

Decoding Biology to Radically Improve Lives



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Such forward-looking statements are based on the current beliefs of Recursion's management as well as assumptions made by and information currently available to them, which are subject to inherent uncertainties, risks and changes in circumstances that are difficult to predict. Actual outcomes and results may vary materially from these forward-looking statements based on a variety of risks and uncertainties. For a complete discussion of factors that could materially affect our financial results and operations, please refer to Recursion's most recent Annual Report on Form 10-K, Recursion's Quarterly Reports on Form 10-Q for the quarterly periods ended March 31, June 30, and September 30, 2024, and the Company's other filings with the U.S. Securities and Exchange Commission (the "SEC"), which can be accessed at https://ir.recursion.com, or www.sec.gov. All forward-looking statements are qualified by these cautionary statements and apply only as of the date they are made. Recursion does not undertake any obligation to update any forward-looking statement, whether as a result of new information, future events or otherwise.

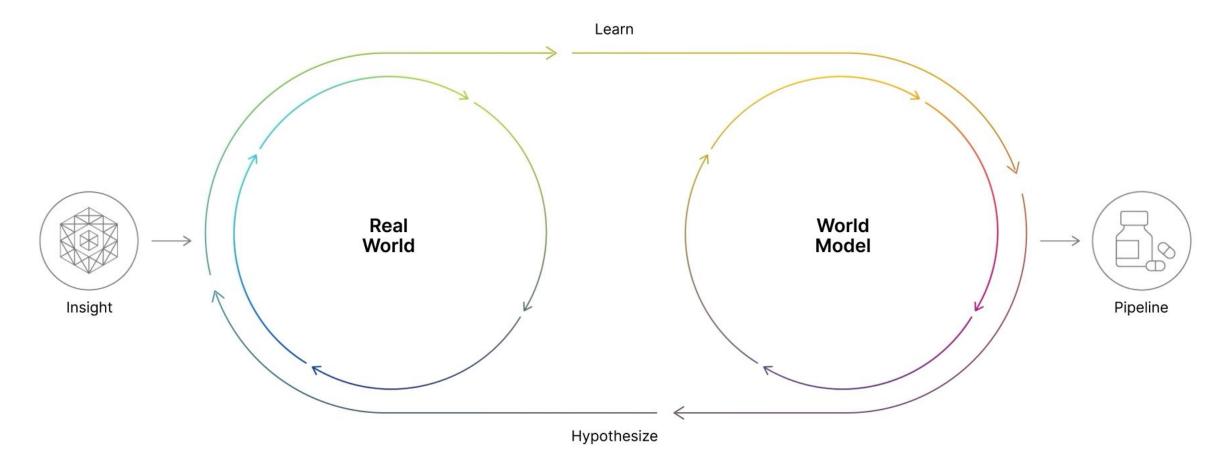
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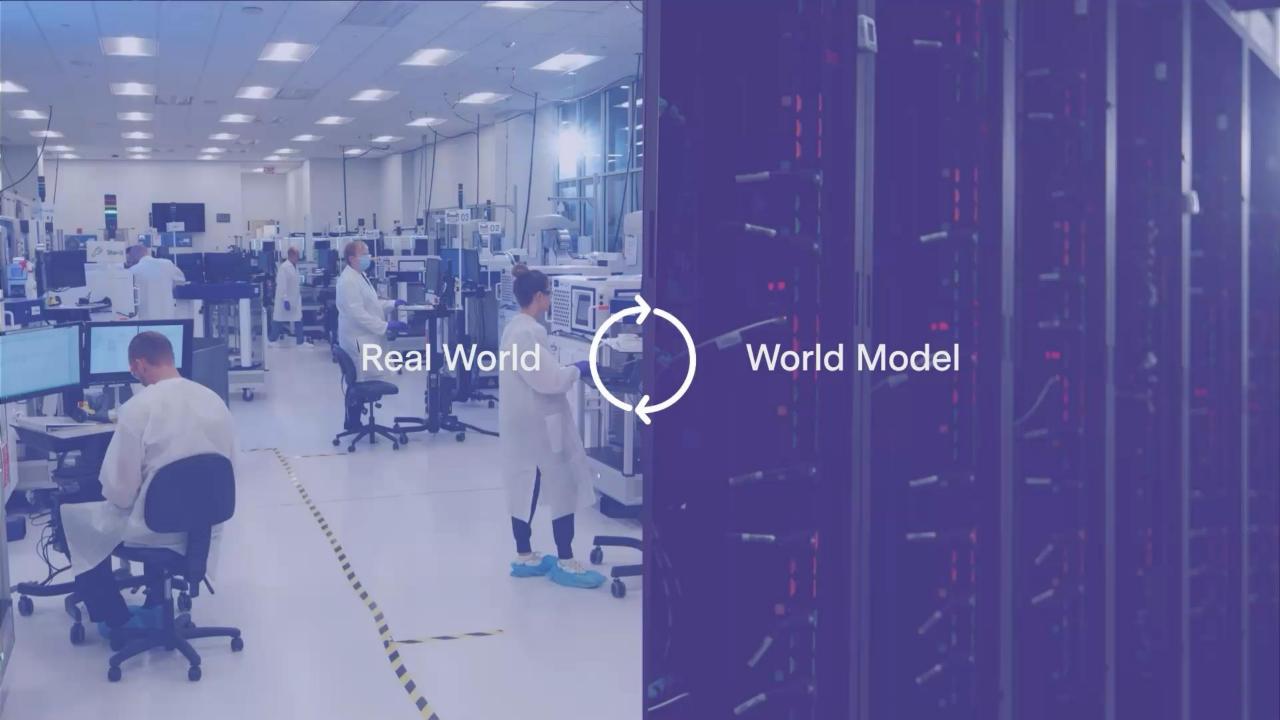
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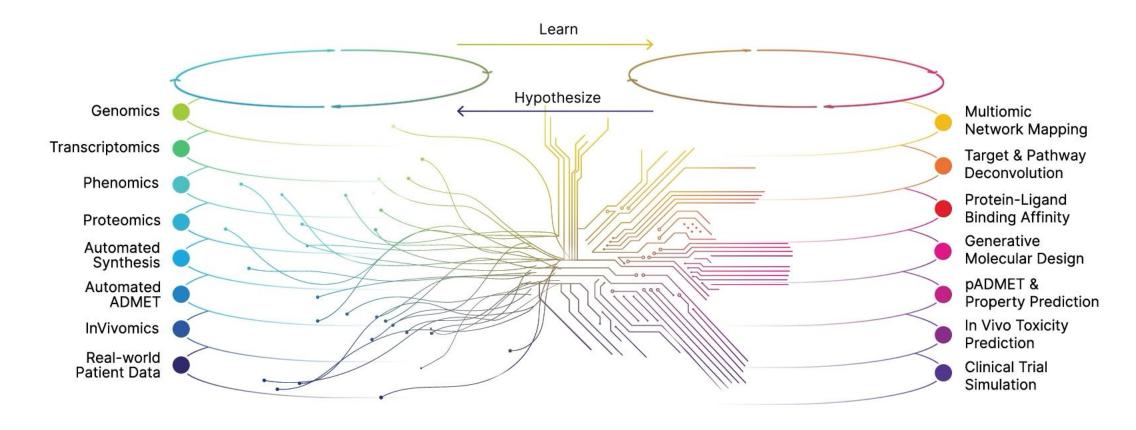
Full-stack Recursion OS is industrializing first-in-class & best-inclass drug discovery







Full-stack Recursion OS is industrializing first-in-class & best-in-class drug discovery





Portfolio poised for value creation with waves of new pipeline and partner programs emerging from Recursion OS

10

Clinical and preclinical programs¹

Oncology, rare diseases, and other high unmet need diseases

 ~ 10

Clinical program milestones over the next 18 months²

 ~ 10

Additional advanced discovery programs

1

Unified Operating System (OS) with both first & best-in-class capabilities 10 +

Partnered programs

Oncology, immunology, and other high unmet need diseases

~\$450M

Upfront and milestone payments earned to-date

~\$20B potential milestone payments

4

Large pharma collaborations







Merck KGaA
Darmstadt, Germany

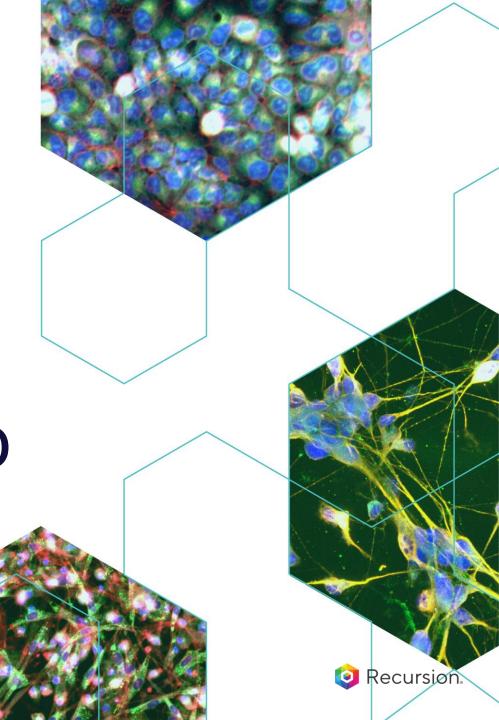
2. Program milestones includes data readouts, preliminary data updates, regulatory submissions, trial initiation, etc.



