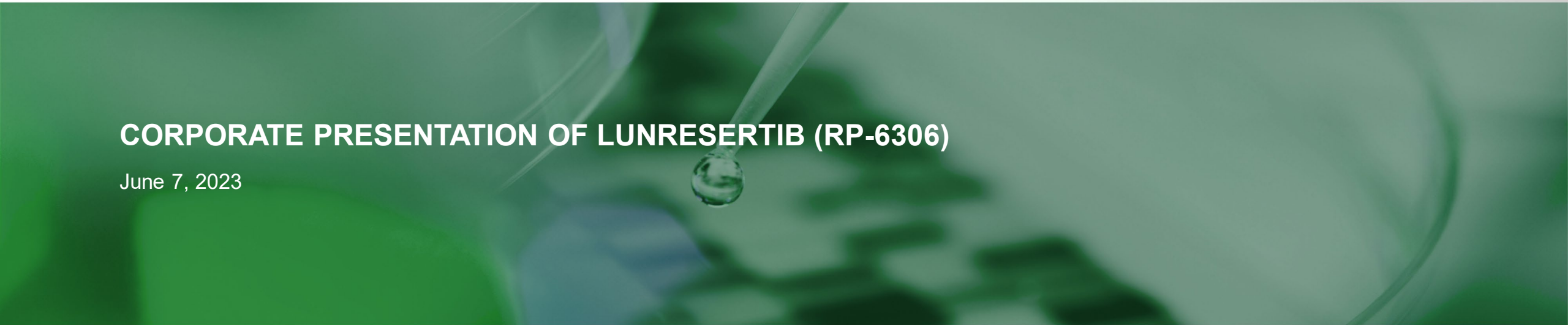
Repare Therapeutics

Insight that enriches. Precision that empowers.



CORPORATE PRESENTATION OF LUNRESERTIB (RP-6306)

June 7, 2023

Introduction

Today's agenda

Welcome
Lloyd M. Segal, President & CEO, Repare Therapeutics

Background on lunresertib (RP-6306)
Mike Zinda, PhD, EVP & CSO, Repare Therapeutics

Lunresertib preliminary monotherapy clinical trial results
Maria Koehler, MD, PhD, EVP & CMO, Repare Therapeutics & **Dr. Timothy Yap, MBBS, PhD, FRCP**, Principal Investigator, MYTHIC Trial

Lunresertib ongoing combination trials
Mike Zinda, PhD, EVP & CSO, Repare Therapeutics & **Maria Koehler, MD, PhD**, EVP & CMO, Repare Therapeutics

Conclusions
Lloyd M. Segal, President & CEO, Repare Therapeutics & **Maria Koehler, MD, PhD**, EVP & CMO, Repare Therapeutics

Q&A

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Repare participants

Lloyd M. Segal
President & CEO

Maria Koehler, MD, PhD
Chief Medical Officer

Mike Zinda, PhD
Chief Scientific Officer

Steve Forte
Chief Financial Officer

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Disclaimer

Statements contained in this presentation regarding matters that are not historical facts are “forward-looking statements” within the meaning of the Private Securities Litigation Reform Act of 1995, as amended. Words such as "anticipates," "believes," "expects," "intends," “plans,” “potential,” "projects,” “would” and "future" or similar expressions are intended to identify forward-looking statements. Each of these forward-looking statements involves substantial risks and uncertainties that could cause actual results to differ significantly from those expressed or implied by such forward-looking statements. Forward-looking statements contained in this presentation include, but are not limited to, statements regarding the initiation, timing, progress and results of our current and future preclinical studies and clinical trials, including specifically our clinical trials of lunresertib and camonsertib; the expected timing of program updates and data disclosures; and the therapeutic potential of our product candidates, including lunresertib (RP-6306) and camonsertib.

These forward-looking statements reflect our current beliefs and expectations. Many factors may cause differences between current expectations and actual results, including the evolving impact of macroeconomic events, including the COVID-19 pandemic, rising inflation, the U.S. Federal Reserve raising interest rates, recent disruptions in access to bank deposits or lending commitments due to bank failures and the Russia-Ukraine war, on our business, clinical trials and financial position, unexpected safety or efficacy data observed during preclinical or clinical studies, clinical site activation rates or clinical trial enrollment rates that are lower than expected, changes in expected or existing competition, changes in the regulatory environment, and unexpected litigation or other disputes. These and other risks are described more fully in our filings with the Securities and Exchange Commission (“SEC”), including the “Risk Factors” section of our Annual Report for the year ended December 31, 2022 filed with the SEC and the AMF on February 28, 2023, our most recently filed Quarterly Report on Form 10-Q, and other documents we subsequently filed with or furnished to the SEC. All forward-looking statements contained in this presentation speak only as of the date on which they were made. Except as required by law, we assume no obligation to update any forward-looking statements contained herein to reflect any change in expectations, even as new information becomes available.

This presentation also contains estimates and other statistical data made by independent parties and by us relating to market size and growth and other data about our industry. This data involves a number of assumptions and limitations, and you are cautioned not to give undue weight to such estimates. Neither we nor any other person makes any representation as to the accuracy or completeness of such data or undertakes any obligation to update such data after the date of this presentation. In addition, projections, assumptions and estimates of our future performance and the future performance of the markets in which we operate are necessarily subject to a high degree of uncertainty and risk.

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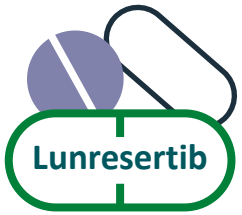
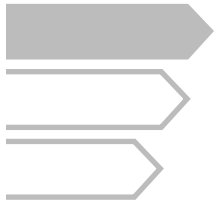
Leading precision oncology company focused on synthetic lethality

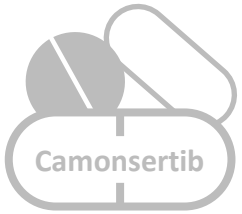


Lunresertib (RP-6306), a first-in-class, oral PKMYT1 inhibitor, drives genomic instability in CCNE1-amplified tumors with Ph 1 monotherapy and multiple combination trials ongoing

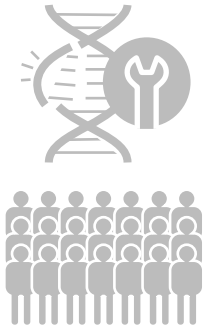


Robust pipeline of SL-based therapeutic opportunities, including Polθ and a pipeline of advanced preclinical opportunities

Balance Sheet of $314M funds Repare through multiple value-creating milestones into 2026



Camonsertib (RP-3500 / RG6526), a potential best-in-class ATR inhibitor with durable responses and clinical benefit in Ph 1/2 and strategic validation through Roche partnership



Proprietary **genome-wide CRISPR-enabled SNIPRx platform,** focused on **genomic instability** and **DNA damage repair**, enabling novel target identification and differentiated patient selection insights

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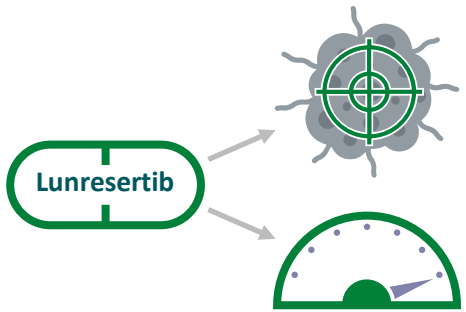
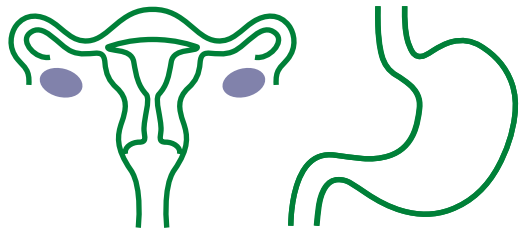
Lunresertib: first-in-class, oral, small molecule, PKMYT1 inhibitor

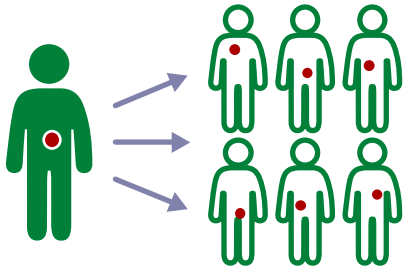
Lunresertib (RP-6306) exploits vulnerabilities caused by increases in *CCNE1*, not previously considered a druggable target



Potent and well tolerated, first in class inhibitor with anti-tumor activity especially in combination



Many affected tumor types, including gynecological and gastrointestinal malignancies



Synthetic lethal combinations with *CCNE1* amplified, *FBXW7* or *PPP2R1A* loss, and other STEP^2 genes aid in patient selection

STEP^2 is the SNIPRx® Targeted Expansion of Patient Populations

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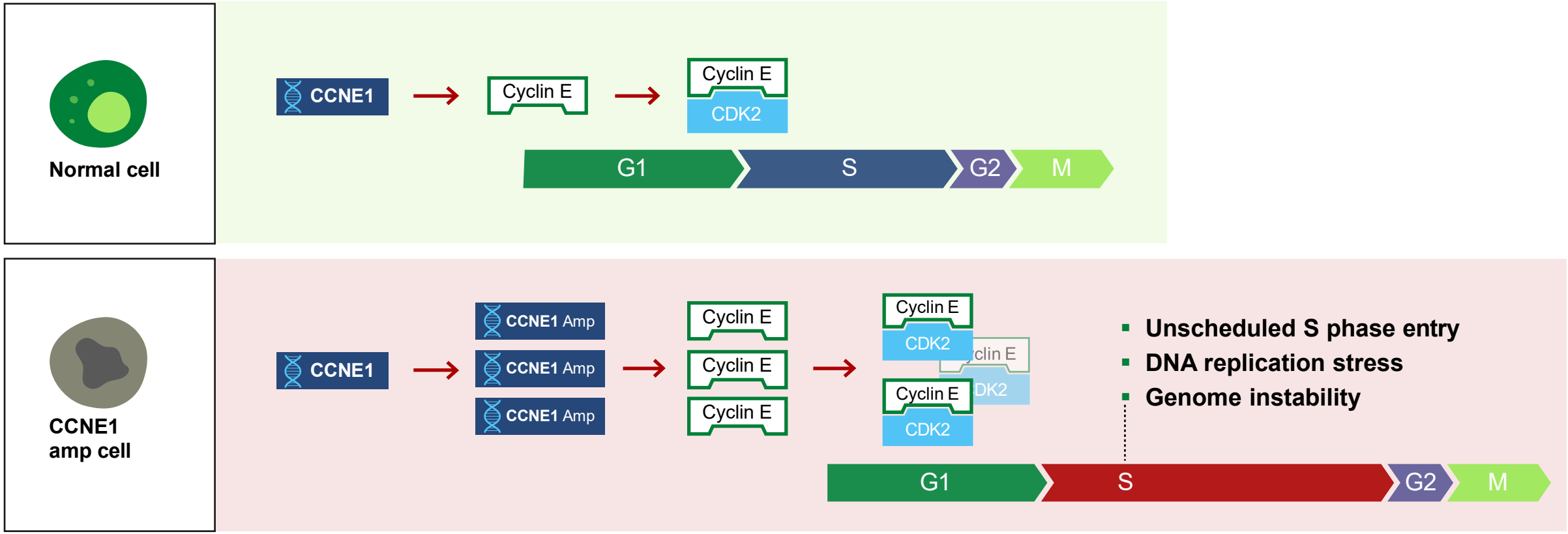
Background on Lunresertib

