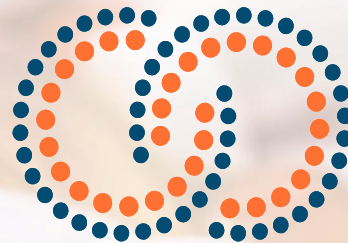


Acrivon

Therapeutics



ACRIVON PREDICTIVE PRECISION PROTEOMICS (AP3)
NEXT GENERATION PRECISION MEDICINE

CORPORATE PRESENTATION
SEPTEMBER 2026

FORWARD-LOOKING STATEMENTS

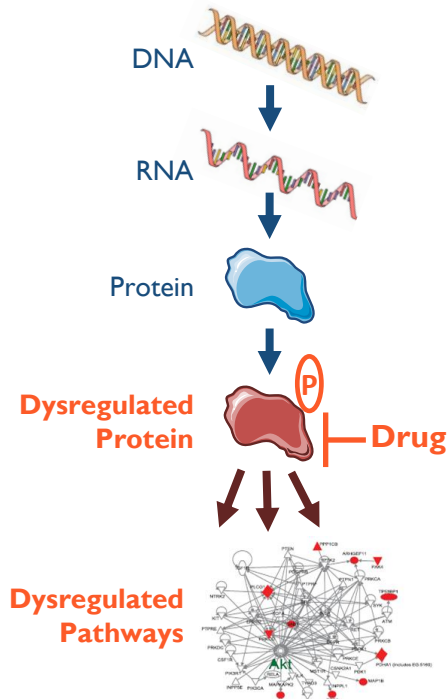
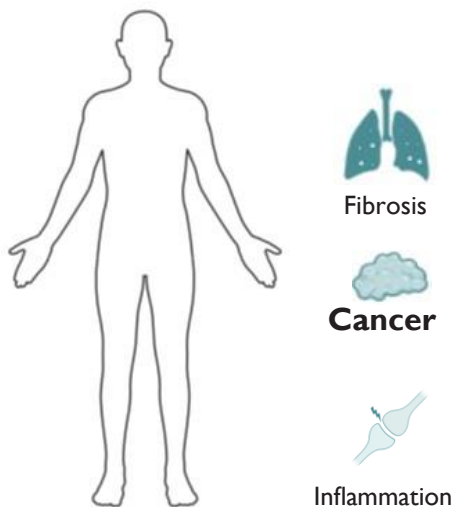
Certain information contained in this presentation includes forward-looking statements within the meaning of the Private Securities Litigation Reform Act of 1995 regarding our future results of operations or financial condition, business strategy and plans and objectives of management for future operations. 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.

Note: Acrivon intends to use its website as a means of disclosing material non-public information and for complying with its disclosure obligations under Regulation FD. For more information, please visit www.acrivon.com and follow the company on LinkedIn.

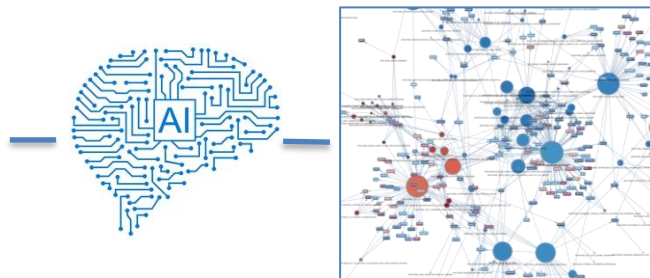
NEXT-GENERATION PRECISION MEDICINE POWERED BY AP3

Current focus oncology,
I&I next



Acrivon Predictive Precision Proteomics (AP3)

- AI-driven, proteomics-based rational drug design and informed clinical development
- AP3 enables an exact match between the disease-driving, dysregulated pathways with drug mechanism of action (Acrivon meaning $\approx$ exact, accurate)



Proprietary tools

- Data portal
- Interactome
- Protein activity predictor

ACRIVON THERAPEUTICS OVERVIEW

Development Site (Boston)

- Drug development and clinical biomarkers
- Clinical leadership and trial oversight
- ML/AI-driven AP3 analyses

HQ LOCATED IN BOSTON - ACCESS TO LEADING DRUG DISCOVERY, BIOTECH, AND PHARMA



Precision Proteomics Site (Lund/Copenhagen)

- Early drug discovery
- BM identification and drug profiling
- AP3 mass spectrometry

PROTEOMIC HUB LOCATED IN MEDICON VALLEY - NORTHERN EUROPE'S LEADING LIFE SCIENCE CLUSTER



Founders



Peter Blume-Jensen,
MD, PhD
CEO, President,
Co-Founder
Inventor of AP3



Kristina Masson
PhD, MBA
EVP, Bus Ops,
Lund Site Head
Co-Founder



Jesper V. Olsen
PhD
Novo-Nordisk Foundation
Protein Center, Cph.
Academic Co-Founder.

Founded 2018, IPO 2022 (NASDAQ:ACRV)

ACCOMPLISHED LEADERSHIP TEAM



Peter Blume-Jensen, M.D., Ph.D.
CEO, President, Co-Founder

- Executive Serono, Merck & Co., Daiichi Sankyo
- CSO Metamark - Marketed prostate proteomic test ProMark®
- Inventor Acrivon Predictive Precision Proteomics (AP3)



Mansoor Raza Mirza, M.D.
Chief Medical Officer

- World-renowned oncology KOL
- Lead investigator for multiple guideline-changing regulatory approvals, including PARP inhibitors for ovarian cancer and new frontline therapy for endometrial cancer



Kristina Masson, Ph.D., M.B.A.
Site Head Acrivon AB, Co-Founder
EVP Business Operations

- Cross-functional Leadership Merrimack Pharmaceuticals, MIT/BROAD
- Founder and CEO, OncoSignature AB (acquired by Acrivon Therapeutics)



Adam Levy, Ph.D., M.B.A.
Chief Financial Officer

- Zentaris Pharmaceuticals, Turning Point Therapeutics, Novartis
- Head of Corporate Strategy Gilead Sciences
- Engagement manager in pharma practice at McKinsey & Company



Eric Devroe, Ph.D.
Chief Operating Officer

- Founder and CEO, Opsonix
- Business executive MD Anderson Cancer Center and Metamark
- EIR Wyss Institute, Harvard
- Associate, Flagship Pioneering



Erick Gamelin, M.D., Ph.D.
Chief Development Officer

- Professor, CEO, national cancer center, appointed by French Minister of Health
- Executive Amgen, Pfizer, Dynavax, MacroGenics; CMO STEP Pharma
- >100 ph I-3 oncology trials

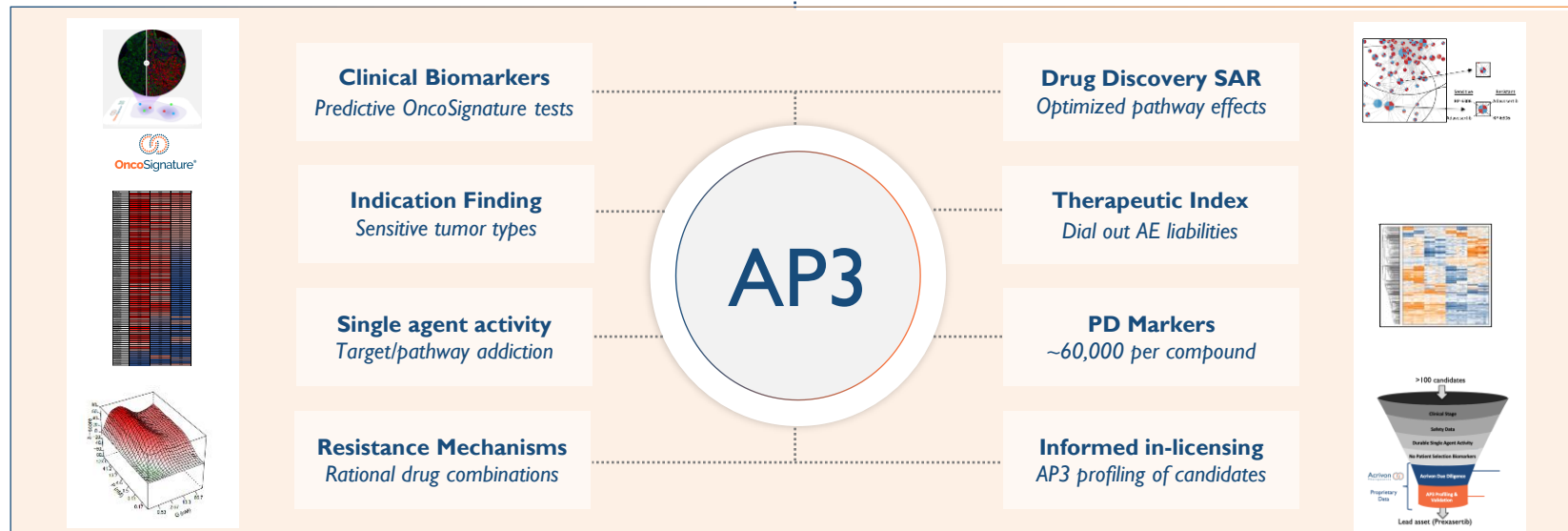


Mary-Alice Miller, J.D.
Chief Legal Officer

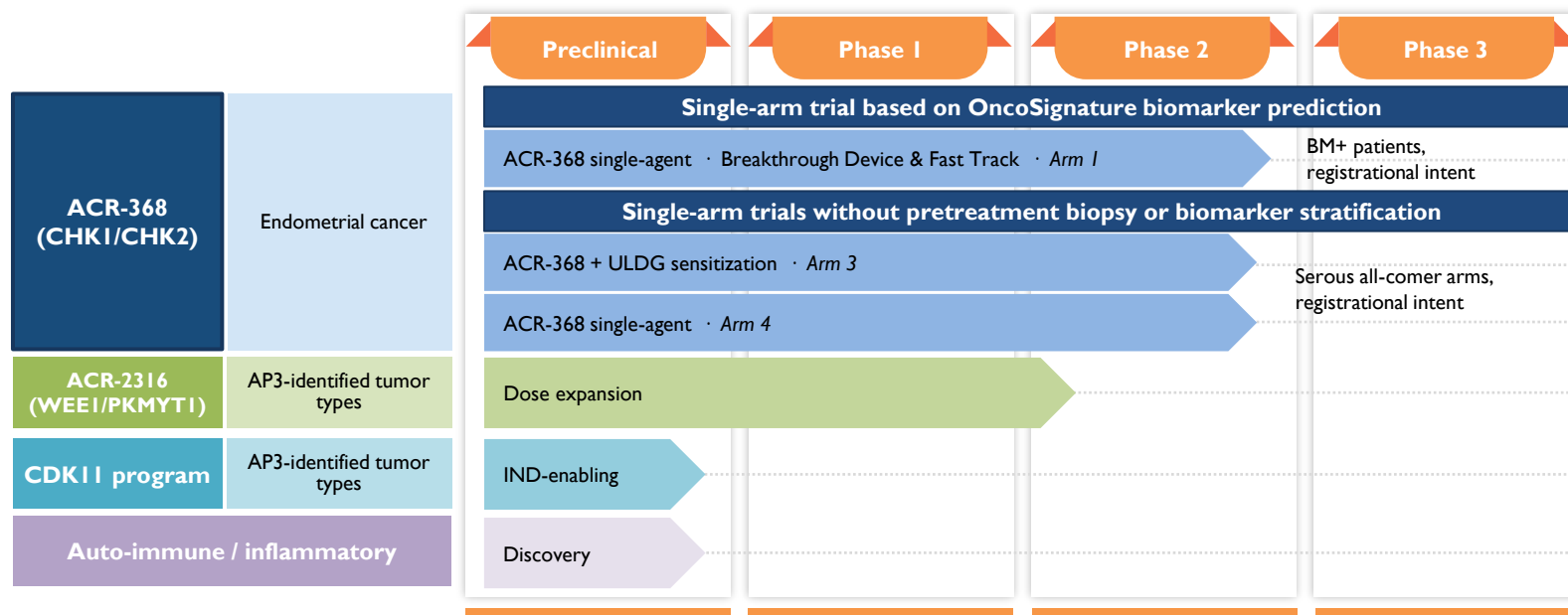
- Over 20 years corporate legal experience
- Served as general counsel of 2 companies taken public
- Boston Business Journal "40 Under 40"

ACRIVON AP3-BASED PRECISION MEDICINE

Novel Target Identification
Activated druggable targets – diseased vs normal



OUR PIPELINE



UPCOMING MILESTONES

ACR-368 (Phase 2b)

- Prespecified simultaneous interim analysis and data update from both all-comer (biopsy-independent) serous EC arms of the ACR-368 Phase 2b study in second half of 2026
- Initiate Phase 3 confirmatory trial for ACR-368 in first half of 2027
- Based on interim data read-out, complete enrollment of the registrational intent all-comer (biopsy-independent) serous EC Arm 3 or Arm 4 by fourth quarter of 2026

ACR-2316 (Phase I/2)

- ✓ Additional ACR-2316 Phase I/2 clinical data for weekly and bi-weekly dosing regimens and transition into dose expansion in AP3-identified tumor types in 2026

Earlier Pipeline

- Submit IND filing to the FDA for CDK11 inhibitor development candidate in first half of 2027
- Initiate additional internal programs utilizing the AP3 platform in 2026

ACRIVON CLIA LABORATORY

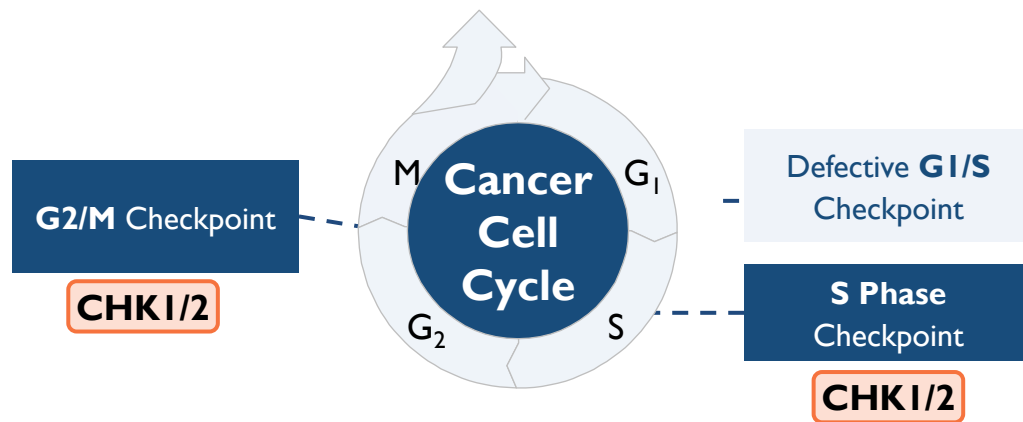
STRENGTHENING ACRIVON PRECISION MEDICINE CAPABILITIES

- Newly licensed CLIA laboratory
- Translates AP3-driven biomarker discoveries into actionable and proprietary CDx for our therapeutics
- Initially focused on ACR-368 OncoSignature testing
- Unique capabilities to support internal pipelines and new BD/partnering opportunities



