APREA THERAPEUTICS

WE CAN'T WAIT TO CURE CANCER

A clinical-stage precision medicine oncology company focused on the discovery and development of targeted therapies for patients with biomarker-defined cancers

August 2026

Forward-Looking Statements

Certain information contained in this presentation includes "forward-looking statements", within the meaning of Section 27A of the Securities Act of 1933, as amended, and Section 21E of the Securities Exchange Act of 1934, as amended related to our study analyses, clinical trials, regulatory submissions, and projected cash position. We may, in some cases use terms such as "future," "predicts," "believes," "potential," "continue," "anticipates," "estimates," "expects," "plans," "intends," "targeting," "confidence," "may," "could," "might," "likely," "will," "should" or other words that convey uncertainty of the future events or outcomes to identify these forward-looking statements. Our forward-looking statements are based on current beliefs and expectations of our management team and on information currently available to management that involve risks, potential changes in circumstances, assumptions, and uncertainties. All statements contained in this presentation other than statements of historical fact are forward-looking statements, including statements regarding our ability to develop, commercialize, and achieve market acceptance of our current and planned product candidates, our research and development efforts, including timing considerations and other matters regarding our business strategies, use of capital, results of operations and financial position, project cash runway, plans and objectives for future operations, the timing, progress, enrollment and completion of our ongoing and planned clinical trials, the timing of expected clinical data updates and medical meeting presentations, the potential expansion of ACESOT-1051, the potential development of APR-1051 in single-agent and combination settings, the potential further development of ATRN-119, and the potential advancement of our early-stage programs. Any or all of the forward-looking statements may turn out to be wrong or be affected by inaccurate assumptions we might make or by known or unknown risks and uncertainties. These forward-looking statements are subject to risks and uncertainties including, without limitation, risks related to the success, timing and cost of our ongoing and anticipated clinical trials for our current product candidates, including the timing of initiation, pace of enrollment and completion of trials, our ability to fully fund our disclosed clinical trials, which assumes no material changes to our currently projected expenses, the receipt and timing of interim or preliminary results, which are not necessarily indicative of final results, our understanding of product candidate mechanisms of action and interpretation of preclinical and early clinical results from our clinical development programs, our ability to predict clinical outcomes based on such preclinical and early clinical results, the timing and outcome of regulatory interactions, and the other risks, uncertainties, and factors described under "Risk Factors," "Management's Discussion and Analysis of Financial Condition and Results of Operations" and elsewhere in the documents we file with the U.S. Securities and Exchange Commission. For all these reasons, actual results and developments could be materially different from those expressed in or implied by our 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 such forward-looking statements for any reason, except as required by law. This presentation shall not constitute an offer to sell or the solicitation of an offer to buy any securities, nor shall there be any sale of any securities in any state or jurisdiction in which such offer, solicitation or sale would be unlawful prior to registration or qualification under the securities laws of any such state or jurisdiction. This presentation may not be reproduced, forwarded to any person or published, in whole or in part.

APR-1051 and ATRN-119 are investigational drugs and have not yet been approved by the U.S. Food and Drug Administration or any other regulatory authority.

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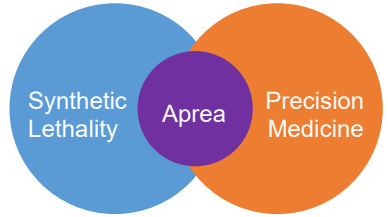
Aprea Therapeutics (NASDAQ: APRE)
One Critical Pathway - Multiple Targets



Positioned at the forefront of synthetic lethality and precision medicine

Targeted Oncology

Transition from broad, toxic chemotherapy to potentially safer, precision-guided targeted therapies

Precision-Driven Development

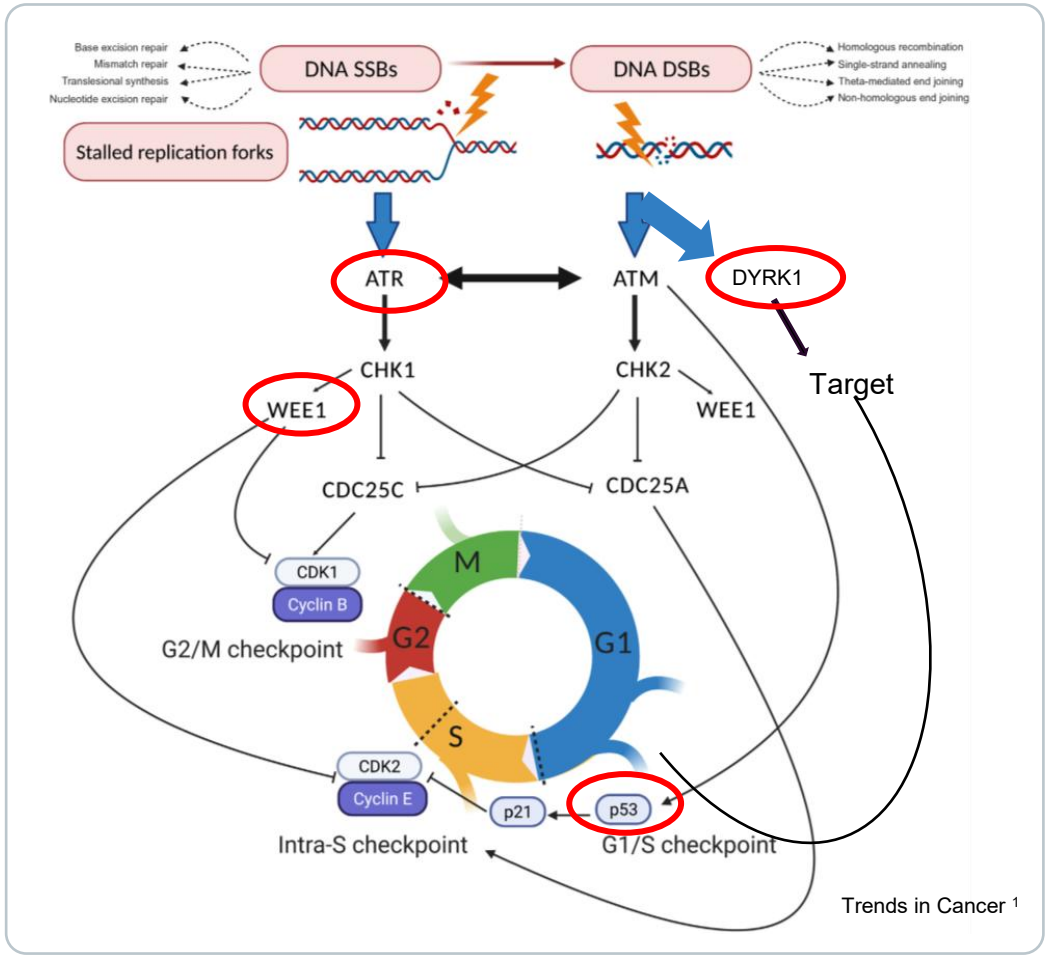
Develop highly selective cancer therapies that exploit tumor-specific mutations to maximize cancer cell killing while sparing healthy tissue

Pipeline with Clinical Momentum

All programs are designed to address significant unmet medical needs across genetically defined cancer populations

Early Clinical Proof-of-Concept

Preliminary signals of monotherapy activity observed in ongoing Phase 1 trial



1. Ngoi N, *et al*. Targeting the replication stress response through synthetic lethal strategies in cancer medicine. *Trends in Cancer*. (2021); 7(10):930-957