^{1.} Includes preclinical programs (programs expected to enter the clinic within the next 18 months).

VALUE CREATION

How the Platform Powers the Portfolio



Pipeline of ~10 clinical and preclinical technology-enabled programs

Candidate	Target	Indication	Preclinical	IND-Enabling	Phase 1/2	Pivotal / Phase 3	Next Anticipated Milestone
REC-617	CDK7	Advanced solid tumors ¹	ELUCIDATE				Combination study initiation – 1H25
REC-1245	RBM39	Biomarker-enriched solid tumors & lymphoma	DAHLIA				• Ph 1 dose-escalation update – 1H26
REC-3565	MALT1	B-cell malignancies	EXCELERIZE				• Ph 1 FPD – 1Q25
REC-4539	LSD1	Small-cell lung cancer (SCLC)	ENLYGHT				• Ph 1 FPD – 1H25
REC-994	Superoxide	Cerebral cavernous malformations (CCM)	SYCAMORE	1			• Ph 2 data – ISC³ – February 5th
REC-4881	MEK1/2	Familial adenomatous polyposis (FAP)	TUPELO				• Ph 1b/2 safety & early efficacy – 1H2
REC-2282	HDAC	Neurofibromatosis type 2 (NF2)	POPLAR				• PFS6 futility – 1H25
REV102 ²	ENPP1	Hypophosphatasia (HPP)					• IND-enabling studies initiation - 2025
REC-3964	TcdB	Prevention of recurrent <i>C. difficile</i> (rCDI)	ALDER				• Ph 2 update – 1Q26
REC-4209	Undisclosed	Idiopathic pulmonary fibrosis (IPF)					IND-enabling studies ongoing
~ 10 advanc	ed discovery pro	ograms including a PI3Kα H1047Ri					

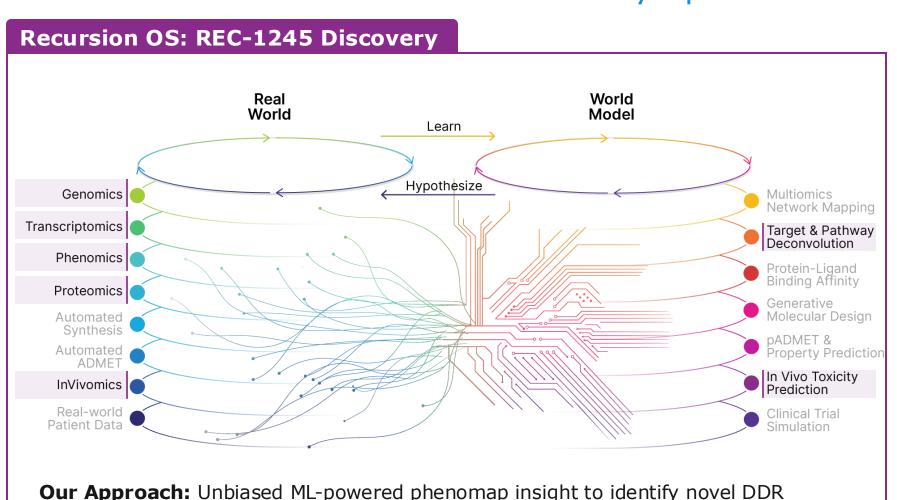


^{8 1.} Includes non-small cell lung cancer (NSCLC), colorectal cancer, breast cancer, pancreatic cancer, ovarian cancer, head and neck cancer

^{2.} Joint venture with Rallybio

^{3.} International Stroke Conference, late breaking oral abstract

REC-1245 (RBM39 degrader): A highly selective RBM39 degrader for biomarker-enriched solid tumors and lymphoma



REC-1245: RBM39

- Movel approach:

 Modulation of DDR

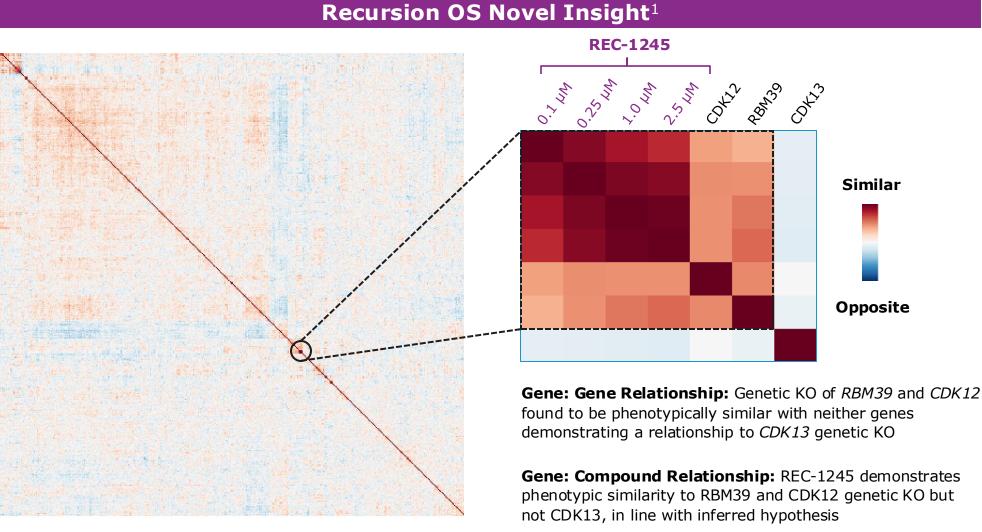
 via RBM39 to

 potentially avoid ontarget toxicities seen
 with cell cycle
 checkpoint inhibitors
 like WEE1, ATR, and
 CHK1/2
- Efficient R&D
 Campaign: 18
 months from target
 ID to IND-enabling
 studies



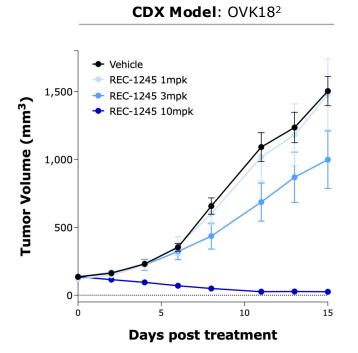
signature and relate cellular phenotypes

REC-1245 (RBM39 degrader): Functional similarity between RBM39 and CDK12 suggesting a potential novel approach to DDR modulation

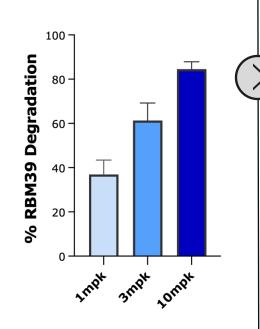


REC-1245 (RBM39 degrader): Potential first-in-class RBM39 degrader in Phase 1 dose-escalation with first patient dosed 4Q24

Compelling **dose-dependent** antitumor activity correlated with **RBM39** degradation in preclinical models¹



PD: Target Engagement³



Product Profile

- First-in-class mechanism

 RNA splicing for targeted tumor control
- **Highly potent and selective** *Minimal off-target liabilities identified*
- Precision based approach
 Biomarker defined population