Preclinical data and rationale for clinical investigation

CCNE1 amplification drives genome instability



Cyclin E-overexpression drives premature entry into S-phase and overloads the DNA replication machinery, resulting in genome instability

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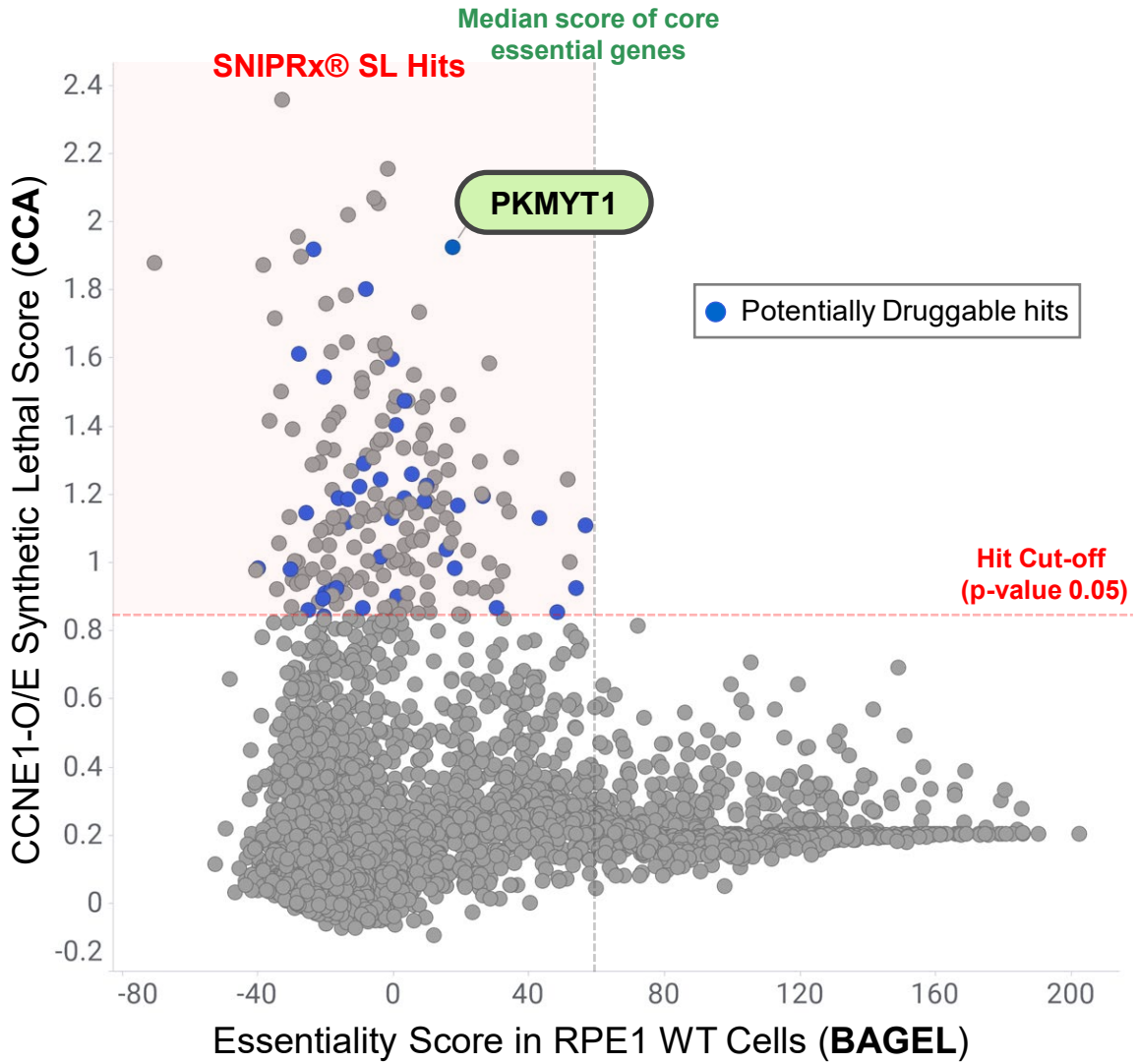
PKMYT1: Strong hit in a CCNE1-O/E SL screen



Genome-wide CRISPR screen

PKMYT1

Highest scoring druggable hit

Also high scoring hit in the DepMap

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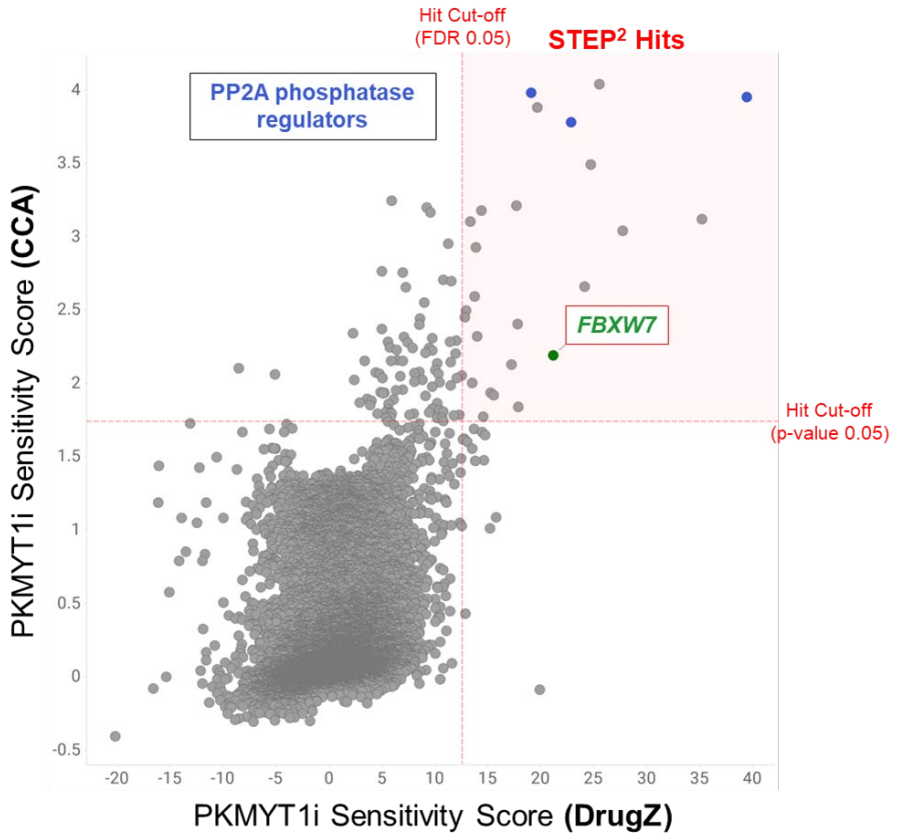
STEP² screen identified new sensitizers to PKMYT1 inhibition



FBXW7 and PP2A Phosphatase Sensitizers

FBXW7

The E3 ubiquitin ligase FBXW7 targets proteins, such as cyclin E, for proteasomal degradation. Therefore, inactivating mutations can increase cyclin E levels and replication stress.

PPP2R1A

The PP2A phosphatase is critical in the response to replication stress. Therefore, hotspot inactivating mutations can increase replication stress.

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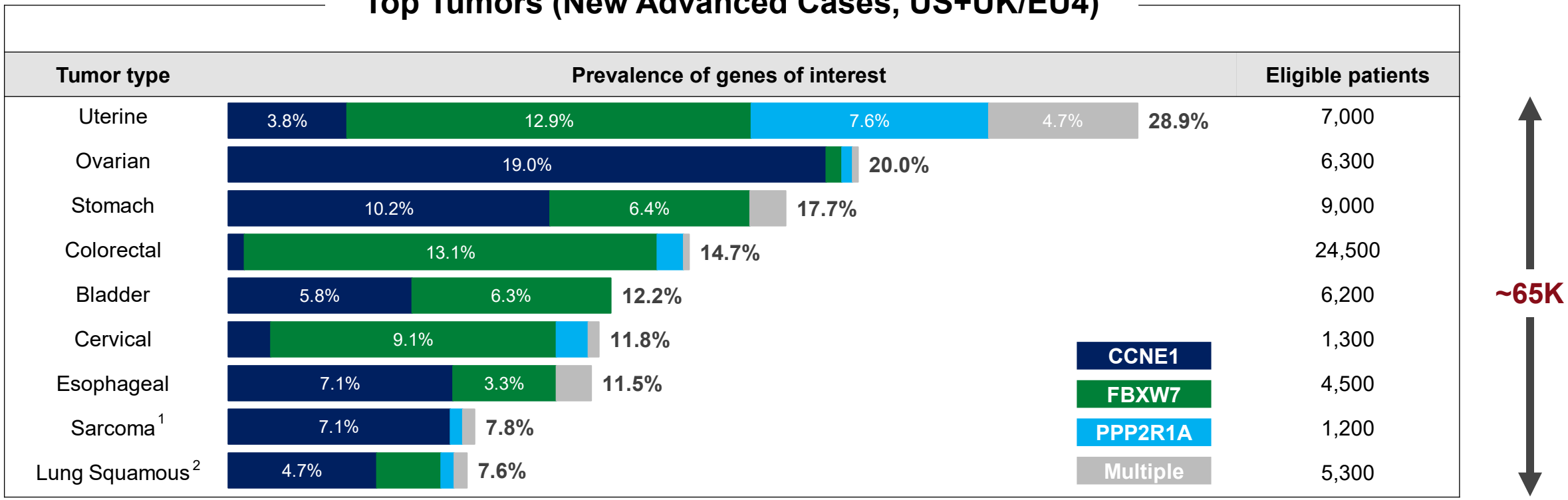
Initial lunresertib addressable patient populations

Top tumor types with highest prevalence of CCNE1 amplification or inactivating mutations in FBXW7 and PPP2R1A include ~65K US+UK/EU4 patients eligible for treatment annually, or ~90K across cancer types

