UNITED STATES
SECURITIES AND EXCHANGE COMMISSION
WASHINGTON, D.C. 20549

FORM 8-K

CURRENT REPORT

Pursuant to Section 13 or 15(d) of the Securities Exchange Act of 1934

Date of Report (Date of earliest event reported): November 20, 2026

Acrivon Therapeutics, Inc.

(Exact name of Registrant as Specified in Its Charter)

Delaware	**001-41551**	**82-5125532**
(State or Other Jurisdiction of Incorporation)	**(Commission File Number)**	**(IRS Employer Identification No.)**

480 Arsenal Way **Suite 100** **Watertown, Massachusetts** **(Address of Principal Executive Offices)**	**02472** **(Zip Code)**

Registrant's Telephone Number, Including Area Code: (617) 207-8979

(Former Name or Former Address, if Changed Since Last Report)

Check the appropriate box below if the Form 8-K filing is intended to simultaneously satisfy the filing obligation of the registrant under any of the following provisions:

☐ Written communications pursuant to Rule 425 under the Securities Act (17 CFR 230.425)

☐ Soliciting material pursuant to Rule 14a-12 under the Exchange Act (17 CFR 240.14a-12)

☐ Pre-commencement communications pursuant to Rule 14d-2(b) under the Exchange Act (17 CFR 240.14d-2(b))

☐ Pre-commencement communications pursuant to Rule 13e-4(c) under the Exchange Act (17 CFR 240.13e-4(c))

Securities registered pursuant to Section 12(b) of the Act:

Title of each class	**Trading Symbol(s)**	**Name of each exchange on which registered**
Common Stock, par value $0.001 per share	ACRV	The Nasdaq Stock Market LLC

Indicate by check mark whether the registrant is an emerging growth company as defined in Rule 405 of the Securities Act of 1933 (§ 230.405 of this chapter) or Rule 12b-2 of the Securities Exchange Act of 1934 (§ 240.12b-2 of this chapter).

Emerging growth company ☒

If an emerging growth company, indicate by check mark if the registrant has elected not to use the extended transition period for complying with any new or revised financial accounting standards provided pursuant to Section 13(a) of the Exchange Act. ☐

Item 7.01 Regulation FD Disclosure.

On November 20, 2026, at the 38th EORTC-NCI-AACR Symposium in Barcelona, Spain, Dr. Timothy A. Yap, MBBS, Ph.D., FRCP, of The University of Texas MD Anderson Cancer Center presented a late-breaker oral plenary presentation that showed results from the dose escalation phase of the Company's ACR-2316 Phase 1/2 trial (the "38th EORTC-NCI-AACR Symposium Deck").

A Copy of the Deck is attached hereto as Exhibit 99.1, and is incorporated by reference into this Item 7.01 of this Current Report on Form 8-K. The Company also posted a copy of the Deck to the "Science & Publications" section of the Company's website at www.acrivon.com.

The information in this Item 7.01, including Exhibit 99.1 attached hereto, shall not be deemed "filed" for purposes of Section 18 of the Exchange Act, or otherwise subject to the liabilities of that section, nor shall it be deemed incorporated by reference in any filing under the Securities Act or the Exchange Act, except as expressly set forth by specific reference in such filing.

Item 9.01 Financial Statements and Exhibits.

Exhibit Number	Description
99.1	38th EORTC-NCI-AACR Symposium Deck
104	Cover Page Interactive Data File (embedded within the Inline XBRL document)

SIGNATURES

Pursuant to the requirements of the Securities Exchange Act of 1934, the registrant has duly caused this report to be signed on its behalf by the undersigned hereunto duly authorized.

Acrivon Therapeutics, Inc.

