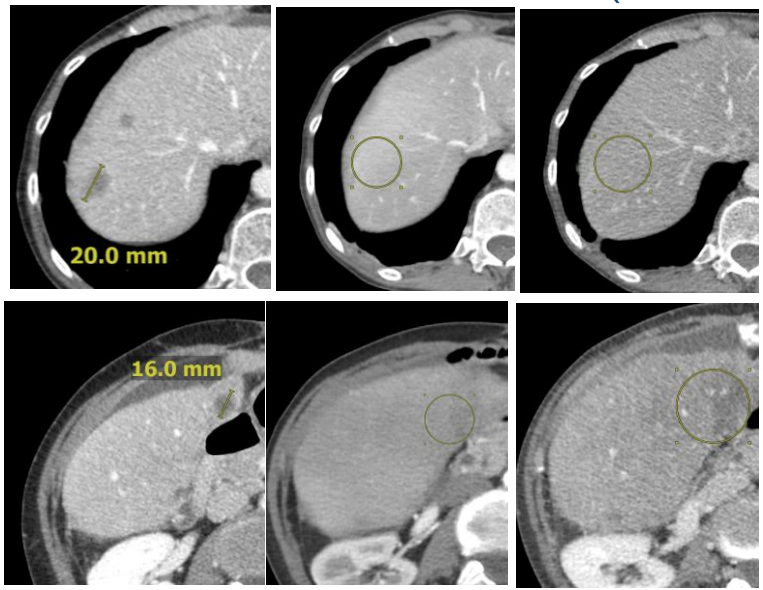
*** 2 patients had PD before first scan

CASE STORY 1: BM+ PATIENT; BOR = CONFIRMED PR

- 67-year-old female with stage IV **serous** endometrial adenocarcinoma
  - **pMMR**
  - **P53 mutant**
- Biopsy of a hepatic metastasis
  - OncoSignature **Positive**
- Tolerated treatment well with only one dosing delay due to mechanism-related reversible hematological AEs
- Patient was REFRACTORY to last prior line, including anti-PD-1

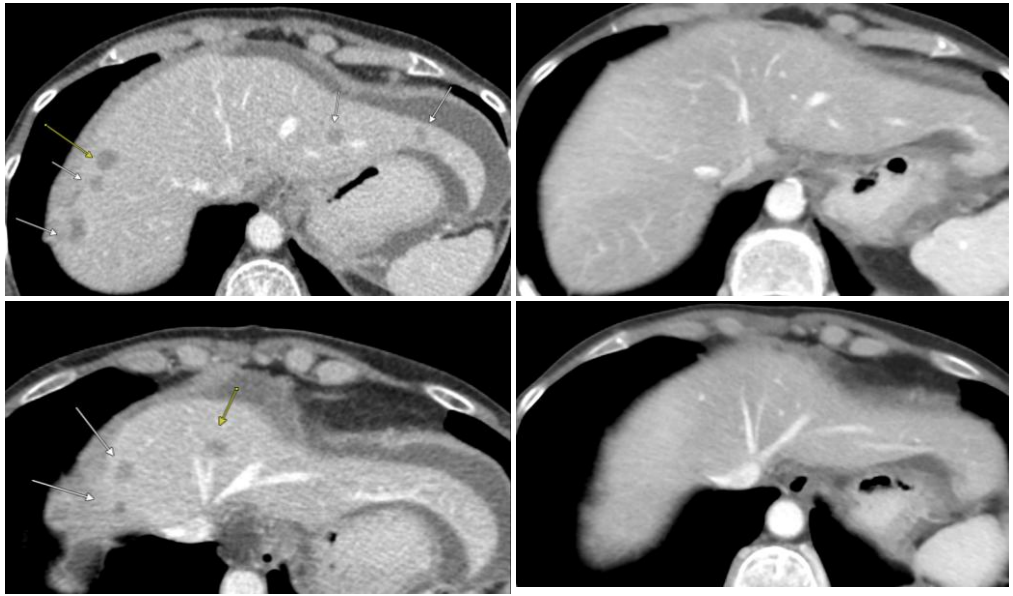
Tx	BOR
Carboplatin Taxol Dostarlimab	CR
Dostarlimab	**PD**

CASE STORY 1 (BM+ PATIENT)



Screening | Week 8 (-100%) | Week 12 (-100%)

Complete resolution of hepatic target lesions



Screening | Week 8 and 12 (-100%)

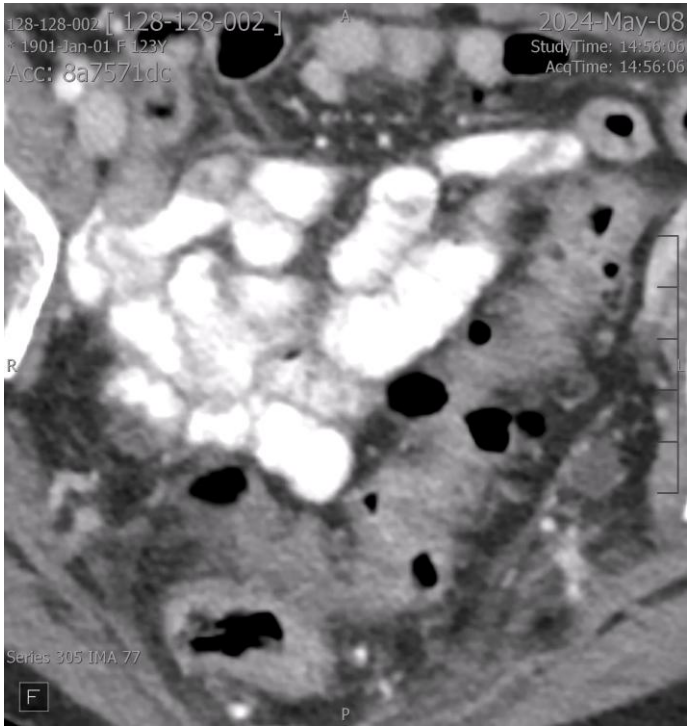
- Confirmed **PR** with **complete** disappearance of *more than 10* additional hepatic metastases spanning both lobes and all hepatic segments
- Demonstrates ACR-368's ability to **fully eradicate** multifocal hepatic disease, even in *high-disease burden* cases