Top Tumors (New Advanced Cases, US+UK/EU4)



- **These lesions are largely mutually exclusive and represent distinct patient populations**

* Based on estimated number of pts treated in 1st line, advanced setting for diagnosed and new recurrent patients (CancerMPact®, Treatment Architecture, United States, 2021; accessed 5/19/23) and lesion prevalence (TCGA). [1] Soft Tissue Sarcoma only; [2] Squamous subtype of Non-Small Cell Lung Cancer only

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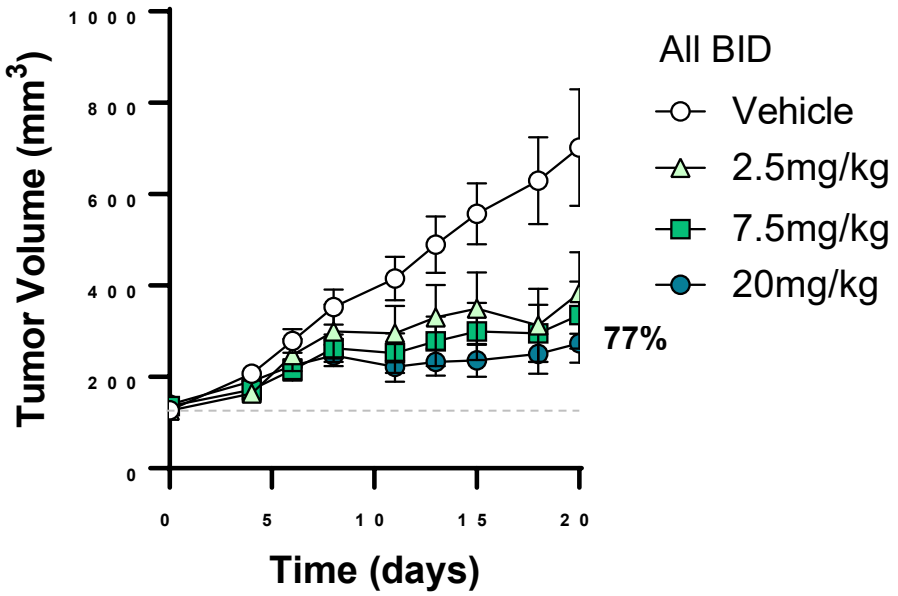
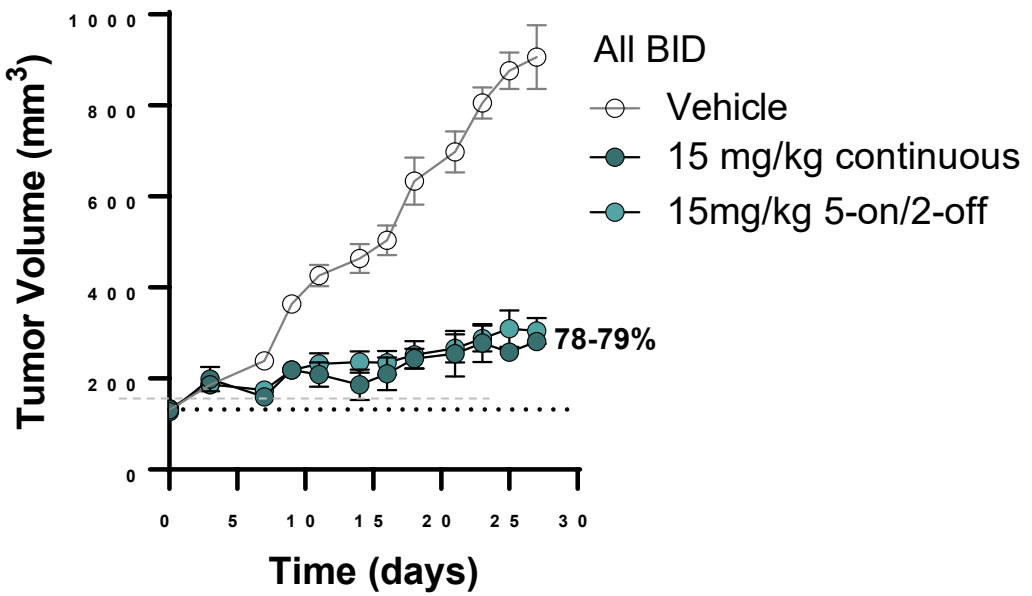
Lunresertib inhibits growth of CCNE1 amplified xenograft tumors

HCC1569 Breast Cancer CDX Model





Robust tumor growth inhibition (TGI) observed at well tolerated doses and exposures

Intermittent dosing delivers equivalent TGI as continuous dosing

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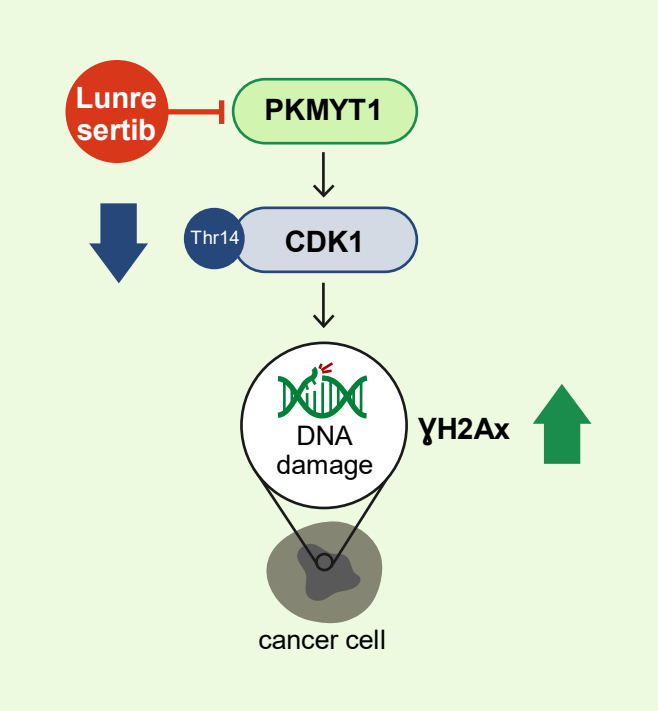
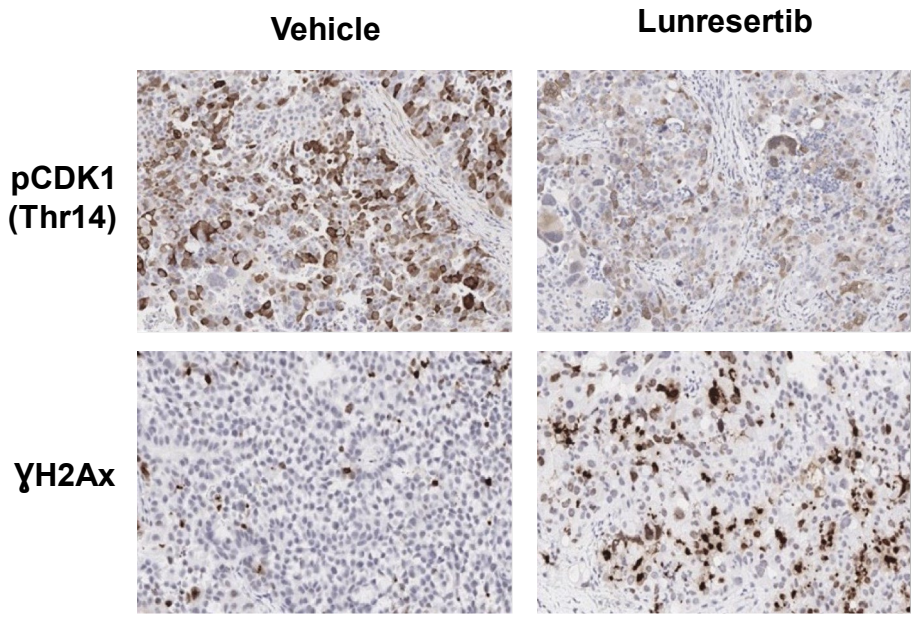
Lunresertib leads to CDK1 activation and induction of DNA damage *in vivo*





- PD biomarkers tested across *CCNE1* amplified and *FBXW7* mutant CDX and PDX *in vivo* models
- ~50% CDK1 dephosphorylation (IHC) and a ~2-fold γH2Ax increase was required for maximal anti-tumor activity across models. Comparable induction observed with DDR targeting agents (e.g., ATRi, PARPi).
- Lunresertib MOA to be confirmed in paired tumor biopsies collected in Phase 1

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Lunresertib initial global clinical trial program

Key inclusion criteria:

Recurrent solid tumors
CCNE1 amplification or
PPP2R1A FBXW7 inactivating mutations

Dose escalation → Dose & schedule

Monotherapy: Lunresertib
Initiated April 2021

Combinations: Lunresertib +

+ Gemcitabine	**MAGNETIC:** Initiated Dec 21
+ Camonsertib	**MYTHIC:** Initiated May 22
+ FOLFIRI	**MINOTAUR:** Initiated Aug 22
+ CCTG [1]	**IST:** Enrolling
+ Carbo/paclitaxel [2]	**IST:** Expected 2023

Determine RP2D dose / schedule

Progress to POC trials

- Selected tumors with amplified CCNE1 — CCNE1
- Selected tumors with FBXW7 Loss — FBXW7
- Tumors with high rate of sensitivity genes — CCNE1, FBXW7, PPP2R1A
- Basket trial — CCNE1, FBXW7, PPP2R1A

FDA/EMA consultation re: further development

[1] Canadian Clinical Trial Group (CCTG) collaborations include NCT05605509 and NCT05601440.
[2] SOC for 1st line ovarian cancer is carbo/paclitaxel (6 cycles) + PARPi maintenance therapy or carbo/paclitaxel with bevacizumab + bev maintenance therapy; this IST supports future potential 1st line combination studies as triplet therapy in patients with CCNE1 amplified tumors.