Date: November 20, 2026

By: /s/ Peter Blume-Jensen, M.D., Ph.D.
Name: Peter Blume-Jensen, M.D., Ph.D.
Title: President and Chief Executive Officer

Exhibit 99.1

ENA 2026
EORTC NCI AACR
38th Symposium

Safety, dose optimization, and early Phase 1/2 clinical results in AP3-predicted tumors for ACR-2316, a first-in-class WEE1/PKMYT1 inhibitor

Timothy A. Yap[1], Mohamad A. Salkeni[2], Neel Gandhi[3], Gerald Falchook[4], David Sommerhalder[5], Benedito A. Carneiro[6], Judy Wang[7], Monica Mita[8], Kamlesh K. Sankhala[9], Jeffery Russell[10], David Levitz[11], Lu Gan[12], Besiana P. Beqo[12], Kristina Masson[12], Christina A. Scherer[12], Erick Gamelin[12], Eric Devroe[12], Peter Blume-Jensen[12], Mansoor R. Mirza[12], Nicole Nevadunsky[11]

[1] MD Anderson Cancer Center, Houston, TX, USA
[2] NEXT Oncology – Virginia, Fairfax, VA, USA
[3] Carolina BioOncology Institute, Huntersville, NC, USA
[4] SCRI HealthONE, Denver, CO, USA
[5] NEXT Oncology – San Antonio, San Antonio, TX, USA
[6] Legorreta Cancer Center, Brown University, Providence, RI, USA
[7] Florida Cancer Specialists / Sarah Cannon Research Institute, Sarasota, FL, USA
[8] Hoag Memorial Hospital Presbyterian, Newport Beach, CA, USA
[9] Precision NextGen Oncology & Research Center, Beverly Hills, CA, USA
[10] Tennessee Oncology, Nashville, TN, USA
[11] Montefiore Medical Center Einstein, Bronx, NY, USA
[12] Acrivon Therapeutics, Inc., Watertown, MA, USA

EORTC European Organisation for Research and Treatment of Cancer
NIH NATIONAL CANCER INSTITUTE
AACR American Association for Cancer Research