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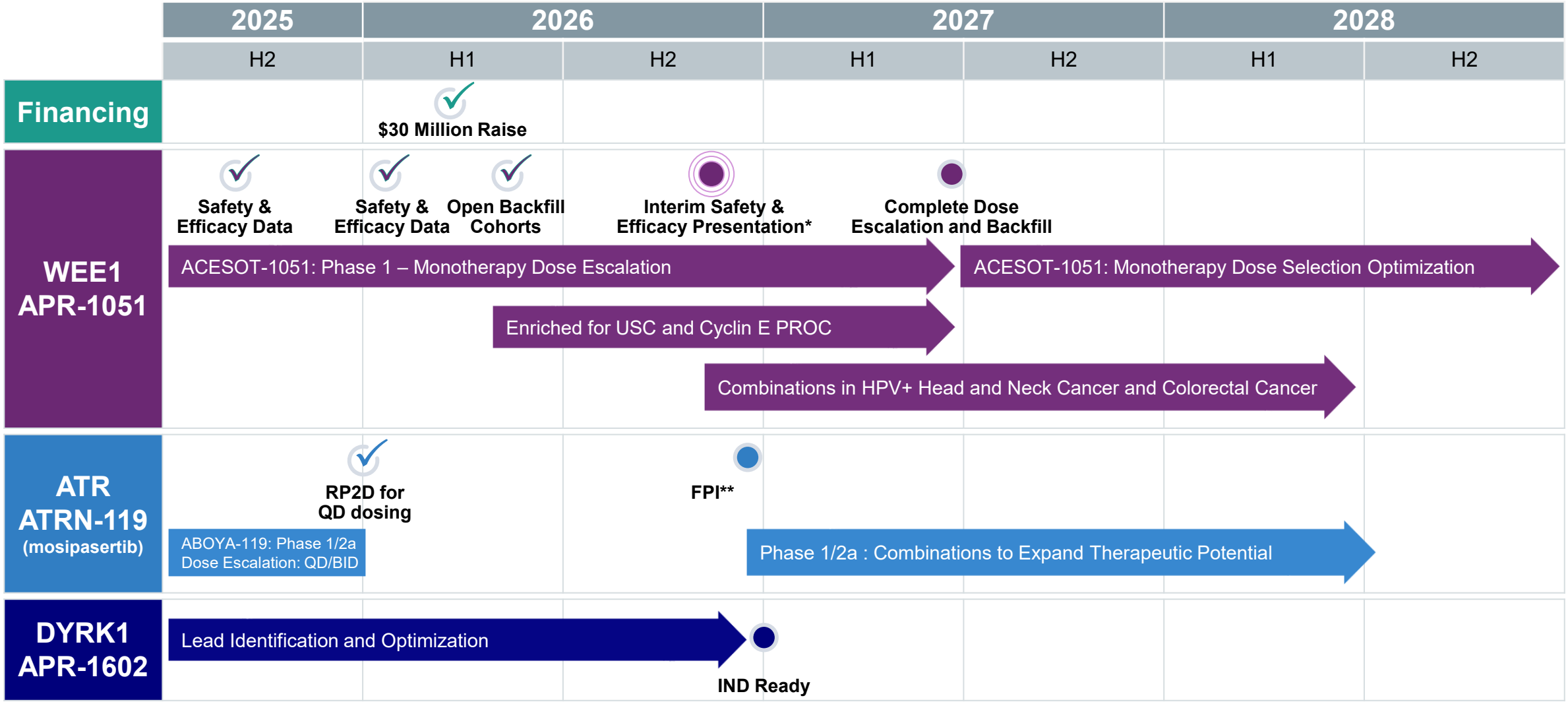
Robust DDR Development Pipeline and Corporate Milestones

2025-2028 Accomplished and anticipated milestones



DNA Damage Response (DDR)
*Planned major medical conference presentation
**FPI contingent upon execution of a research collaboration or partnering agreement; study initiation is expected to be externally supported without use of current company resources.

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Strong Drug Development and Commercial Expertise

Experienced team in synthetic lethality and targeted therapy

Management

Oren Gilad, PhD President and CEO	John P. Hamill SVP and CFO	Eugene Kennedy, MD Chief Medical Advisor	Ze'ev Weiss, CPA, BSc Chief Business Advisor	Mike Carleton, PhD Translational Medicine Advisor	Brian Wiley SVP, Corporate Strategy
ATRIN Pharmaceuticals Perelman School of Medicine, University of Pennsylvania	Windtree Therapeutics Trevena Savient Pharmaceuticals, Inc. NephroGenex pharmanet	carisma Therapeutics Galera Therapeutics, Inc ICT Innovative Cellular Therapeutics NewLink Genetics	ATRIN Pharmaceuticals INTEC PHARMA pwc	inipharm ATRIN Pharmaceuticals MERCK Bristol Myers Squibb presage Mavu Pharma	Boston BioConsulting NewLink Genetics Gloucester Pharmaceuticals Celgene Aventis Millennium

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WEE1 Inhibitor: APR-1051

ACESOT-1051:

Clinical Proof-Of-Concept

WEE1 Has Emerged as a Therapeutic Target of Significant Industry Interest

Clinically validated, prior WEE1 inhibitors have been challenged by narrow therapeutic windows
Aprea is applying key insights to advance APR-1051 as a potentially best-in-class WEE1 inhibitor

Program	Clinical Limitations	Strategic Outcome	What It Signals
Adavosertib (AstraZeneca)	Hematologic & GI toxicity limited dose intensity	Terminated further clinical development Returned by AstraZeneca to Merck & Co.	Biology works, narrow therapeutic window
Azenosertib (Zentalis)	Continuous dosing not tolerated[1]	TRAEs leading to dose reductions, interruptions and discontinuation	Biology works, therapeutic window still being defined
Debio 0123 (Debiopharm)	QT prolongation liability at high doses[2]	Limited single-agent activity – no responses up to MTD[3]; activity seen in combination with PKMYT1 inhibitor[4]	Potential future as combination agent
Program	**Engineered Profile**	**Strategic Outcome**	**What It Signals**
APR-1051 (Aprea Therapeutics)	Structurally differentiated, highly potent, limited off-target inhibition	Preliminary signals of monotherapy activity without class-limiting tox to date	Potential to expand therapeutic window

No head-to-head studies have been conducted. Trial information is based on publicly available data and should be interpreted cautiously

TRAEs – Treatment related adverse events

1. Zentalis Corporate Presentation, April 2026
2. Debio 0123-101, A Phase 1 Trial of Debio 0123 In Combination With Carboplatin In Advanced Solid Tumors: Safety, Pharmacokinetic, And Preliminary Antitumor Activity Data, Poster ASCO 2023
3. Abstract 3120, ASCO 2024
4. Poster CT022, AACR 2026