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Study Principal Investigator: Timothy Yap, MBBS, PhD, FRCP

Medical Oncologist and Physician-Scientist at the University of Texas, MD Anderson Cancer Center

- Associate Professor, Department for Investigational Cancer Therapeutics
- Medical Director of the Institute for Applied Cancer Science
- Associate Director of Translational Research in the Institute for Personalized Cancer Therapy
- Primary research focuses on the first-in-human and combinatorial development of molecularly targeted agents and immunotherapies, and their acceleration through clinical studies using novel predictive and pharmacodynamic biomarkers
- Main interests include the targeting of the DNA damage response with novel therapeutics, such as ATR and PARP inhibitors, as well as the development of novel immuno-therapeutics
- BSc degree in Immunology and Infectious Diseases and MD from Imperial College London, UK

Speaker disclosures: Timothy Yap, MBBS, PhD, FRCP

I have the following financial relationships to disclose:

Employment

University of Texas MD Anderson Cancer Center, where I am Medical Director of the Institute for Applied Cancer Science, which has a commercial interest in DDR and other inhibitors (IACS30380/ART0380 was licensed to Artios)

Grant/Research support (to the Institution)

Acrivon, Artios, AstraZeneca, Bayer, Beigene, BioNTech, Blueprint, BMS, Boundless Bio, Clovis, Constellation, Cyteir, Eli Lilly, EMD Serono, Forbius, F-Star, GlaxoSmithKline, Genentech, Haihe, Ideaya ImmuneSensor, Ionis, Ipsen, Jounce, Karyopharm, KSQ, Kyowa, Merck, Mirati, Novartis, Pfizer, Ribon Therapeutics, Regeneron, Repare, Rubius, Sanofi, Scholar Rock, Seattle Genetics, Tesaro, Vivace and Zenith

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Lunresertib Preliminary Monotherapy Clinical Trial Results

First-in-human biomarker-driven Phase 1 MYTHIC trial of PKMYT1 inhibitor lunresertib (RP-6306) in patients with advanced solid tumors harboring *CCNE1* amplification or *FBXW7* or *PPP2R1A* genomic alterations

Timothy A. Yap, MBBS, PhD, FRCP
University of Texas MD Anderson Cancer Center, Houston, TX;

On behalf of MYTHIC study Investigators

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First-in-human MYTHIC study

PK**MYT**1 In**HI**bition for the treatment of **C**ancers: study design

Phase 1 MYTHIC study NCT04855656 (accruing)

Inclusion criteria:

Pts ≥12yo with solid tumors resistant or intolerant to standard therapy

Tumors centrally reviewed* with **CCNE1** amplification**, deleterious **FBXW7** or **PPP2R1A** alterations

ECOG 0, 1 or 2

Hgb ≥9.0 g/dL,
Platelets ≥100 K/uL,
ANC ≥1.5 K/uL

Module 1:
Single agent lunresertib

N=63 pts enrolled

Study is ongoing

28 April 2021 — Initiated

28 April 2023 — Data cut-off date

✓ **Primary endpoints:**

- Safety and tolerability
- Recommended phase 2 dose (RP2D), schedule

✓ **Other endpoints:**

- Pharmacokinetics
- Pharmacodynamics in paired tumor biopsies
- Preliminary antitumor activity
- Kinetics of circulating tumor DNA (ctDNA)

*Central review by Precision Oncology Decision Support (PODS) Group at MDACC
**CCNE1 amplification (Copy number ≥6)

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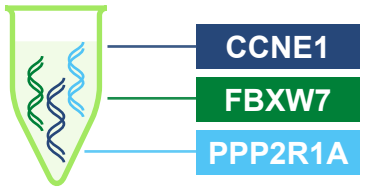
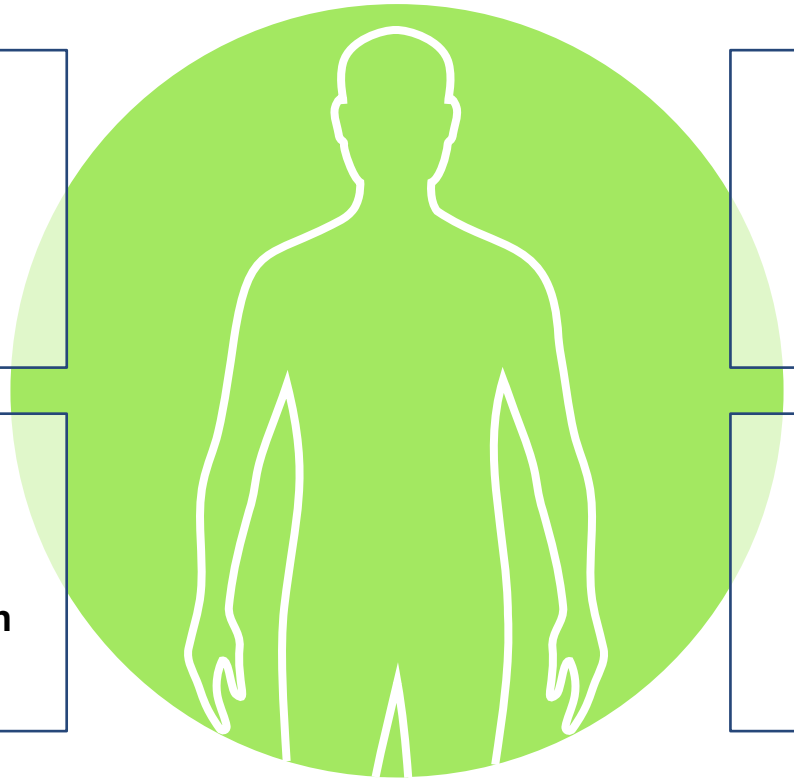
Comprehensive biomarker analyses for first in class PKMYT1i

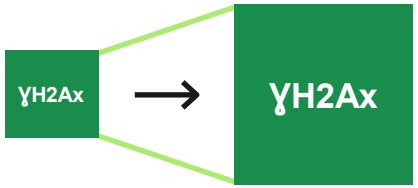


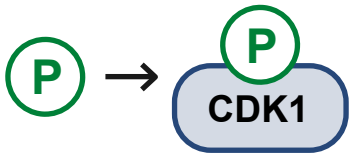
Targeting 3 genes

Copy Number alterations and mutation profile



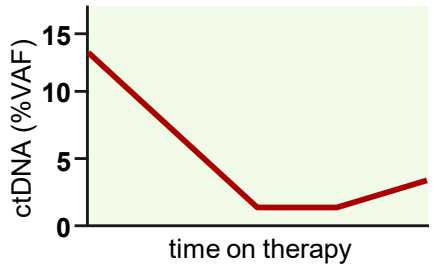
Proof of principle - DNA damage





Proof of mechanism - PKMYT1 inhibition evidence

Molecular response monitoring (ctDNA)



- Thorough understanding of predictive biomarkers for lunresertib
- Assessment of PK/PD relationship
- Early efficacy readout via ctDNA

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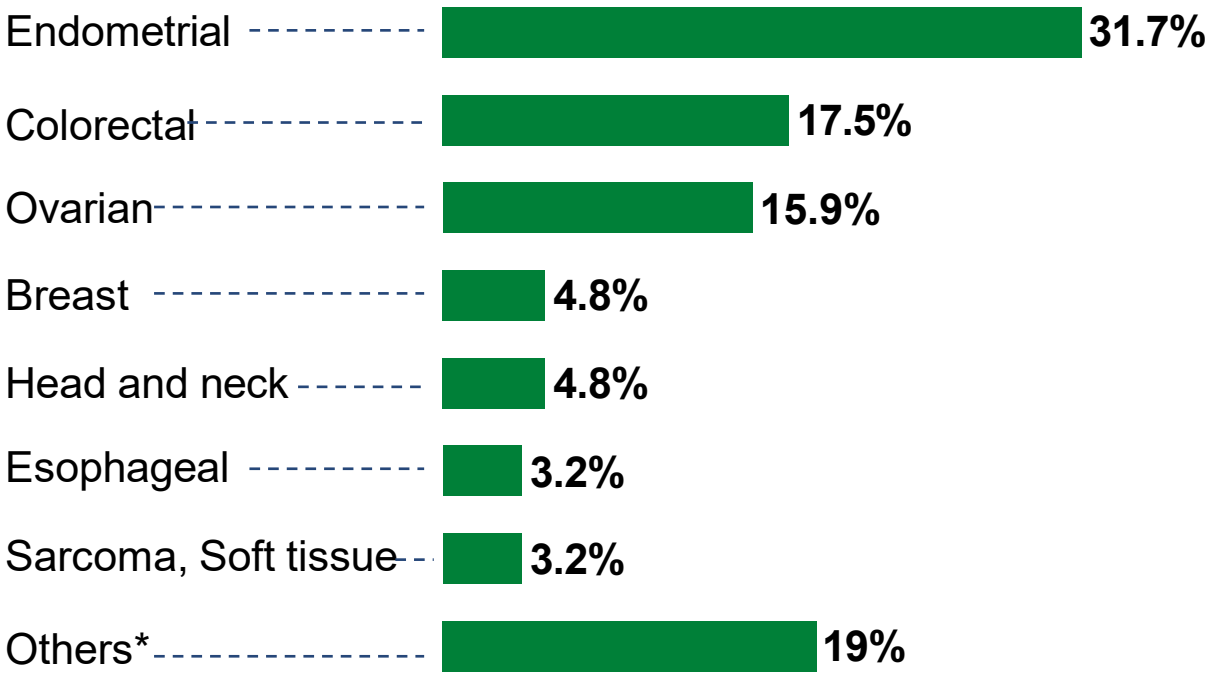
FIH Phase 1 MYTHIC study N=63, tumors and genotypes

Tumor types:



* Uterine Carcinosarcoma, Bladder, Brain, Cervical, Gastroesophageal Junction, Gastrointestinal, Melanoma, Gallbladder, Vulvar, Sarcoma

Most common genotypes:

CCNE1 amplification	46.0%
FBXW7	31.7%
PPP2R1A	17.5%
FBXW7/PPP2R1A	1.6%
Endometrial cancer without above mutations	3.2%