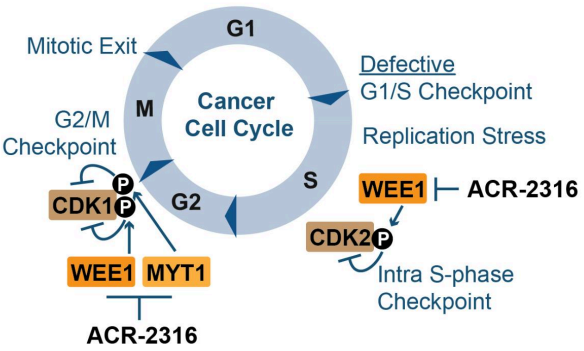
Disclosures

Timothy A. Yap

- **Employee of:** University of Texas MD Anderson Cancer Center, where I am Vice President, Head of Clinical Development in the Therapeutics Discovery Division, which has a commercial interest in IACS-6274 and other inhibitors (IACS30380/ART0380 was licensed to Artios)
- **Consultant for**: AbbVie, Acrivon, Adagene, Almac, Aduro, Amphista, Artios, Astex, AstraZeneca, Athena, Atrin, Avenzo, Avoro, Axiom, Baptist Health Systems, Bayer, BeiGene, BioCity Pharma, Blueprint, Boxer, Bristol Myers Squibb, C4 Therapeutics, Calithera, Cancer Research UK, Carrick Therapeutics, Circle Pharma, Clovis, Cybrexa, Daiichi Sankyo, Dark Blue Therapeutics, Diffusion, Duke Street Bio, 858 Therapeutics, EcoR1 Capital, Ellipses Pharma, EMD Serono, Entos, F-Star, Genesis Therapeutics, Genmab, Glenmark, GLG, Globe Life Sciences, GSK, Guidepoint, Ideaya Biosciences, Idience, Ignyta, I-Mab, ImmuneSensor, Impact Therapeutics, Institut Gustave Roussy, Intellisphere, Jansen, Kyn, MEI pharma, Mereo, Merck, Merit, Monte Rosa Therapeutics, Nested Therapeutics, Nexys, Nimbus, Novocure, Odyssey, OHSU, OncoSec, Ono Pharma, Onxeo, PanAngium Therapeutics, Pegascy, PER, Pfizer, Piper-Sandler, Pliant Therapeutics, Prolynx, Radiopharma Theranostics, Repare, resTORbio, Roche, Ryvu Therapeutics, SAKK, Sanofi, Schrodinger, Servier, Synnovation, Synthis Therapeutics, Tango, TCG Crossover, TD2, Terremoto Biosciences, Tessellate Bio, Theragnostics, Terns Pharmaceuticals, Tolremo, Tome, Thryv Therapeutics, Trevarx Biomedical, Varian, Veeva, Versant, Vibliome, Voronoi Inc, Xinthera, Zai Labs, and ZielBio
- **Grant/Research support from:** Acrivon, Aprea, Artios, AstraZeneca, Bayer, BeiGene, BioNTech, Blueprint, Bristol Myers Squibb, Boundless Bio, Clovis, Constellation, Cyteir, Eli Lilly, EMD Serono, Forbius, F-Star, GlaxoSmithKline, Genentech, Haihe, Ideaya ImmuneSensor, Insilico Medicine, Ionis, Ipsen, Jounce, Karyopharm, KSQ, Kyowa, Merck, Mirati, Novartis, Pfizer, Ribon Therapeutics, Regeneron, Repare, Rubius, Sanofi, Scholar Rock, Seattle Genetics, Tango, Tesaro, Vivace, and Zenith.

WEE1 and PKMYT1 - critical cell cycle checkpoints in human cancer

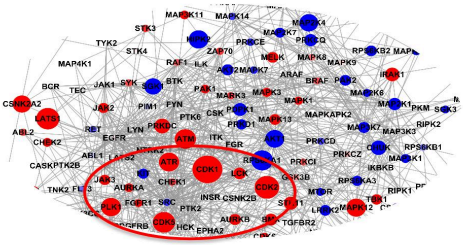
- Defective DNA repair is highly prevalent in cancers, creating a dependency on S- and G2/M checkpoint proteins, such as WEE1 and PKMYT1
- Acrivon Predictive Precision Proteomics (AP3) measures the global cellular effects of a drug on the protein signaling network in an unbiased manner
- AP3 was applied to design a highly differentiated, first-in-class WEE1/PKMYT1 inhibitor that activates CDK1, CDK2 as well as PLK1 to achieve potent single agent activity and high selectivity for improved Therapeutic Index



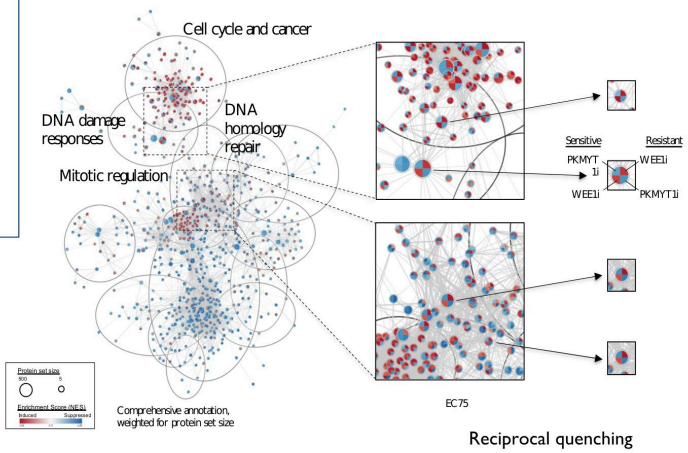
ACR-2316: uniquely designed by AP3 for superior therapeutic index over WEE1 and PKMYT1 inhibitors

A differentiated, potentially first-in -class WEE1/PKMYT1 inhibitor

- ✓ Designed for high selectivity for WEE1 and PKMYT1 to overcome resistance and to optimize safety
- ✓ Potent activation of CDK1, CDK2 as well as PLK1 to ensure strong mitotic catastrophe and tumor cell killing
- ✓ Complete tumor regression across all preclinical *in vivo* models