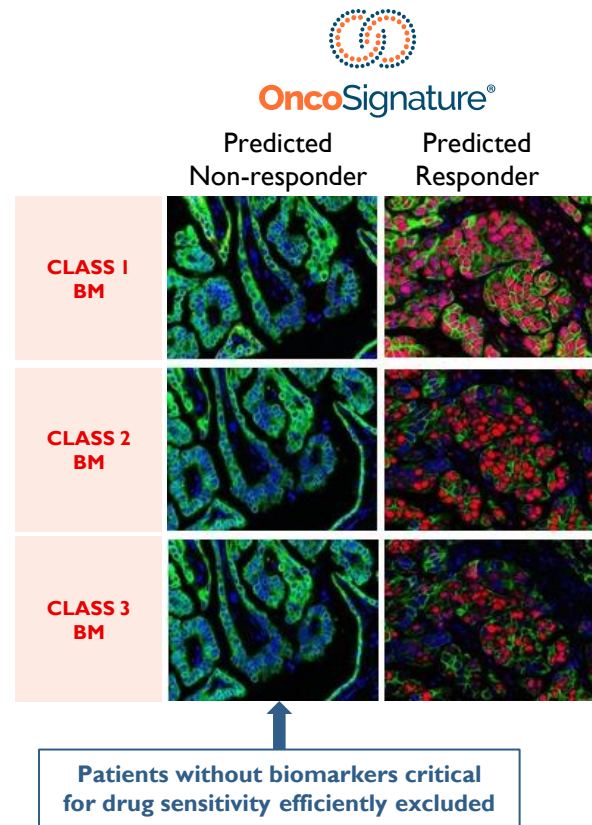
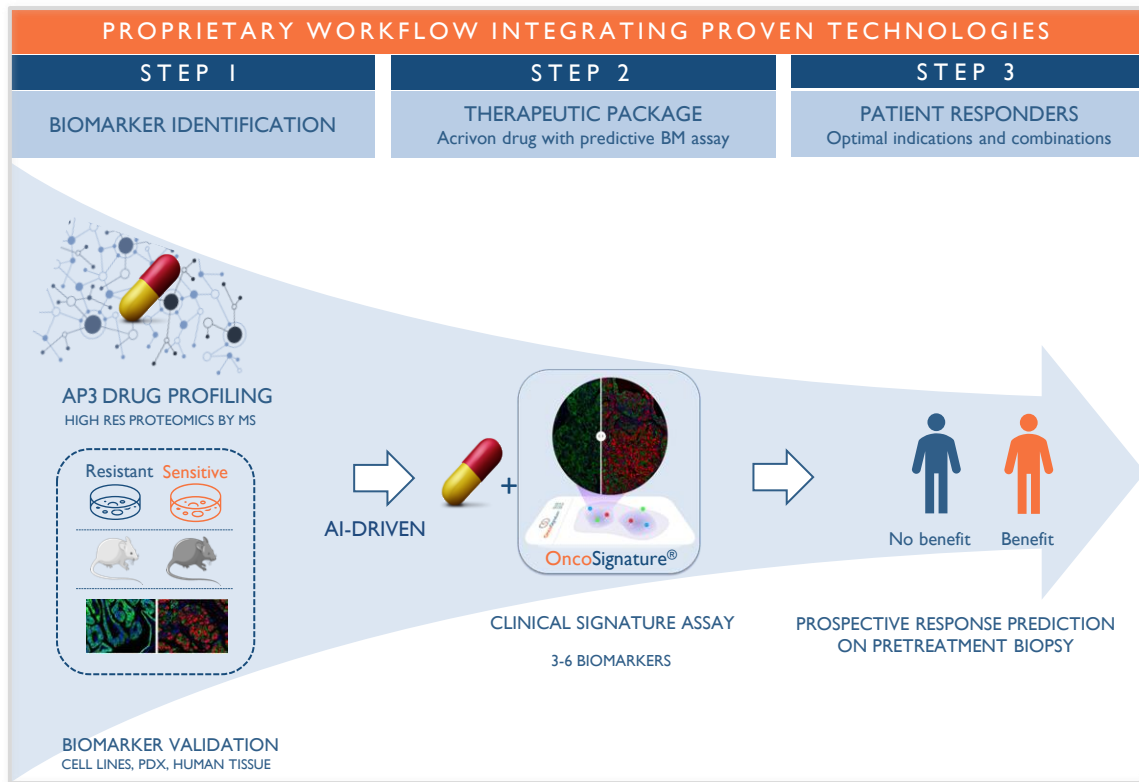
ACR-368: A POTENT SELECTIVE CHK1/2 INHIBITOR

- **Clinical efficacy and safety:** Durable monotherapy activity across multiple cancers with favorable tolerability and absence of non-hematological, severe toxicities
- **AP3 Indication Finding:** Endometrial cancer was identified as a tumor type predicted to be sensitive to ACR-368
- **Synergy with both immune checkpoint inhibitors and TOPOI inhibitors:** Strong preclinical data and rationale support potential for confirmatory Phase 3 combination studies



- **Potent inhibition:** Balanced potent (nM) inhibition of CHK1 and CHK2 enabling monotherapy activity
- **IP:** Protected by composition-of-matter and lactate form patents through 2030 and 2037, ensuring exclusivity

ONCOSIGNATURE: DRUG RESPONSE PREDICTION IN INDIVIDUAL PATIENTS



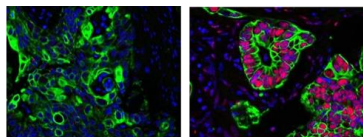
"Disease Pathway-Based Method to Generate Biomarker Panels Tailored to Specific Therapeutics for Individualized Treatments": EP 2 229 589, issued June 10, 2015; US2017/0067877A9, pending. OncoSignature® is a Registered Trademark: US Reg. No. 5,718,472; Int. Cl. 5, 42. Intl. Reg. 1382289

AP3 INDICATION FINDING IDENTIFIES ENDOMETRIAL CANCER AS A NEW TUMOR TYPE SENSITIVE TO ACR-368

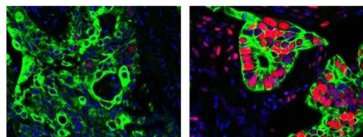
ACR-368 OncoSignature-based indication finding prior to trial entry



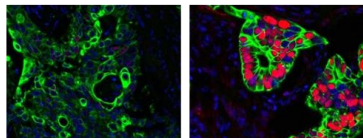
Endometrial patient tumor samples



BM1



BM2

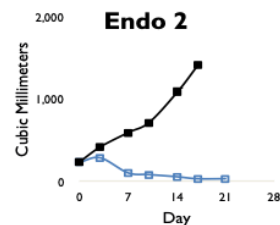
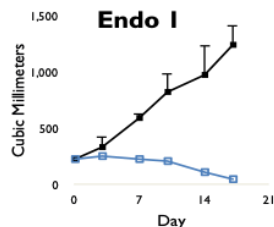


BM3

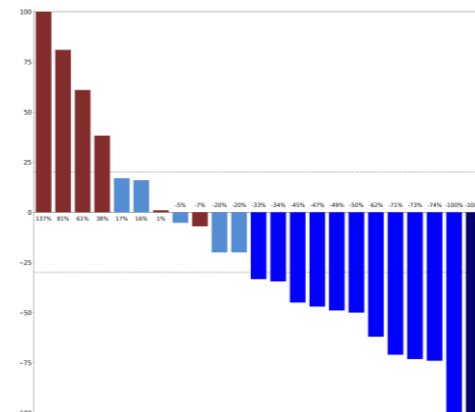
Predicted
Non-Responder

Predicted
Responder

Functional validation in preclinical models

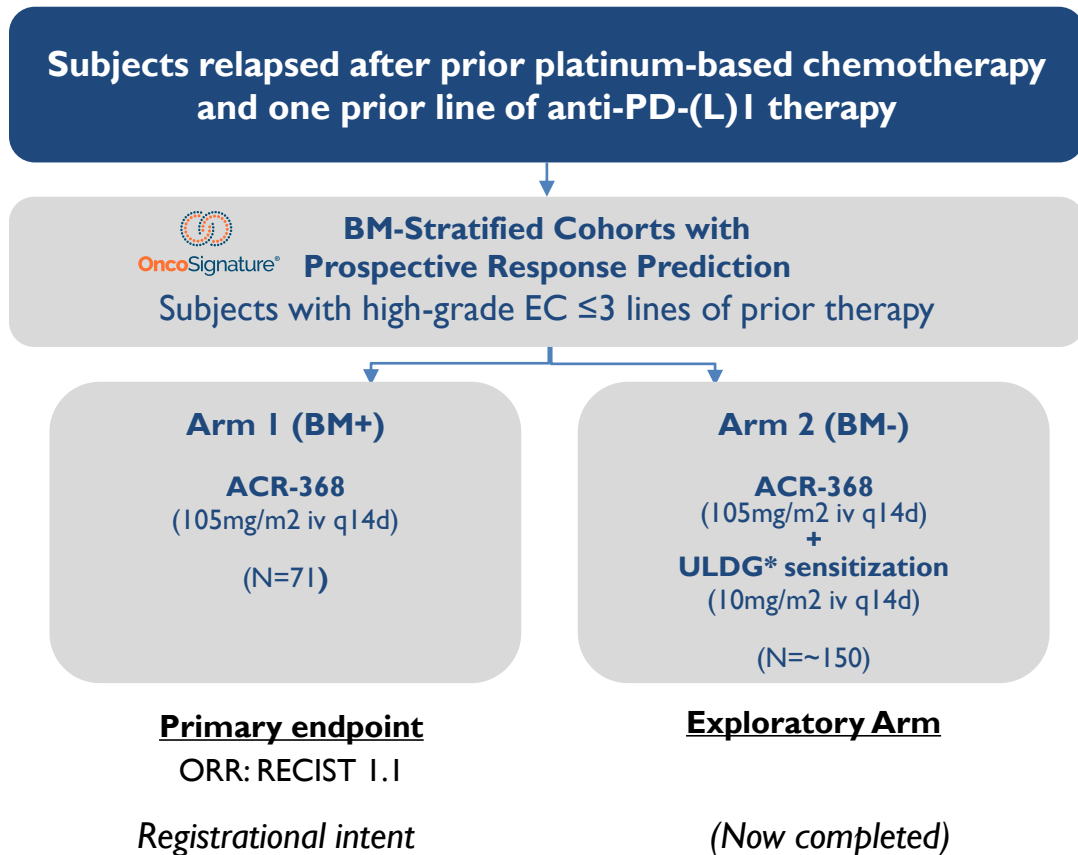


Clinical validation in ongoing Phase 2b study



INITIAL TRIAL DESIGN

- The ACR-368 OncoSignature is a tumor agnostic Biomarker (BM) test designed to prospectively predict benefit from ACR-368 (prexasertib), a potent, selective CHK1/2 inhibitor.
- It measures the tumor's addiction to CHK1/2-mediated DNA repair independent of genetic alterations.
- Screening with Acrivon's OncoSignature BM test across routine-processed (FFPE) human tumor types, predicted endometrial cancer (EC) to be particularly sensitive to ACR-368.



*ULDG = ultra low dose gemcitabine

FAVORABLE SAFETY PROFILE

- Limited, transient, mechanism-based hematological AEs
- **Notable Absence of**
 - GI toxicities, ILDs, stomatitis, ocular toxicity, peripheral neuropathy, etc.

Treatment-Related Adverse Events	Arm 1 (ACR-368) N=40 Grades 3/4	Arm 2 (ACR-368 + ULDG) N=48 Grades 3/4
<i>N = number of subjects (%)</i>		
Thrombocytopenia	9 (22)	17 (34)
Anemia	11 (27)	22 (46)
Leukopenia	6 (15)	11 (23)
Neutropenia	10 (25)	16 (33)
Febrile neutropenia	2 (5)	4 (8)
Acute kidney injury	2 (5)	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 ACR-368 + ULDG

ENDOMETRIAL PATIENT DEMOGRAPHICS AND BASELINE CHARACTERISTICS

Subject Demographics	BM+ N = 40	BM- N = 48
Median Age (range)	66.0 (40, 77)	66.0 (49, 80)
Race, n (%)		
White	29 (72.5)	29 (60.4)
Black/African American	6 (15)	10 (20.8)
Asian	1 (2.5)	4 (8.3)
American Indian or Alaska Native	0	0
Native Hawaiian or other pacific islander	1 (2.5)	0
Other/Unknown	3 (7.5)	5 (10.4)
Stage, n (%)		
III	10 (25)	18 (37.5)
IV	30 (75)	29 (60.4)
Unknown	0	1 (2.1)
Histology, n (%)		
Serous	20 (50)	16 (33.3)
Clear-cell carcinoma	2 (5)	4 (8.3)
Carcinosarcoma	6 (15)	10 (20.8)
Endometrioid, G3	9 (22.5)	15 (31.3)
Other	3 (7.5)	3 (6.3)
ECOG Status at Baseline, n (%)		
0	20 (50)	25 (52)
I	20 (50)	23 (48)

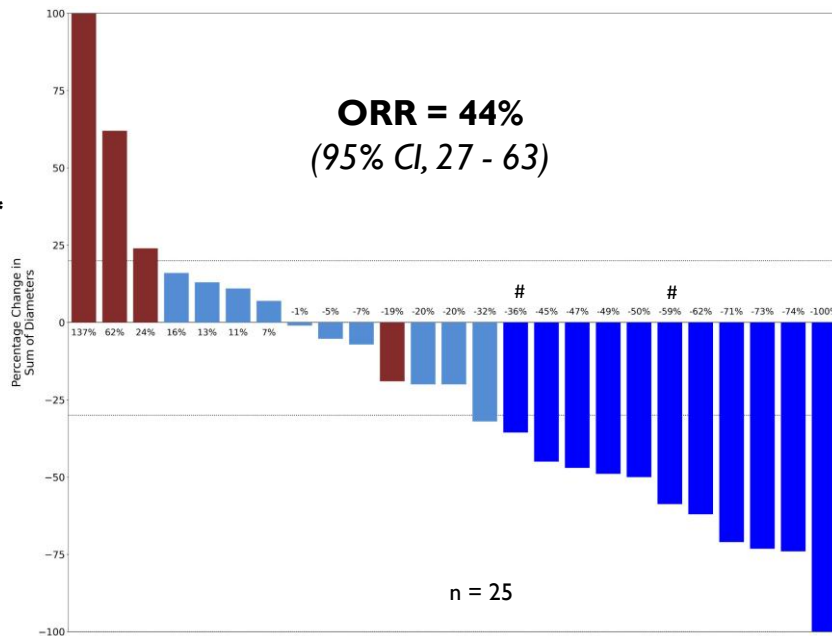
Subject Demographics	BM+ N = 40	BM- N = 48
Prior Lines of Therapy, Median (Mean)	2 (2)	2 (2)
Best Overall Response to Last Prior Line		
Refractory	9 (22.5)	15 (31.3)
Relapsed disease	31 (77.5)	33 (68.7)
Unknown	0 (0)	0 (0)
MMR Status, n (%)		
pMMR	28 (70)	29 (60.4)
dMMR	1 (2.5)	6 (12.5)
Unknown	11 (27.5)	13 (27.1)
TP53 Status, n (%)		
Mutant	24 (60)	15 (31.3)
Wildtype	6 (15)	11 (22.9)
Unknown	10 (25)	22 (45.8)
Prior exposure to PD-1/PD-L1, n (%)		
Yes	39 (97.5)	46 (95.8)
No	1 (2.5)	2 (4.2)
Prior exposure to Pembro/Len, n (%)		
Yes	19 (47.5)	20 (41.7)
No	21 (52.5)	28 (58.3)

Non QC'ed data based on EDC data extract as of 01/13/2026

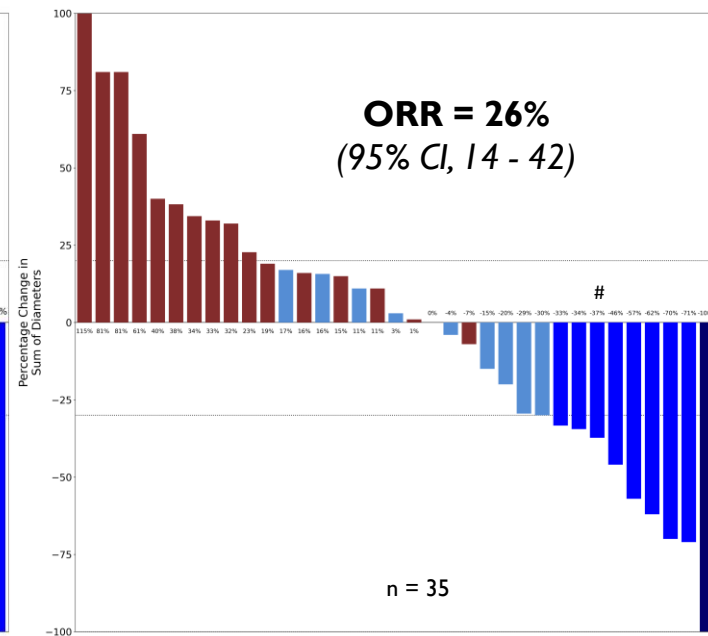
BETTER ORR OBSERVED IN SUBJECTS WITH ≤ 2 PRIOR LINES OF THERAPY

Arm 1 (BM+)
ACR-368

BOR by RECIST 1.1
on study treatment*



ARM 2 (BM-)
ACR-368 + ULDG



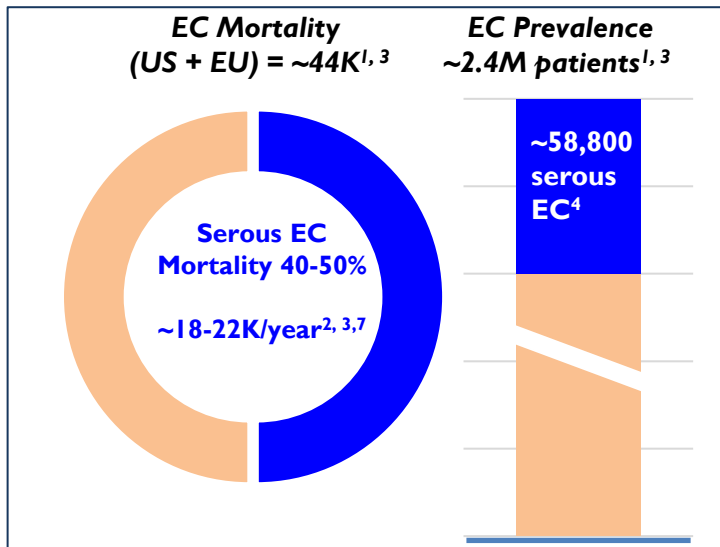
* Best of BICR and/or PI
Unconfirmed PR

OncoSignature BM levels and the BM+ fraction were noted to be significantly higher in serous

Non QC'd data based on EDC data extract as of 12/04/2025



SEROUS ENDOMETRIAL CANCER - A SIGNIFICANT UNMET NEED



Disproportionate Mortality

- Accounts for ~40-50% of all endometrial cancer deaths.^{5, 8}

Limited Effective Treatment Options

- After frontline IO + chemotherapy, pem + len no longer viable option in $\geq 2L$ ⁶
- Chemotherapy responses short-lived. Rapid resistance, early recurrence.
- HER2-targeting benefits smaller proportion, no TP53-directed therapies

Almost all serous patients progress to $\geq 2L$ of therapy

SOC in $\geq 2L$ post-IO/platinum ~13% ORR and ~3.6 months PFS and 10 months OS (single agent chemotherapy)^{5, 7, 9}

Large addressable total annual market (TAM) opportunity (US + EU)

- Serous patients: ~(20,000 per year x >6 months mDoT)
- Existing high unmet need prevalence pool: additional ~60,000 patients

¹SEER database

²<https://pmc.ncbi.nlm.nih.gov/articles/PMC9445918>

³Concin, C. et al, ESGO-ESTRO-ESP 2025 Guidelines; Lian Y., Luo P. Annals of Global Health (2025).