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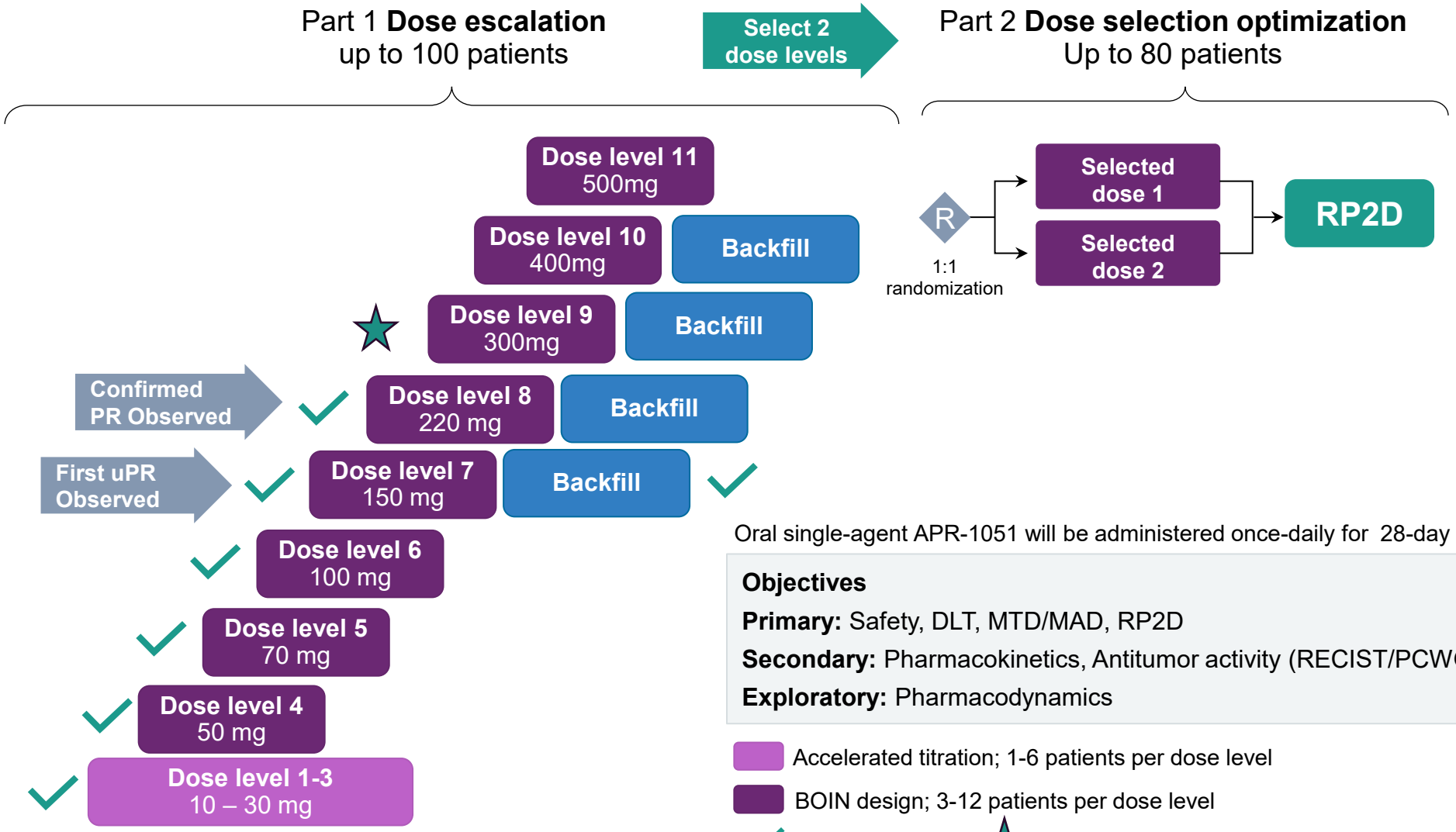
ACESOT-1051: Phase 1 Study Design

Multi-center, open-label Phase 1 single-agent dose escalation and dose selection optimization

Eligible patients

≥ 18 yo with advanced solid tumor harboring cancer-associated gene alterations

- **USC regardless biomarker status**
- **Cyclin E-overexpressing platinum-resistant ovarian cancer (PROC)**
- CCNE1, CCNE2, FBXW7 or PPP2R1A
- HPV+ oropharyngeal squamous cell carcinoma, cervical, vaginal, or vulvar carcinoma
- KRAS-GLY12/GLY13 & TP53 colorectal cancer



Oral single-agent APR-1051 will be administered once-daily for 28-day cycles

Objectives

Primary: Safety, DLT, MTD/MAD, RP2D

Secondary: Pharmacokinetics, Antitumor activity (RECIST/PCWG3)

Exploratory: Pharmacodynamics

Accelerated titration; 1-6 patients per dose level

BOIN design; 3-12 patients per dose level

= cleared = currently enrolling

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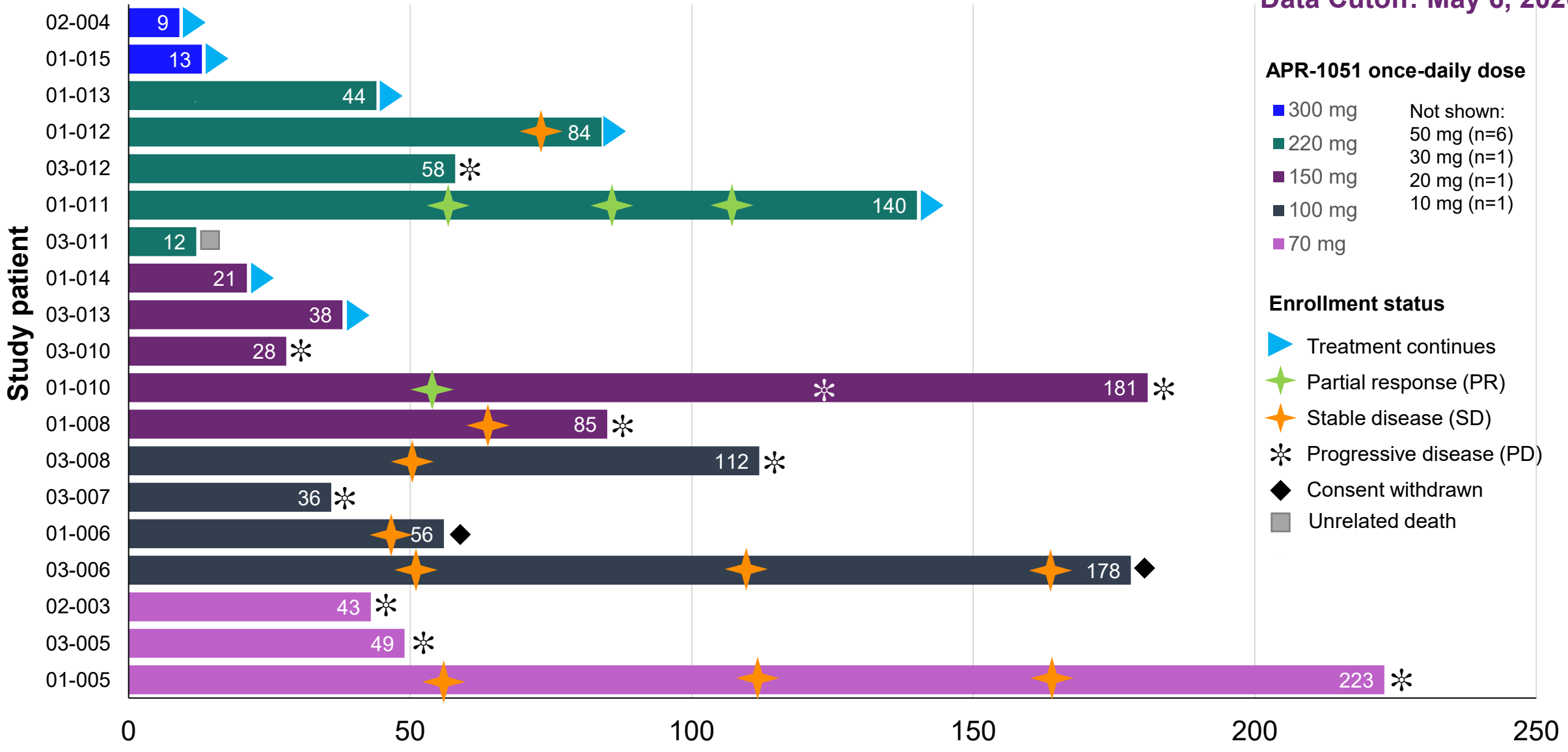
APR-1051 Summary of Duration of Treatment (n=28 for all dose levels)

Data Cutoff: May 6, 2026



Not all data source verified

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Treatment-related AEs in Patients Treated with APR-1051 (N=28)