Intracellular activation (red) of CDK1, 2, and PLK1

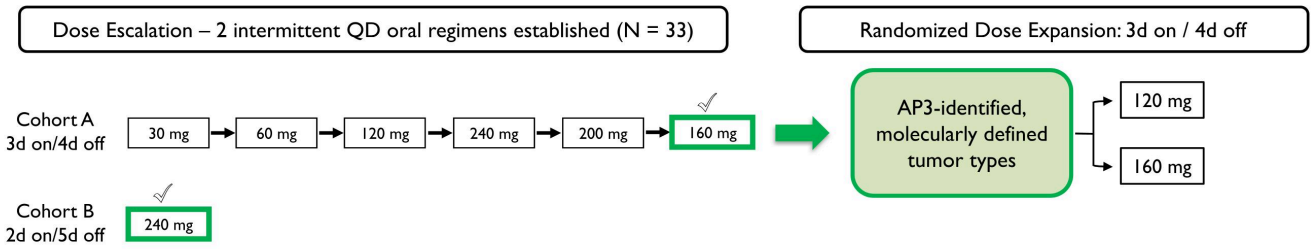


Reciprocal quenching

Drug Designed by AP3 to suppress resistance mechanisms

Study design and objectives

Phase 1/2, first-in-human, open-label, multicenter study of oral ACR-2316 in advanced solid tumors



Objectives (Dose escalation)

- **Primary:** MTD during 1st dosing cycle (21 days Cohort A/B)
- **Secondary:** safety and tolerability (TEAEs/TRAEs, CTCAE v5.0); PK of ACR-2316 and its metabolites
- **Exploratory:** antitumor activity – confirmed ORR (RECIST v1.1), DOR, CBR; PD markers in PBMCs; exposure–safety

Key eligibility (Dose escalation)

- Advanced solid tumor progressed after ≥1 prior line; ≤3 prior lines for recurrent disease
- ECOG PS 0–1; measurable disease (RECIST v1.1); adequate organ function
- Tumor types: SCLC, sqNSCLC, a denoNSCLC, high-grade endometrial, ovarian, cervical, anal, vulvar, HPV+ oropharyngeal, small-cell neuroendocrine prostate; other tumors if p53 mutated

Oral, once daily, 3-week cycles, until progression or unacceptable toxicity. Tumor assessments per RECIST v1.1 every 6 weeks.

ACR-2316 Patient demographics and baseline characteristics

Subject Demographics	N = 33
Median age, years (range)	63 (35–77)
Sex, n (%)	
Female	20 (60.6)
Male	13 (39.4)
Race, n (%)	
White	26 (78.8)
Black/African American	3 (9.1)
Asian	1 (3.0)
Native Hawaiian/Pacific Islander	1 (3.0)
Other/Unknown/Not Reported	2 (6.1)

Cancer Type, n (%)	N = 33
Endometrial	5 (15.2)
Ovarian / fallopian tube	4 (12.1)
Breast	4 (12.1)
Adenocarcinoma NSCLC	3 (9.1)
Colorectal	3 (9.1)
Head & Neck SCC	3 (9.1)
Squamous NSCLC	3 (9.1)
Pancreatic / ampullary	2 (6.1)
Prostate	2 (6.1)
Small Cell Lung Cancer	2 (6.1)
Urothelial	1 (3.0)
Vulvar	1 (3.0)

ECOG at Baseline, n (%)	N = 33
0	10 (30.3)
1	23 (69.7)
Prior Therapy Lines, n (%)	
1	1 (3.0)
2	5 (15.2)
3	14 (42.4)
4	7 (21.2)
5	1 (3.0)
≥6*	5 (15.2)
Median prior lines (range)	3 (1–8)
Prior IO Therapy, n (%)	
Yes	25 (75.8)
No	8 (24.2)

* ≥6 prior lines: 3 with 6, 1 with 7, 1 with 8.

Data cut-off 02-Nov-2026; non-QC'd EDC data.