⁴Based on internal estimates of approximately 2.4% serous in the prevalence pool given survival approximations

⁵Bogani et al, Gynecol Oncol. 2021 July ; 162(1): 226-234. doi:10.1016/j.ygyno.2021.04.029.

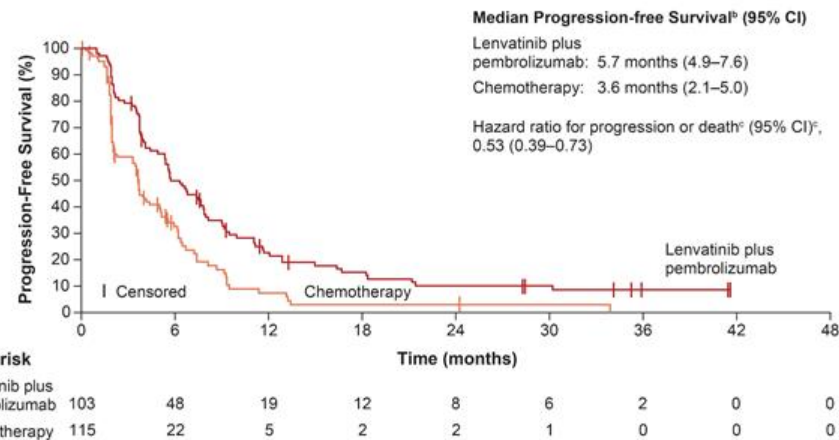
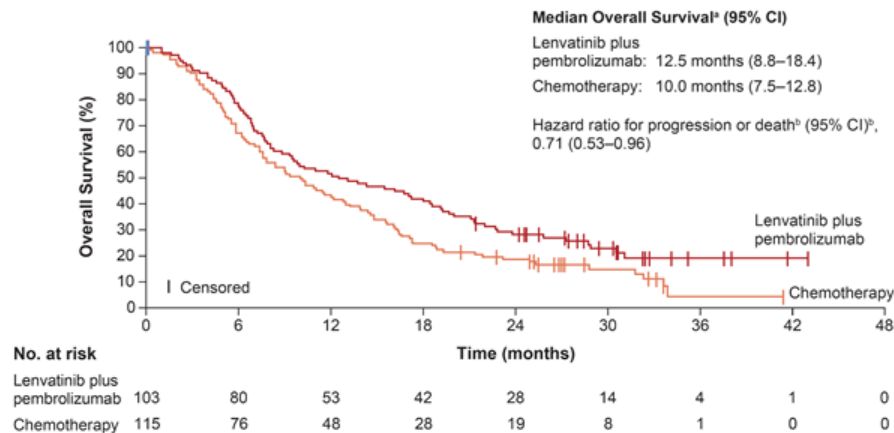
⁶Live KOL event during ESGO (See <https://ir.acrivos.com/events/event-details/endometrial-cancer-kol-panel-discussing-acrivons-phase-2b-acr-368-study>)

⁷Makker et al, NEJM; 2022; 386:437-48

⁸KOL estimates

⁹Sub-group analysis of KEYNOTE 775 data: Lorusso, Makker, et al data from ESGO 2022

SEROUS EC ALL-COMER POPULATION (KEYNOTE-775) SHOWS 3.6 MONTHS PFS AND 10 MONTHS OS IN 2ND LINE



Control arm from KEYNOTE-775 study (N = 115)

Lorusso et al, 2022

^aFrom product-limit (Kaplan-Meier) method for censored data; ^bbased on Cox regression model with Efron's method of tie handling with treatment as a covariate. Analyses were conducted using an unstratified Cox model.

SEROUS EC ALL-COMER POPULATION (KEYNOTE-775 CONTROL ARM) SHOWS 13% ORR

	Low-grade endometrioid		Endometrioid not further defined/not defined as low grade		Serosus		Clear cell	
Parameter ^a	LEN + pembro (n = 59)	Chemotherapy (n = 54)	LEN + pembro (n = 185)	Chemotherapy (n = 200)	LEN + pembro (n = 103)	Chemotherapy (n = 115)	LEN + pembro (n = 30)	Chemotherapy (n = 17)
ORR, % (95% CI ^b)	42.4 (29.6–55.9)	11.1 (4.2–22.6)	32.4 (25.7–39.7)	18.5 (13.4–24.6)	36.9 (27.6–47.0)	13.0 (7.5–20.6)	26.7 (12.3–45.9)	0 (0–19.5)
CR, %	13.6	3.7	5.9	2.5	8.7	3.5	3.3	0
PR, %	28.8	7.4	26.5	16.0	28.2	9.6	23.3	0
mDOR, mo range	20.0 (1.6–35.5+)	4.3 (3.7–31.3+)	12.7 (2.6–32.7+)	5.8 (0.0+ –37.1+)	9.0 (2.4–39.5+)	5.6 (3.2–30.2)	8.4 (4.2–28.3+)	---
DCR ^c , % (95% CI ^b)	81.4 (69.1–90.3)	44.4 (30.9–58.6)	72.4 (65.4–78.7)	54.0 (46.8–61.1)	73.8 (64.2–82.0)	35.7 (26.9–45.1)	56.7 (37.4–74.5)	35.3 (14.2–61.7)

Lorusso et al, 2022

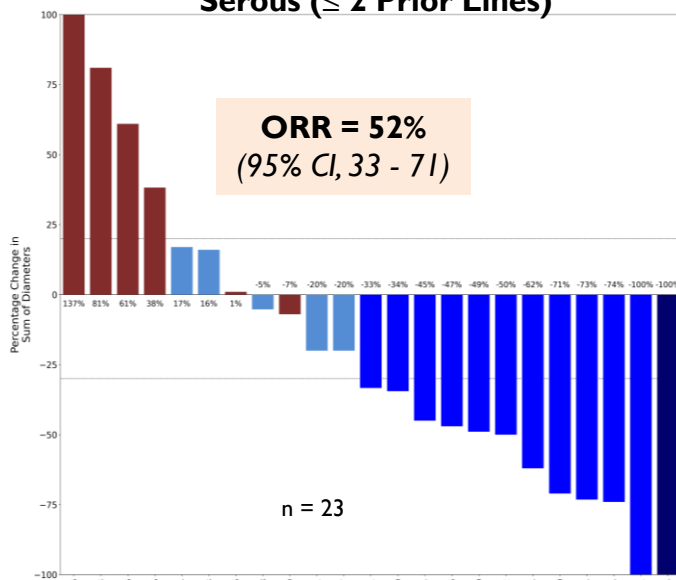
^aBy BICR per RECIST v1.1; ^bbased on binomial exact confidence interval method; ^cCR + PR + SD ≥ 7 weeks.



23rd European Congress
on Gynaecological Oncology
October 27–30, 2022 | Berlin, Germany

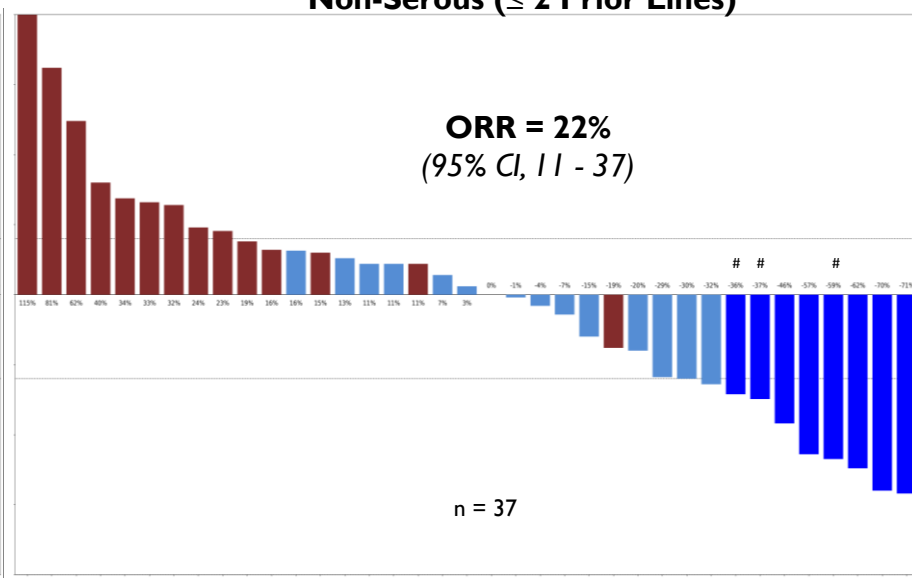
SIGNIFICANT ORR IN SEROUS ALL-COMER POPULATION WITH ≤ 2 PRIOR LINES OF THERAPY

Serous (≤ 2 Prior Lines)



DCR: 74%, CBR (16 weeks): 65%

Non-Serous (≤ 2 Prior Lines)

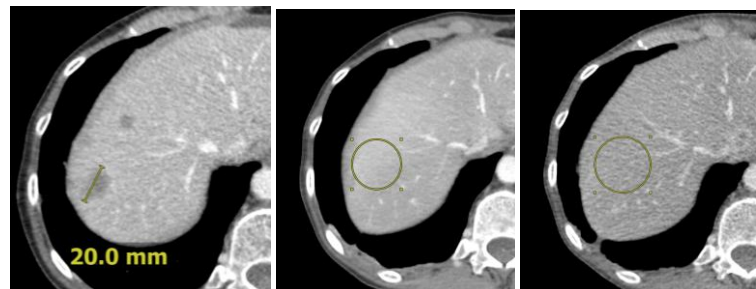


* Best of BICR and/or PI
Unconfirmed PR

Non QC'ed data based on EDC data extract as of 12/04/2025

DEEP, RAPID RESPONSES IN PATIENTS WITH AGGRESSIVE SEROUS ENDOMETRIAL TUMORS

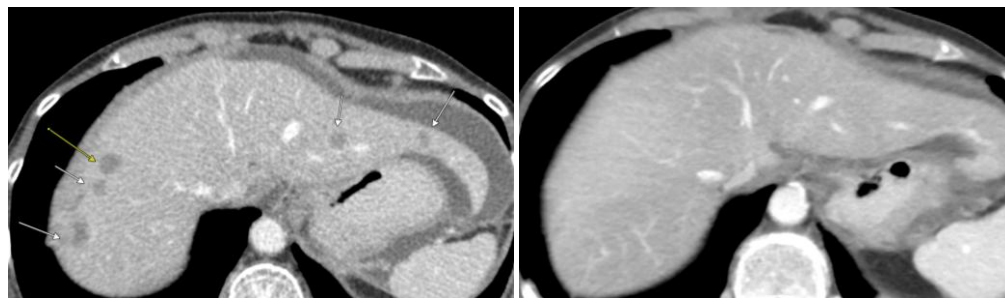
67 y old patient with stage IV serous cancer, pMMR, p53-mutated; patient was refractory to last prior line, including anti-PD-1



Screening

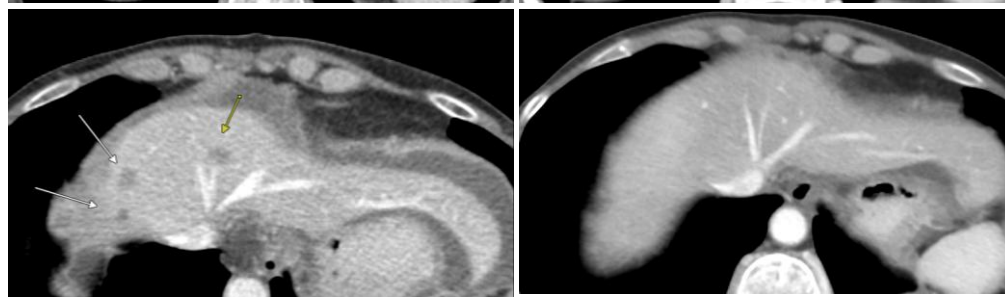
Week 8
(-100%)

Week 12
(-100%)



Screening

Week 8 and 12
(-100%)

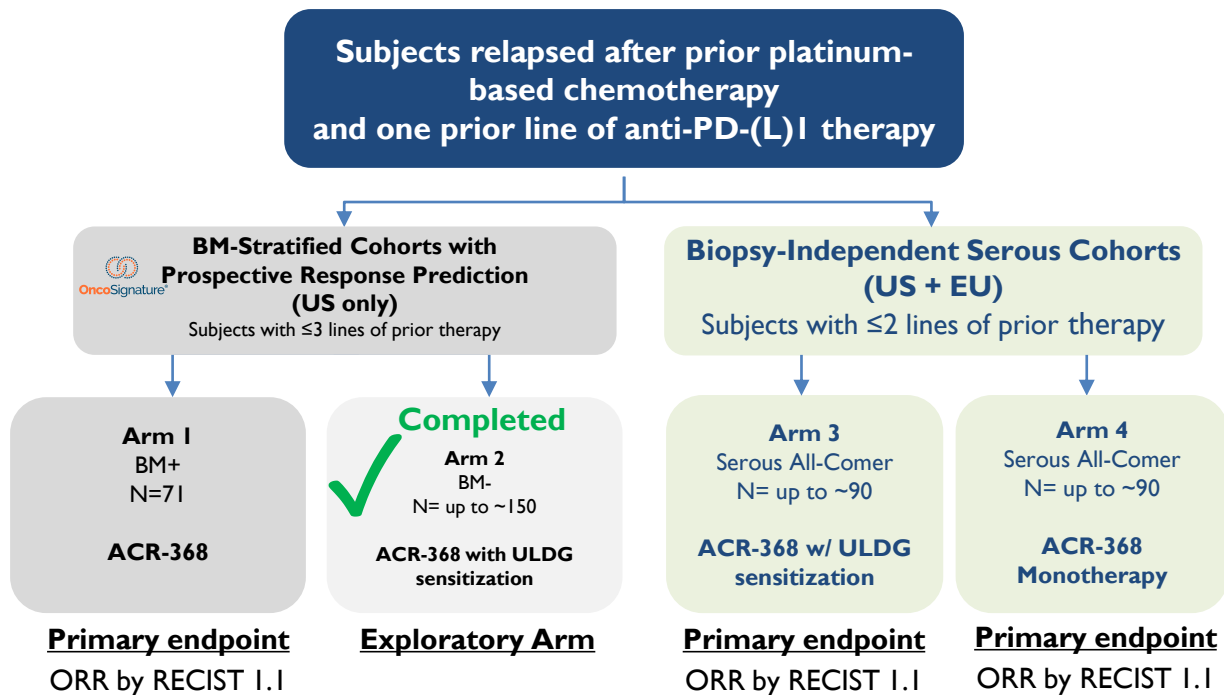


Complete resolution of hepatic target lesions

- Confirmed **PR** with **complete** disappearance of >10 additional hepatic metastases spanning both lobes
- ACR-368 single agent activity in multifocal hepatic disease, even in *high-disease burden* cases