Data Cutoff: May 6, 2026

MedDRA Preferred Term	APR-1051 All dose levels (N=28)	
Treatment-related AEs, n (%)[a]	**All Grades**	**Grade ≥ 3[b]**
Nausea	10 (35.7)	0 (0)
Fatigue	4 (14.3)	0 (0)
Vomiting	3 (10.7)	0 (0)
Alanine aminotransferase increased	1 (3.6)	1 (3.6)[c]
Anemia	1 (3.6)	0 (0)
Aspartate aminotransferase increase	1 (3.6)	1 (3.6)[c]
Blood bilirubin increased	1 (3.6)	0 (0)
Constipation	1 (3.6)	0 (0)
Dehydration	1 (3.6)	0 (0)
Dysgeusia	1 (3.6)	0 (0)
Dyspepsia	1 (3.6)	0 (0)
Eczema	1 (3.6)	0 (0)
Gastroesophageal reflux disease	1 (3.6)	0 (0)
Hypokalemia	1 (3.6)	0 (0)
Lymphocyte count decreased	1 (3.6)	1 (3.6)
Platelet count decreased	1 (3.6)	0 (0)

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a A patient may have more than one AE and/or have the same AE more than once
b Grade 3 unless otherwise indicated
c Increased alanine aminotransferase and aspartate aminotransferase occurred in the same patient, was determined to be serious, and considered one DLT event

Not all data source verified

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Confirmed Partial Response in Patient 01-011

220mg QD – Currently on treatment

Demographics: 63-year-old Black Female
Site: MD Anderson Cancer Center
Diagnosis: Uterine carcinosarcoma
Key Mutations: PPP2R1A

Treatment History (4 prior lines)

- Line 1: Carboplatin + Paclitaxel → 126 days, PD
- Line 2: Doxorubicin → 56 days, PD
- Line 3: Topotecan → 70 days, PD
- Line 4: Pembrolizumab + Lenvatinib → 5months, PD

APR-1051 – Treatment Outcome

- C1D1: Dec 18, 2025
- Current Status: **On treatment 140 days (C6D1) – May 6, 2026**
- Best Response: **Confirmed Partial Response (PR)**
  - PR (-50%) at first assessment Feb 10, 2026
  - Confirmed PR with additional -9.5% reduction from C3D1 Mar 10, 2026; scan April 8, 2026 showed 0% change
- Tumor marker: CA-125 reduction from BL 362.4 U/mL to C3D1 46.8 U/mL (87% decrease); 40.2 U/mL Mar 11, 2026; 53 U/ml April 08, 2026
- Adverse Events: C1D22 Gr 1 rash. C1D15 Gr1 thrombocytopenia , possibly related to IP; intermittent nausea Gr1 probably related; amylase increase Gr1 unlikely related. No DLT

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Disease Control Observed in Early Patient Outcomes

APR-1051 shows preliminary evidence of disease control in rectal cancer with mutated FBXW7

100 mg Cohort Case Report

Stable disease maintained for 178 days in patient with FBXW7 mutation (100 mg QD) (elected to stop study participation)

Patient: 86-year-old Asian Female
Diagnosis: Rectal Cancer
Key Mutations: FBXW7 (Drives Cyclin E accumulation and overexpression)

Treatment History: 5 prior lines - heavily pretreated

- **Line 1:** Capecitabine/oxaliplatin → 191 days, PD
- **Line 2:** Capecitabine/oxaliplatin/bevacizumab → 45 days, PD
- **Line 3:** FOLFIRI + bevacizumab → 43 days, PD
- **Line 4:** Local XRT (lung mets) → 12 days, not evaluable
- **Line 5:** Tretinoin/bevacizumab/Tecentriq (ATRT trial) → 50 days, PD

APR-1051 Activity:

- **Current Status:** Consent withdrawn after 178 days
- **Best Response:** SD at third scan (-15% tumor response)

Notes: Durable SD maintained 181 days in a heavily pretreated 86-year-old patient; well tolerated with minimal toxicity. FBXW7 mutation may be relevant to response

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Disease Control Observed in Early Patient Outcomes

APR-1051 shows preliminary evidence of disease control in HPV+ head and neck cancer

70 mg Cohort Case Report

Stable disease maintained for 223 days in patient 01-005 HPV+ head and neck cancer (70 mg QD) (PD)

Patient: 62-year-old White Male
Diagnosis: HPV+ Oropharyngeal Squamous Cell Carcinoma (base of tongue)

Key Mutations: P16+

Treatment History: 3 prior lines

- **Line 1:** Concomitant cisplatin/XRT → 49 days, PD
- **Line 2:** Pembrolizumab→ 84 days, PD
- **Line 3:** Paclitaxel/carboplatin → 184 days, PD

APR-1051 Activity

- **Current Status:** PD after 223 days of SD treatment
- **Best Response:** SD at first scan (-5% tumor response)

Notes: Stable disease maintained for 223 days.

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Biomarker Defined Registration Path

Preliminary signals of monotherapy activity across cohorts in genomically defined tumors including uterine cancer, endometrial cancer, CRC and HNSCC

Clinical Activity by Indication and Biomarker

Indication	Biomarker / Altered Genes	Clinical Activity in ACESOT-1051*
Uterine/ Endometrial Cancer	PPP2R1A	2 PR 2 SD
	CCNE1 overexpressed TP53	
CRC	KRAS & TP53	3 SD
	FBXW7	
HNSCC	HPV+	1 SD

Path to Registrational Cohort

1. Expand USC and CycE-PROC
2. Add additional cohorts
   - CRC FBXW7-mutated
   - HPV+ cancers
   - PPP2R1A mutated
3. Confirm durability, consistency of response and safety

- Responses across dose levels support a biomarker enriched expansion path
- Activity beyond endometrial cancer supports additional biomarker-defined cohorts

*Data as of May 6, 2026 and are preliminary from an ongoing dose-escalation study. Responses and stable disease require confirmation in additional patients and may change as follow-up matures. Safety and efficacy outcomes may vary by dose, schedule, and patient characteristics.

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APR-1051:

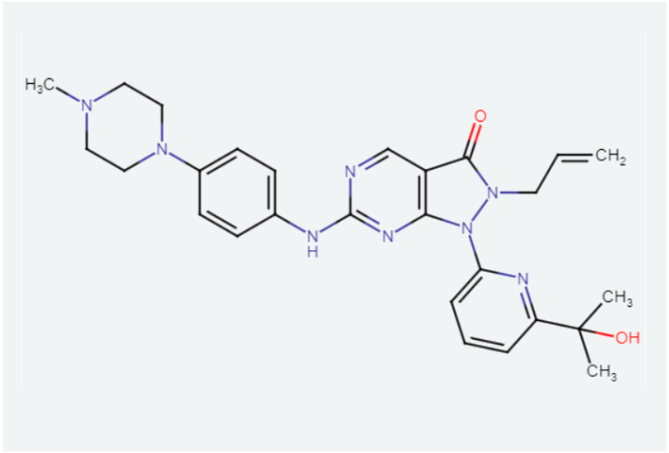
Potentially Differentiated WEE1 Inhibitor

Pre-Clinical

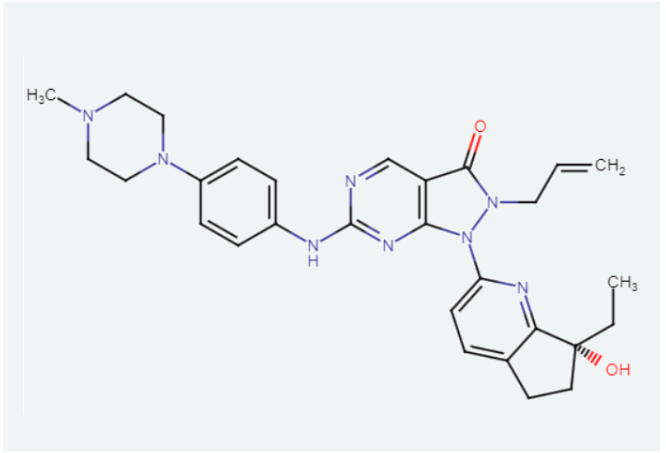
APR-1051: Potentially Best-in-Class WEE1 Inhibitor