ACR-2316 Safety: treatment-related adverse events

Grade ≥3 TRAEs (≥5% overall) – Dose-escalation population, Cohort A 120 mg and 160 mg

Grade ≥3 TRAEs	Overall N = 33 (Cohorts A and B)			Cohort A 120 mg (n = 4)			Cohort A 160 mg (n = 4)		
	Grade 3 n (%)	Grade 4 n (%)	Grade ≥3 n (%)	Grade 3 n (%)	Grade 4 n (%)	Grade ≥3 n (%)	Grade 3 n (%)	Grade 4 n (%)	Grade ≥3 n (%)
Neutropenia	5 (15.2)	10 (30.3)	15 (45.5)	1 (25.0)	0	1 (25.0)	1 (25.0)	0	1 (25.0)
Leukopenia	3 (9.1)	4 (12.1)	7 (21.2)	0	0	0	0	0	0
Anemia	6 (18.2)	0	6 (18.2)	1 (25.0)	0	1 (25.0)	0	0	0
ALT increased	2 (6.1)	0	2 (6.1)	0	0	0	1 (25.0)	0	1 (25.0)
Lymphopenia	2 (6.1)	0	2 (6.1)	0	0	0	0	0	0
TOTAL unique subjects	**15 (45.5)**	**11 (33.3)**	**19 (57.6)**	**2 (50.0)**	**0**	**2 (50.0)**	**2 (50.0)**	**0**	**2 (50.0)**

Highly differentiated, favorable safety profile

Grade ≥3 AEs were predominantly asymptomatic, transient, mechanism-based neutropenia and leukopenia

No non-hematological DLTs, no febrile neutropenia, and no Grade 5 AEs were observed

TRAE = treatment-related adverse event (related or possibly related); worst CTCAE v5.0 grade per subject.

Data cut-off 02-Nov-2026; non-QC'd EDC data.

Tolerability of ACR-2316

Dose-escalation population (N = 33), Cohort A 120 mg and 160 mg

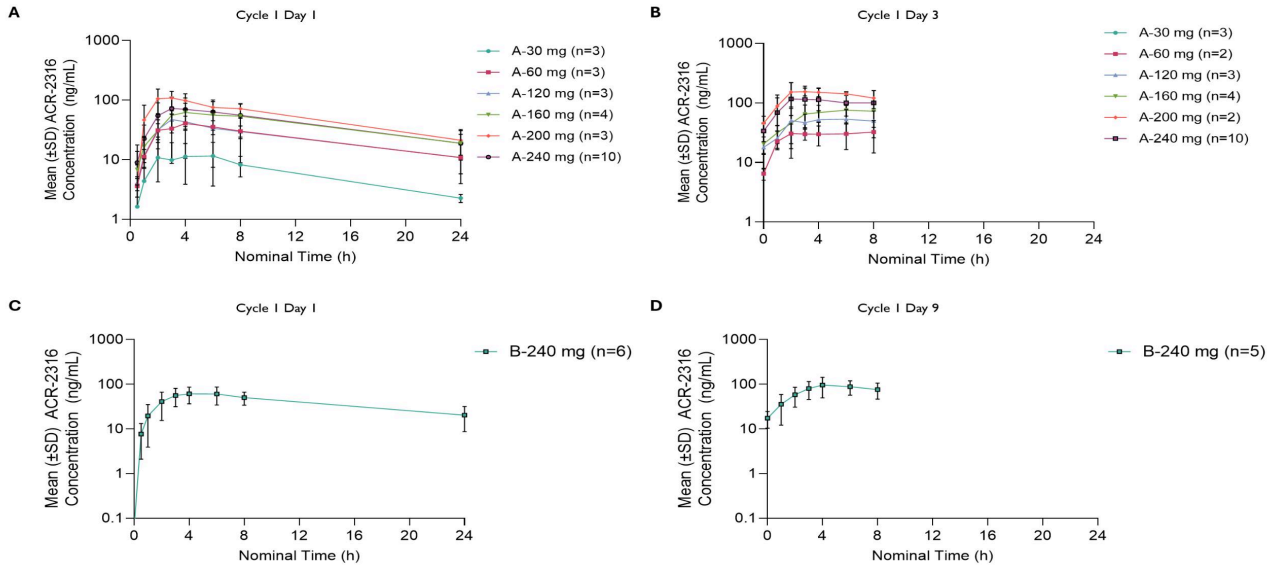
Tolerability endpoints n (%)	Overall N = 33	120 mg n = 4	160 mg n = 4
Any TRAE	26 (78.8)	3 (75.0)	3 (75.0)
Grade 3 TRAE	8 (24.2)	2 (50.0)	2 (50.0)
Grade 4 TRAE	11 (33.3)	0	0
Serious TRAE	5 (15.2)	0	0
TRAE → dose interruption	19 (57.6)	2 (50.0)	2 (50.0)
TRAE → dose reduction	8 (24.2)	0	1 (25.0)
TRAE → discontinuation	1 (3.0)	0	0
DLT /DLT-evaluable	8/29 (27.6)	0/4 (0.0)	0/3 (0.0)