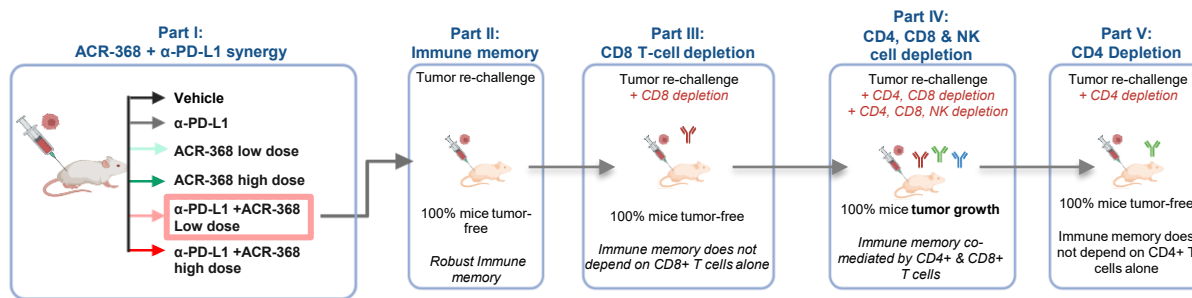
ACR-368 PROGRAM INCREASINGLY FOCUSED ON SEROUS ENDOMETRIAL CANCER

- Serous EC shows particularly high ORR in both BM+ and BM- tumors
- ACR-368 has a favorable safety profile
- Arm 3 and Arm 4 are evaluating ACR-368 + ULDG and ACR-368, respectively, in serous EC all-comer (biopsy-independent) subjects
- The study is being expanded to >20 EU sites

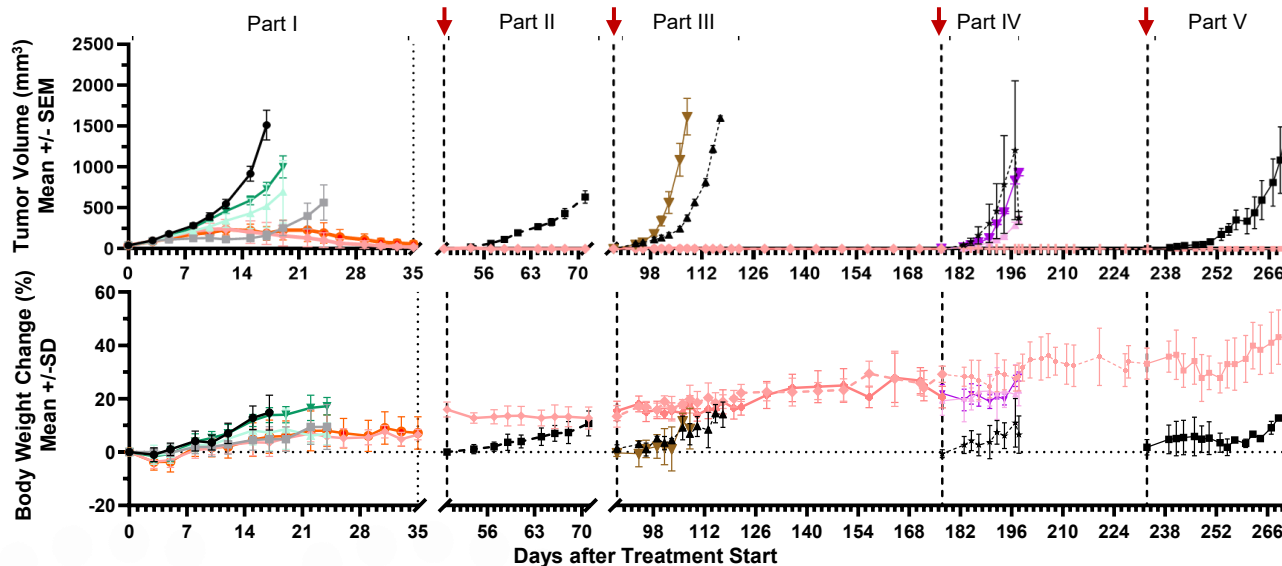


**Prespecified interim
analysis 2H 2026**

POTENT ACR-368 + ANTI-PD-L1 SYNERGY WITH COMPLETE TUMOR REGRESSION AND IMMUNE MEMORY MEDIATED BY CD4+ AND CD8+ CELLS



↓ Tumor cell re-challenge



AACR 2026: ACR-368 SYNERGY WITH CHECKPOINT INHIBITION

Poster # LB152

ACR-368 synergizes with PD-L1 blockade by coordinated activation of adaptive and innate immunity pathways to achieve robust anti-tumor efficacy

Amira Elbakry¹, Taronish Dubash¹, Joelle Baddour-Sousounis¹, Jessica Hopkins¹, Subodh Kumar¹, Ahmed Yousef¹, Yingchun Spring Liu¹, Kate Rappard¹, Calvin Yang¹, Ignacio Arribas Diez², Marc Isaksson¹, Luka Romero¹, Zachary Best¹, Nina Lipjancic¹, Sofija Skoric¹, Courtney Cooper¹, Emma Ahman¹, Valentina Sino¹, Portia Lombardo¹, Corey Xu¹, Magnus E. Jakobsson¹, Helen Nilsson¹, Lei Shi¹, Ayesha Murshid¹, Michal Shipilini¹, Joon Jung¹, David Proia¹, Erick Gamelin¹, Kristina Masson¹, Peter Blume-Jensen¹

¹Actron Therapeutics Inc., Watertown MA, USA; ²Actron AB, Medicin Village, Lund, Sweden



Introduction

- ACR-368 is a potent and selective CHK1/2 inhibitor with demonstrated durable clinical activity which is currently being evaluated as monotherapy and in combination regimens in Actron Therapeutics' ongoing registrational-intent Phase 2 endometrial cancer clinical trial.
- Predictive studies indicate that ACR-368 activates multiple anti-tumor immune responses, supporting its combination with immune checkpoint inhibitors (ICIs).
- ACR-368 in combination with anti-PD-L1 led to complete tumor regression in a syngeneic colorectal cancer mouse model and long-lasting immune memory driven by anti-tumor innate and adaptive immune responses.

Potent synergy of ACR-368 with anti-PD-L1 and induction of durable immune memory mediated by CD4⁺ and CD8⁺ cells

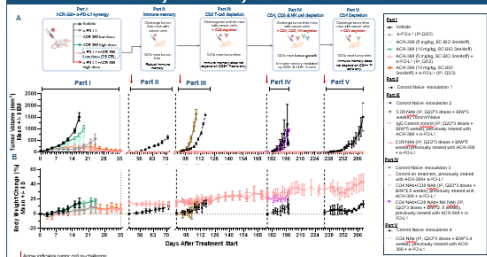


Figure 1: Complete tumor regression and durable immune memory mediated by adaptive immunity. (A) In a syngeneic colorectal cancer mouse model, ACR-368 monotherapy resulted in 74% tumor growth inhibition and anti-PD-L1 resulted in complete tumor regression in 7/8 mice. Robust synergy was observed with the combination of ACR-368 with anti-PD-L1, which led to complete tumor regression in 7/8 mice (Part I), and generated durable immune memory persisting beyond 200 days after four sequential tumor challenges (Part II-V). Systemic depletion of immune cell subpopulations (Parts II-V) showed that the absence of either CD4⁺ or CD8⁺ T-cells alone did not lead to tumor growth upon rechallenge, while co-depletion of both resulted in tumor formation, indicating that immune memory is driven by adaptive immune responses. (B) The combination of ACR-368 and anti-PD-L1 was well-tolerated, consistent with non-overlapping toxicities.

ACR-368 activates ssDNA/dsRNA sensing and innate immune signaling pathways

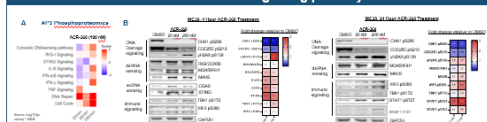


Figure 2: Activation of innate immune signaling and nucleic acid sensing pathways in vitro. (A) Actron Predictive Precision Proteomics (AP3) analysis of MCF8 cells showed a time-dependent increase in phosphorylation of proteins associated with multiple innate immune signaling and DNA damage pathways after ACR-368 treatment in DMSO. (B) Western blot analysis of selected markers in ACR-368 treated cells (200nM, 200nM) showed reduction in CHK1 activity accompanied by an increase in DNA damage, ssDNA/dsRNA sensing (d), and subsequent activation of innate immunity pathways (d).

ACR-368 activates DNA damage responses and induces apoptotic cell death in vivo

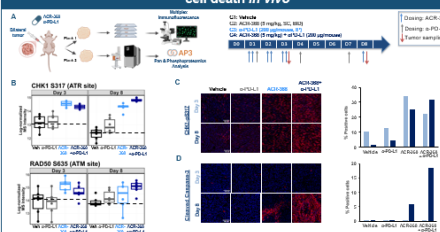


Figure 3: Robust induction of DNA damage response signaling by ACR-368 in vivo. (A) Mice with bilateral syngeneic tumors were treated with vehicle, ACR-368 (5 mg/kg), anti-PD-L1 (200 µg/mouse), or the combination. Tumors from either flank were collected for immunostaining (IF) or AP3 profiling. AP3 analysis confirms drug activity as indicated by the strong induction of ATM (RAD50/5635) and ATR (CHK1/S177) phosphorylation (blue at early and late timepoints lower panels). (C) Spatial IF analysis of Day 3 and Day 8 samples confirms ACR-368 driven ATR activation and subsequent apoptosis in Day 8, as indicated by cleaved caspase-3 staining (D).

Enhanced innate immune signaling and modulation of immune pathways by ACR-368 and anti-PD-L1 combination

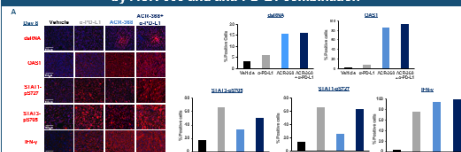


Figure 4: ACR-368 and anti-PD-L1 combination treatment activates immune sensing and upregulates multiple immune pathways. (A) IF analysis of MCF8 tumors shows that ACR-368 (single agent or in combination) induces dsRNA and its sensor DAB1 with enhanced downstream immune signaling via pSTAT1-S727, pSTAT3-Y705, and pIRAK4. (B) AP3 pan-protein profiling shows modest upregulation of immune pathways at Day 3 by ACR-368 and anti-PD-L1 that is more pronounced at Day 8, with maximal effects observed in the combination treatment, consistent with the observed in vivo synergy. (C) Further dissection of AP3 data shows a time dependent increase in macrophage-associated proteins at Day 8, but not Day 3, including the combination, based on drug-regulated individual protein changes. (D) Simplified illustration of macrophage polarization towards anti- or pro-inflammatory phenotypes.

ACR-368 in combination with anti-PD-L1 promotes pro-inflammatory macrophage phenotypes and enhances cytotoxic innate and adaptive immune responses

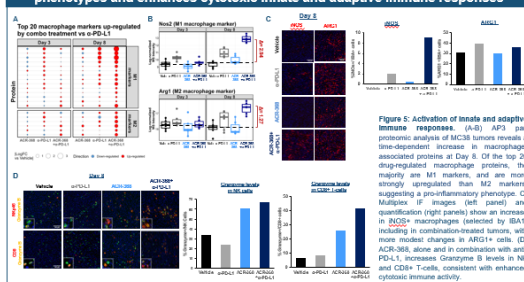


Figure 5: Activation of innate and adaptive immune response. (A-B) AP3 pan-proteomic analysis of MCF8 tumors reveals a time-dependent increase in macrophage-associated proteins at Day 8. Of the top 20 drug-regulated macrophage proteins, the majority are M1 markers and are more strongly upregulated than M2 markers, suggesting a pro-inflammatory phenotype. (C) Multiplex IF images (left panel) and quantification (right panel) show an increase in CD8⁺ macrophages (selected by BBAT) including in combination-treated tumors, with modest changes in CD4⁺ cells. (D) ACR-368, alone and in combination with anti-PD-L1, increases Granzyme B levels in NK and CD8⁺ T-cells, consistent with enhanced cytotoxic immune activity.

ACR-368 is associated with reduced T-cell exhaustion marker expression in combination with anti-PD-L1

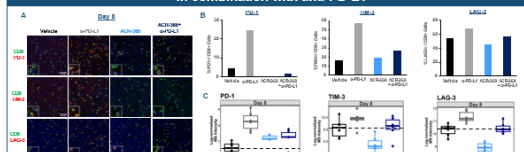
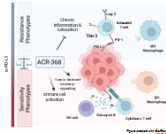


Figure 6: Suppression of T-cell exhaustion markers by ACR-368. (A) Multiplex IF analysis of MCF8 tumors at Day 8 shows expression of PD-L1, TIM-3, and LAG-3 across treatment groups. (B-C) IF quantifications (upper panels) and AP3 proteomic analysis (lower panels) demonstrate that anti-PD-L1 treatment is associated with increased exhaustion marker expression, while ACR-368, alone and in combination, is associated with lower levels of PD-L1, TIM-3, and LAG-3.

Conclusions

- ACR-368 activates the DNA damage response accompanied by robust innate immune signaling in MCF8 cells in vitro and in vivo.
- AP3 pan- and phospho-proteomics coupled with spatial IF immune profiling demonstrate activation of both the innate and adaptive components of the immune system, together with suppression of T-cell exhaustion markers.
- ACR-368 combined with anti-PD-L1 provides a balanced, sustainable inflammatory response leading to anti-tumor efficacy and long-term immune memory.
- These data provide a strong mechanistic rationale for clinical evaluation of ACR-368 in combination with ICIs to enhance the clinical efficacy of these agents.



AACR 2026: ACR-368 SYNERGY WITH TOPOISOMERASE I

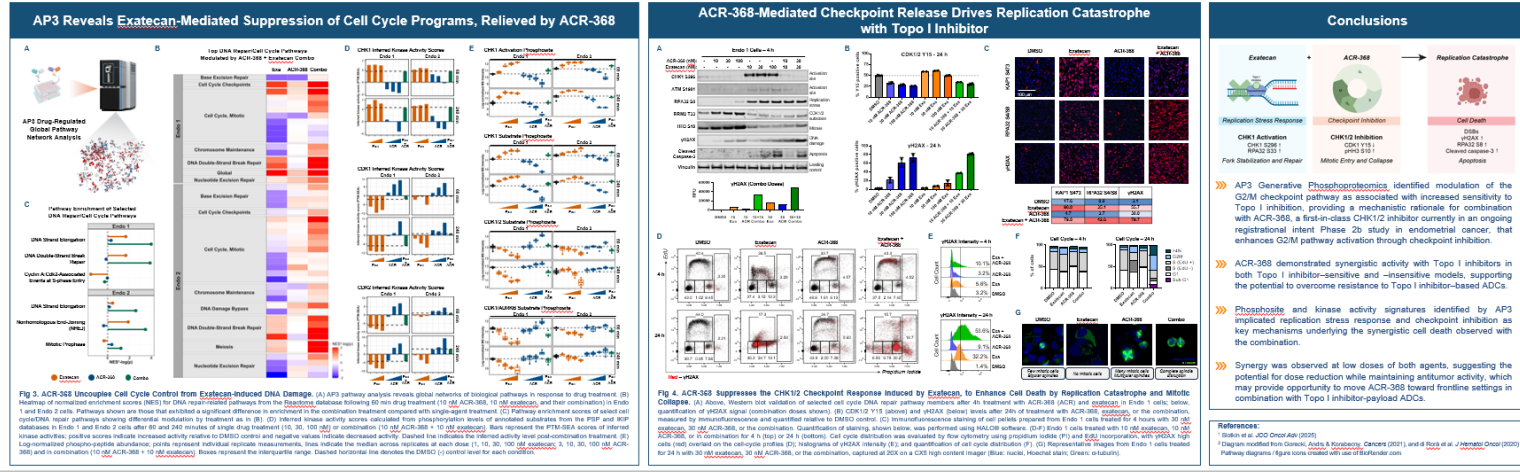
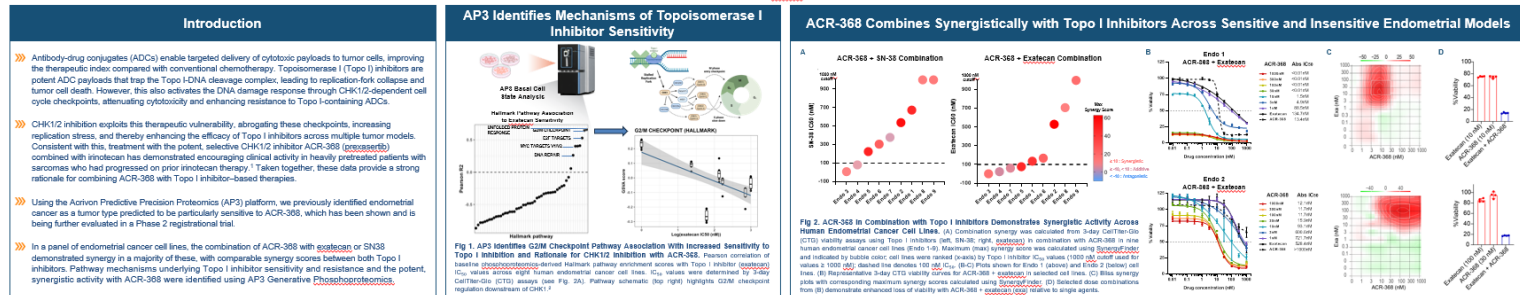
Poster # 239