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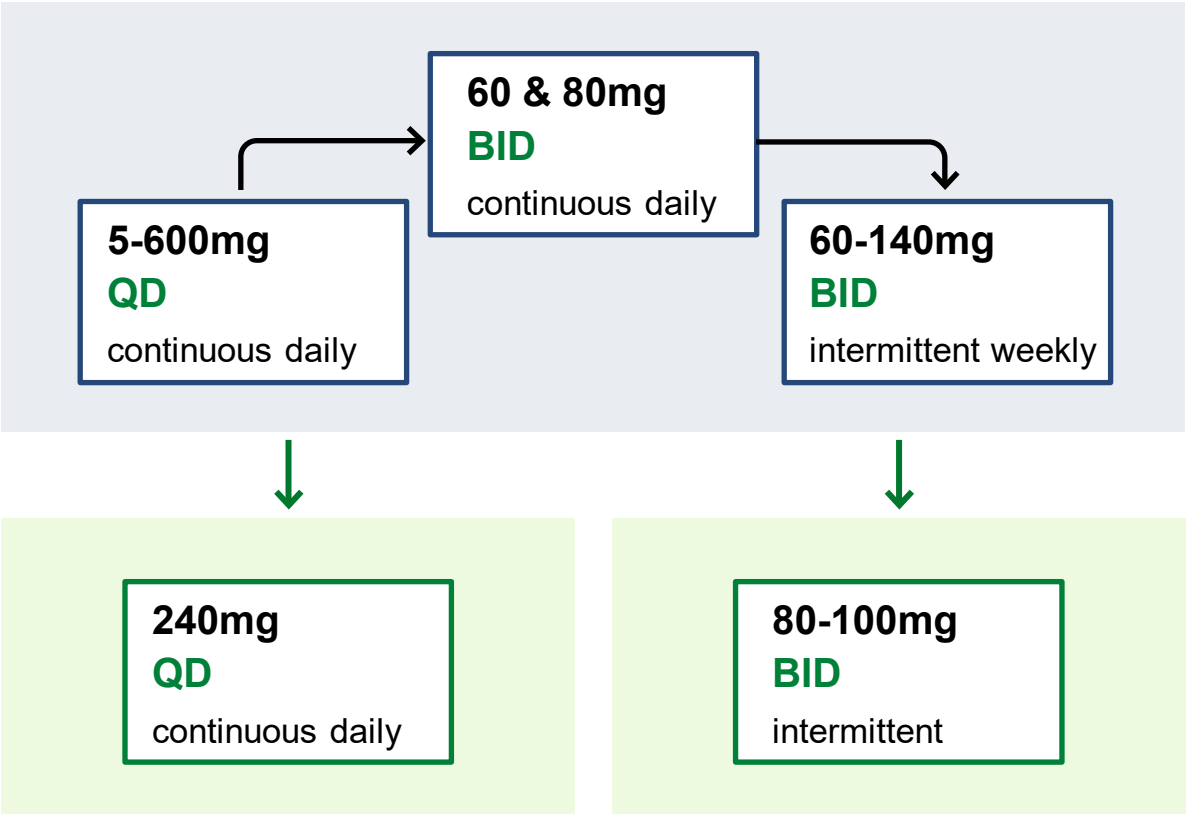
Single agent lunresertib tested at multiple doses/schedules



- Adaptive BOIN design and sufficient cohort sizes to ensure robust MTD/RP2D decision
  - Continuous daily 240mg QD and 80-100mg BID intermittent weekly schedules proposed for future combination use
- DLT: Reversible rash
- Rash any grade reported in 36.5% of patients (7.9% grade 3)
- Well managed with dose modification, topical steroids, emollients and oral antihistamines
- Rechallenge at reduced dose well tolerated, intermittent schedule prevents G3 rash

Investigation of the mechanism of rash ongoing.
MTD: maximum tolerated dose, RP2D: recommended Phase 2 dose

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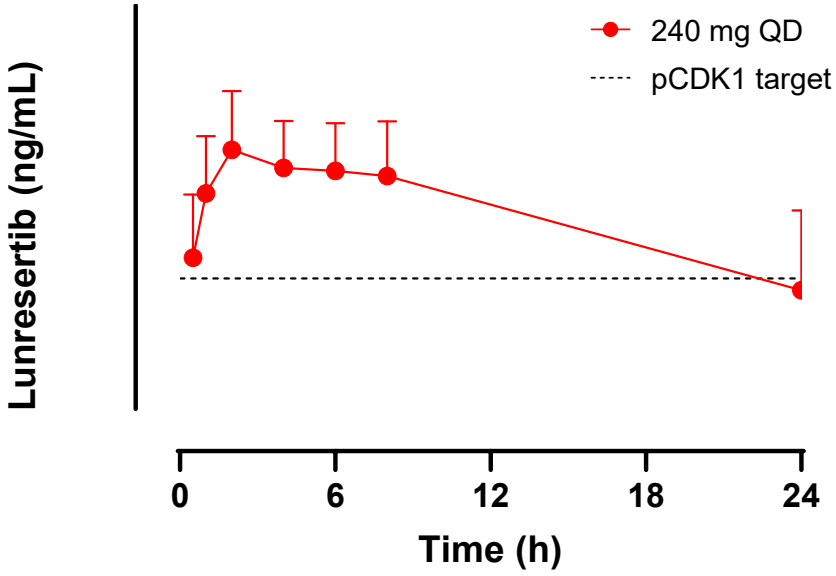
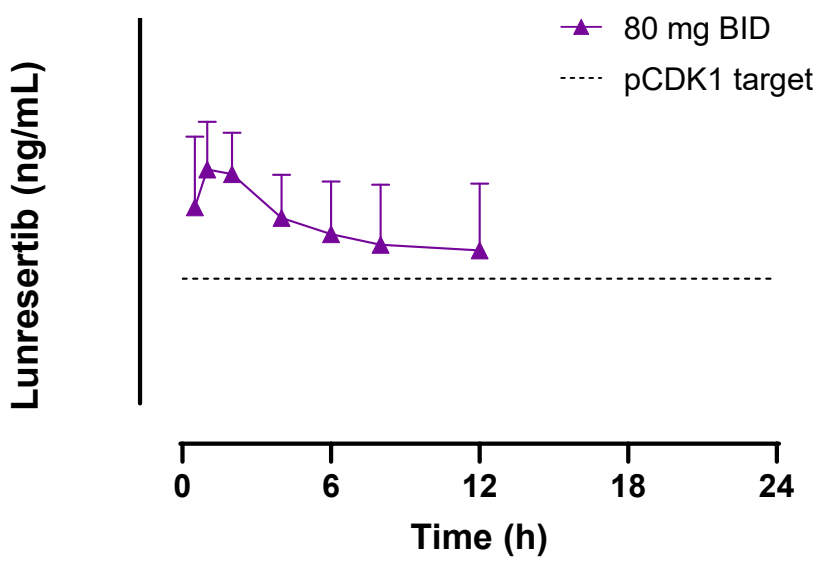
Human PK achieves preclinical target coverage at recommended doses

Cycle 1-Day 1 PK at 240 mg QD



Cycle 1-Day 1 PK at 80 mg BID



Human lunresertib PK is linear up to daily doses of 160-240 mg with a half-life of ~9 hours

Exploration of QD and BID regimens to maximize target coverage

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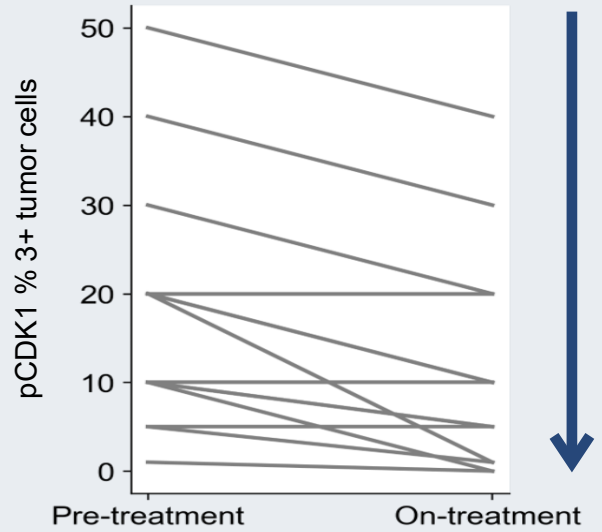
Clinical mechanism of action confirmed in paired biopsies

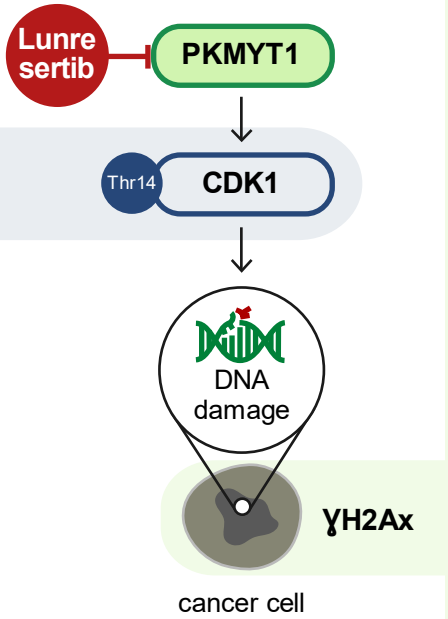
pCDK1-Thr14
(Direct Target Inhibition)



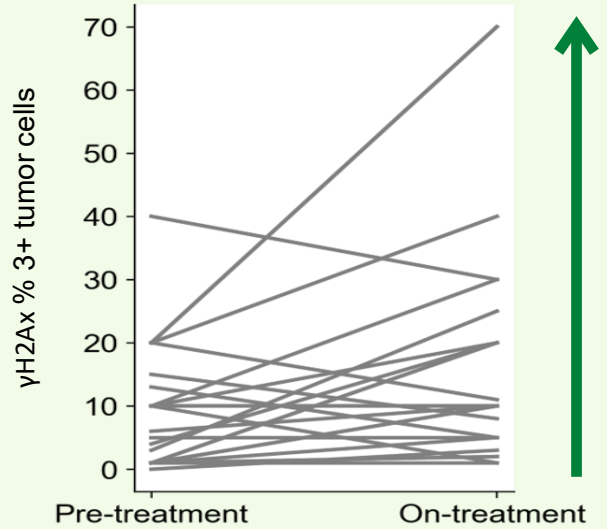
CDK1 -50%

	Pre-treatment (n=17)	On-treatment (n=17)
Median	20	10
p-value	-	**0.001**

Interim trial data



γH2Ax
(Induction of DNA Damage)



γH2Ax ~2-fold

	Pre-treatment (n=25)	On-treatment (n=25)
Median	6	10
p-value	-	**0.022**

Interim trial data

Preclinical PD targets that drive maximal activity (~50% CDK1 dephosphorylation and ~2-fold induction of γH2AX) achieved in paired tumor biopsies acquired pre- and post- lunresertib treatment

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Lunresertib related Treatment Emergent Adverse Events

Distinct profile from cell cycle inhibitors currently in clinic: limited myelotoxicity

All grades, occurring in ≥10% of patients

	All Patients N=63			Recommended Doses 80-100mg BID-I & 240mg QD-C, N=17		
Adverse event:	**All Grades (%)**	**G3 (%)**	**G4 (%)**	**All Grades (%)**	**G3 (%)**	**G4 (%)**
Rash*	36.5%	7.9%	**0**	47.1%	5.9%	**0**
Nausea/Vomiting	33.3%	1.6%	**0**	29.4%	0	**0**
Fatigue	23.8%	1.6%	**0**	29.4%	0	**0**
Anemia	20.6%	6.3%	**0**	23.5%	11.8%	**0**
Decreased appetite	15.9%	0	**0**	0	0	**0**

- **Safety profile unremarkable**
- **Favorable tolerability profile:**
  - manageable adverse events
  - events of interest: rash, nausea/vomiting
  - grade 3 toxicity infrequent
  - no grade 4 toxicity

Proposed dose/schedule and food mitigated both rash and nausea/vomiting

* Rash terms included: Rash Maculopapular, Pruritis, Rash, Skin Exfoliation, Erythema, Dermatitis Contact, Eczema, Flushing, Rash Erythematous, Rash Pruritic