No Grade 4 TRAEs or DLTs observed at 120 or 160 mg (Cohort A; 3 on / 4 off)
All DLTs were neutropenia at doses >160 mg (no febrile neutropenia)
120 and 160 mg selected for randomized dose expansion (Project Optimus)

TRAE = treatment-related adverse event; DLT = dose-limiting toxicity. Dose interruption includes delays and holds.

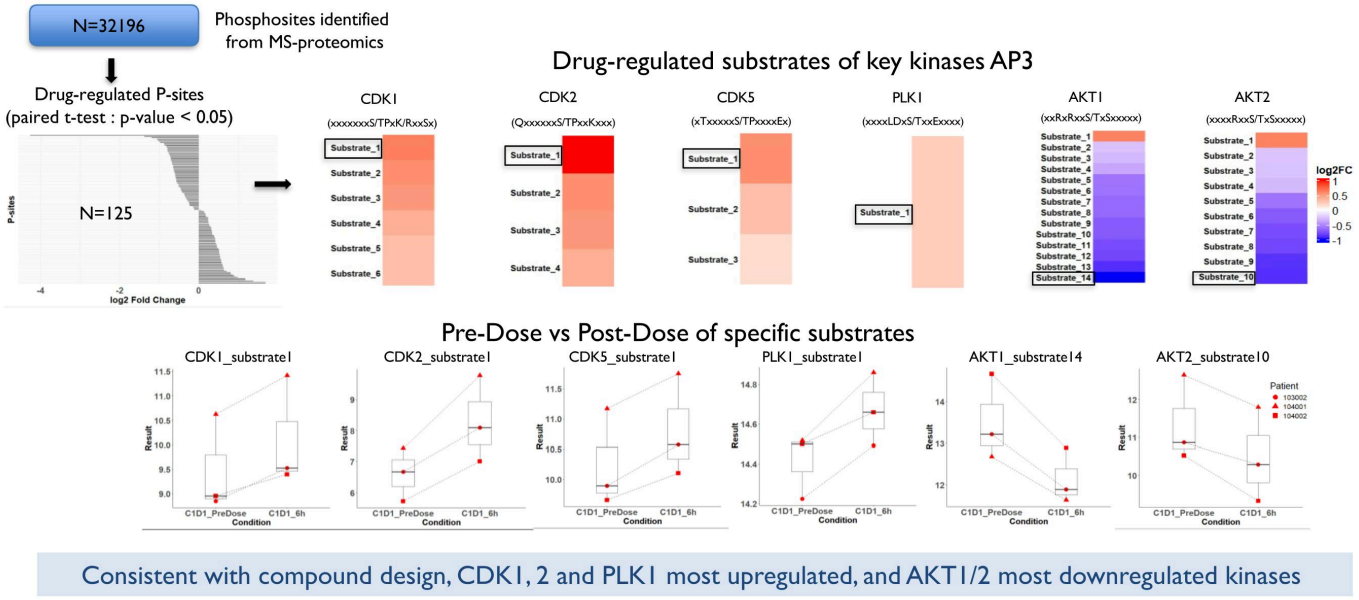
Data cut-off 02-Nov-2026; non-QC'd EDC data.