Potent Synergy Between CHK1/2 Inhibitor ACR-368 and the ADC Payload Topoisomerase I Inhibitor: Rationale for ADC + ACR-368 Combination Therapy

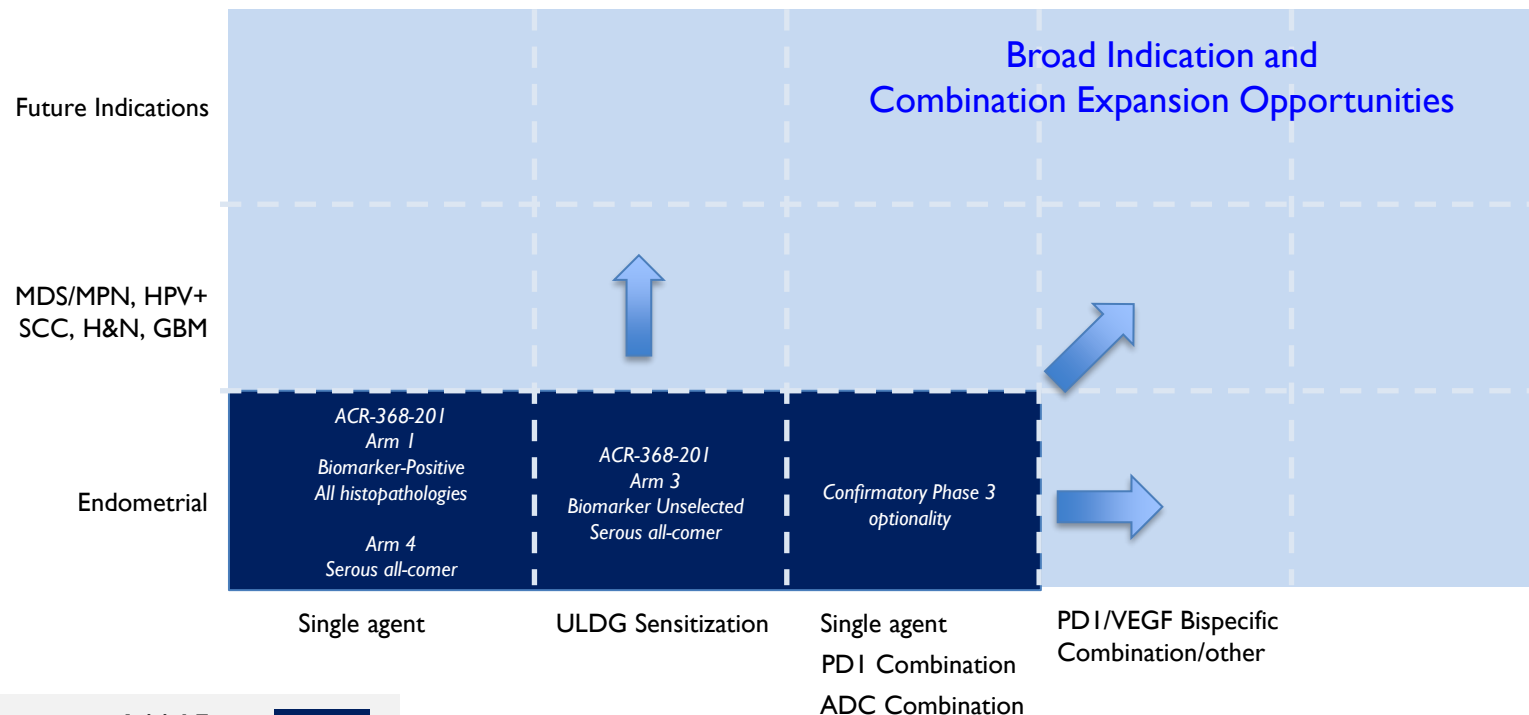
Portia Lombardi¹, Ahmed Yousef², Mohamed Eldeeb³, Nina Lipinski⁴, Martina Pasello⁵, Ruben Comas⁶, Sofja Skoric⁷, Ignacio Ambis Diaz⁸, Zachary Best⁹, Anna-Marie Alavi¹⁰, Subodh Kumar¹¹, Kate Rappaport¹², Calvin Yang¹³, Corey Xu¹⁴, Emma Ahn¹⁵, Valentina Siro¹⁶, Tarnish Dubash¹⁷, Maria Rodriguez-Zabala¹⁸, Joelle Baidou-Sassouni¹⁹, Reina Impropo²⁰, Magnus E. Jakobsson²¹, Aysha Muzaffar²², Michael Shigabov²³, Joon Jung²⁴, David Pross²⁵, Erik Camdemil²⁶, Mansoor Mirza²⁷, Kristina Masson²⁸, Peter Blume-Jensen²⁹
¹Acvion Therapeutics Inc., Watertown MA, USA; ²Acvion AB, Medicin Village, Lund, Sweden; ³Authors contributed equally



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ACR-368 OFFERS MULTIPLE ATTRACTIVE OPPORTUNITIES



Initial Focus
Expansion Opportunities

INTERNAL PIPELINE: AP3-BASED DRUG DISCOVERY

POTENTIAL FIRST- AND BEST IN CLASS CANDIDATES

ACR-2316: Clinical stage, novel WEE1/PKMYT1 inhibitor

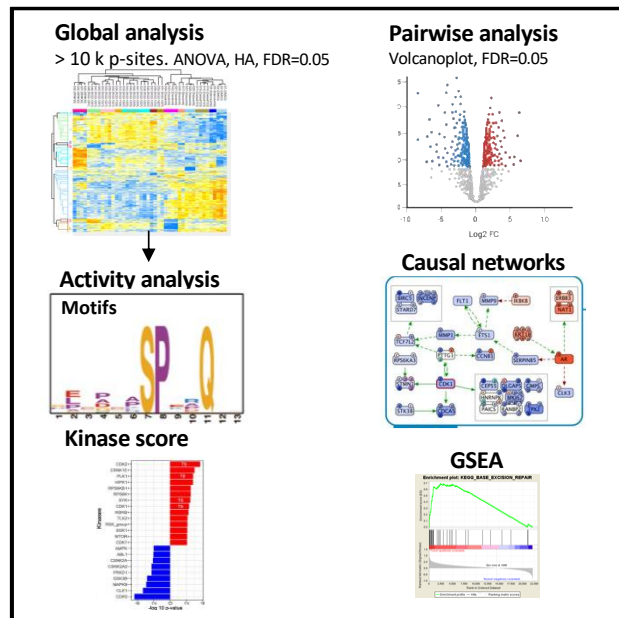
- Additional clinical data and transition to dose expansion 2026

Development candidate, novel CDK11 inhibitor

- Anticipated IND filing 1H 2027
- Multiple equally promising back-up series being pursued in parallel

Additional AP3-based internal programs

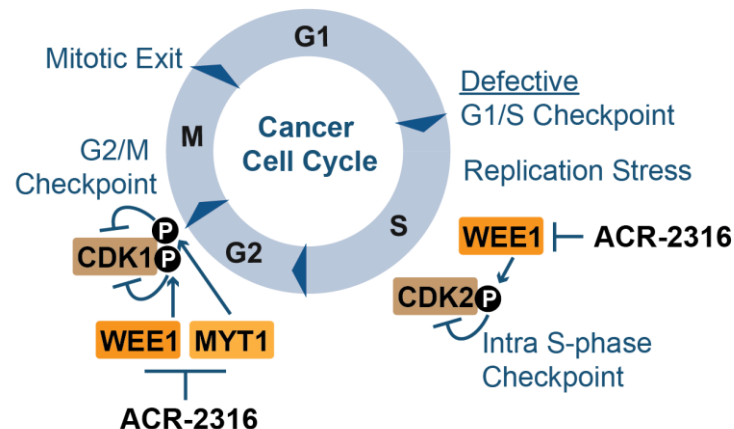
Streamlined AP3-based drug discovery



AP3 used for biologically relevant selectivity profiling

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
- Demonstrated clinical activity, but the Therapeutic Index has been challenging
- AP3 was applied to design a molecule with potent single agent activity and exquisite selectivity to achieve expanded Therapeutic Index



ACR-2316 IS A POTENTIALLY BEST- AND FIRST-IN-CLASS AGENT – FIRST POC FOR AP3 PLATFORM

Program Goals

- 1 Superior single agent activity
- 2 High selectivity and potency
- 3 Favorable ADME-PK, Therapeutic Index, and tolerability profile
- 4 Streamlined clinical development

AP3-Enabled SAR

Demonstrated Results

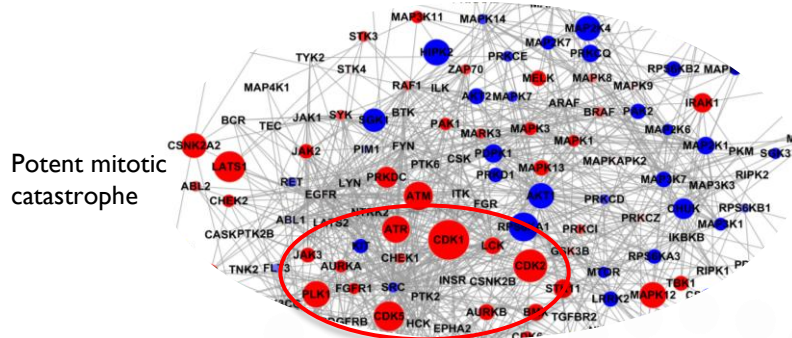
- Superior anti-tumor activity* (robust CDK1, CDK2, and PLK1 activation; inhibition of PKMYT1 resistance)
- 5-20-fold more potent* in preclinical models than clinical benchmarks; complete tumor regression
- Potent tumor cell killing*, short elim. $T_{1/2}$; only transient AEs
- Broad preclinical T.I. and complete tumor regression
- Advanced from lead to oral dosing in clinic in ≤ 15 month
- AP3-based Indication Finding
- AP3 MS-based PD assay developed for human PBMCs
- Two weekly oral dosing regimens established
- Durable clinical activity in AP3-predicted tumor types, including SCLC, sqNSCLC
- Favorable, differentiated safety, primarily only neutropenia

*Head-to-head preclinical studies against benchmarks with clinical data

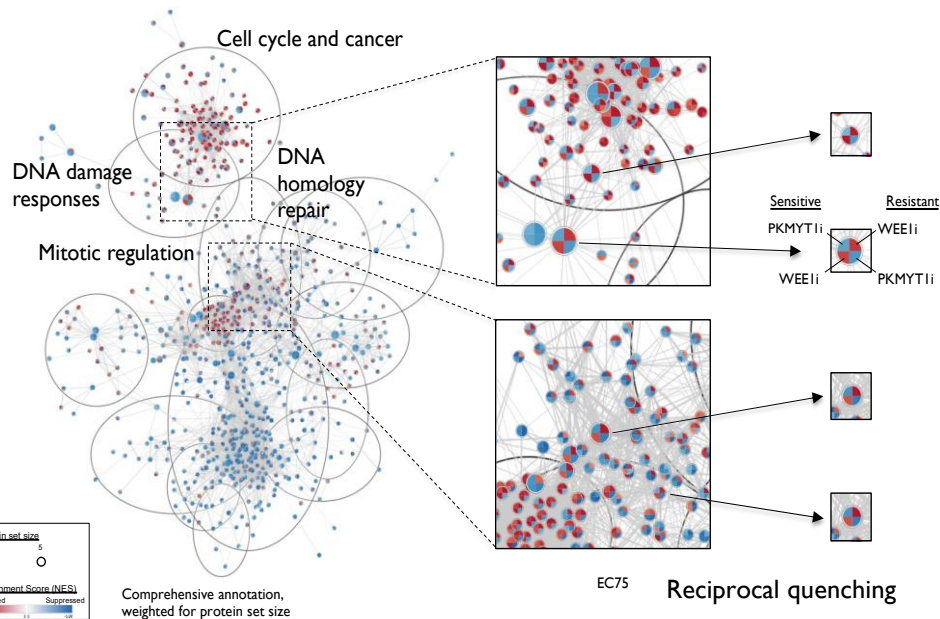
ACR-2316: UNIQUELY DESIGNED BY AP3 FOR SUPERIOR THERAPEUTIC INDEX OVER WEE1 AND PKMYT1 INHIBITORS

ACR-2316: Novel WEE1/PKMYT1 inhibitor with potent single agent activity

- ✓ Complete tumor regression across all 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
- ✓ Clinical activity observed in AP3-predicted tumor types, including SCLC, SqNSCLC and adenoNSCLC

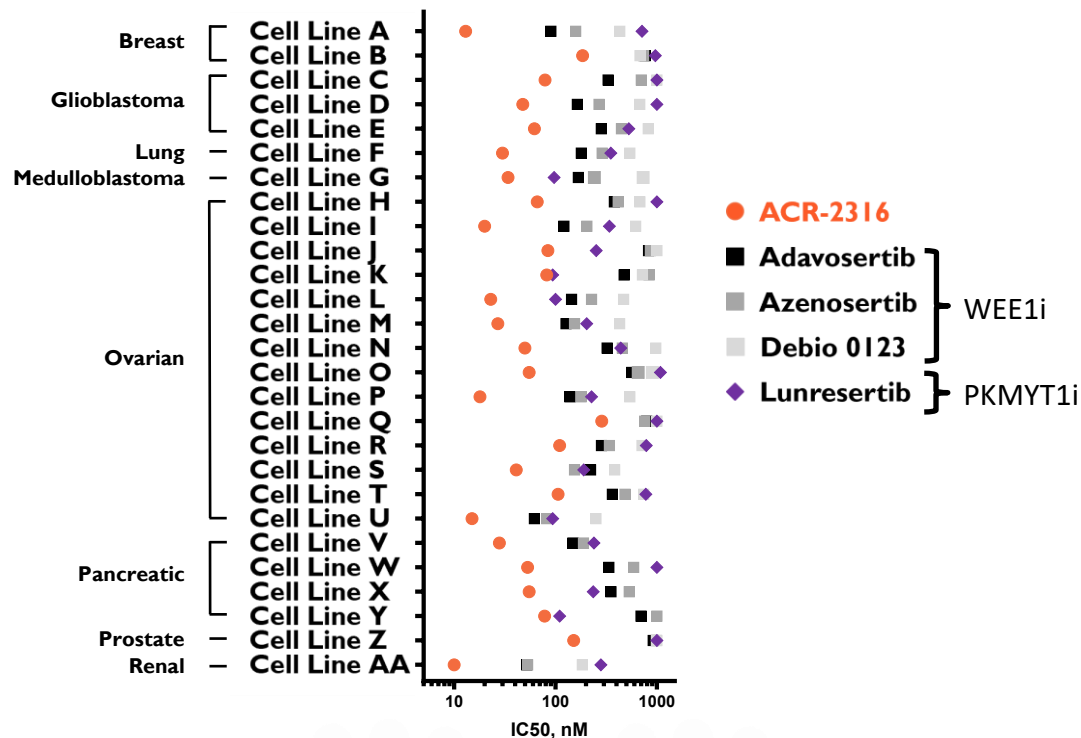


Drug Design by AP3-based SAR for desirable pathway effects and suppressing resistance mechanisms