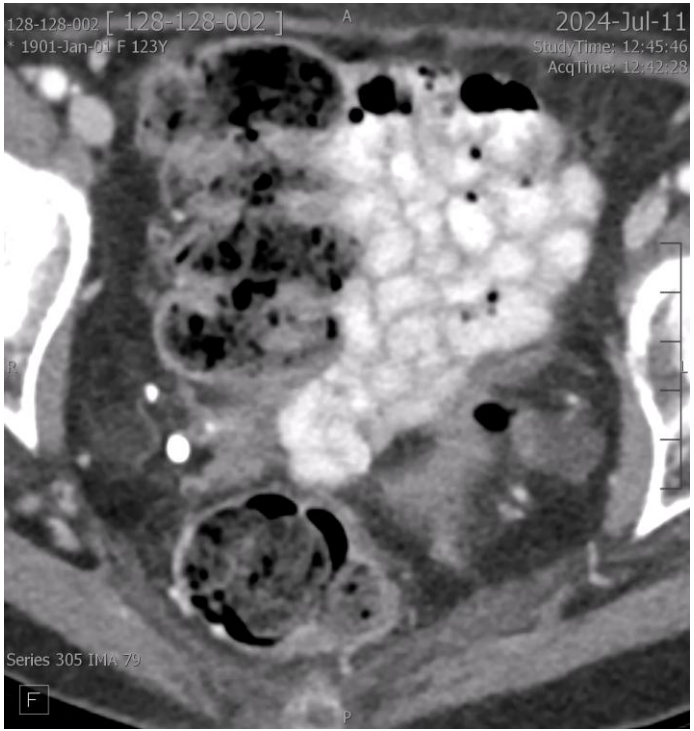
CASE STORY 2: BM+ PATIENT; BOR = SD

- 67-year-old female with stage III **endometroid** endometrial adenocarcinoma
  - **pMMR**
  - **P53 mutant**
- Biopsy of a vaginal mass
  - OncoSignature **Positive**
- Dose reduction and delay due to primarily mechanism related reversible hematological AEs
- Patient was REFRACTORY to prior lines, including anti-PD-1

Tx	BOR
Carboplatin Taxol Pembrolizumab	**PD**
Pembrolizumab Lenvatinib	**PD**

CASE STORY 2 (BM+ PATIENT)





Large volumetric shrinkage (44%) yet not meeting RECIST criteria

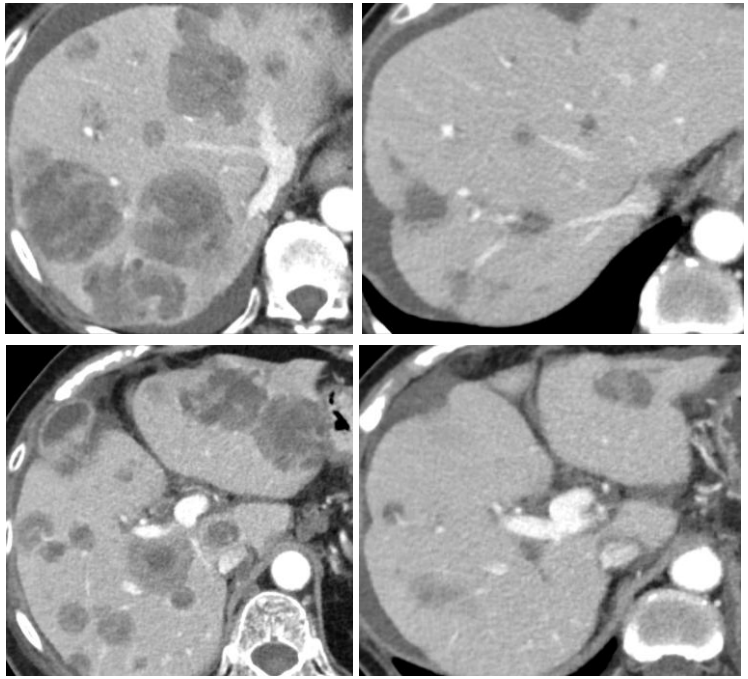
Screening	Week 8	
Longest Diameter: 4.8 cm	Longest Diameter: 4.6 cm	(-4.2%)
Volume: 37.2 cm^3	Volume 20.9 cm^3	**(-44%)**

CASE STORY 3: BM- PATIENT; BOR = CONFIRMED PR

- 73-year-old female with stage IV **serous** endometrial adenocarcinoma
  - **dMMR**
  - **P53 mutant**
- Biopsy of a hepatic metastasis
  - Oncosignature **Negative**
- Tolerated treatment well at reduced dosage with mechanism related reversible hematological AEs
- BOR in last prior line was PD