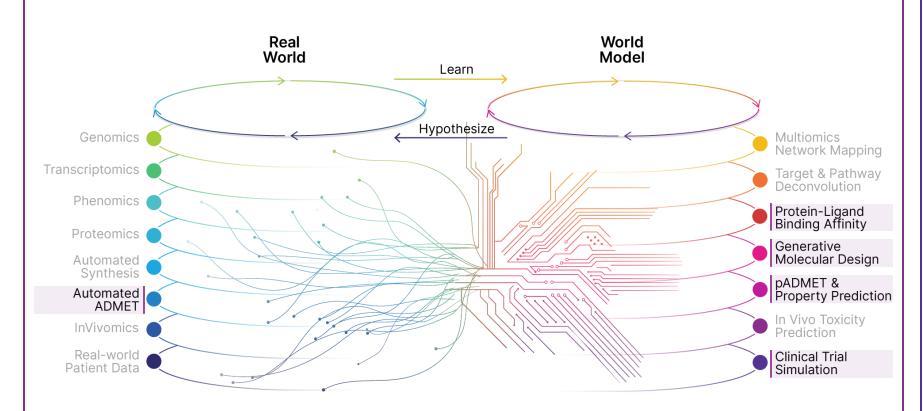
Clinical Updates

- First patient dosed 4Q24
- Recruitment active and ongoing
- Phase 1 dose-escalation update 1H26



REC-617 (CDK7 inhibitor): Precision design for optimizing therapeutic index with CDK7

Recursion OS: REC-617 Discovery



Our Approach: Generative AI and active learning to optimize molecule design, including non-covalent binding and ADME/PK for rapid absorption

REC-617: CDK7

- Recursion Approach:
 AI-powered precision
 design with reduced
 transporter
 interactions intended
 to minimize GI adverse
 events seen with prior
 molecules in class
- Rapid Design Cycle:

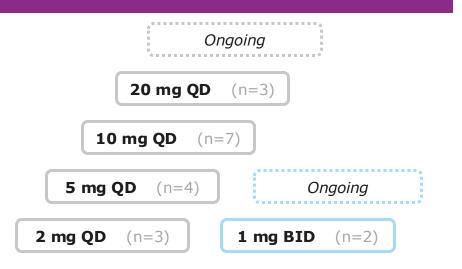
 136 novel compounds
 synthesized and under
 11 months from hit to candidate ID



REC-617 (CDK7 inhibitor): first-in-human study in solid tumors enrolled a heterogenous and heavily pretreated population

Phase 1 Design

- Unresectable, locally recurrent, or metastatic cancer
- Progressed following, or intolerant to, available standard of care treatments
- ECOG PS 0-1



Endpoints

Primary:

 Monotherapy safety and recommended Phase 2 dose (RP2D)

Key Secondary:

Pharmacokinetics

Exploratory:

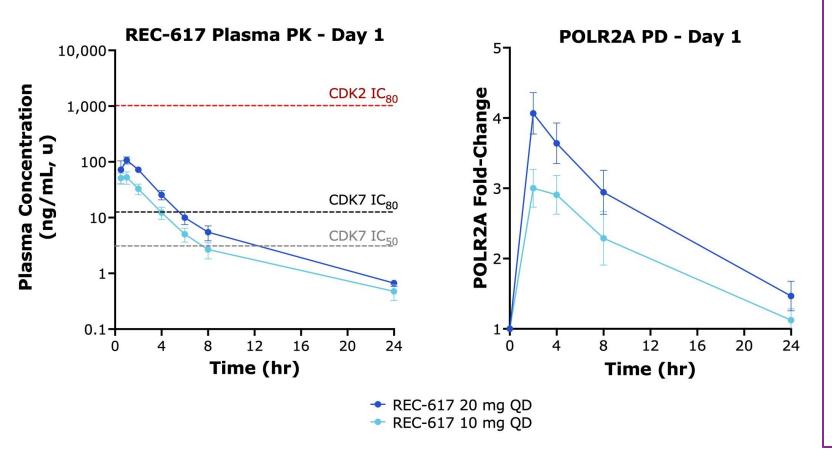
- Preliminary antitumor activity (RECIST v1.1)
- Pharmacodynamics: POLR2A gene expression in blood

Baseline Demographics

- N=19
- Median of 4 prior lines of therapy in advanced setting
- Heterogenous population, including:
 - Colorectal cancer
 - Breast cancer
 - Ovarian cancer
 - NSCLC
 - Pancreatic cancer



REC-617 (CDK7 inhibitor): REC-617 achieves dose dependent PK/PD and strong target modulation in the clinic

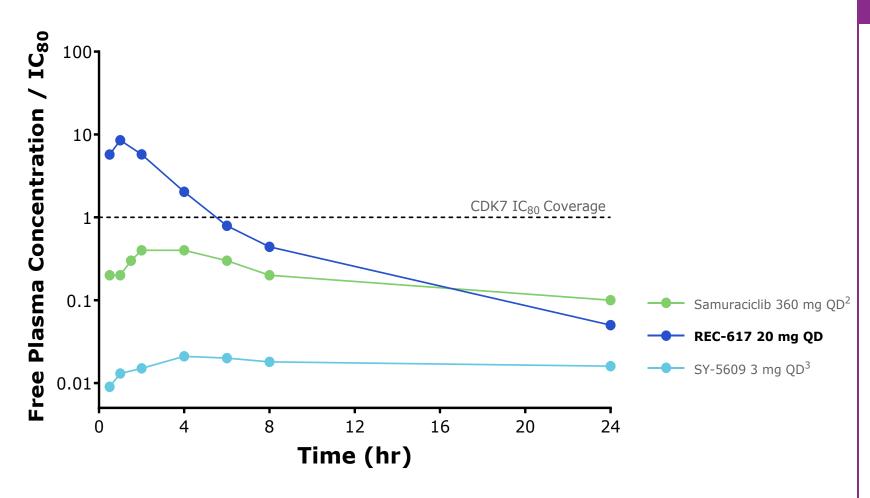


PK/PD Summary

- **Dose-Linear PK**: REC-617 exceeds CDK7 IC₈₀ with rapid absorption (Tmax 0.5–2h) and short t½ (5–6h)
- Robust Target Engagement: Early POLR2A 3-4x modulation suggests ~80-90% target engagement¹
- Rapid Transient Modulation:
 Quick, time-limited target engagement with POLR2A normalization in 24h
- BID Evaluation: Twice-daily dosing under investigation



REC-617 (CDK7 inhibitor): REC-617 offers a competitive and unique profile that potentially improves the therapeutic index



Key Differentiation

- Data suggests superior target coverage for REC-617¹ compared to two clinical CDK7 inhibitors
- REC-617 is more rapidly absorbed (earlier Tmax) compared to reported PK from two CDK7 inhibitors^{2,3} suggesting a reduction in localized GI residence time
- A shorter half-life would allow for flexible target modulation, which may improve the therapeutic index in the clinic



^{1.} CDK7 IC₈₀ reflects biochemical in vitro potencies on file

^{2.} Coombes, RC, Nat Comms (2023)

^{3.} Papadopoulos KM, et al. ENA (2020)

REC-617 (CDK7 inhibitor): Durable monotherapy PR observed in a metastatic ovarian cancer patient after 4 prior lines of therapy

One confirmed, durable partial response (PR)¹

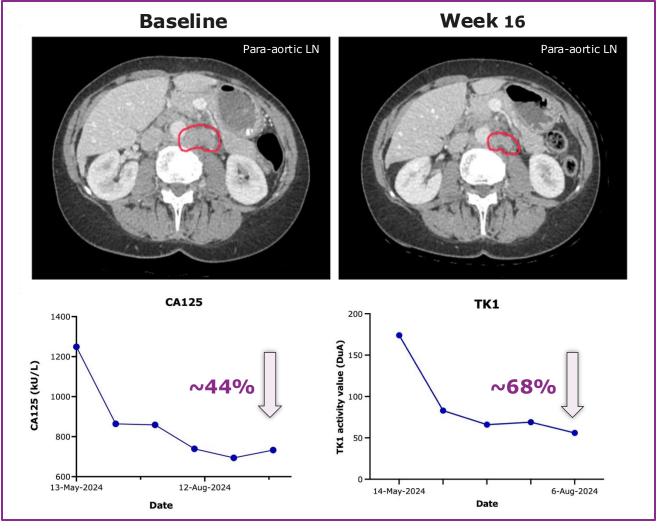
- Partial response (-34%) achieved at Week 16
- Meaningful reduction of tumor markers
- Response ongoing after 6+ months treatment

Early data indicates favorable safety profile

Maximum tolerated dose (MTD) not reached

Clinical Updates

- Continue dose escalation (QD & BID)
- Initiate combination study in 1H25
- Leverage new tech and clinical data partnerships for patient stratification