REPARE THERAPEUTICS

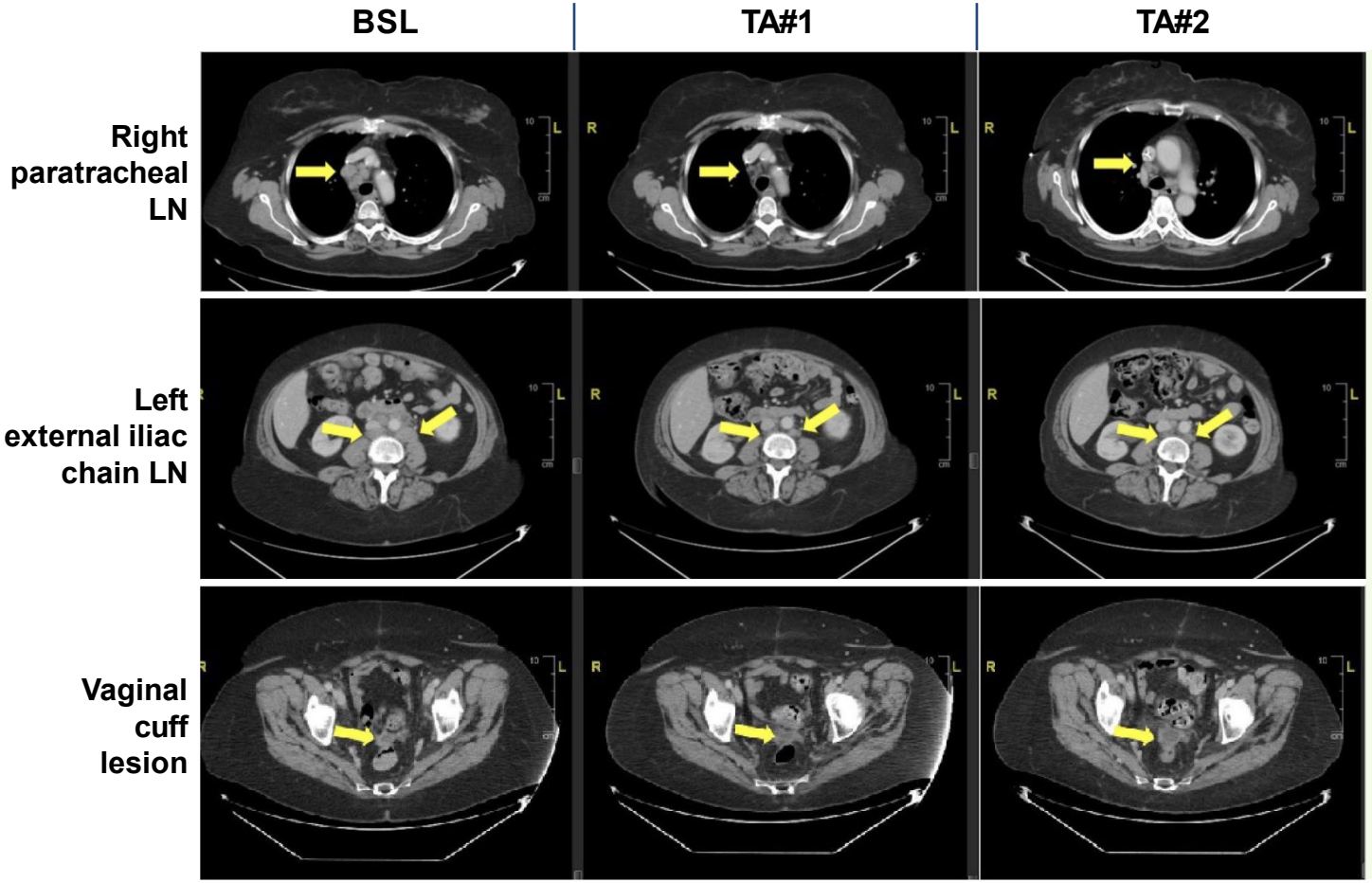
Tumor response to lunresertib monotherapy at intermittent RP2D

Female
73 years old

Metastatic recurrent uterine carcinosarcoma

FBXW7 & PPP2R1A

Received 3 prior lines of therapy



- **Overall response: cPR (RECIST)**
- **Max. tumor burden decrease - 41%**
- **On therapy 7+ months ***

Several patients with <30% tumor shrinkage and long stable disease ongoing up to >11 months

* As of June 1, 2023

REPARE THERAPEUTICS

Lunresertib: preliminary MYTHIC trial monotherapy conclusions

- ✓ **Proof of concept established in clinic**
- ✓ **Monotherapy appears safe and well tolerated**
  - Potentially suitable for maintenance therapy
- ✓ **Preliminary antitumor activity observed, including:**
  - Confirmed RECIST partial response
  - Several patients with <30% tumor shrinkage and long stable disease ongoing up to >11 months
- ✓ **Preclinical findings translated into the clinic**
  - Confirmed PKMYT1 inhibition and DNA damage at active doses
- ✓ **Intermittent & continuous schedules enable combinations**

Achieved monotherapy objectives and continuing to move forward on monotherapy and combination opportunities

REPARE THERAPEUTICS

Lunresertib initial global clinical trial program

Key inclusion criteria:

Recurrent solid tumors
CCNE1 amplification or
PPP2R1A FBXW7 inactivating mutations

Dose escalation → Dose & schedule

Monotherapy: Lunresertib
Initiated April 2021

Preliminary data today

Combinations: Lunresertib +

+ Gemcitabine	MAGNETIC: Initiated Dec 21	Preliminary data Q4 2023
+ Camonsertib	MYTHIC: Initiated May 22	Preliminary data Q4 2023
+ FOLFIRI	MINOTAUR: Initiated Aug 22	
+ CCTG [1]	IST: Enrolling	
+ Carbo/paclitaxel [2]	IST: Expected 2023	

Determine RP2D dose / schedule

Progress to POC trials

- Selected tumors with amplified CCNE1 — CCNE1
- Selected tumors with FBXW7 Loss — FBXW7
- Tumors with high rate of sensitivity genes — CCNE1, FBXW7, PPP2R1A
- Basket trial — CCNE1, FBXW7, PPP2R1A

FDA/EMA consultation re: further development

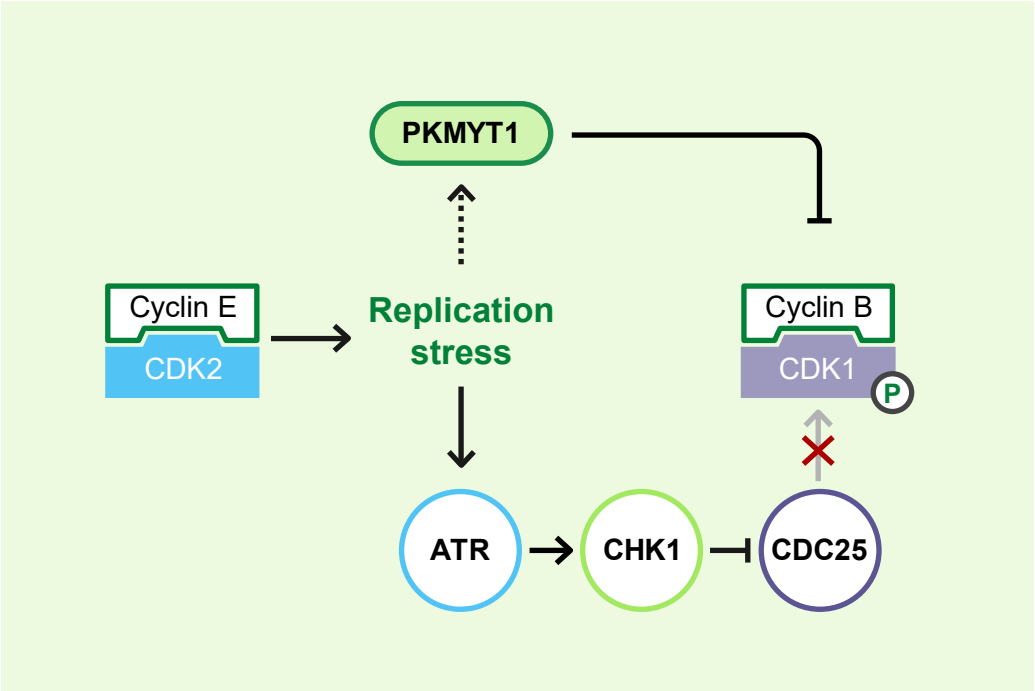
[1] Canadian Clinical Trial Group (CCTG) collaborations include NCT05605509 and NCT05601440.
[2] SOC for 1st line ovarian cancer is carbo/paclitaxel (6 cycles) + PARPi maintenance therapy or carbo/paclitaxel with bevacizumab + bev maintenance therapy; this IST supports future potential 1st line combination studies as triplet therapy in patients with CCNE1 amplified tumors.