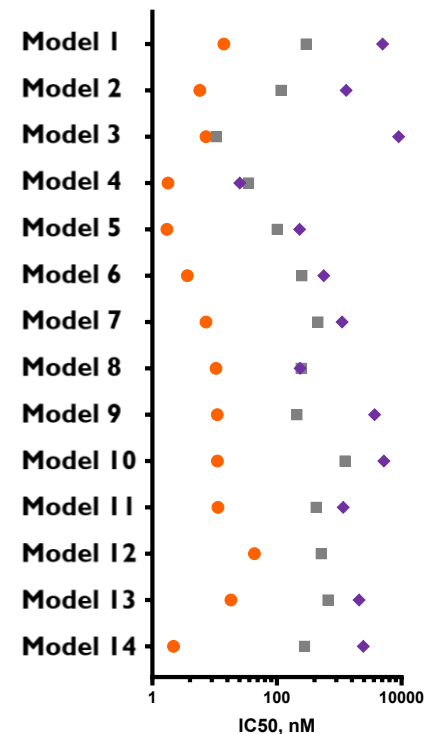


ACR-2316 IS HIGHLY POTENT ACROSS HUMAN TUMOR CELL LINES AND PATIENT-DERIVED *EX VIVO* TUMOR MODELS

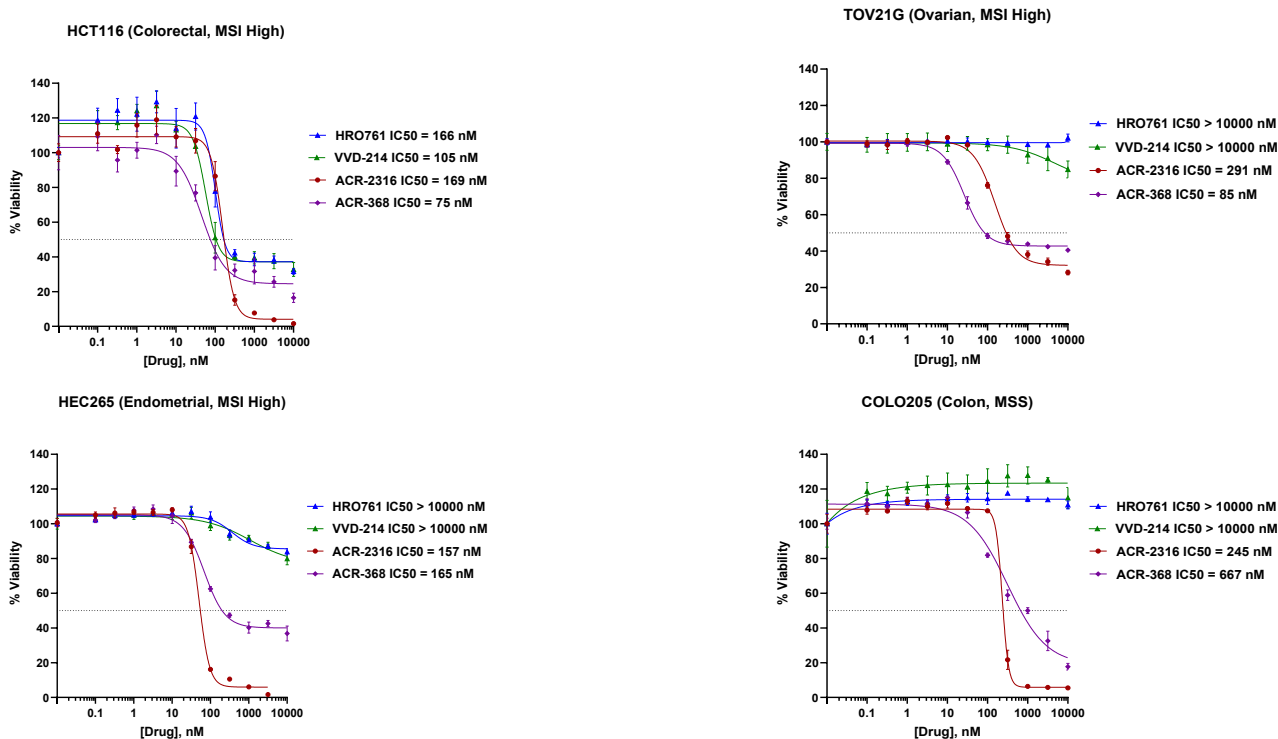
Human tumor cell lines (not genetically selected)



Patient-derived *ex vivo* ovarian tumor models

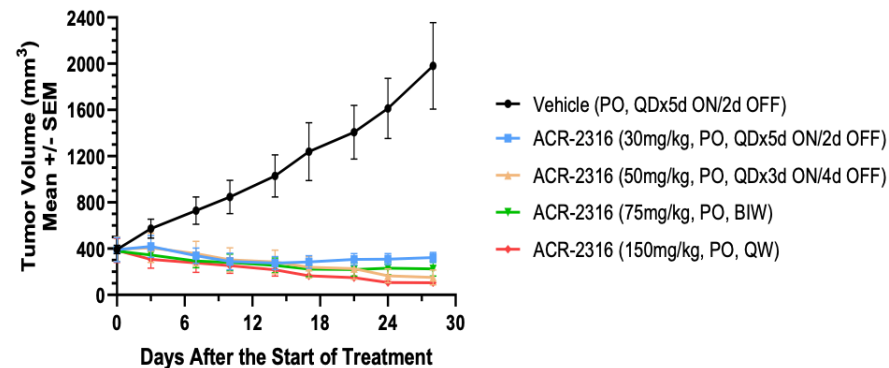
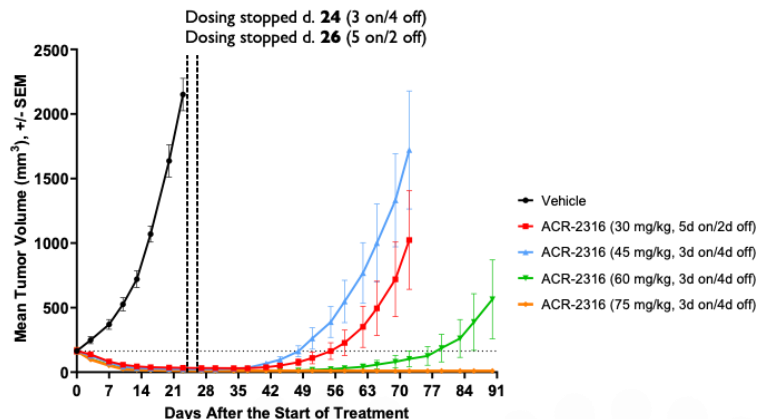
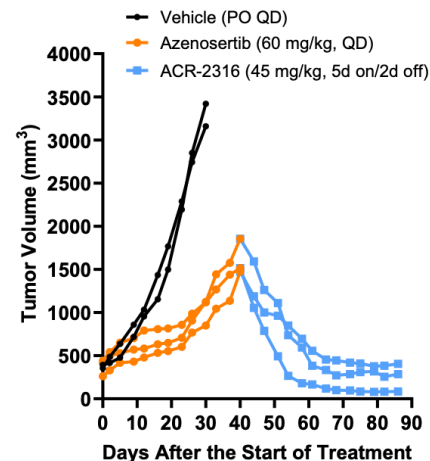
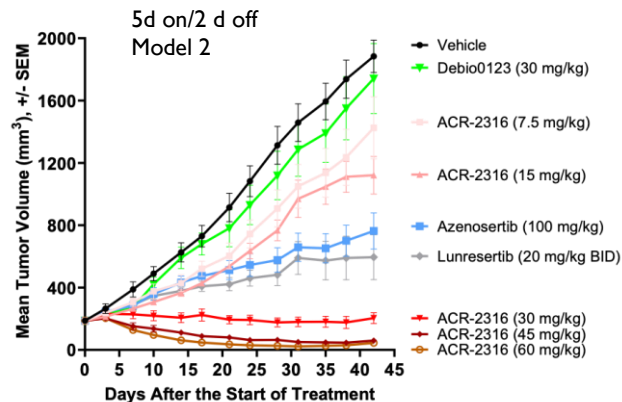
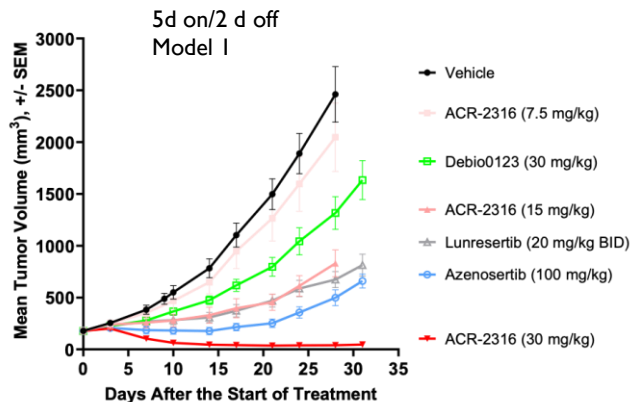


SUPERIOR ACTIVITY OF ACR-2316 OVER WRN INHIBITORS IN BOTH MSI HIGH AND MSS CELL LINES



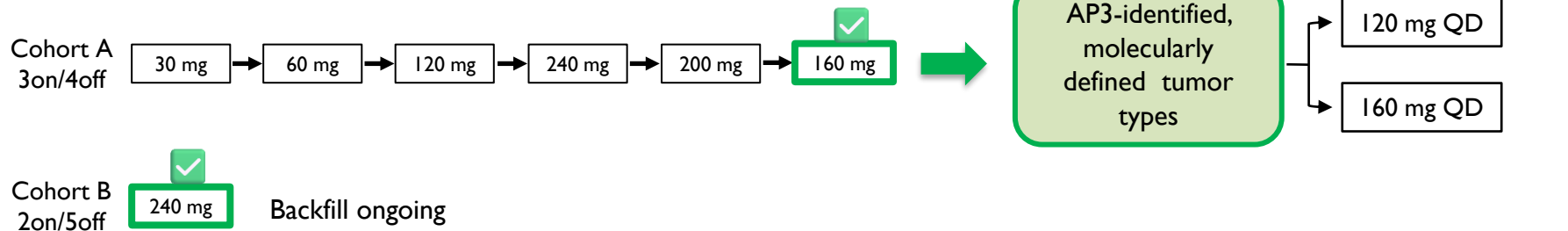
HRO761 is a noncovalent allosteric inhibitor of WRN (Novartis); VVD-133214/ RO7589831 is a covalent allosteric WRN inhibitor (Roche); ACR-2316 is more potent than both WRN inhibitors even in the optimal MSI high context, associated with strong dependency on WRN activity

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



ACR-2316 DOSE EXPANSION IN AP3-DEFINED TUMOR TYPES

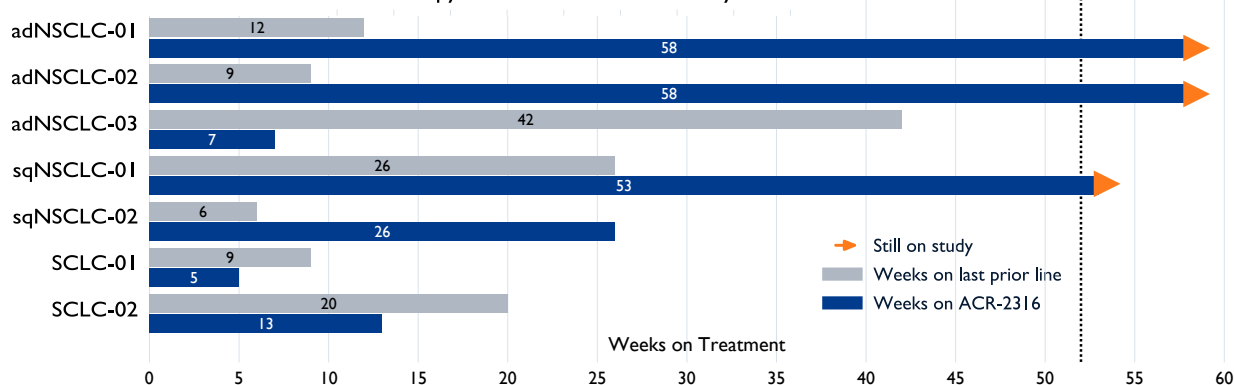
Dose Escalation – 2 weekly oral regimens established (N = 35)



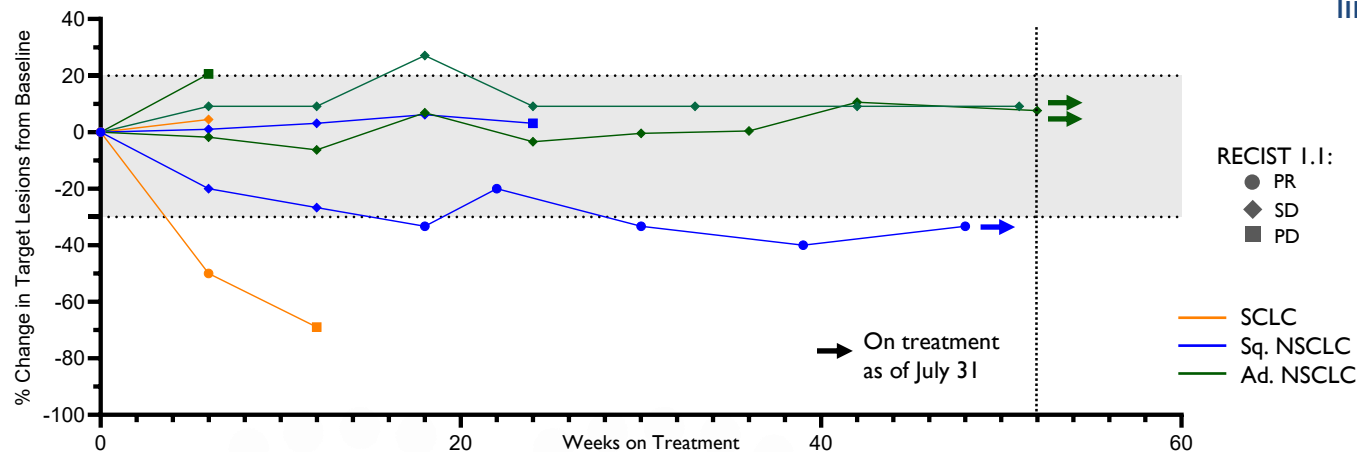
- Favorable differentiated safety profile, mainly transient heme AEs (predominantly neutropenia)
- Durable clinical activity observed during dose escalation, including in heavily pretreated lung cancer subjects
- Stratification into molecularly defined lung vs non-lung cancer types with 1:1 randomization into 2 dose levels

DURABLE CLINICAL ACTIVITY IN AP3-PREDICTED TUMOR TYPES, INCL. HEAVILY PRETREATED LUNG CANCER PATIENTS, DURING DOSE ESCALATION

Duration of Last Prior Therapy and Time on ACR-2316 by Patient



- Durable single-agent activity, including tumor shrinkage and PRs
- Clinical benefit over 1 year in several heavily pretreated subjects (median 3 prior lines, incl. ICI and chemo)



Non QC'd data based on EDC as of 7/31/2026

DIFFERENTIATED, FAVORABLE SAFETY PROFILE, PRIMARILY LIMITED TO TRANSIENT NEUTROPENIA

Established weekly dosing regimens; N = 22

Event	Grade 3 n (%)	Grade 4 n (%)	Grade ≥3 n (%)
Neutropenia	4 (18.2)	2 (9.1)	6 (27.3)
Leukopenia	1 (4.5)	1 (4.5)	2 (9.1)
TOTAL (unique subjects)	5 (22.7)	2 (9.1)	6 (27.3)

Grade ≥3 treatment-related AEs with incidence ≥5% shown; no Grade 5 TRAEs.

TOTAL: Subjects counted individually in Grade 3 and Grade 4 columns; same subject counted only once in Grade ≥3 column

- **Primarily transient, mechanism-based hematological AEs, mainly neutropenia**
- **No other types of toxicities observed above (N = 13) tolerated weekly doses**
- **Notable absence of GI toxicities, long-lasting myelosuppression or more severe non-hematological AEs commonly seen with ADCs and chemotherapy**

Non QC'd data based on EDC as of 7/31/2026

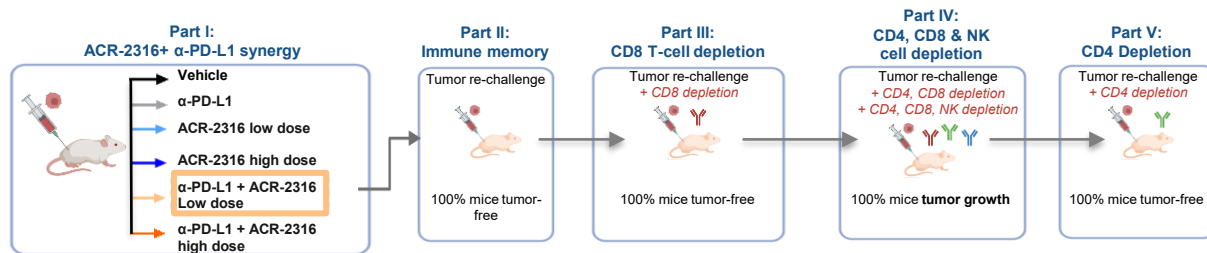
ACR-2316-101 DEMOGRAPHICS AND BASELINE CHARACTERISTICS

Subject Demographics	N = 35	Cancer Type, n (%)	N = 35	ECOG Status at Baseline, n (%)		
Median Age, years (range)	63.0 (35–77)	Endometrial	6 (17.1)	0	11 (31.4)	
Sex, n (%)		Ovarian Carcinoma	5 (14.3)	1	24 (68.6)	
Female	22 (62.9)	Breast	4 (11.4)	Prior Lines of Therapy, n (%)		
Male	13 (37.1)	Adenocarcinoma NSCLC	3 (8.6)		1	1 (2.9)
Race, n (%)		Colorectal	3 (8.6)		2	5 (14.3)
White	27 (77.1)	Head & Neck SCC	3 (8.6)		3	16 (45.7)
Black/African American	4 (11.4)	Squamous NSCLC	3 (8.6)		4	7 (20.0)
Asian	1 (2.9)	Pancreatic	2 (5.7)	5	1 (2.9)	
Native Hawaiian/Pacific Islander	1 (2.9)	Prostate (Neuroendocrine)	2 (5.7)	≥6*	5 (14.3)*	
Other/Unknown/Not Reported	2 (5.7)	Small Cell Lung Cancer	2 (5.7)	* ≥6 prior lines: 3 with 6; 1 with 7; 1 with 8		
		Bladder	1 (2.9)	Prior Exposure to IO Therapy, n (%)		
		Cervical	1 (2.9)	Yes	26 (74.3)	
				No	9 (25.7)	