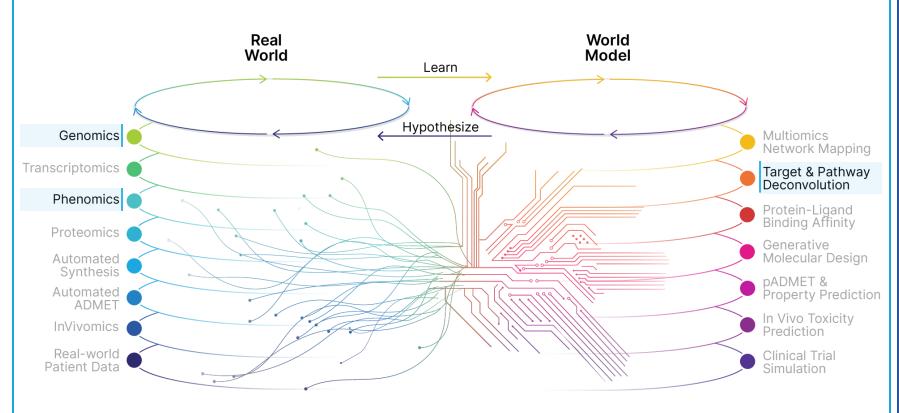


^{1.} By RECIST 1.1. Response evaluation criteria in solid tumors, PR: decrease of more than 30% in the sum of the longest diameters of target lesions + no new lesions + no progression of non target lesions



REC-994 (superoxide scavenger): A safe and well tolerated superoxide scavenger for the treatment of Cerebral Cavernous Malformation (CCM)

Recursion OS: REC-994 Discovery



Our Approach: Unbiased phenotypic screen to identify cellular and structural changes associated with CCM2 loss, a pathogenic mutation in CCM

REC-994 (superoxide)

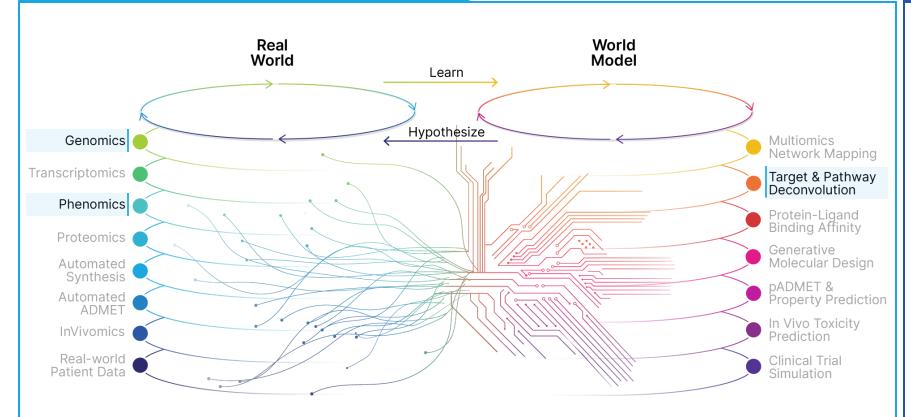


- Met primary endpoint of safety/tolerability in Phase 2 study
- Time- and dosedependent trends in reduced lesion volume and hemosiderin ring size compared to placebo
- **80%** of Phase 2 patients continued to LTE
- Full results to be presented at ISC as a late-breaking oral abstract on February 5, 2025



REC-4881 (MEK1/2 inhibitor): Modeling FAP-relevant biology to discover REC-4881, a potential best-in-class MEK1/2 inhibitor

Recursion OS: REC-4881 Discovery



Our Approach: Unbiased phenotypic screen to identify cellular and structural changes associated with APC loss, the driving genetic mutation in FAP

REC-4881 (MEK 1/2)

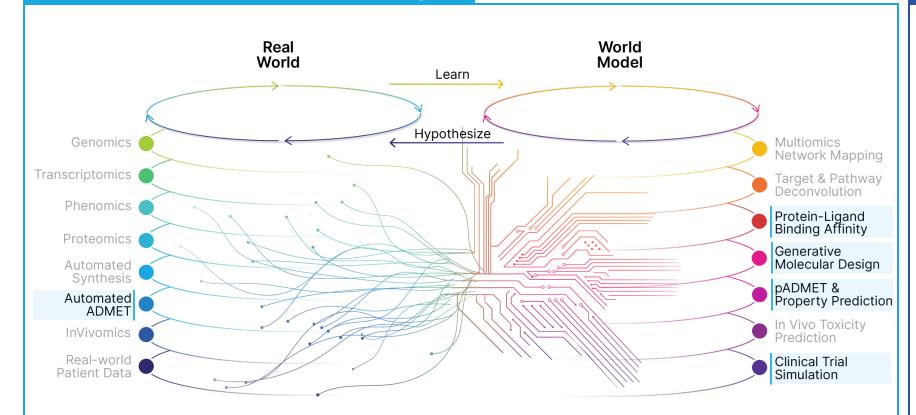


- 4 mg QD dose is pharmacologically active and well-tolerated
- Differentiated ADME profile may enhance exposures at the site of GI adenomas
- FTD (US) and ODD (US,EU)
- Ph 1b/2 safety and early efficacy data expected in 1H25



REV102 (ENPP1 inhibitor): Identified novel chemical space to enable the design of a potential first-in-class and best-in-class therapy for HPP

Recursion OS: REV102 Discovery



Our Approach: Generative AI-powered fragment-based screen to achieve selectivity and improve physiochemical properties compared to prior molecules in the class

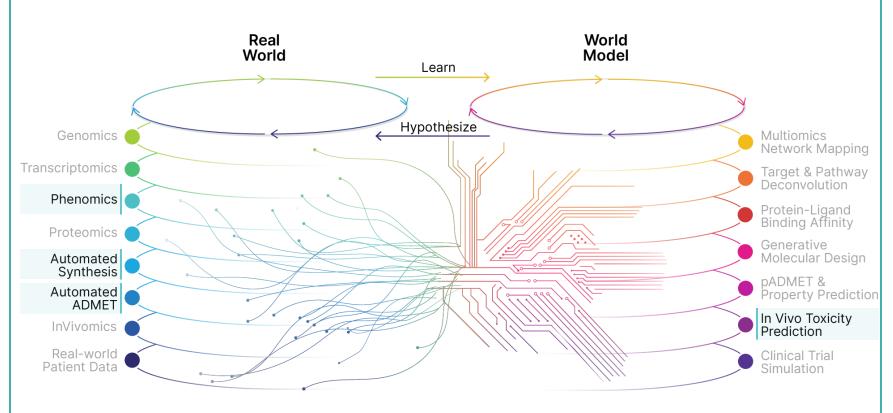
REV102 (ENPP1)

- Structurally distinct vs competitor ENPP1 inhibitors
- Dose-dependent normalizations in PPi and other disease relevant markers observed in genetic models of HPP
- Suitable for chronic dosing with no significant in vitro safety concerns
- IND-enabling studies to start in 2025



REC-3964 (CDI TcdB inhibitor): A safe and well tolerated *C. difficile* toxin B selective inhibitor for the prevention of recurrent *C. difficile* (rCDI)

Recursion OS: REC-3964 Discovery



Our Approach: Unbiased ML-aided conditional phenotypic drug screen in human cells to identify novel mechanisms that rescue the effect of *C. diff.* toxin B treatment

REC-3964 (TcdB)



- Potential first-in-class non-antibiotic oral toxin B selective inhibitor for prevention of rCDI
- Well-tolerated with no treatment-related discontinuations or SAEs in Phase 1
- Recruitment ongoing with Phase 2 update expected in 1Q26
- 30+ new trial sites identified using RWD/ML

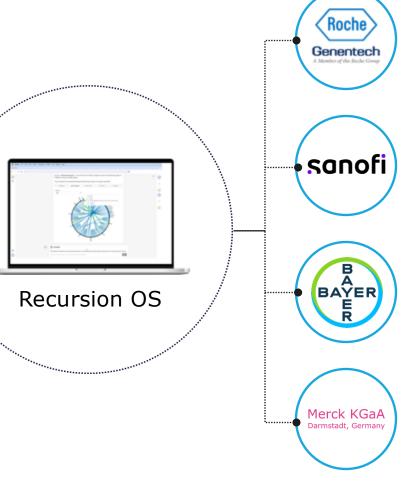


Pipeline

Partnership Pipeline



Pharma partnerships with approximately \$450M¹ earned to date and potential to receive more than \$20B in additional milestones



NEUROSCIENCE & ONCOLOGY

- \$150M upfront | Up to \$300M per program for 40 programs
- Identifying novel targets and small molecules, with the potential to advance up to 40 programs in neuroscience & a GI oncology indication.