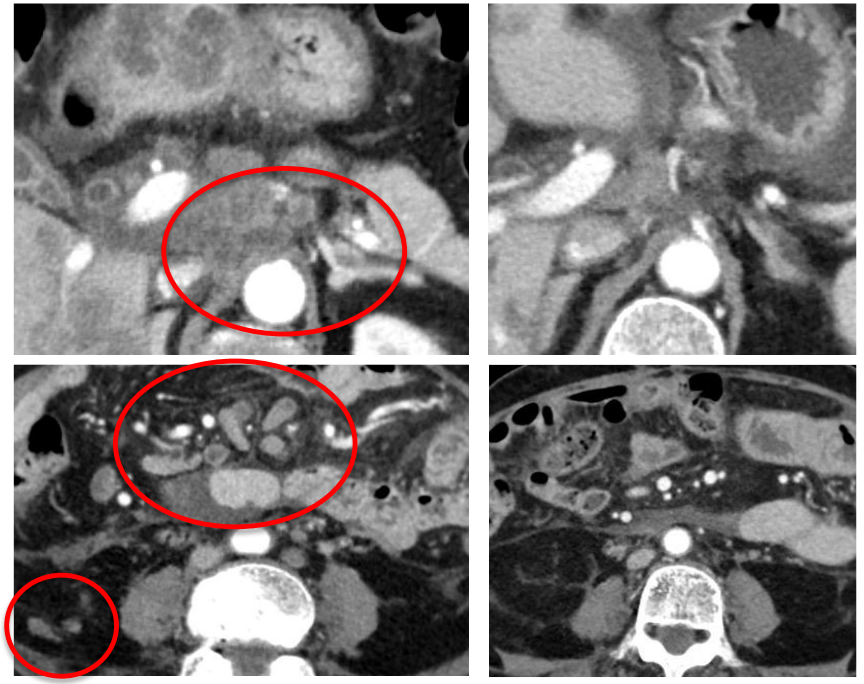
Tx	BOR
Carboplatin Taxol	PR
Pembrolizumab	**PD**

CASE STORY 3 (BM- PATIENT)



Screening Week 16

- ***>50*** hepatic metastases occupying 40% of liver
- Dramatic reduction in size and number
- Remaining areas consist of residual fibrosis and necrosis, indicating a strong treatment response



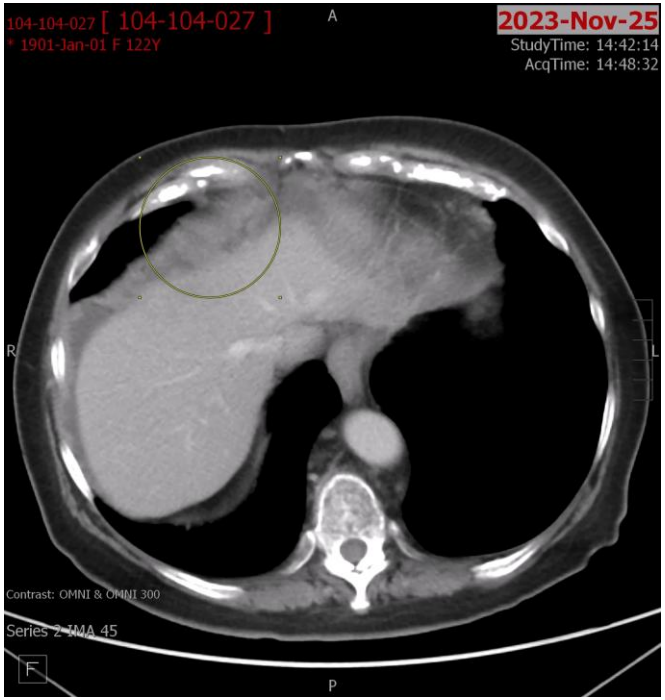
- Confirmed PR with equally strong regression in necrotic lymphadenopathy and peritoneal disease
- Supports LDG's role in sensitizing tumors to ACR-368, driving a robust treatment effect