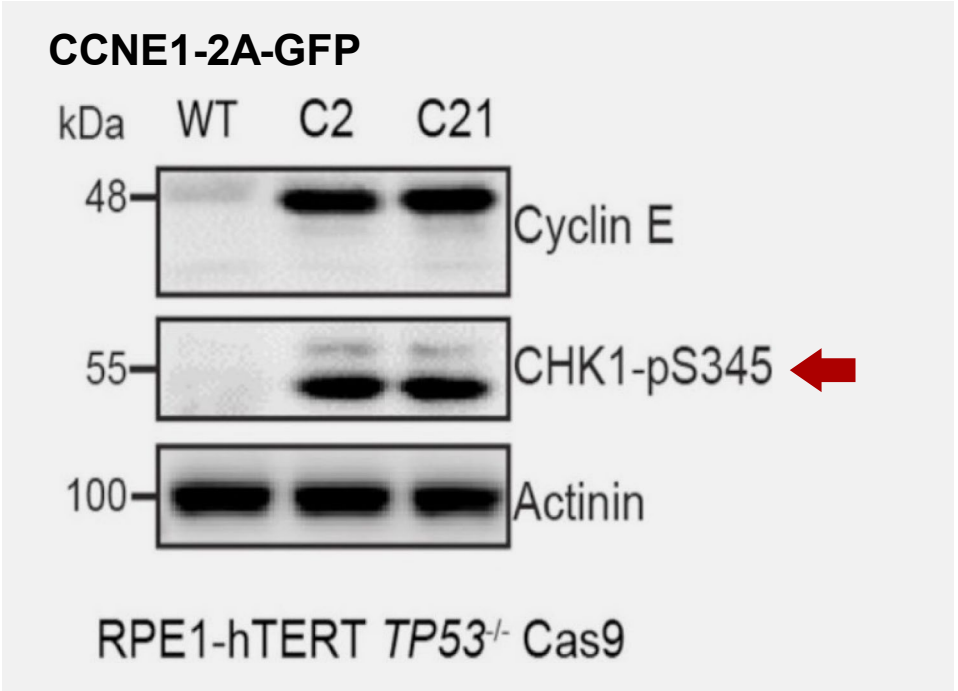
REPARE THERAPEUTICS

Lunresertib Ongoing Combination Trials

Preclinical rationale for testing lunresertib with camonsertib

Replication stress caused by CCNE1 overexpression engages ATR

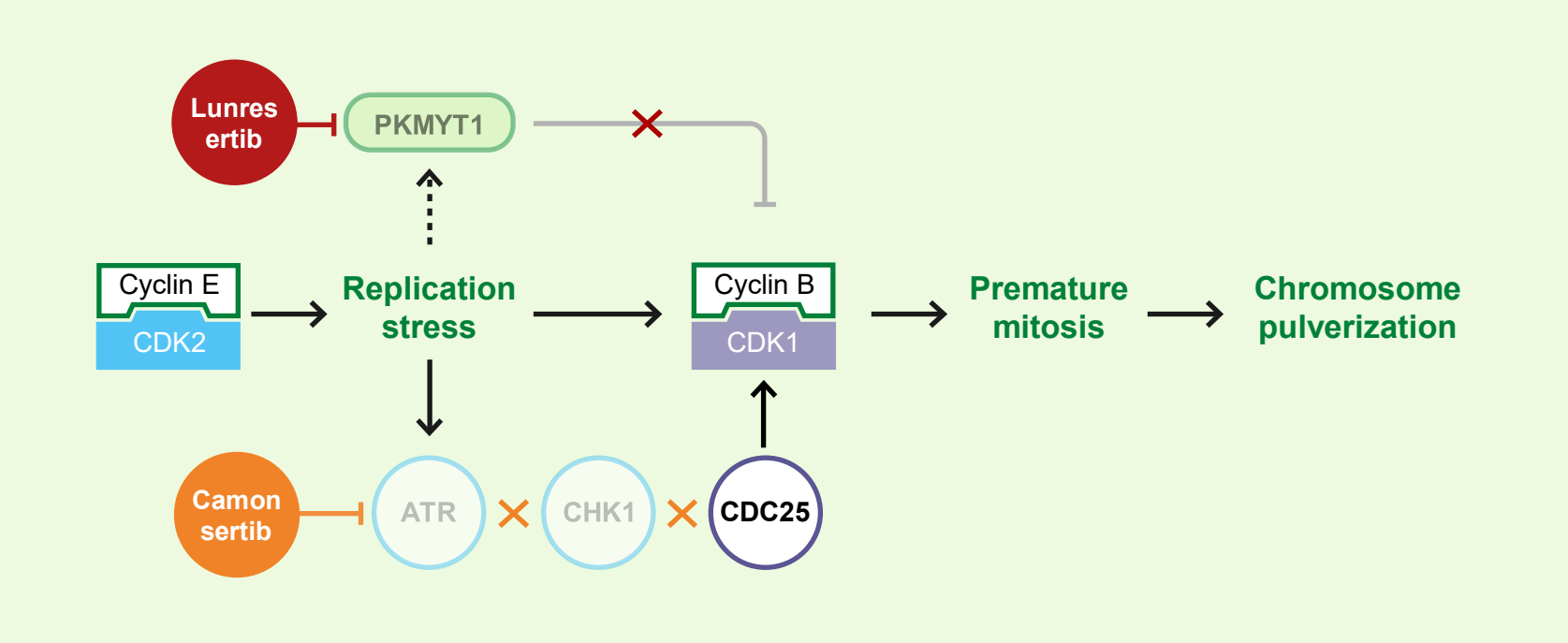




- *CCNE1* amplification activates the ATR pathway
- ATR (CHK1-mediated inhibition of CDC25) and PKMYT1 inhibition result in inactive CDK1
- These findings provide a rationale for combination of ATR and PKMYT1 inhibition

REPARE THERAPEUTICS

Lunresertib + camonsertib combination MoA hypothesis

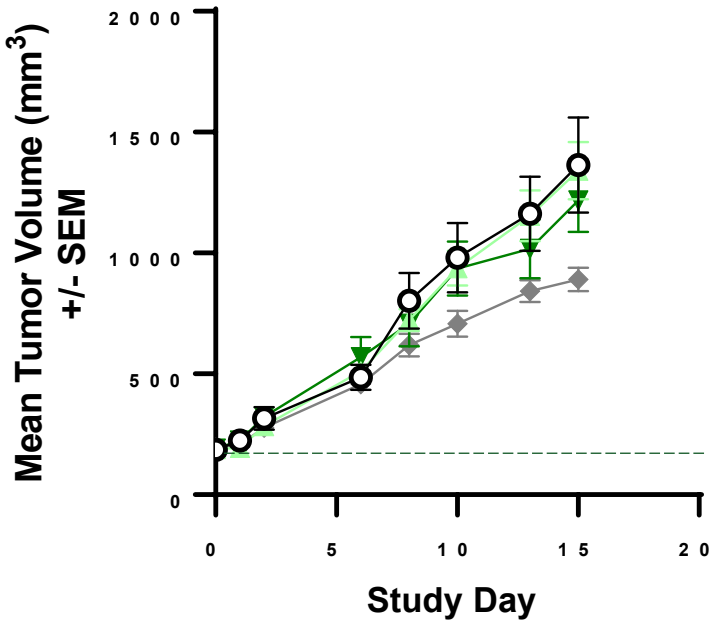


- Addition of camonsertib to lunresertib exacerbates premature mitosis
- Camonsertib synergizes with lunresertib in tumor models with CCNE1 amplification, FBXW7 or PPP2R1A loss

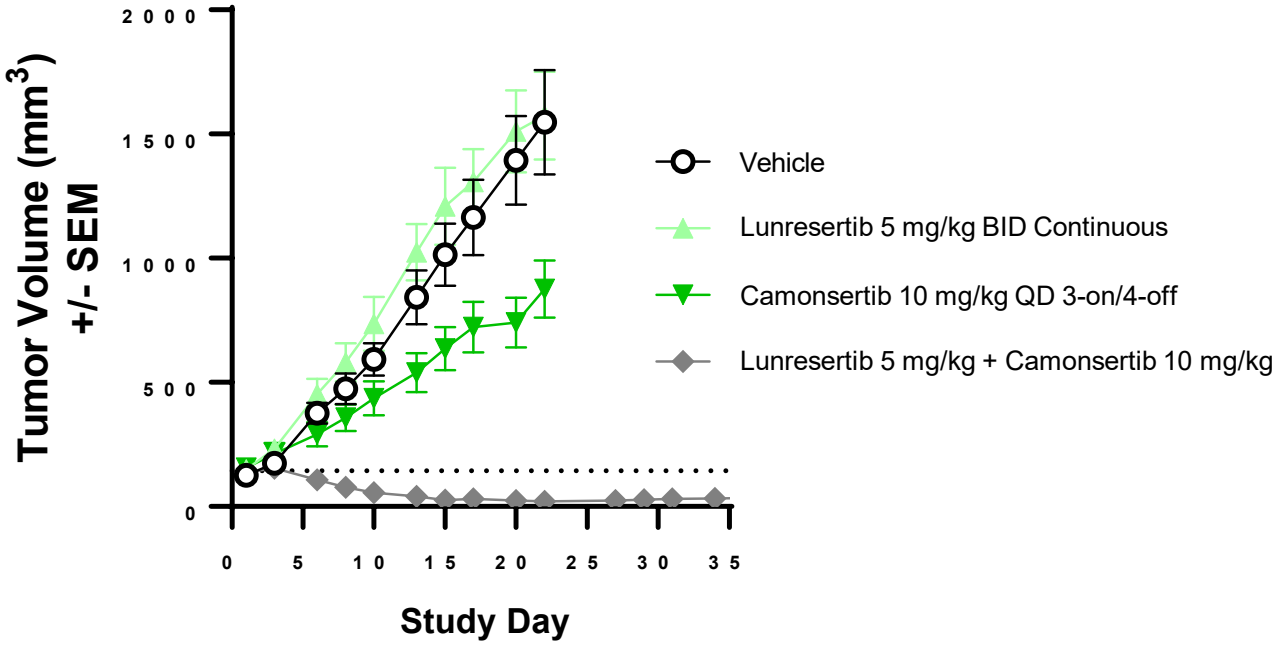
REPARE THERAPEUTICS

Lunresertib + camonsertib drives regression in *FBXW7 KO* models

DLD1 (*FBXW7 Wild Type*)



DLD1 (*FBXW7 Knockout*)



Lunresertib + camonsertib combination leads to significant tumor regressions at doses showing minimal single-agent activity