Favorable dose-proportional PK of ACR-2316 with minimal accumulation



T1/2 ~ 12h

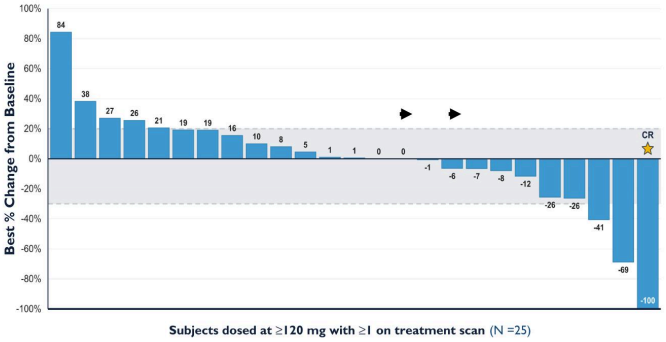
Drug target engagement of ACR-2316 in patient PBMCs from 1st dose level (AP3 MS assay)



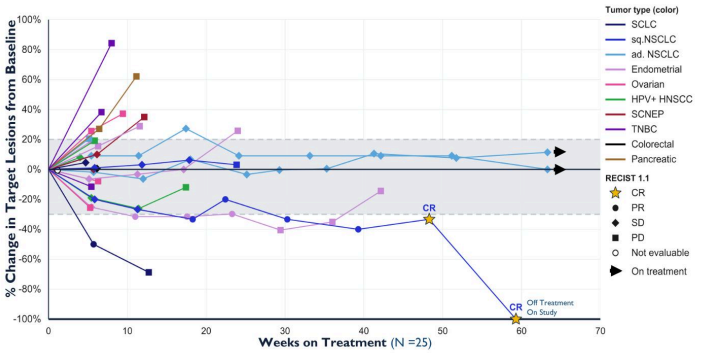
Consistent with compound design, CDK1, 2 and PLK1 most upregulated, and AKT1/2 most downregulated kinases

Promising clinical activity of ACR-2316 observed at doses ≥120 mg

Best Tumor Shrinkage



Target Lesion Change Over Time



SqNSCLC

Confirmed CR · 160 mg

Molecular profile: BRCA1 p.L392fs (germline susp.), TP53 p.H179L, FGFR1 gain; PD-L1 TPS <1%, MSS, TMB 4.5 mut/Mb

Prior lines (3) 1) carbo/tax + RT → durva 2) pembro → nivo + ipi 3) PD-1×PD-L1 bispecific

Endometrial

Confirmed PR (-41%) · 120 mg

Molecular profile: MSS, PTEN, BRCA1/2$_{wt}$, TP53$_{wt}$, POLE$_{wt}$; no tumor NGS