Structurally differentiated: high potency, limited off-target inhibition design compared to other molecules

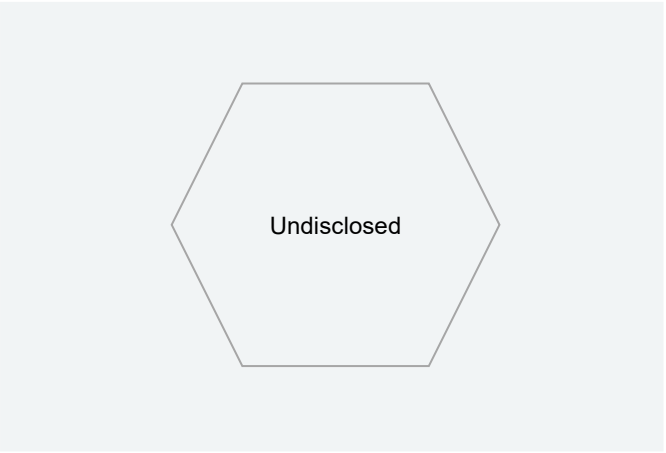
APR-1051 is based on a different molecular structure than AZD-1775 and ZN-c3 (not an analogue)



AstraZeneca
Adavosertib (AZD-1775)



Zentalis
Azenosertib (ZN-c3)



Aprea
APR-1051

No head-to-head clinical studies have been conducted.

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APR-1051: Potentially Best-in-Class WEE1 Inhibitor

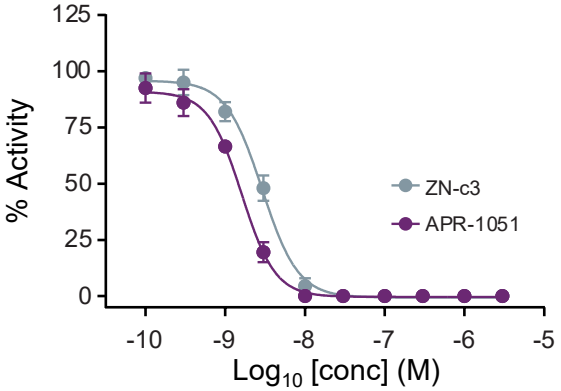
Potent inhibitor of WEE1

Does not substantially inhibit structurally and functionally related PLK1, PLK2 or PLK3

On-target WEE1 potency[1]

Important difference in off-target inhibition between APR-1051 and ZN-c3[1]

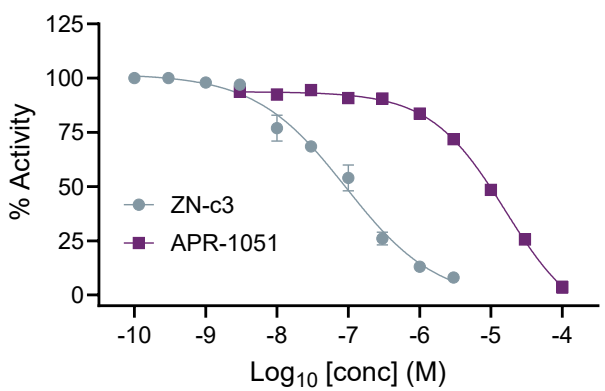
WEE1 IC_{50} Determination



ZN-c3 = 2.9 nM
APR-1051 = 1.6 nM

WEE1 Inhibition
IC50 similar to ZN-c3

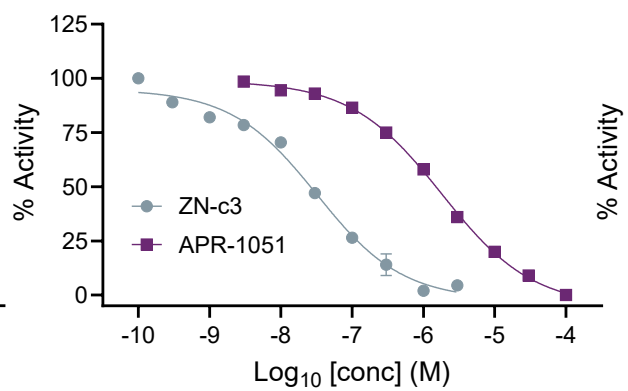
PLK1 IC_{50} Determination



ZN-c3 = 92.1 nM
APR-1051 = 15,900 nM

PLK1 Inhibition
IC50 >150-fold difference

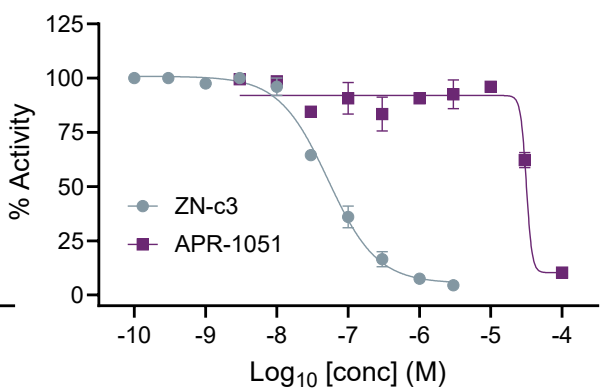
PLK2 IC_{50} Determination



ZN-c3 = 32.0 nM
APR-1051 = 1,800 nM

PLK2 Inhibition
IC50 >50-fold difference

PLK3 IC_{50} Determination



ZN-c3 = 52.2 nM
APR-1051 = 31,600 nM

PLK3 Inhibition
IC50 >600-fold difference

APR-1051 specificity for WEE1 opens potential for greater therapeutic window

[1]Data from exploratory in-vitro studies. No head-to-head clinical studies have been conducted.

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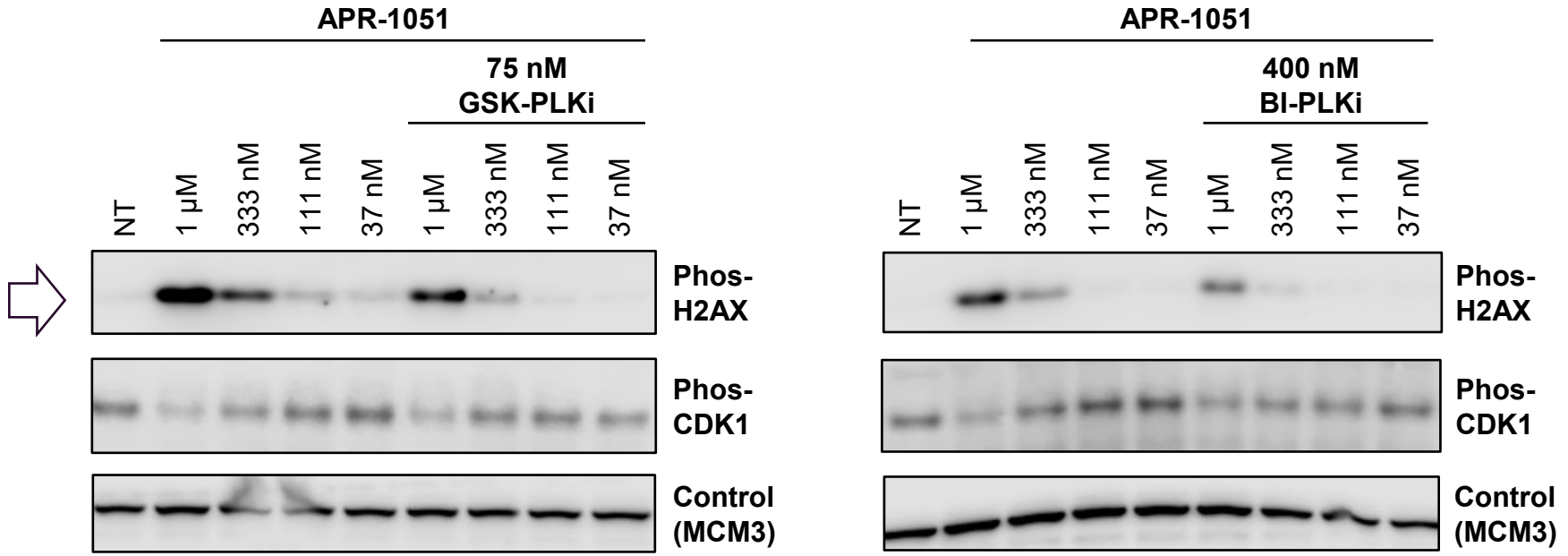
PLK1 Inhibition Counteracts Effect of WEE1 Inhibitors[1]