ONCOLOGY & IMMUNOLOGY

- \$100M upfront | >\$5B total value
- Focusing on up to 15 novel small molecule candidates across oncology & immunology

ONCOLOGY

- \$80M upfront + investment | ~\$1.5B total value
- Focusing on up to seven programs in oncology

ONCOLOGY & IMMUNOLOGY

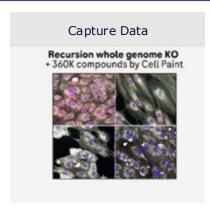
- \$20M upfront | >\$650M deal value
- Focused on three projects in oncology & immunology

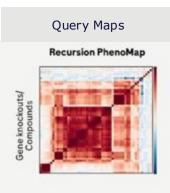


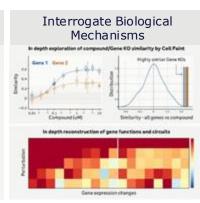


GI Oncology

- Multiple whole-genome phenomaps with chemical perturbations completed in disease relevant cell types
- First validated hit series now in hit-to-lead
- Additional near-term program options

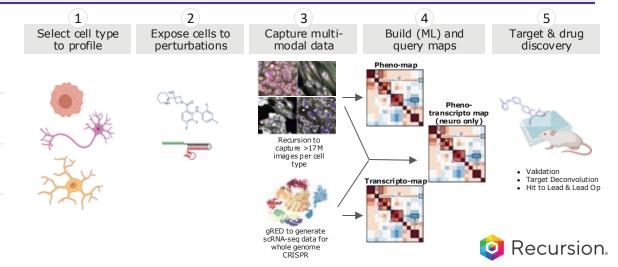






Neuroscience

- World's first neuro-specific whole-✓ genome arrayed CRISPR KO phenomap optioned for \$30M
- ✓ Target validation packages underway
- Additional neuroscience phenomap options
- Additional near-term program options



sanofi

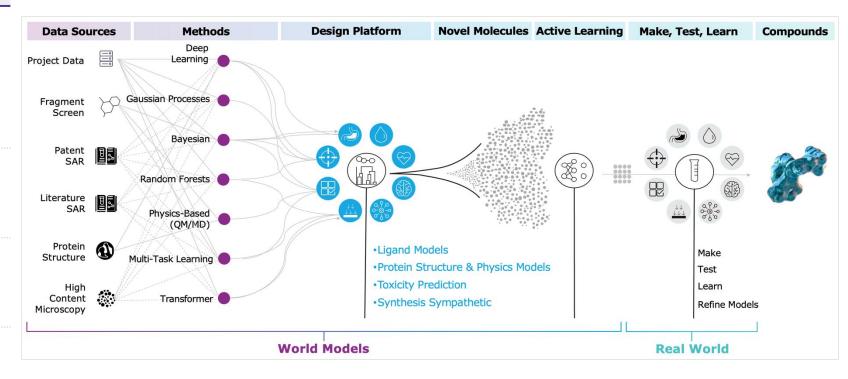
Oncology and Inflammation

Three programs have advanced through initial milestones; aggregate 2024 milestones of \$15m for two programs

Expansion of collaboration✓ with internally-discovered program

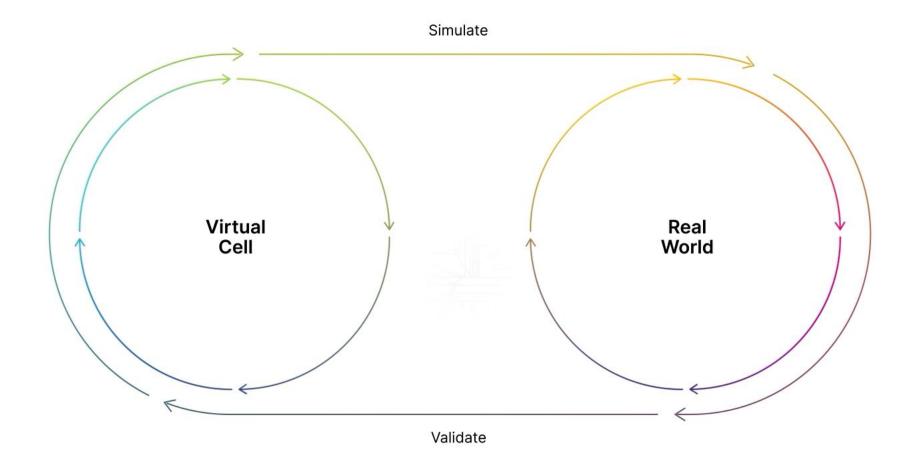
Identification of new targets and initiation of multiple new programs

Advance additional programs into lead optimization



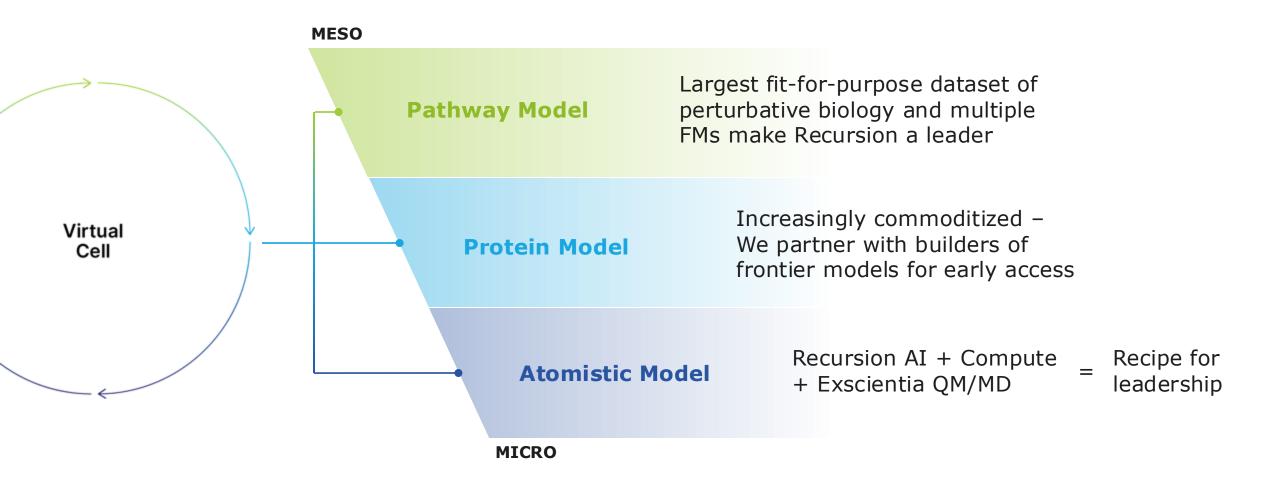


Full-stack Recursion OS is industrializing first-in-class & best-inclass drug discovery





Full-stack Recursion OS is industrializing first-in-class & best-in-class drug discovery





Business Updates



Our leadership brings together experience & innovation to advance TechBio

Executive Team



Chris Gibson, **PHD** Co-Founder, & Chief Executive Officer



Najat Khan, PHD Chief R&D Officer & Chief Commercial Officer



Erica Fox Chief People & Impact Officer

Goode :: PRIMER



David Hallett, PHD Chief Scientific Officer

🔪 evotec 🕝 MERCK



Nathan Hatfield Chief Legal Officer WILSON SONSINI



Matt Kinn Chief Business Officer







Ben Mabev Chief Technology Officer





David Mauro, MD PHD Chief Medical Officer







Lina Nilsson, PHD SVP, Head of Platform





Kristen Rushton **Chief Operations** Officer **Wriad** genetics



Ben Taylor Chief Financial Officer & President Recursion UK

Goldman AETION

Board of Directors



Rob Hershberg, MD PHD Co-Founder, CEO, & Chair of HilleVax; Former EVP, CSO, & CBO of Celgene







Zachary Boque Co-Founder & Partner of Data Collective





Blake Borgeson, PHD Co-Founder of RXRX





Zavain Dar Co-Founder & Partner of Dimension







Chris Gibson, PHD Co-Founder & Chief **Executive Officer**



Najat Khan, PHD Chief R&D Officer & Chief Commercial Officer Johnson&Johnson



Dean Li, MD PHD Co-Founder of RXRX, President of Merck Research Labs







Franziska Michor, PHD Chair at Dana-Farber Cancer Institute & Professor at Harvard University