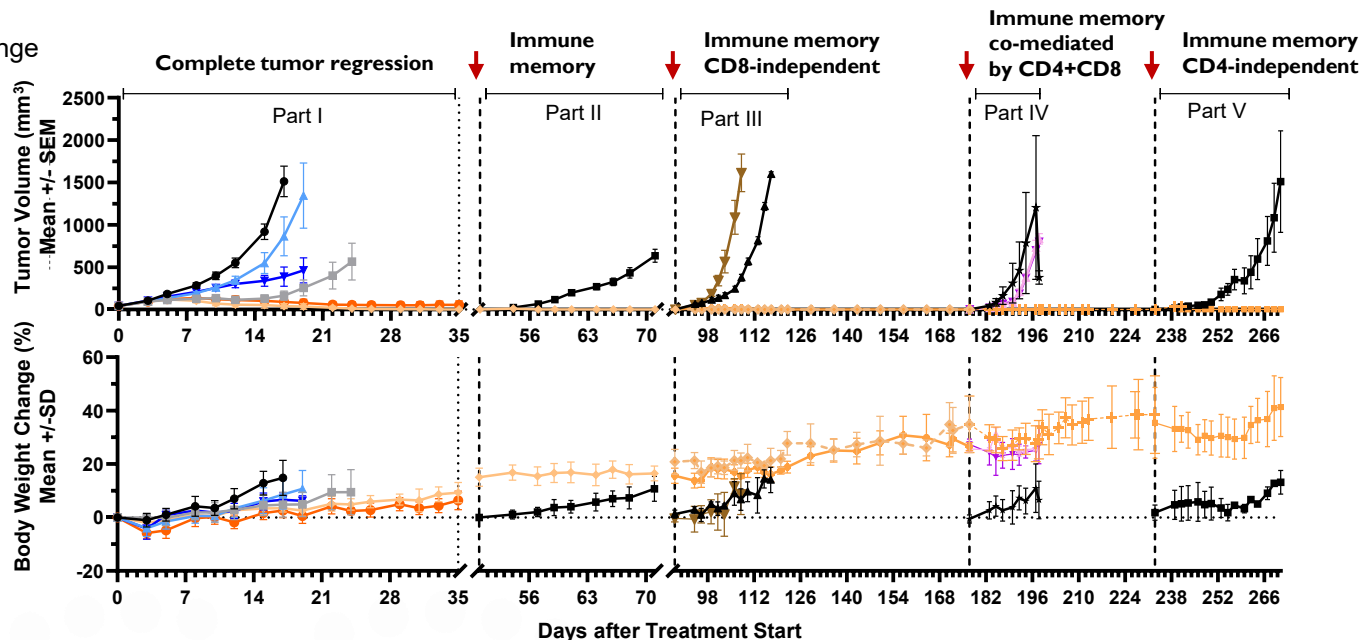
NSCLC = non-small cell lung cancer.
SCC = squamous cell carcinoma.

Non QC'd data based on EDC as of 07/31/2026

POTENT ACR-2316 + ANTI-PD-L1 SYNERGY WITH COMPLETE TUMOR REGRESSION AND IMMUNE MEMORY MEDIATED BY CD4+ AND CD8+ CELLS



↓ Tumor cell re-challenge



AACR 2026: ACR-2316 SYNERGY WITH CHECKPOINT INHIBITION

Poster # 3789

Treatment with ACR-2316, a potential first- and best-in-class WEE1/PKMYT1 inhibitor, combined with anti-PD-L1 induces complete tumor regression with durable immune memory in a preclinical syngeneic model

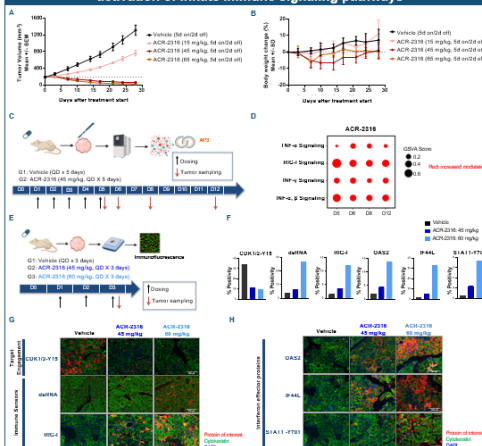
Taronish Dubashi¹, Joelle Baddour-Sousounis¹, Amira Elbakry¹, Jessica Hopkins¹, Suboth Kumar¹, Yingchun Spring Liu¹, Ahmed Youssef¹, Kate Rappard¹, Ignacio Arribas Diez¹, Georgina Mitsa¹, Marc Jansson¹, Francisco Santana¹, Maria Rodriguez-Zabala¹, Luka Romero¹, Zachary Best¹, Nina Lipjanick¹, Anna Maria Alves¹, Daphne Garcia-Lopez¹, Portia Lombardo¹, Calvin Yang¹, Emma Ahlman¹, Valentina Sino¹, Reina Imogho¹, Magnus E. Jakobsson¹, Helen Nilsson¹, Jaysha Mumbesh¹, Lei Shi¹, Caroline Wigenrup¹, Michail Shiptsin¹, Joon Jung¹, David Proia¹, Enck Gamelin¹, Kristina Masson¹, Peter Blume-Jensen¹
¹Acvion Therapeutics Inc., Watertown MA, USA; ²Acvion AB, Medicon Village, Lund, Sweden



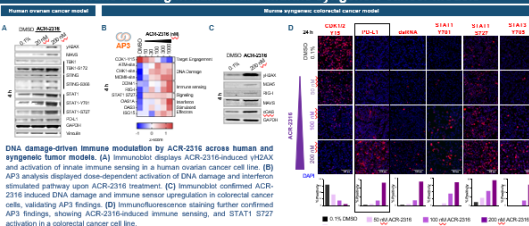
Introduction

- WEE1 and PKMYT1 have critical roles in the S and G2M checkpoint and replication stress response. ACR-2316 is a potent WEE1/PKMYT1 inhibitor designed by Acvion's Predictive Precision Proteomics (AP3) for superior pro-apoptotic tumor cell killing through blockade of PKMYT1, uncovered by AP3 as a dominant resistance mechanism to WEE1 inhibition, resulting in potent activation of not only CDK1 and 2, but importantly also PLK-1.
- Based on this mechanism, ACR-2316 is expected to exacerbate tumor-intrinsic DNA damage and induce strong mitotic catastrophe in genetically unstable cancers, resulting in activation of innate immune sensing pathways, providing a mechanistic link between replication stress and adaptive anti-tumor immunity.
- Here, we evaluated ACR-2316 in human and murine syngeneic cancer models to characterize its effects on DNA damage response signaling and immune activation. We further assessed combination with anti-PD-L1 to determine whether DDR-driven tumor priming enhances tumor regression and induces durable immune memory, and profiled associated signaling using our AP3 platform.

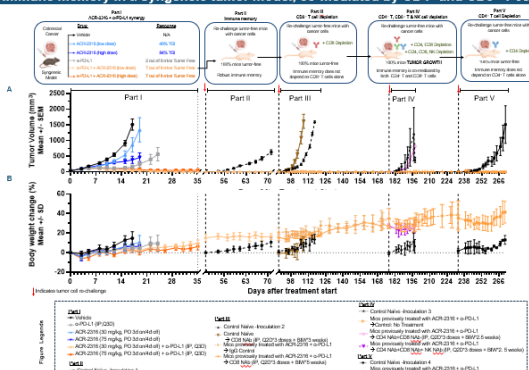
AP3 profiling of ACR-2316-treated tumors reveals activation of innate immune signaling pathways



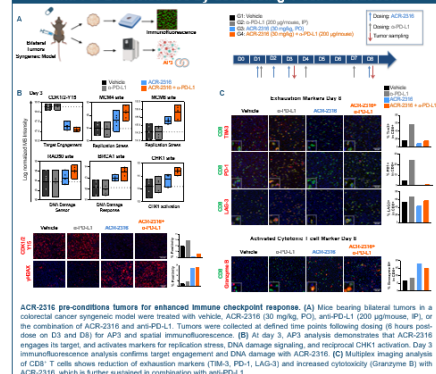
ACR-2316 induces tumor-intrinsic DNA damage and activates multi-dimensional immune sensing in human and murine syngeneic cancer models



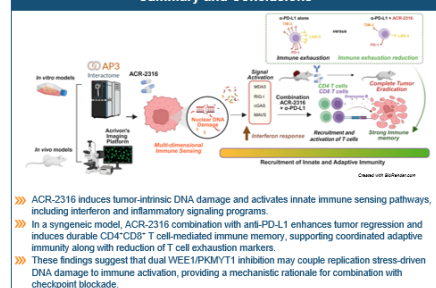
ACR-2316 synergizes with anti-PD-L1 to induce complete response and durable immune memory in a syngeneic tumor model, co-mediated by CD4⁺ and CD8⁺ T cells



ACR-2316 boosts immune-mediated tumor killing and overcomes anti-PD-L1 resistance by decreasing T-cell exhaustion

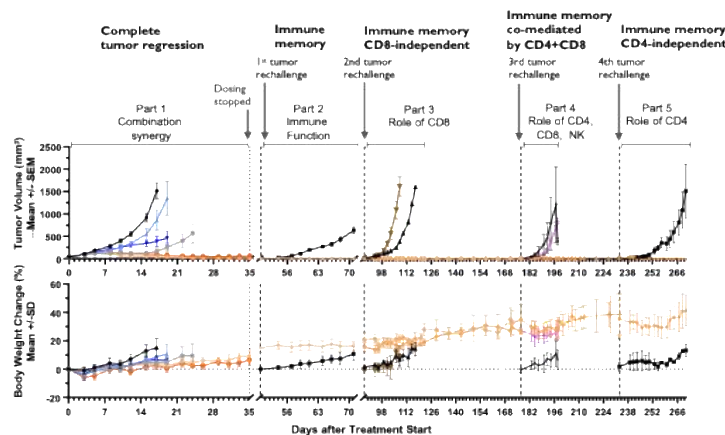


Summary and Conclusions

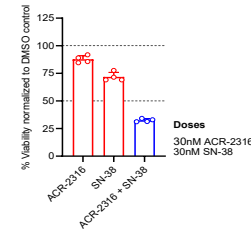
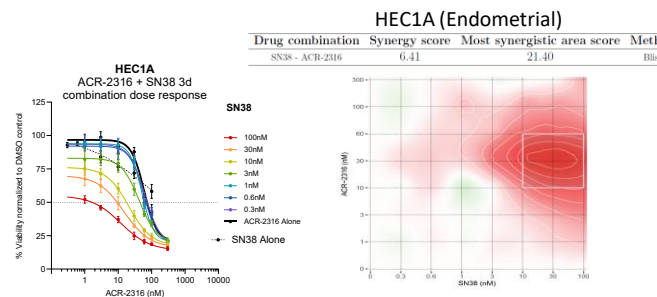


POTENTIAL DEVELOPMENT PATHS FOR ACR-2316

- AP3-predicted solid tumor types of high unmet need, e.g. SCLC, sqNSCLC, HPV+ tumors, etc., in $\geq 2^{\text{nd}}$ line
 - Single agent
 - Chemo combination (DDR stress sensitization)
- Just like for ACR-368, the weekly and bi-weekly dosing regimens of ACR-2316 provide flexible opportunities to move towards frontline combinations with both anti-PD(L)I and TOPOI payload ADCs based on strong synergy observed across preclinical models in AP3-predicted solid tumor types



ACR-2316 + anti-PD-L1 therapy results in complete regression and permanent CD4 + CD8-mediated immune memory

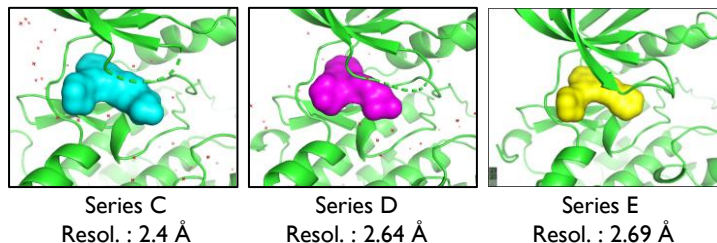


Max synergy score = 34

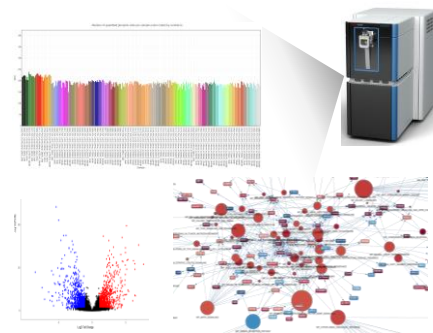
ACR-2316 + TOPOI payload ADCs show strong synergy in preclinical models

CDK11 – AN ESSENTIAL REGULATOR OF RNA TRANSCRIPTION AND SPLICING

- No clinical-stage competitors
- Attractive cancer cell cycle drug target, well-suited for AP3 platform, multiple protein isoforms
- Broad role in cell cycle control and oncogenesis; CDK11 controls transcription, pre-mRNA processing and splicing of mitotic genes, and is predicted to be a key driver in aggressive AML
- AP3 profiling (benchmarks/leads) for MOA-based SAR and lead optimization
- **Development candidates: ACR-6840 + equally promising back-up series**
 - **Small molecule CDK11 inhibitor, highly selective (kinome scan), orally available, potent (single digit nM cellular target engagement; <30 nM EC50 viability), preclinical activity with complete tumor regression**



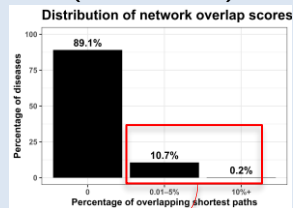
All AP3-driven programs deploy co-crystallography



AP3-based screening funnel guides intracellular protein SAR

AP3-BASED INDICATION FINDING IDENTIFIES AML AS ONE OF THE TOP INDICATIONS FOR CDK11 INHIBITION

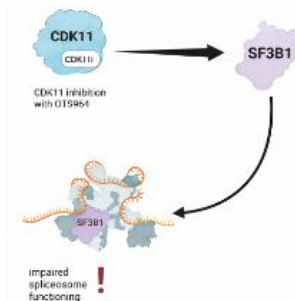
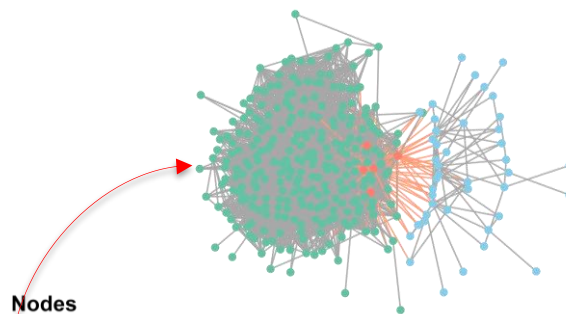
AP3 Indication Screening for CDK11 inhibitor (>2000 diseases)



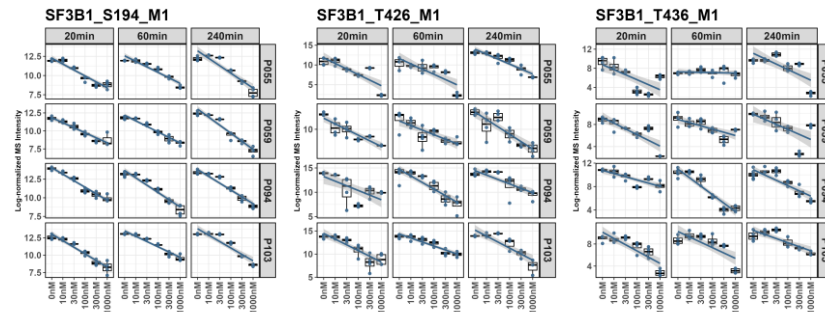
Top 10 indications matching AP3 profile

Disease	Classification	Rank
T-cell non-Hodgkin lymphoma	Hematologic	1
Cutaneous T-cell lymphoma	Hematologic	2
Malignant glioma	Solid tumor	3
Peripheral T-cell lymphoma	Hematologic	4
Lysosomal storage disease	Metabolic	5
T-cell acute lymphoblastic leukemia	Hematologic	6
Chronic lymphocytic leukemia	Hematologic	7
Parkinson disease	Neurodegenerative	8
Acute myeloid leukemia	Hematologic	9
Alzheimer disease	Neurodegenerative	10