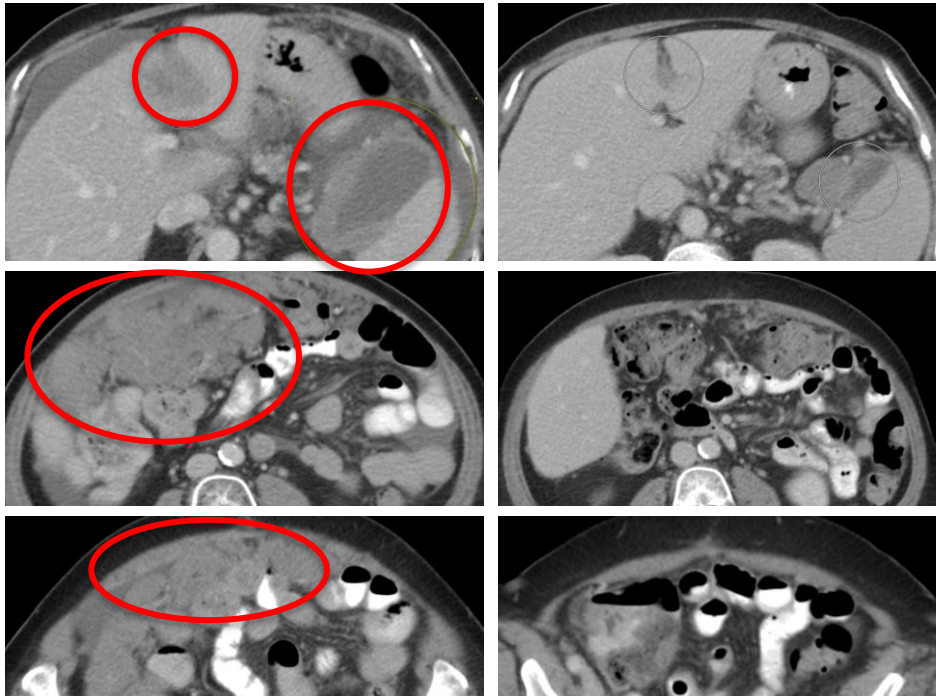
CASE STORY 4: BM- PATIENT; BOR = SD

- 69-year-old female with stage IV endometrial adenocarcinoma
  - **MMR status unknown**
  - **P53 status unknown**
- Biopsy of a peritoneal lesion
  - OncoSignature **Negative**
- Tolerated treatment well with dose reduction in last 2 doses and only mechanism related reversible hematological AEs
- Patient was REFRACTORY to prior lines, including anti-PD-1

Tx	BOR
Carboplatin Taxol	PD
Pembrolizumab Lenvatinib	PD
Doxil Bevacizumab	PD

CASE STORY 4 (BM- PATIENT)





- Massive peritoneal disease burden at Screening
- Volume of disease **1020 cm³**

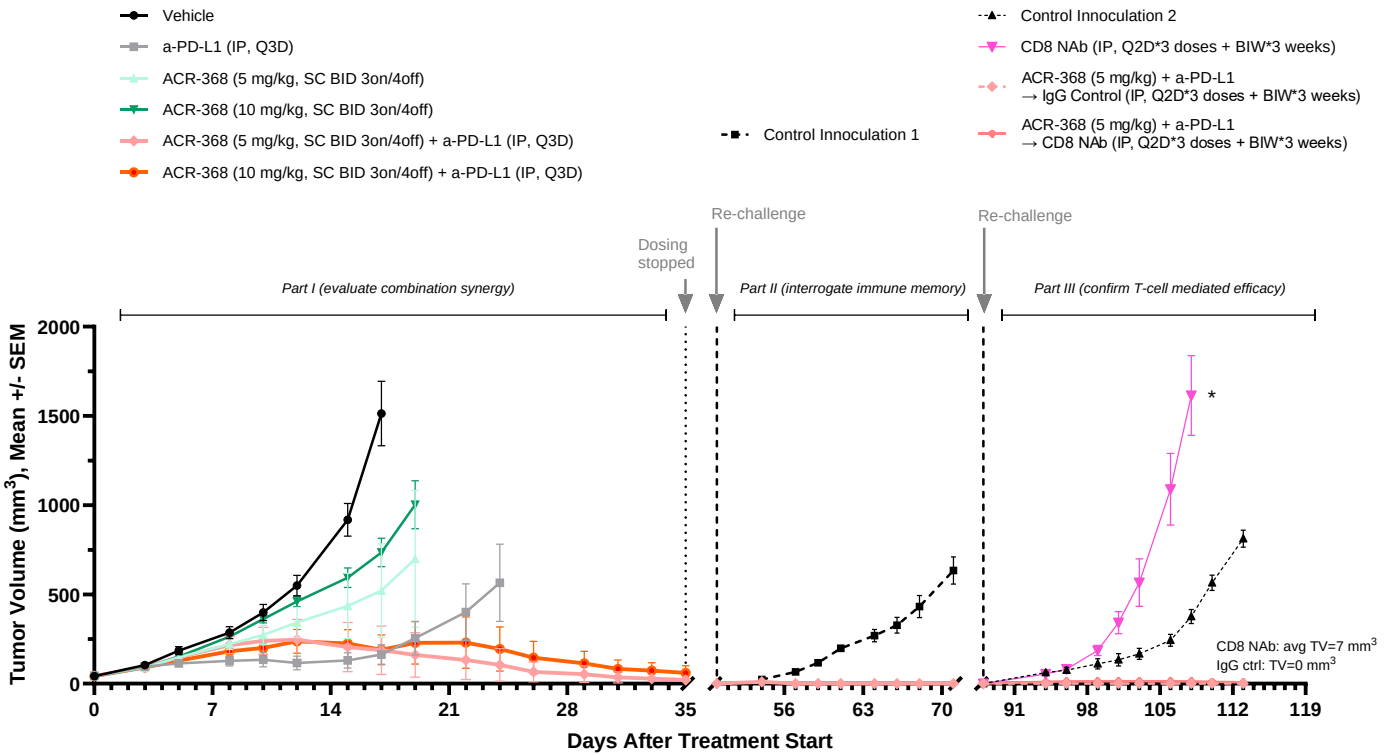
- Profound volumetric reduction in peritoneal disease (1020 cm³ to 168 cm³)
- RECIST (SD) failing to capture true extent of tumor shrinkage
- Strongly supports LDG's role in sensitizing tumors to ACR-368, enabling significant treatment response