Deeply focused on maximizing return post business-combination

Status

- Hosted successful in-person multi-day onboarding event in London
- Low post-close regrettable turnover
- Senior-leadership teams combined and operating
- Company-wide processes and platforms review underway with focus on highest impact operations
- Integration of EXAI precision design platform into Recursion OS underway

Next steps

- 90-day goals update at YE24 earnings
- Actively progressing with combination synergies additional guidance to be provided in 2Q25

Pro Forma Cash¹: \$752 million



Positioned for a catalyst-rich 2025

Pipelines							
☐ REC-994 (Superoxide Scavenger) in CCM	Late breaker oral presentation (Phase 2) at International Stroke Conference	Feb 5 th , 2025					
☐ REC-3565 (MALT-1i) in B-cell malignancies	Phase 1 first patient dosed	1Q25					
☐ REC-617 (CDK7i) in advanced solid tumors	Initiation of combination studies	1H25					
□ REC-4881 (MEK1/2i) in FAP	Phase 1b/2 safety and early efficacy data	1H25					
□ REC-2282 (HDACi) in NF2	PFS6 futility analysis	1H25					
□ REC-4539 (LSD-1i) in SCLC	Phase 1 first patient dosed	1H25					
☐ REC-617 (CDK7i) in advanced solid tumors	Additional Phase 1 data from ELUCIDATE	2H25					
☐ Advancement of discovery programs including	g a PI3Kα H1047Ri	FY25					

P	Partnerships					
	Potential for additional phenomap options					
	Potential for multiple new project initiations					
	Potential for multiple programs optioned by partners					

Platform				
	Updates on early clinical development AI build in Recursion OS			
	Updates on industry-leading foundation models at multiple biological levels			
	Integration of technology and autonomous workflows to support best- and first-in-class programs			



Recursion

Appendix



Culture and Team



Our people are the most important ingredient for our mission



~800 employees

- Technology data science, software engineering, automation, etc.
- Life Sciences biology, chemistry, development, etc.
- Strategic Operations

Parity Pledge Signer:
Gender parity and people
of color parity



Headquartered in **Salt Lake City, Utah** with other primary locations in:

- · Milpitas, California
- New York, New York
- · Toronto, Ontario
- · Montréal, Québec
- London, England
- · Oxford, England



ESG Highlights







Learn more about Recursion's ESG stewardship: www.recursion.com/esq

Community Impact

altitude _ lab



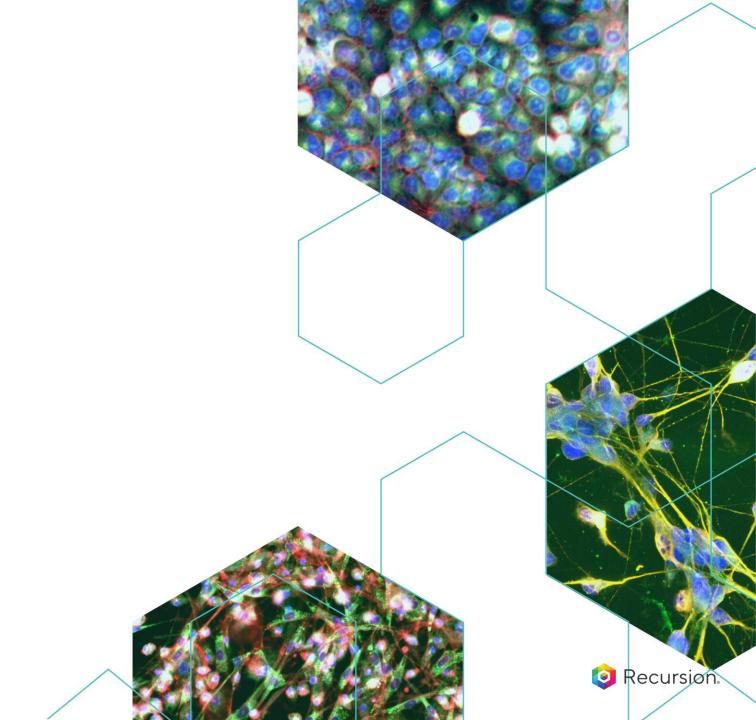
Founding Partner, Life Science Accelerator

Founding Member, Life Science Collective



APPENDIX

Pipeline Details



PIPELINE

Oncology



REC-617: CDK7 Inhibitor

A precision designed highly selective CDK7 inhibitor for Relapsed and/or Refractory (R/R) Solid Tumors

Program Status

- Potential Best-in-Class and First-in-Class CDK7 inhibitor
- Phase 1/2 study in advanced solid tumors ongoing
- Initial Phase 1 monotherapy safety, PK/PD update presented at AACR
 Special Conference in Cancer Research held on December 9, 2024

Mechanism of Action

 Reversible CDK7 inhibitor that targets <u>both</u> cell cycle progression and transcriptional regulation

Thesis & Differentiation

- Non-covalent binding and improved selectivity to decrease offtarget toxicity
- 8-10 hours of therapeutic coverage at IC₈₀ with a **short half-life** to reduce on-target toxicity
- Rapid absorption and permeability at lowest possible dose

Unmet Need¹

- Multiple cancer indications that have the potential to address ~185,000 patients annually
- R/R solid tumors including breast, NSCLC, ovarian, pancreatic, colorectal, and head & neck

- AI-powered precision design to optimize PK/PD to maximize potential therapeutic index
- 136 novel compounds synthesized to candidate ID



REC-617: Robust antitumor activity demonstrated in disease relevant preclinical tumor models

Initial clinical safety and PK/PD presented at AACR Special Congress in December 2024

Key Preclinical Data

REC-617 has Best-in-Class potential¹

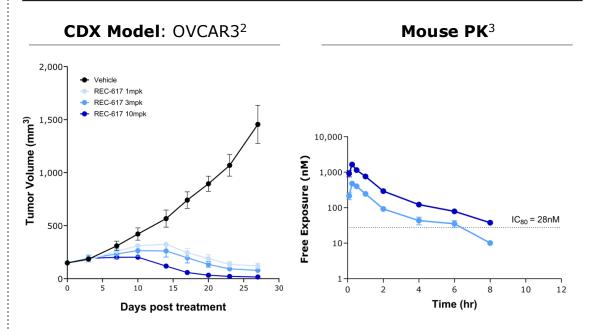
Designed to avoid efflux transporter substrate to minimize GI adverse events

Assay	DC Criteria	Ph 1 Competitor	Ph 1/2 Competitor	REC-617
CDK7 IC50 (nM)	<10			
CDK family selectivity	>100-fold			
HCC70 (breast cancer) IC50 (nM)	<100			
Caco-2 A2B (efflux) 10 ⁻⁶ cm/s	>5 (<3)			
Predicted human half-life (hr)	<15			
Meets or exceeds criteria Minor deviation Major deviation				

Development Candidate (DC) Criteria:

- CDK7 IC50: green <10nM; yellow 10-30nM; red >30nM
- CDK7 selectivity: green >100-fold; yellow 30-100-fold; red <30-fold
- **HCC70 IC50:** green <100nM; yellow 100-500nM; red >500 nM
- Caco-2 A2B (efflux): green >5(<3); yellow >1.5 (<10); red <1.5 (>30)
- **Half-life**: green <15, yellow <24, red >24

Potent tumor regression with minimal IC₈₀ exposure



 REC-617 demonstrates potent tumor regression with less than 10 hours of exposure above IC₈₀ to optimize benefit-risk



REC-617 (CDK7 inhibitor): Study Design and Next Steps

Development Strategy

ELUCIDATE Phase 1/2 study design

CURRENT STAGE

Phase 1

Dose Escalation (N≤60)

Part A

Monotherapy

Part B

 Initial combination with SERD in HR+/HER2- post CDK4/6 inhibitor population

Primary Endpoint:

- Safety and Tolerability
- Recommended Dose

RP2D ······→

Phase 2 Dose Expansion

- N = 30-60 patients in combination
- N will depend on number of disease specific cohorts

Primary Endpoint:

ORR

REC-617 Competitive Profile

- Potential Best-in-Class CDK7 inhibitor
- Reduced risk of off-target toxicity
- Highly selective & potent