Prior lines (4) 1) carbo/tax 2) carbo/tax 3) tamoxifen 4) pembro + lenva

SCLC

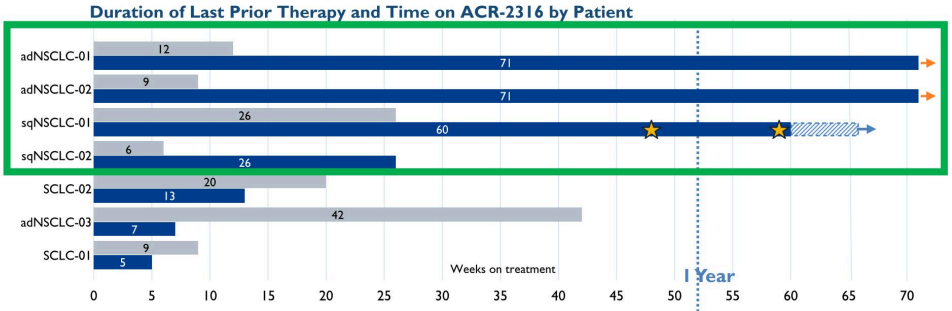
Unconfirmed PR (-69%) · 160 mg

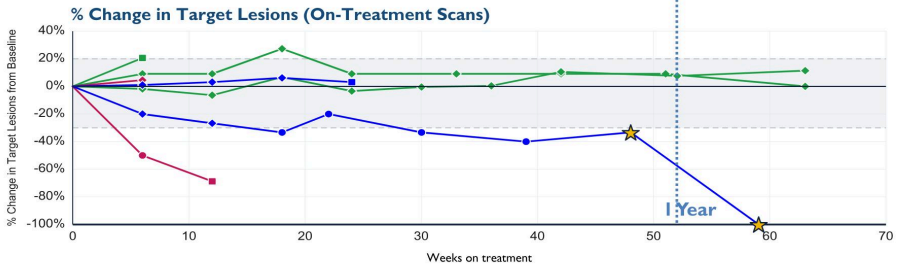
Molecular profile: TP53 p.E286V, RB1 loss, FGFR2 p.M538I (activating); MSS, TMB 7.4 mut/Mb (low)

Prior lines (3) 1) cis/etop + RT 2) durva 3) tarlatamab

Data cut-off 02-Nov-2026; non-QC'd EDC data.

Durable clinical activity of ACR-2316 in heavily pretreated lung cancer patients







7 Lung Cancer Patients (≥120 mg)

- **Squamous NSCLC:** confirmed CR and CMR (FDG-PET) on study >65 weeks
- **AdenoNSCLC:** 2 patients with SD, on treatment >71 weeks
- **Squamous NSCLC:** SD >25 weeks
- **SCLC:** deep tumor shrinkage (uPR)



Data cut-off 02-Nov-2026; non-QC'd EDC data.

ACR-2316: Conclusions

Highly differentiated, potentially first-in-class oral WEE1/PKMYT1 inhibitor

- Rationally designed with the AP3 platform for superior single-agent activity through dual Wee1/PKMYT1 inhibition and activation of not only CDK1 and CDK2, but also PLK1

Favorable safety profile

- Predominantly limited to transient, asymptomatic, mechanism-based neutropenia – with no Grade 4 or 5 treatment-related events and no DLTs at the selected 120 mg and 160 mg doses

Durable single-agent activity with long-lasting clinical benefit

- 5/7 pts with lung cancer, an AP3-predicted tumor type not typically sensitive to WEE1i or PKMYT1i, experienced clinical benefit
  - 1 confirmed CR (SqNSCLC; on study >65 wks)
  - 3 SD (2 adenoNSCLC; on treatment >71 wks; 1 SqNSCLC; >25 weeks)
  - 1 unconfirmed PR with deep tumor shrinkage (SCLC)
- Confirmed PR >40 weeks in pt with endometrial cancer

Two optimized doses advancing in randomized expansion in AP3-predicted tumor types

- 120 mg and 160 mg, 3 days on / 4 days off, selected to support RP2D and further development (NCT06667141)

ACR-2316: Acknowledgments

To our patients and their families

Our deepest gratitude goes to every patient who has taken part, and continues to take part, in this study, and to the families and caregivers who stand beside them. Your trust, courage and generosity are the foundation of everything we are learning. We are humbled to share this journey with you!

To our investigators and site teams

We are sincerely grateful to all the principal investigators, sub-investigators, study coordinators, research nurses, pharmacists and data managers at every participating site. Their dedication and meticulous work make this trial possible. Thank you for your continued partnership!

To the entire Acrivon team for their commitment and hard work in bringing ACR-2316 to patients. Thank you!

This study (NCT06667141) is ongoing and currently enrolling in the dose-expansion phase.
Sponsored by Acrivon Therapeutics, Inc.