FULL FOCUS ON ENDOMETRIAL CANCER; OVARIAN AND BLADDER CANCER BEING DEPRIORITIZED

- Based on our emerging positive clinical data, competitive positioning, and attractive commercial opportunity, endometrial cancer is prioritized as first potential registration opportunity
  - Tumor type predicted sensitive by AP3 Indication Finding prior to clinical entry
  - Breakthrough Device Designation (BDD) received January 21, 2025
  - Huge unmet need in 2nd line (ORR ~12% and PFS ~3 months)
  - ~27,000 est. new cases/year in 2nd line stage III/IV recurrent or locally advanced, all histopathologies*
  - Preclinical data show LDG sensitization also in BM+ tumors opening for potential 2nd line all-comer ACR-368 + LDG
- **Acrivon continues pursuing registrational intent for ACR-368 in ≥2nd line BM+ patients (post new frontline) and our confirmatory trial strategy (switch maintenance w/ anti-PD-1) in frontline, supported by strong preclinical data**
- Ovarian and bladder cancer deprioritized
  - Due to increased competition and small market opportunity, the clinical bar for ovarian cancer is high and based on our preliminary data after 23 BM+ patients it is not met
  - In bladder cancer, we observed a lower than predicted BM+ fraction resulting in challenging enrollment
- All clinical resources are now focused on ACR-368 in endometrial cancer and ACR-2316

* Blinded, proprietary third-party market research with endometrial KOLs conducted August-September 2024

POTENT SYNERGY OF ACR-368 WITH ANTI-PD-L1 DISPLAYS IMMUNE MEMORY IN 100% OF IMMUNOCOMPETENT MICE

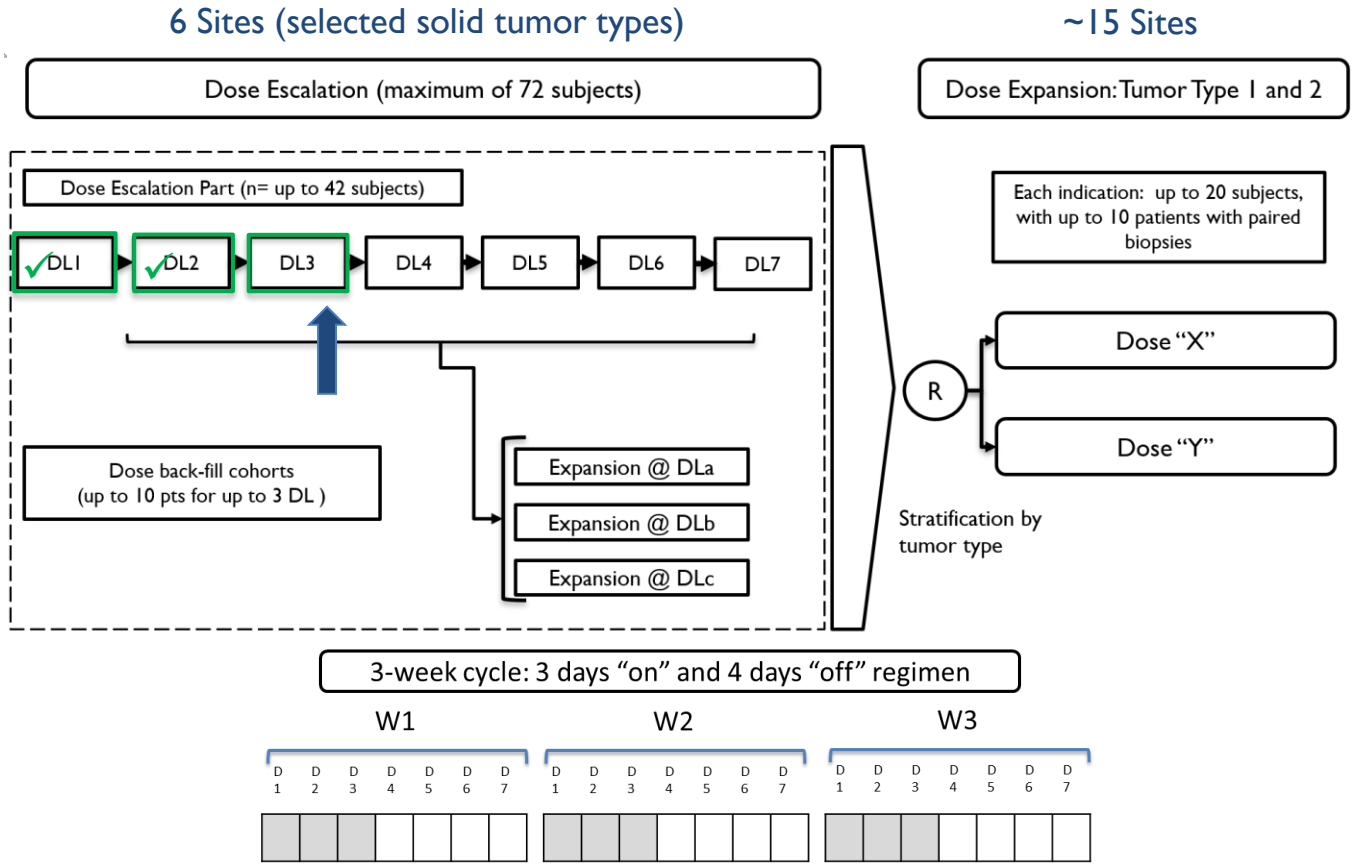


Average TV at start of treatment in Part I: 43 mm³ (N=8 mice/group)
7 CRs in Part I with combination treatment of ACR-368 + anti-PD-L1
7 CRs in Part II post-rechallenge with MC38 tumor cell inoculation (off-treatment)
3 CRs from Part II → received IgG control in Part III and continue to be CRs post-second rechallenge with MC38 tumor cell inoculation (off combo treatment)
4 CRs from Part II → received CD8Nab in Part III and have average TV of 7 mm³ on day 113 post-second rechallenge with MC38 tumor cell inoculation (off combo treatment)