Trial Update

- Phase 1 monotherapy preliminary safety and PK/PD data update presented at AACR Special Conference in Cancer Research in December 2024
- Continue dose escalation (QD & BID)
- Initiate combination study in 1H25
- Leverage new tech and clinical data partnerships for patient stratification



REC-1245: RBM39 Degrader

A highly selective RBM39 degrader for Biomarker-Enriched Solid Tumors and Lymphoma

Program Status

- Potential First-in-Class RBM39 degrader in solid tumors
- First patient dosed in 4Q24
- Phase 1 monotherapy update on dose-escalation expected in 1H26

Mechanism of Action

- Molecular glue that degrades RBM39 via E3 ligase adaptor DCAF15
- Disrupts RNA splicing to downregulate cell cycle checkpoints and DDR networks

Thesis & Differentiation

- RBM39 phenotypically mimics CDK12 and is distinct from CDK13 in Recursion OS
- Novel approach to target DDR biology via RBM39 avoids on-target toxicities associated with cell cycle checkpoint inhibitors (e.g., CDK12, WEE1, ATR, ATM, PLK1)
- **Selective** RBM39 degrader with **minimal ITGA2 liability** to limit thrombocytopenia

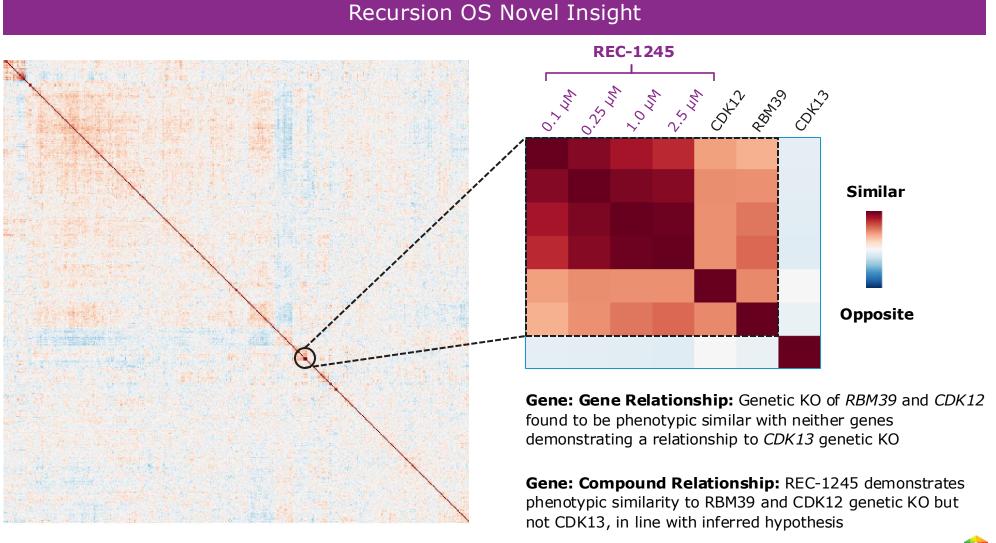
Unmet Need¹

- >100,000 patients with solid tumor or lymphoma experience disease progression while on frontline therapies
- Potential to be used as a single agent or in combination with chemo/IO

- Unbiased ML-aided genomics screen to identify biological signature and relate cellular phenotypes
- Progressed REC-1245 from target biology to IND-Enabling studies in under 18 months (vs. 42 months in industry²)



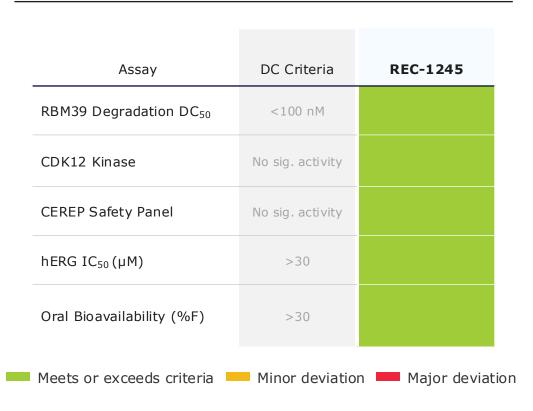
REC-1245 (RBM39 degrader): Platform inferred a functional similarity between RBM39 and CDK12 biology suggesting a novel approach to potential DDR modulation



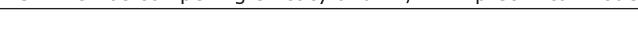
REC-1245 (RBM39 degrader): Robust efficacy/PK/PD in biomarker-positive disease relevant preclinical tumor models – Phase 1 initiated in 4Q24

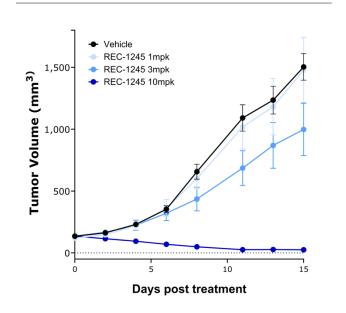
Key Preclinical Data¹

REC-1245 is highly selective and potent¹

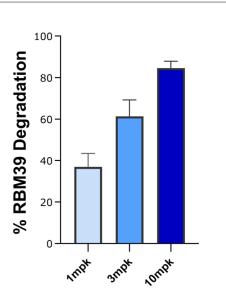


REC-1245 has compelling efficacy and PK/PD in preclinical models





CDX Model: OVK18²



PD: Target Engagement³

- REC-1245 shows significant monotherapy regressions
- Dose-dependent antitumor activity correlates with PD

REC-1245 (RBM39 degrader): Study Design and Next Steps

Development Strategy



DAHLIA Phase 1/2 study design

CURRENT STUDY

Phase 1

Dose Escalation (N~55)

Part A

Monotherapy dose-finding

Part B

 Monotherapy doseconfirmation



Phase 2 Dose Expansion

Key Study Characteristics

Study objectives:

- Safety/Tolerability
- RP2D
- ORR

Eliaibility:

 Select histologies including a biomarker population and R/R lymphomas

REC-1245 Competitive Profile

- **Highly potent**, potential **First-in-Class** RBM39 degrader (<100nM DC50)
- No significant in vitro safety concerns (CEREP, hERG)
- No significant activity in CDK12 kinase assay
- Minimal ITGA2 liability to limit thrombocytopenia
- High oral bioavailability

Trial Update

- First patient dosed in 4Q24
- Trial active and enrolling at 5 US sites



REC-3565*: MALT1 Inhibitor

A precision designed selective MALT1 inhibitor for B-Cell Malignancies

Program Status

- Potential Best-in-Class MALT1 inhibitor
- Phase 1 initiation in B-Cell Malignancies (e.g., chronic lymphocytic leukemia), expected 1Q25

Mechanism of Action

- Reversible allosteric MALT1 inhibitor that can dampen NF-κB signaling
- Selectively inhibits CLL proliferation with limited impact on T-Cell viability

Thesis & Differentiation

- Low UGT1A1 liability with potential for reduced risk of hyperbilirubinemia
- Potential for reduced liver toxicity and enhanced efficacy in combination with BTK and BCL2 inhibitors
- Low predicted human clearance and high oral bioavailability

Unmet Need¹

- Current monotherapy treatments in B-cell malignancies not curative and prone to resistance
- ~41,000 patients with R/R B-cell malignancies (treatable in US and EU5) targeting CLL combination therapy

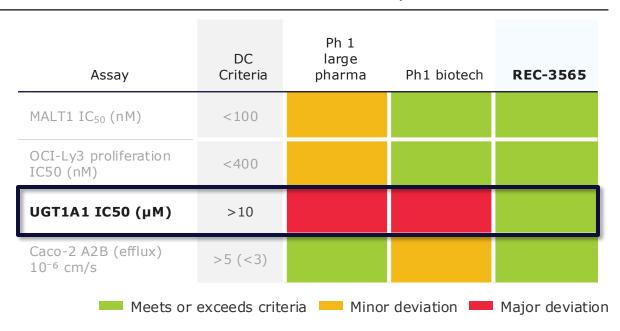
- AI powered precisiondesigned novel molecule using molecular dynamics and hotspot analysis
- 344 novel compounds synthesized to candidate ID
- Maintain selectivity and deliver a candidate with lower predicted safety risk in the clinic



REC-3565 (MALT1 inhibitor): Minimal UGT1A1 liability vs competitors and significant tumor regression observed in vivo with Phase 1 initiation anticipated in 1Q25