Overlap of AML disease network with CDK11 inhibitor AP3 profile

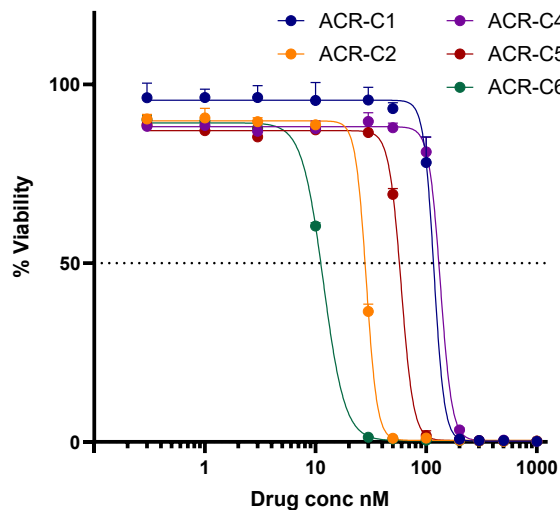


Dose-dependent regulation of SF3B1 phosphorylation sites (AP3 experiments, n=4)

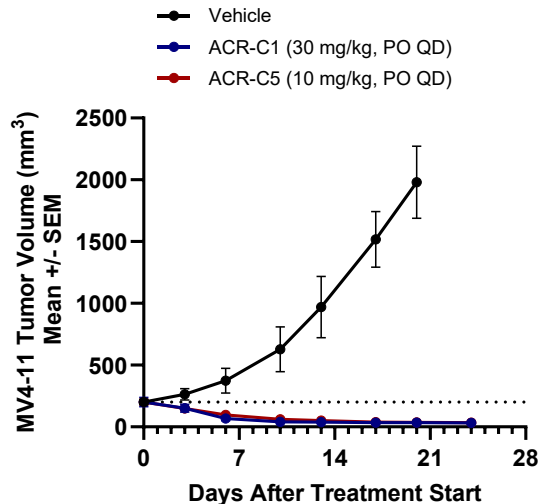


CDK11 INHIBITOR LEAD SERIES SHOWS COMPLETE AML TUMOR REGRESSION AND FAVORABLE TOLERABILITY

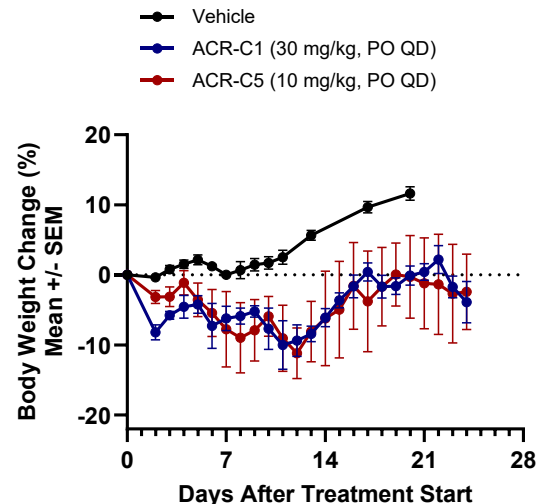
Multiple Lead Compounds



Complete Regression In Vivo



Favorable Safety Profile

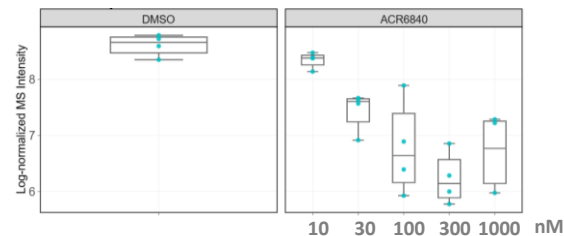


CDK11 INHIBITOR ACTIVITY ALONE AND IN COMBINATION WITH BCL-2 FAMILY INHIBITORS IN HEMATOLOGICAL MALIGNANCIES

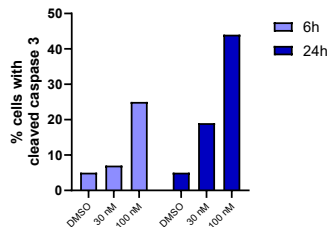
Rationale: CDK11 inhibition sensitizes tumors to BCL-2 family inhibitors through MCL1 downregulation and dysregulated splicing

- Dysregulated splicing of anti-apoptotic transcripts to pro-apoptotic (Bcl-xL to Bcl-xS) providing strong rationale for combination with BCL2 inhibitors

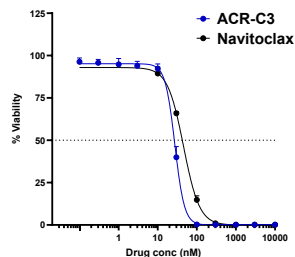
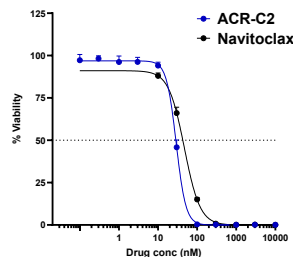
ACR CDK11 inhibitors reduce levels of anti-apoptotic MCL1 (AP3 global proteomics analysis)



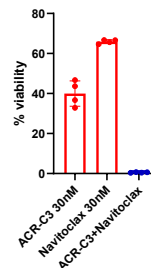
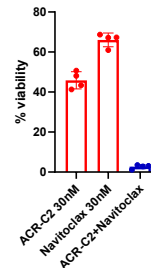
CDK11 inhibitors induce apoptosis in aggressive AML cell line MV4-I1



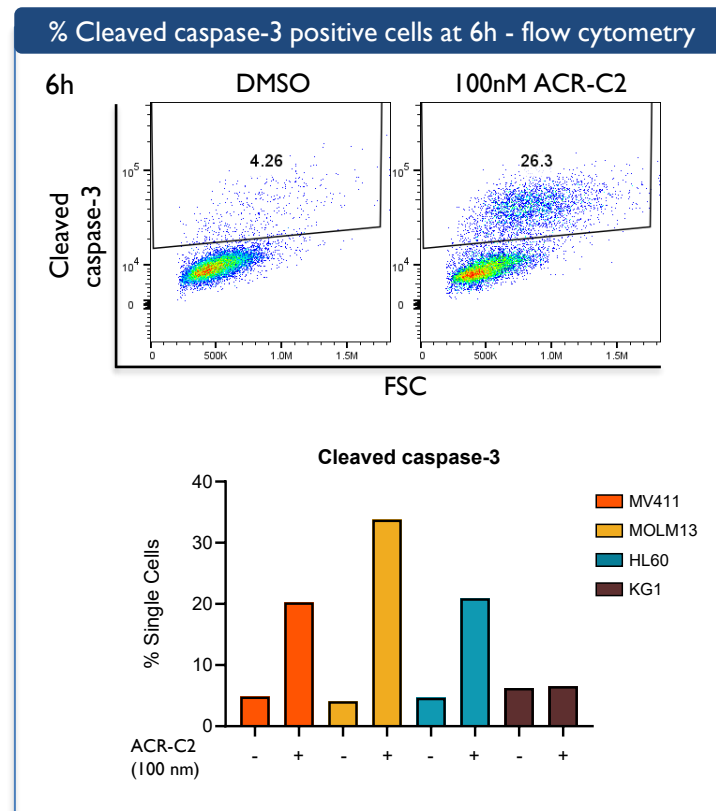
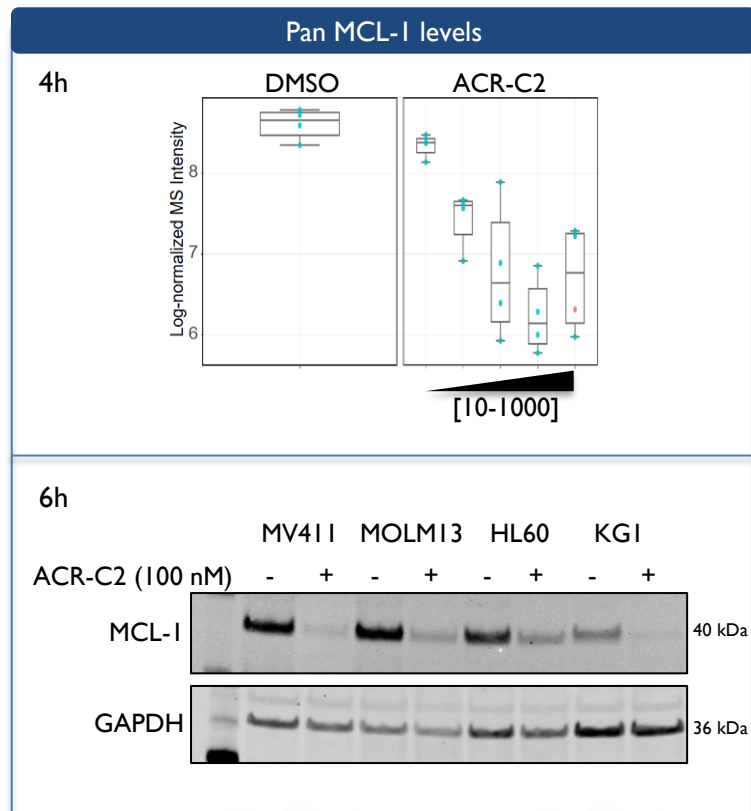
ACR CDK11 inhibitors are as potent as BCL-2 inhibitor in AML cell line



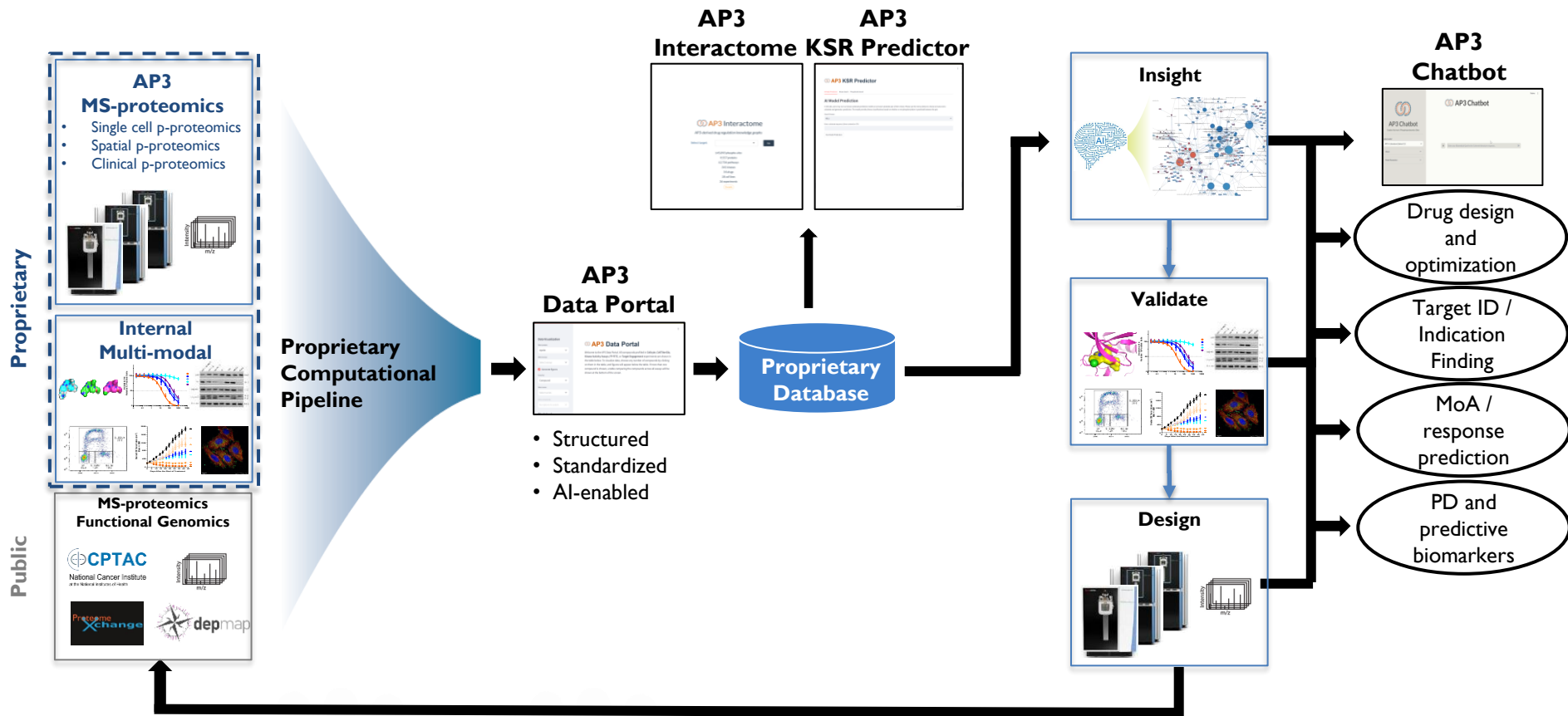
ACR CDK11 inhibitors exhibit synergistic activity with BCL-2 inhibitor in AML cell line



ACR-C2 DOWNREGULATES ANTI-APOPTOTIC MCL-1 PROTEIN AND INDUCES CELL DEATH AFTER 6H TREATMENT



AP3 GENERATIVE PHOSPHOPROTEOMICS PLATFORM

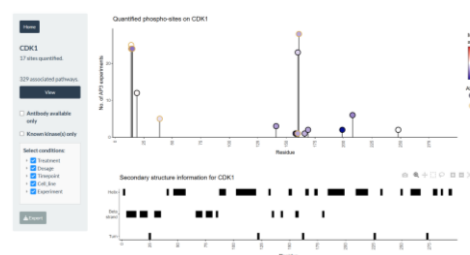
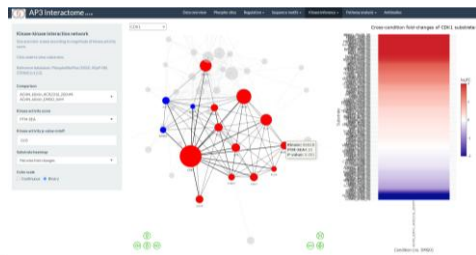
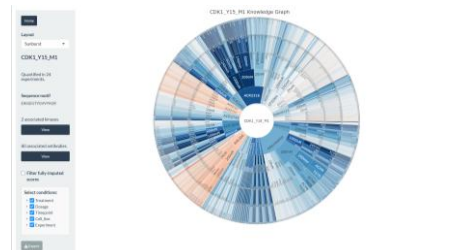


AP3 INTERACTOME: PROPRIETARY INTERACTIVE DATA ANALYSIS INFRASTRUCTURE FOR ALL ACRIVON DATA AND EXPERIMENTS

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

~164,000 phosphosites

~50,000 phosphosites



DATA DRIVEN EXECUTION AND VALUE CREATION

Recent Accomplishments (Last 12 Months)

- ✓ Continued positive ACR-368 data in ongoing registrational intent EC trial (April 2025 and January 2026 Corporate R&D Events)
- ✓ Strong enthusiasm for promising ACR-368 clinical data at 2026 ESGO late breaking oral presentation and corporate live KOL expert panel (Q1 2026)
- ✓ cORR of 52% observed in serous EC, a high unmet need disease w/ limited treatment options contributing to ~50% of all EC mortality
- ✓ Ongoing prospective enrollment (US + EU) in serous EC (biopsy independent) Arm 3 (ACR-368 + ULDG) in Phase 2b trial (Q4 2025)
- ✓ Ongoing prospective enrollment (US + EU) in serous EC (biopsy independent) Arm 4 (ACR-368) in Phase 2b trial (Q2 2026)
- ✓ 4 presentations showcasing expanded AP3 capabilities and compelling ACR-2316 preclinical data (AACR Q2 2025 and AACR-NCI-EORTC Q4 2025)
- ✓ Provided ACR-2316 Phase 1/2 dose escalation clinical data and transitioned into randomized dose expansion in AP3-identified solid tumor types (Q3 2026)

Anticipated Next Milestones

ACR-368 (Phase 2b)

- Prespecified simultaneous interim analysis and data update from both all-comer (biopsy-independent) serous EC arms of the ACR-368 Phase 2b study in second half of 2026
- Initiate Phase 3 confirmatory trial for ACR-368 in first half of 2027
- Based on interim data read-out, complete enrollment of the registrational intent all-comer (biopsy-independent) serous EC Arm 3 or Arm 4 by fourth quarter of 2026

ACR-2316 (Phase 1/2)

- ✓ Additional ACR-2316 Phase 1/2 clinical data for weekly and bi-weekly dosing regimens and transition into dose expansion in AP3-identified tumor types in 2026

Earlier Pipeline

- Submit IND filing to the FDA for CDK11 inhibitor development candidate in first half of 2027
- Initiate additional internal programs utilizing the AP3 platform in 2026

FINANCIAL HIGHLIGHTS

Cash and Investments

\$90.0M

Balance sheet
30-Jun-2026

Projected runway into

Q4 2027

Current operating plan, assuming
no additional financing

Fully Diluted Shares Outstanding

52.0M

Shares and equity grants
outstanding 30-Jun-2026

Note: unaudited