**1 mouse euthanized on D106 due to ulceration and hypothermia, 1 mouse found dead on D110*

ACR-2316 PHASE 1 UPDATE

PHASE I DOSE ESCALATION AND EXPANSION DESIGN



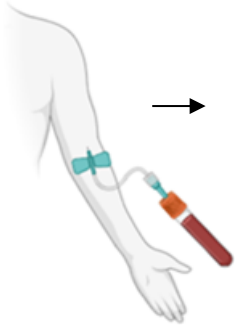
ACR-2316 PROGRAM STATUS

- Dose level (DL) 1 and DL2 cleared without safety concerns or DLTs by the safety review committee
- Preliminary PK data indicate approximate dose proportionality in the first 2 DL cohorts
- Significant drug target engagement observed in human PBMCs already at DL1 using our proprietary AP3 MS PD method
- Development focused on selected high unmet need solid tumor types (beyond endometrial and ovarian cancer) predicted sensitive by AP3 Indication Finding
- DL3 fully enrolled and DLT period anticipated completed by April 1
- Initial clinical activity observed in DL3
  - Prior chemotherapy and anti-PD-1; 3 prior lines of therapy
  - Baseline tumor burden: 3 index lesions each between 3 to 4.4 cm
  - Significant decreased size of metastatic lesions throughout the chest, abdomen and pelvis
  - Shrinkage (% RECIST) on first scan = -23.07%
  - Patient remains on therapy

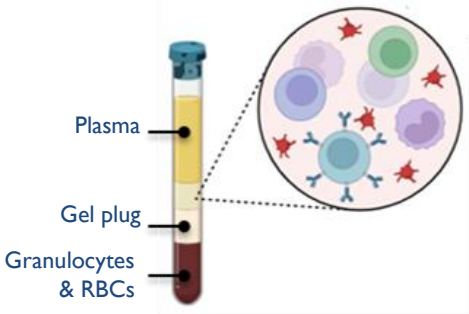
AP3 MS PHARMACODYNAMIC (PD) PBMC ASSAY ESTABLISHED TO SUPPORT ACR-2316 PHASE I STUDY

- PBMCs isolated from ACR-2316 Phase I trial patients
  - Non-invasive liquid biopsy for pharmacodynamic biomarkers
  - Exhaustive depth: ~8,000 proteins and ~ 20,000 phospho-sites
  - Rapid turn-around: ~ 1 week from sample shipment to data output
  - Quantitative measurement of drug PD effects

Blood draw

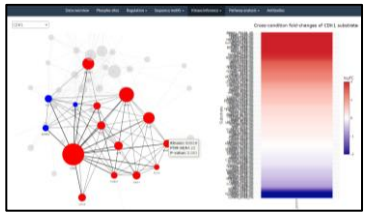


PBMC isolation



LC-MS analysis

Quantitative PD result

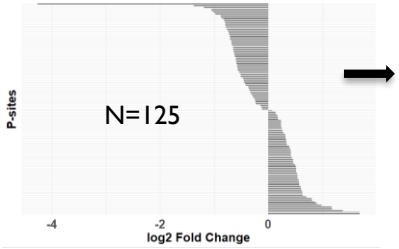


SIGNIFICANT DRUG TARGET ENGAGEMENT OBSERVED IN PBMCS ALREADY AT DOSE LEVEL 1

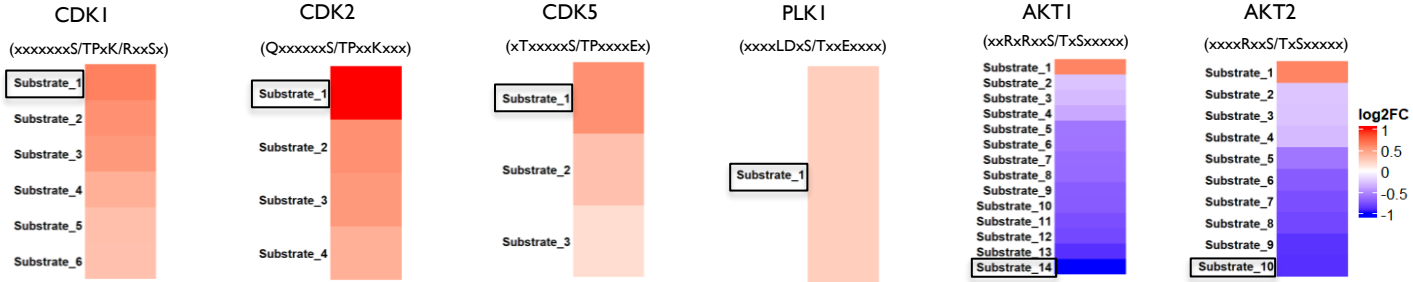
N=32196 Phosphosites identified from MS-proteomics

N=2229 Phosphosites measured in all samples (no imputation)

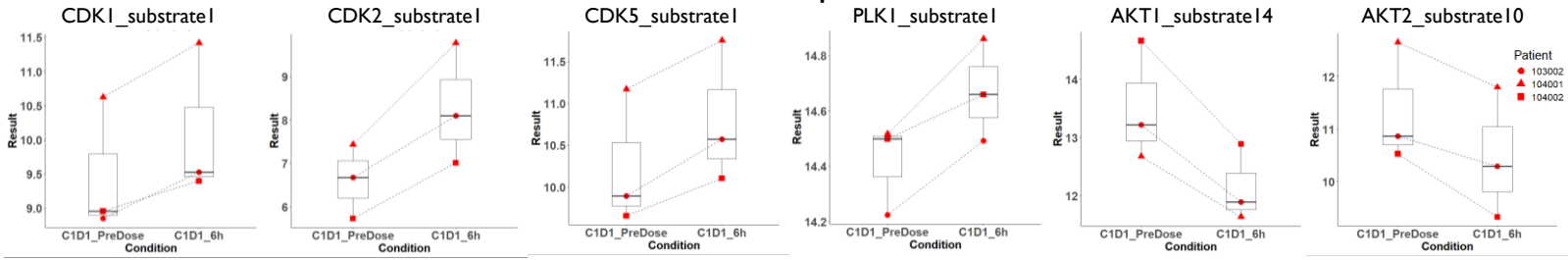
Drug-regulated P-sites (paired t-test : p-value < 0.05)