Minimal PLK1 co-inhibition enhances therapeutic window for APR-1051

Evidence of DNA damage allowed by WEE1 inhibition. PLK1 reduces functional potency of WEE1 inhibition



PLK inhibitor, GSK-461364, interferes with the effects of APR-1051 in OVCAR-3 cells

PLK inhibitor, BI-2536, interferes with the effects of APR-1051 in OVCAR-3 cells

Inhibition of PLK1 reduces efficacy of WEE1 inhibition. Results in requiring higher doses of WEE1 inhibitors and introduces PLK1 related toxicity

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[1]Data from exploratory in-vitro studies

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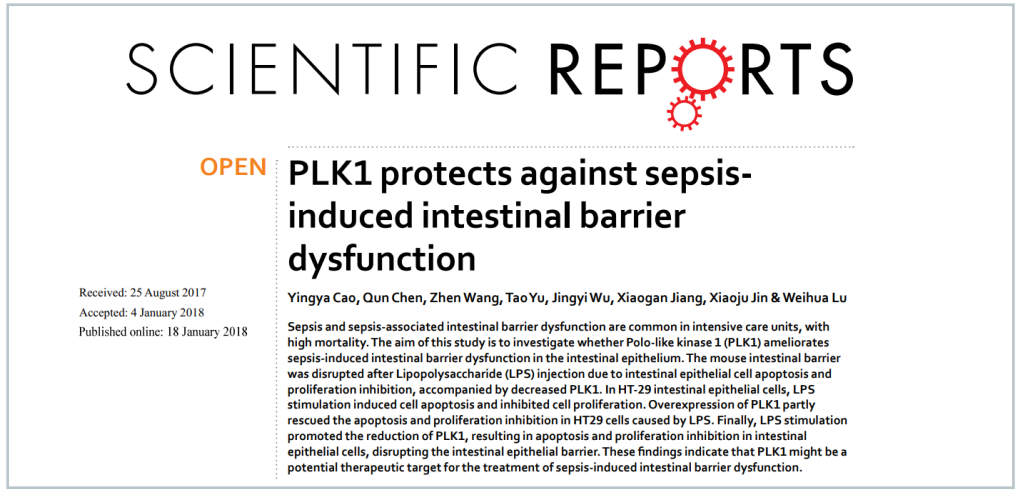
Studies Show PLK1 Suppression is Associated with Sepsis-Induced Loss of Intestinal Barrier Function



SCIENTIFIC REPORTS

OPEN

PLK1 protects against sepsis-induced intestinal barrier dysfunction

Received: 25 August 2017
Accepted: 4 January 2018
Published online: 18 January 2018

Yingya Cao, Qun Chen, Zhen Wang, Tao Yu, Jingyi Wu, Xiaogan Jiang, Xiaoju Jin & Weihua Lu

Sepsis and sepsis-associated intestinal barrier dysfunction are common in intensive care units, with high mortality. The aim of this study is to investigate whether Polo-like kinase 1 (PLK1) ameliorates sepsis-induced intestinal barrier dysfunction in the intestinal epithelium. The mouse intestinal barrier was disrupted after Lipopolysaccharide (LPS) injection due to intestinal epithelial cell apoptosis and proliferation inhibition, accompanied by decreased PLK1. In HT-29 intestinal epithelial cells, LPS stimulation induced cell apoptosis and inhibited cell proliferation. Overexpression of PLK1 partly rescued the apoptosis and proliferation inhibition in HT29 cells caused by LPS. Finally, LPS stimulation promoted the reduction of PLK1, resulting in apoptosis and proliferation inhibition in intestinal epithelial cells, disrupting the intestinal epithelial barrier. These findings indicate that PLK1 might be a potential therapeutic target for the treatment of sepsis-induced intestinal barrier dysfunction.

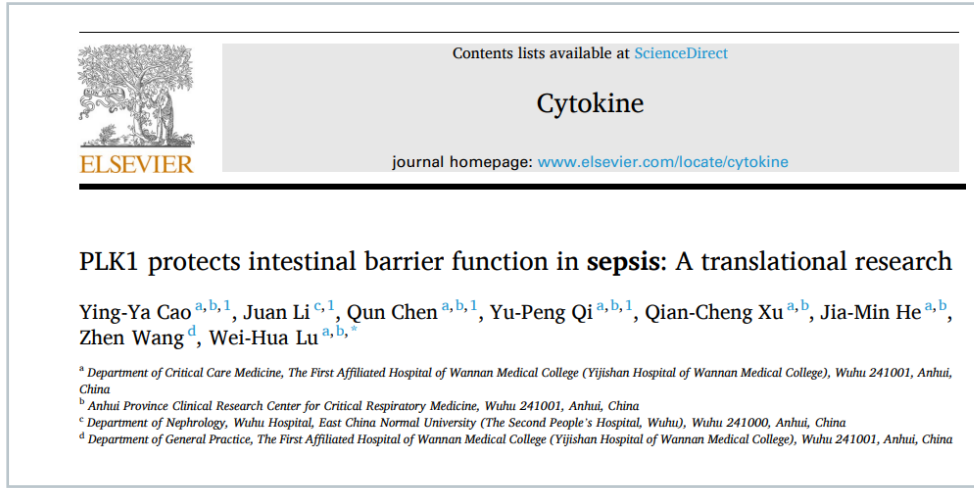
Cao *et al. Molecular Medicine* (2022) 28:163
https://doi.org/10.1186/s10020-022-00597-z

Molecular Medicine

RESEARCH ARTICLE Open Access

PLK1 protects intestinal barrier function during sepsis by targeting mitochondrial dynamics through TANK-NF-κB signalling

Ying-Ya Cao[1,2†], Yuan Zhang[1†], Wuyun Gerile[1†], Yan Guo[1], Li-Na Wu[1], Li-Li Wu[1], Kai Song[1], Wei-Hua Lu[2] and Jian-Bo Yu[1*]



Contents lists available at ScienceDirect

Cytokine

journal homepage: www.elsevier.com/locate/cytokine

PLK1 protects intestinal barrier function in **sepsis**: A translational research

Ying-Ya Cao [a,b,1], Juan Li [c,1], Qun Chen [a,b,1], Yu-Peng Qi [a,b,1], Qian-Cheng Xu [a,b], Jia-Min He [a,b], Zhen Wang [d], Wei-Hua Lu [a,b,*]