REPARE THERAPEUTICS

MYTHIC Module 2: Lunresertib with Camonsertib

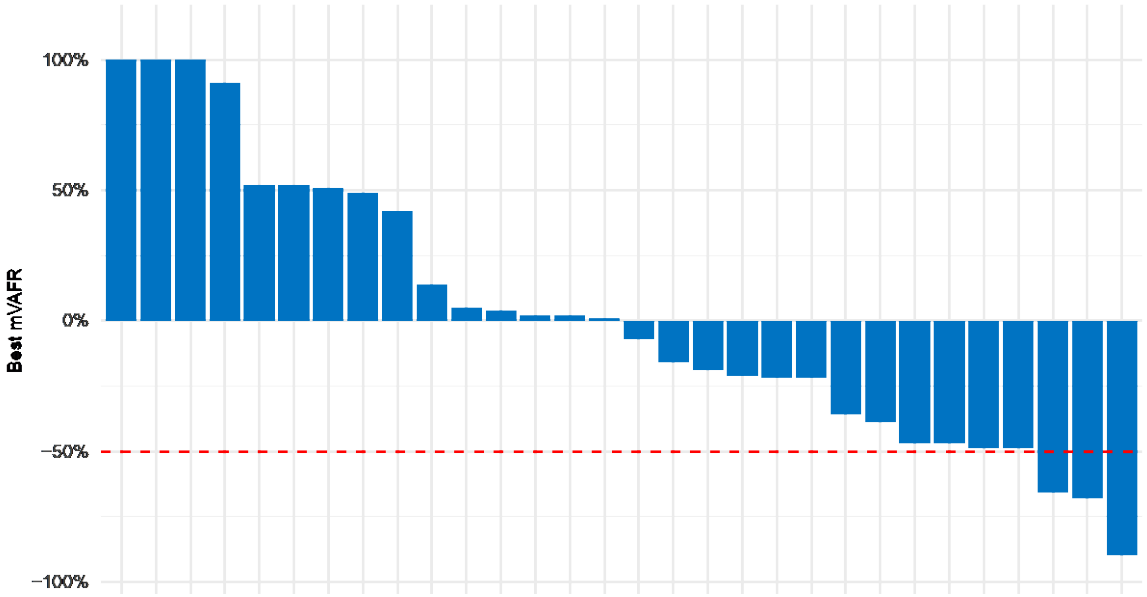
Brief preview of clinical data

Greater anti-tumor activity of lunresertib + camonsertib than lunresertib

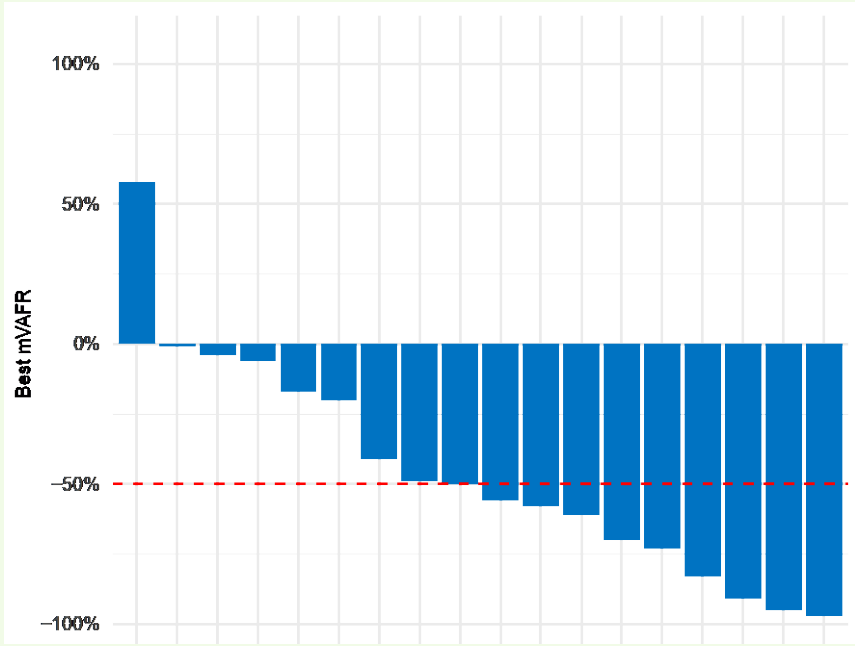
Monitoring of Molecular Response

Analysis of ctDNA changes at baseline, 3 weeks, 6 weeks (interim trial data)

MYTHIC Module 1: Lunresertib monotherapy



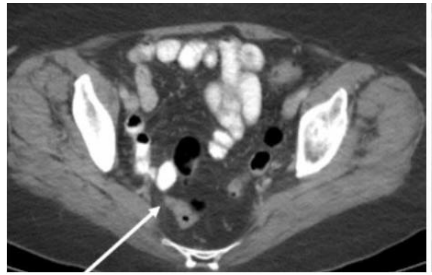
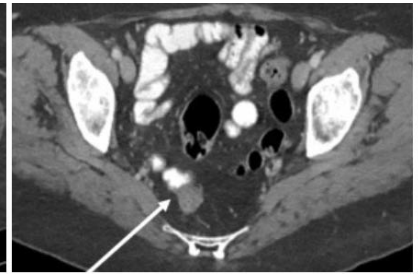
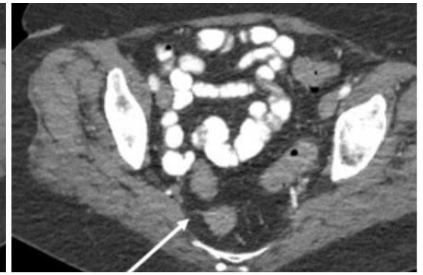
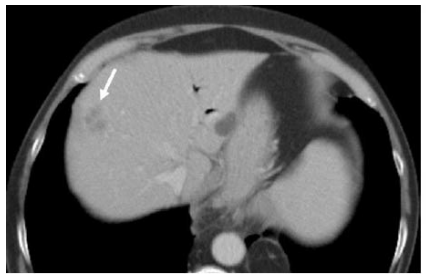
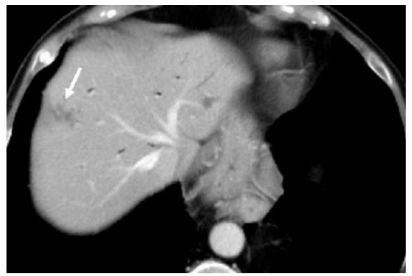
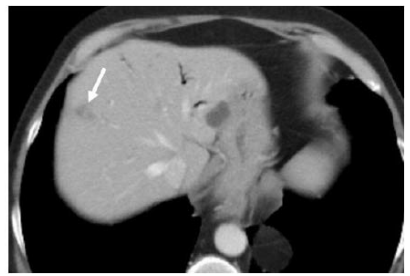
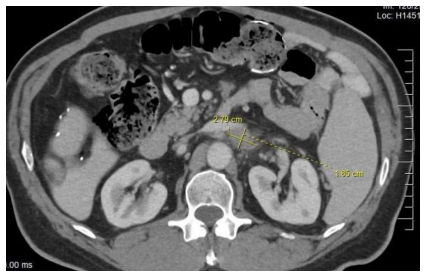
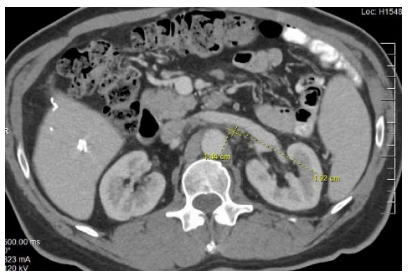
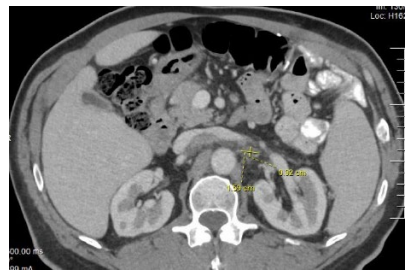
MYTHIC Module 2: Lunresertib + camonsertib



Greater depth and higher frequency of ctDNA changes in combination vs. monotherapy are expected to predict similar trend in clinical results in combination therapy

REPARE THERAPEUTICS

Lunresertib + camonsertib case studies: confirmed responses

		BSL	TA#1	TA#2	
♀ 66 y.o. Female Endometrial adenocarcinoma with **FBXW7** & **PPP2R1A** mutation (MSS)	Perirectal implant				Overall response: cPR (RECIST) Max tumor burden decrease: **-56%** On therapy for 7 months *
♀ 67 y.o. Female Cholangiocarcinoma with **CCNE1** amplification	Liver segment VIII				Overall response: cPR (RECIST) Max tumor burden decrease: **-35%** On therapy for 6 months *
♂ 63 y.o. Male CRC with **FBXW7** mutation	Left para-aortic lymph node				Overall response: cPR (RECIST) Max tumor burden decrease: **-43%** On therapy for 5+ months *

Confirmed RECIST responses observed across genotypes and tumor types for lunresertib + camonsertib combination

* As of June 1, 2023

REPARE THERAPEUTICS

Conclusions from today's presentation

- ✓ **Potent and well tolerated, first in class PKMYT1 inhibitor**
- ✓ **Proof of concept established in clinic**
- ✓ **Monotherapy tolerability differentiated from WEE1 and CDK2 inhibitors**
- ✓ **Early efficacy signals in monotherapy and combinations**
  - Monotherapy cPR and several patients with long stable disease >11+ months
  - Combination cPRs, including in tumors not expected to respond to ATR inhibition
- ✓ **Recommended range of monotherapy doses and schedules**

Clear clinical understanding of the PKMYT1 target and lunresertib performance

Substantial evidence to support ongoing combinations to be presented in Q4 2023

REPARE THERAPEUTICS

Combination outlook for lunresertib

Encouraging early responses across gemcitabine, camonsertib, and FOLFIRI clinical combinations – in multiple tumor types and genotypes

Near-term guidance:

- MYTHIC (+ camonsertib) and MAGNETIC (+ gemcitabine) planned disclosure in 4Q 23
- MINOTAUR (+ FOLFIRI) to be disclosed subsequently
- Princess Margaret Cancer Center sponsored study start 2023 (lunresertib + carboplatin/paclitaxel)[1] in recurrent *TP53* mutated ovarian and uterine cancer
- Canadian Cancer Trials Group sponsored Phase 2 Basket Study is enrolling and second study of lunresertib + gemcitabine in patients with CDK4/6 inhibitor treated ER+/HER2- metastatic breast cancer is active
- WEE1 combination – evaluating *in vitro* and *in vivo* data to assess combination potential

[1] SOC for 1st line ovarian cancer is carbo/paclitaxel (6 cycles) + PARPi maintenance therapy or carbo/paclitaxel with bevacizumab + bev maintenance therapy; this IST supports future potential 1st line combination studies as triplet therapy in patients with CCNE1 amplified tumors

Q&A

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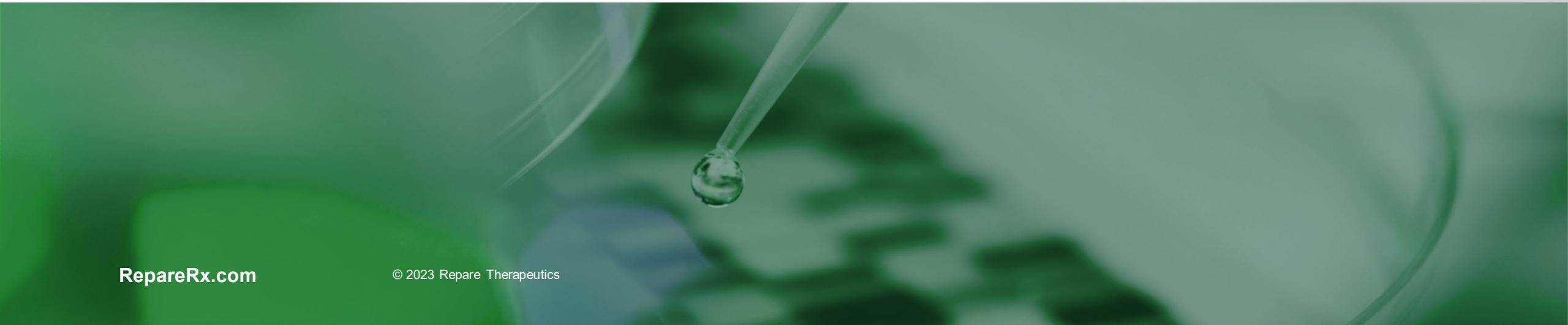

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