Drug-regulated substrates of key kinases (consensus motifs)



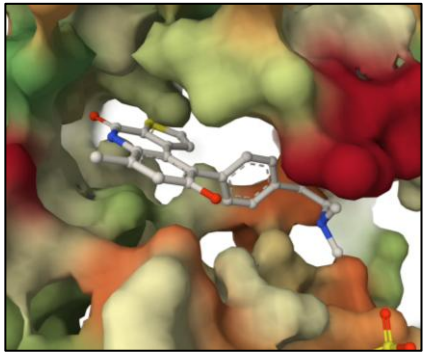
Pre-Dose vs Post-Dose of specific substrates



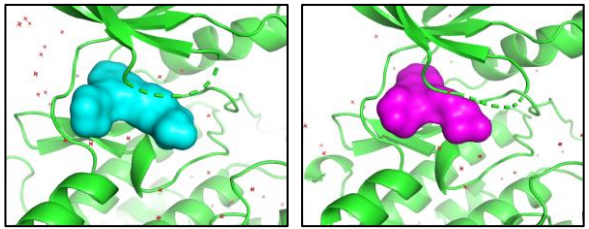
PRECLINICAL PROGRAM

PRECLINICAL CELL CYCLE PROGRAM (UNDISCLOSED TARGET)

- Target X – Attractive cancer drug target, no/minimal competitor programs, well-suited for AP3 platform
- DepMap data suggests 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
- AP3 profiling of benchmarks and lead compounds ongoing to enable MOA-based SAR
- Co-crystallography done for multiple lead series with compounds progressing towards in vivo profiling



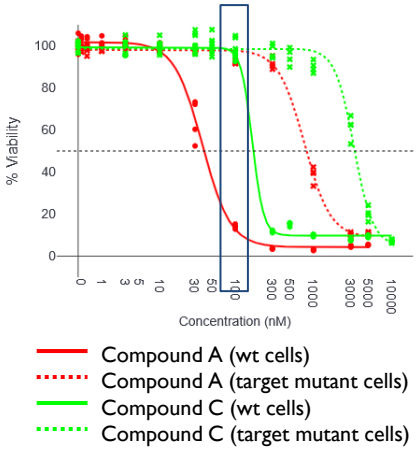
Tool compound is a selective target X inhibitor
(originally believed to be inhibitor for another target)



Series C
Resol. : 2.4 Å

Series D
Resol.: 2.64 Å

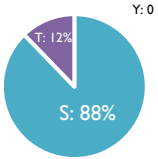
Development candidate 2025



AP3-PREDICTED TARGET X SUBSTRATES AND PATHWAYS

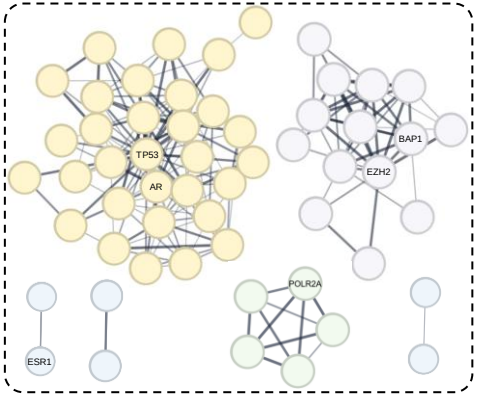
Clusters of selected Target X substrates