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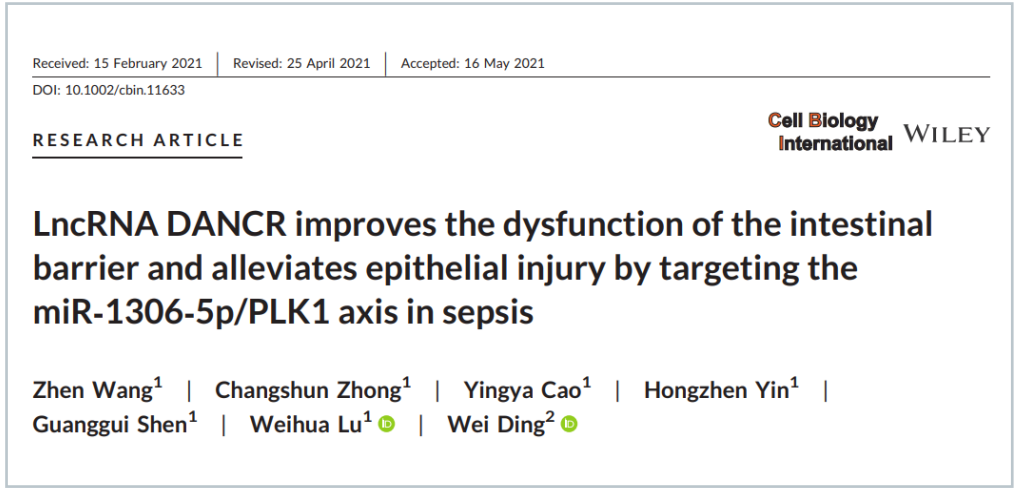

Received: 15 February 2021 | Revised: 25 April 2021 | Accepted: 16 May 2021
DOI: 10.1002/cbin.11633

RESEARCH ARTICLE

Cell Biology International WILEY

LncRNA DANCR improves the dysfunction of the intestinal barrier and alleviates epithelial injury by targeting the miR-1306-5p/PLK1 axis in sepsis

Zhen Wang[1] | Changshun Zhong[1] | Yingya Cao[1] | Hongzhen Yin[1] | Guanggui Shen[1] | Weihua Lu[1] | Wei Ding[2]

1 PLK1 protects against sepsis-induced intestinal barrier dysfunction, Cao *et al, Scientific Reports* (2018).
2 PLK1 protects intestinal barrier function in sepsis: A translational research, Cao *et al*, *Cytokine* (2023).
3 PLK1 protects intestinal barrier function in sepsis: A translational research, Cao *et al*, *Molecular Medicine* (2022).
4 LncRNA DANCR improves the dysfunction of the intestinal barrier and alleviates epithelial injury by targeting the miR-1306-5p/PLK1 axis in sepsis, Wang *et al., Cell Biology International* (2021).

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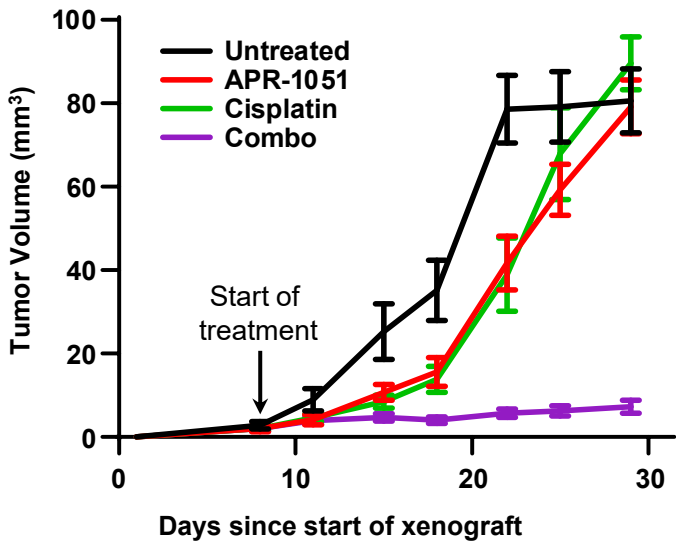
APR-1051 Showed Preclinical Activity in Combination with Chemo, IO and ATRi Across Multiple Cancer Models

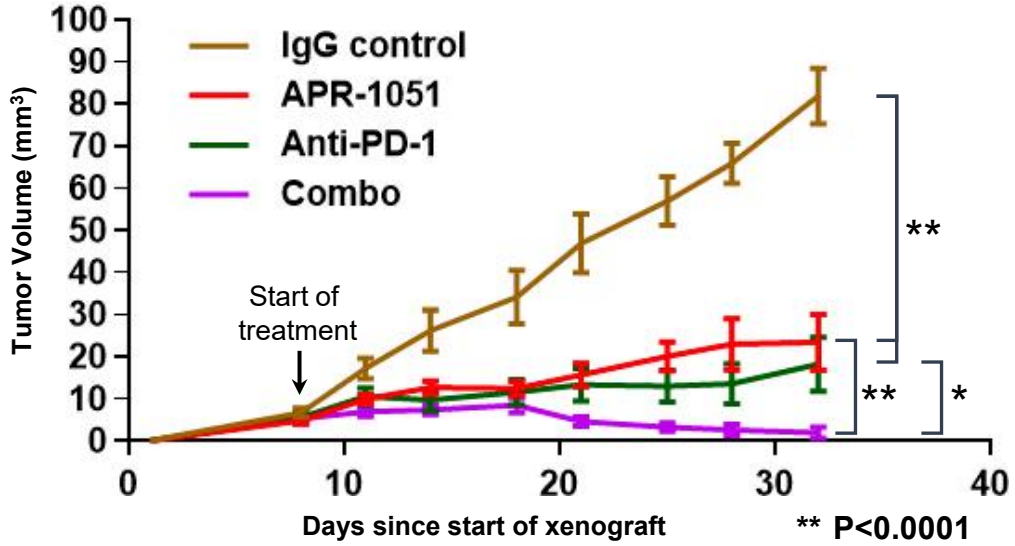
APR-1051 suggests synergistic potential preclinically with standard oncology agents

Chemotherapy



UMSCC47 tumor cells

Immuno-oncology

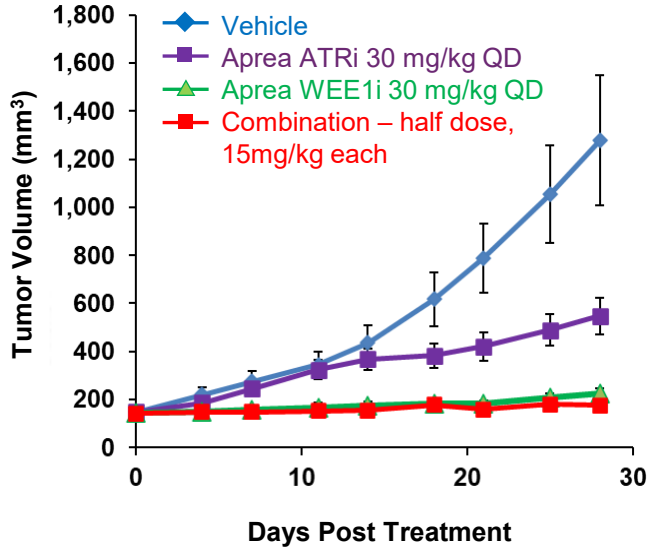


** P<0.0001
* P=0.0067

mEER tumor cells

HPV+ Cancer – Collaboration with MD Anderson

DDR combination



OVCAR3 tumor xenograft

Ovarian Cancer*

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* Data on file.