Target X is a protein Serine kinase

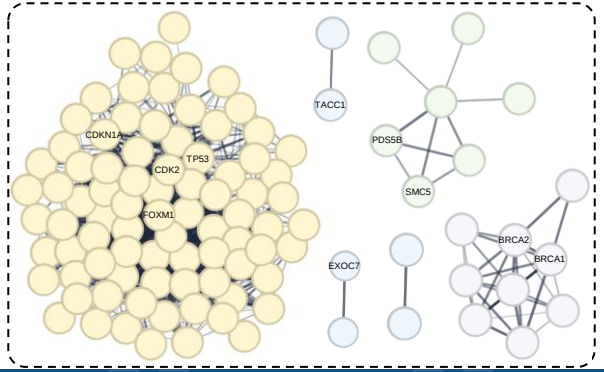


Phosphorylation residues Total: 3265

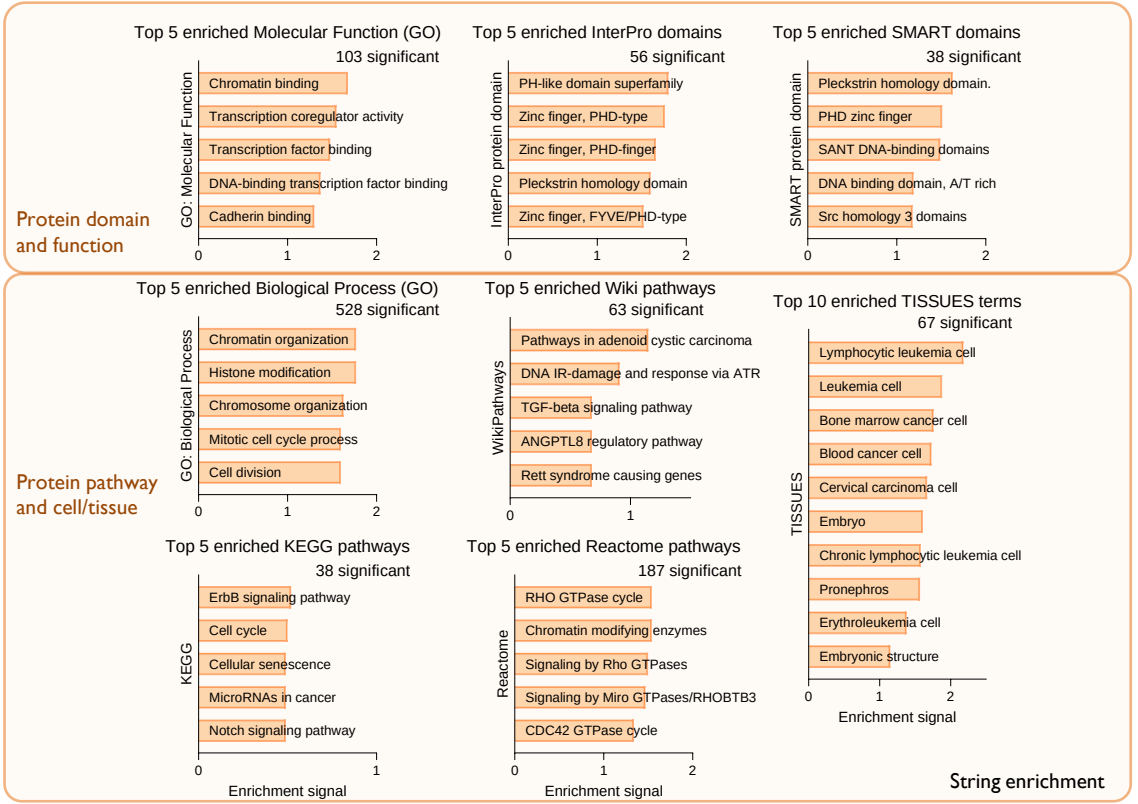
GO:0003682, **Chromatin binding**, FDR: 4.77E-24



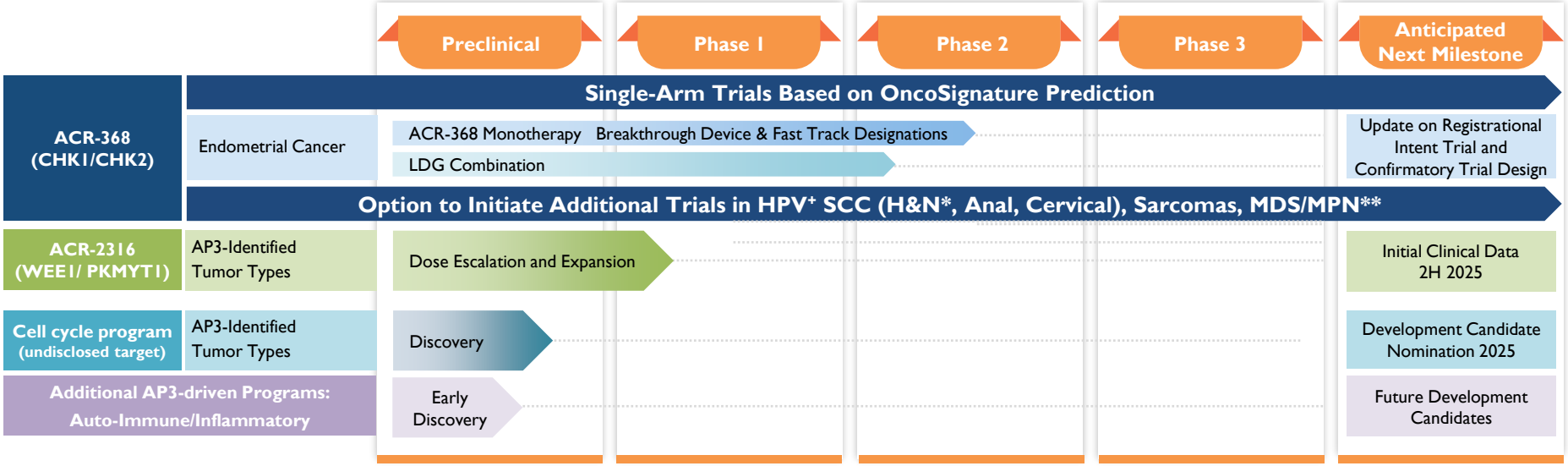
GO:1903047, **Mitotic cell cycle process**, FDR: 2.66E-21



Pathway enrichment of key Target X substrates



ACRIVON PIPELINE



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

*Investigator-Initiated Trial (IIT) activated at Moffitt Cancer Center
**Myelodysplastic syndrome and myeloproliferative neoplasms

FINANCIAL HIGHLIGHTS

Cash and Investments	Projected runway into	Fully Diluted Shares Outstanding
$184.6M	2027	43.8M
Balance sheet 31-Dec-2024	**Current operating plan, assuming no additional financing**	**Including shares, pre-funded warrants, and equity grants outstanding 31-Dec-2024**

Additional runway compared to previous guidance