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APR-1051 WEE1 Summary

A next-generation WEE1i with the potential to deliver meaningful clinical benefit and durable value

1

Clinically validated target

- WEE1 inhibitors have shown promising activity in genomically defined tumors
- Competitor programs constrained by low therapeutic window

2

APR-1051 clinical highlights

- Preliminary clinical proof-of-concept at 150 mg and 220 mg dose levels with two partial responses and six patients with stable disease presented at ASCO 2026
- Potentially favorable safety profile at active dose levels
- Enrollment accelerating with expansion from three to ten active clinical sites
- **Interim clinical data to be presented Q4 2026**

3

APR-1051 preclinical differentiation and potentially best-in-class opportunity

- Novel structure with high potency, limited off-target inhibition design
- Minimal PLK1 inhibition enhances therapeutic window
- Potential for synergy demonstrated in combination with standard oncology agents

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WE CAN'T WAIT TO CURE CANCER

Intellectual Property Portfolio

Financial Summary & Capitalization

Investment Highlights

Robust Global Intellectual Property Protection

Family 1	**Ataxia Telangiectasia and Rad3-Related (ATR) Protein Kinase Inhibitors**	• Macrocyclic inhibitors of ATR & methods of using them to treat various cancers, filed on Oct. 13th, 2015 • Patents granted in AU, BR, CA, CN, EP, IL, IN, JP, KR, MX, HK. • 1.1: Issued on May 30, 2017 as U.S. Patent 9,663,535; 1.2: Issued on May 29, 2018 as U.S. Patent 9,981,989; 1.3: Issued on Feb. 5, 2019 as U.S. Patent 10,196,405
Family 2	**ATR Inhibitors and Methods of Use**	• Carboxylic acid-containing macrocyclic ATR inhibitors, and prodrugs; methods of using these inhibitors to treat various cancers; filed on Apr. 12, 2017 • Issued on May 28th, 2019 as U.S. Patent 10,301,324
Family 3	**ATR Inhibitor Pharmaceutical Composition and Methods**	• International application filed on Apr. 14, 2023 • Pharmaceutical formulation and composition of our lead ATR inhibitor in the clinic • Patent granted in JP and EA; Applications pending US, AU, BR, CA, CN, EP, HK, IL, IN, KR, MX, NZ, PH, SG, ZA
Family 4	**WEE1 Inhibitor Pharmaceutical Compositions and Methods**	• International Application filed on Jun. 3, 2022 • Composition of our lead WEE1 inhibitor compounds • Patent granted in AU; Applications pending in US, AU, BR, CA, CN, EP, HK, IL, IN, JP, KR, MX, ZA
Family 5	**Methods of Treating Cancer**	• Clinical methods of treating advanced solid cancer tumors using lead ATR inhibitor • International application filed on Sept. 19, 2025
Family 6	**Macrocyclic DYRK1A-B Inhibitors and Methods of their Preparation and Use**	• International application filed on Jan. 22, 2026 • Macrocyclic inhibitors of DYRK1A-B and methods of using them to treat disease
Family 7	**Macrocyclic DDR Target Inhibitors and Methods of their Preparation and Use**	• International application filed on Jan. 25, 2026
Family 8	**Methods of Treating Cancer**	• Provisional application filed on Jan. 9, 2026 • Clinical methods of treating cancer using lead Wee1 inhibitor

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Aprea Therapeutics (NASDAQ: APRE)

Financial Summary and Capitalization

Cash and Equivalents of ~$41.2M as of June 30, 2026

$30.0M in gross proceeds raised in private placement that closed on March 31, 2026

Securities	Common Equivalents as of August 12, 2026
Preferred Stock (as converted)	15,596
Common Stock (1)	12,591,136
Warrants (2)	89,438,517
Options	1,137,333
Restricted Stock Units	65,438
Fully Diluted Equivalents	103,248,020

1. 400,000,000 common shares authorized
2. Total warrants include pre-funded, Tranche A, Tranche B and Purchase

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Investment Highlights

Technology developed by pioneers in synthetic lethality

- Management with strong drug development and commercial expertise
- Focused on addressing unmet needs for patients with biomarker defined cancers

Highly potent and selective design, potential best in class inhibitors

- Diversified portfolio including WEE1 (APR-1051) and ATR (ATRN-119) inhibitors
- Preliminary evidence of clinical activity including PRs with APR-1051
- Single agent and combination potential therapies

Near term catalysts

- APR-1051: Q4 2026 Interim clinical data; Q2 2027 Complete dose escalation
- ATRN-119: October 2025 RP2D ✓ H2 2026 Potential collaborations on combinations

Expected cash runway into Q1 2028

- Achieve near term inflection points and catalysts
- Evaluate optimal strategic partnerships

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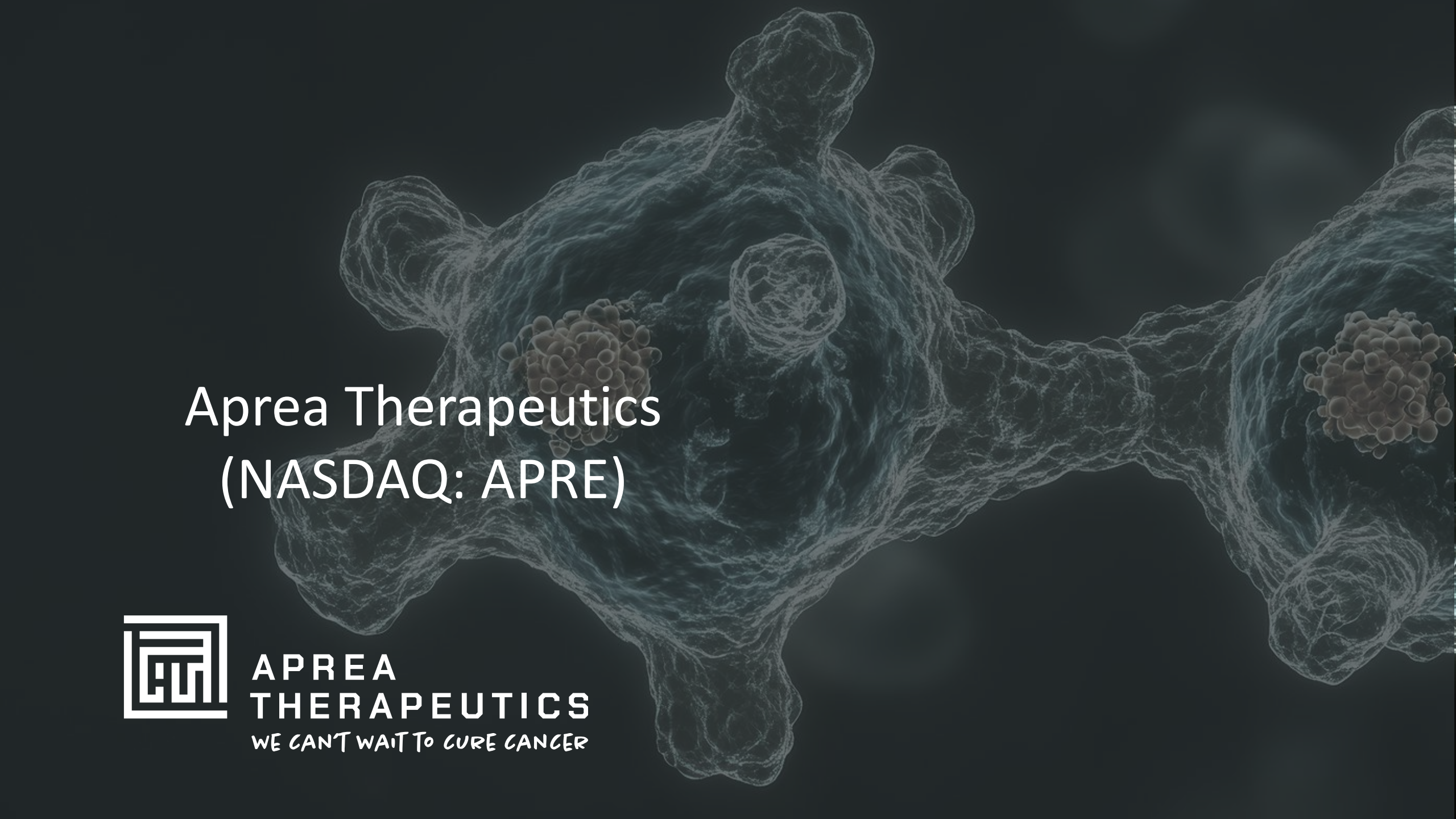

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