Key Preclinical Data

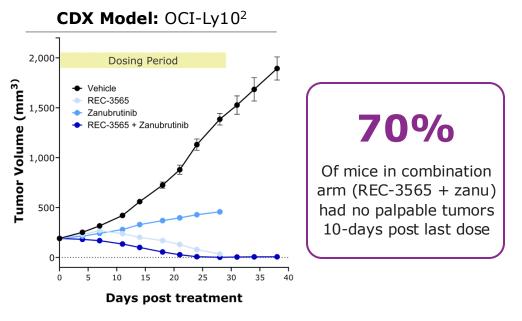
REC-3565 has Best-in-Class potential¹



Development Candidate (DC) Criteria:

- MALT1 IC50 nM: green <100 nM; yellow >100-<300 nM; red>300 nM
- OCI-Ly3 IC50 nM: green <400 nM; yellow >400-<1000 nM; red>1000 nM
- UGT1A1 IC50 uM: green >10 uM; yellow <10->1 uM; red<1 uM
- Caco-2 A2B (efflux): green >5(<3); yellow >1-<5(>3-<10); red <1(>10)

Single-agent and synergistic activity in vivo²



- OCI-Ly10 and Rec-1 cells are sensitive to both MALT1i and zanubrutinib in vitro
- Administration of REC-3565 as a single agent showed tumor growth regression
- Durable tumor growth regression observed when REC-3565 was combined with zanubrutinib

REC-3565 (MALT1 inhibitor): Study Design and Next Steps

Development Strategy EXCELERIZE Phase 1 study design Phase 1 Dose Escalation **Q1** 2025 Part A Part B RD Monotherapy Combination • N ~30 R/R B-Cell Malignancies REC-3565 PO OD or BID **Primary Endpoint:** Safety / tolerability RD for combination

REC-3565 Competitive Profile

- Low predicted human clearance and high oral bioavailability
- No unexpected in vitro or in vivo safety concerns identified
- Well tolerated in rat/dog dose range finding (DRF) studies
- GLP-tox studies completed with suitable noobserved-adverse-effect level (NOAEL) enabling clinical trials

Trial Update

• Trial initiation expected 1Q25



REC-4539: LSD1 Inhibitor

A precision designed unique LSD1 inhibitor with CNS penetrance

Program Status

- Potential Best-in-Class LSD1 inhibitor
- Phase 1 initiation in SCLC expected 1H25

Mechanism of Action

- Reversible LSD1 inhibitor that can selectively upregulate NOTCH signaling
- Promotes differentiation of neuroendocrine cancer cells
- **Impairs DNA repair pathways** sensitizing SCLC cells to immune checkpoint inhibitors

Thesis & Differentiation

- LSD1 inhibitor designed to be reversible and brain penetrant
- Shorter-predicted half life versus competitors to manage on-target toxicity
- Highly selective to reduce off-target toxicity
- Preclinical data shows therapeutic exposures have minimal effects on platelets, suggesting potential reduced risk of thrombocytopenia

Unmet Need¹

- >45,000 patients with treatable Stage III/IV SCLC
- Limited treatment options post progression on frontline therapies

- Precision design using active learning to select most information rich compounds
- 414 novel compounds synthesized to candidate ID
- Used multiparameter optimization to design a unique candidate combining reversibility with CNS penetration



REC-4539 (LSD1 inhibitor): Sufficient CNS exposures vs competitors and compelling dose-response demonstrated in vivo with Phase 1 initiation anticipated in 1H25

Key Preclinical Data

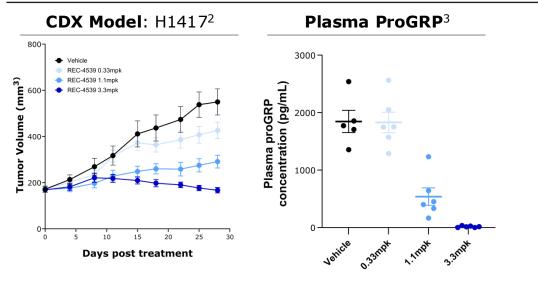
REC-4539 has Best-in-Class potential¹



Development Candidate (DC) Criteria:

- **Brain:plasma ratio:** green >0.5; red <0.5
- MDCK-MDR1 efflux ratio (Pgp): green <2; yellow >2-<10; red >10
- **Predicted half-life:** green <24 hours; yellow 24-48h hours; red >48 hours

REC-4539 highly efficacious in SCLC xenograft model²



- Dose-dependent regression
- Well-tolerated with limited impact on platelet levels

Trial Update

Phase 1 First Patient Dosed in SCLC expected 1H25



PIPELINE

Rare disease



REC-994: Superoxide Scavenger

A safe and well tolerated superoxide scavenger for the treatment of Cerebral Cavernous Malformation (CCM)

Program Status

- First therapeutic candidate advanced to an industry-sponsored Phase 2 trial
- Phase 2 primary endpoint of safety met with similar AE profile across arms
- Meeting with FDA anticipated in 2H25 to discuss plans for additional clinical study

Mechanism of Action

- Selective, orally bioavailable, redox-cycling nitroxide
- Promotes the metabolism of ROS to reduce oxidative stress within cells
- Stabilizes endothelial barrier function

Thesis & Differentiation

- Develop the first oral therapy for the treatment of symptomatic CCM
- Target the underlying genetic mechanisms that drive the disease pathophysiology of CCM

Unmet Need¹

- ~360,000 symptomatic CCM patients with **no approved therapies**
 - ~63,000 patients harboring brainstem lesions and elevated bleeding risk
 - ~36,000 patients with cavernoma-related epilepsy^{2,3}

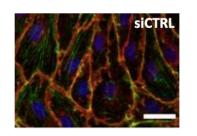
- Unbiased ML-aided phenotypic drug screen to identify effective therapeutics driving CCM
- In vivo POC demonstrated lesion reductions that were also observed in the Ph2 trial

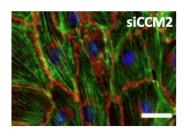


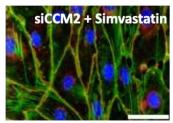
REC-994 (Superoxide Scavenger): Preclinical studies showing reduction of lesion burden de-risked the first industry-sponsored Phase 2 study in CCM

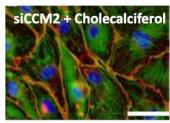
Recursion OS Insight

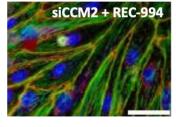
Identified REC-994 as potential rescue molecule in phenotype associated with CCM2 loss of function





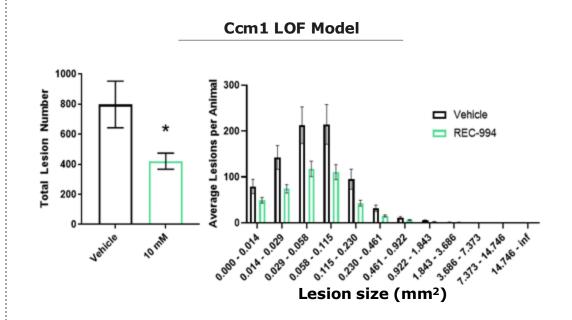






Key Preclinical Data¹

Reduces lesion number & size in *Ccm1* and *Ccm2*² loss of function (LOF) mouse models





REC-994 (Superoxide Scavenger): Topline Phase 2 data in September demonstrated encouraging signals of efficacy

Trial Update



- · Randomized, double-blind, placebo-controlled Phase 2 study
- Primary endpoint of safety and tolerability met September 2024
- Encouraging trends observed in objective MRI-based exploratory efficacy measures observed
- Time- and dose-dependent trends in reduced lesion volume and hemosiderin ring size compared to placebo
- 80% of Phase 2 study participants remain on the long-term extension phase of the study

Next Steps

- Meeting with FDA to define regulatory path and Phase 2/3 study under development
- Data to be presented at a late breaking oral abstract session at the International Stroke Conference on Feb 5, 2025



REC-4881: MEK1/2 Inhibitor

A highly selective and potent MEK1/2 inhibitor for chemoprevention of Familial Adenomatous Polyposis (FAP)

Program Status

- First-in-Disease and best-in-class potential for the treatment of FAP
- Phase 1b safety and futility analysis (polyp burden) anticipated in 1H25

Mechanism of Action

- Loss of APC drives FAP disease progression through aberrant MAPK signaling
- REC-4881 is a highly potent, non-competitive, allosteric MEK1 and MEK2 inhibitor
- Selectively blocks the activation of ERK (MAPK pathway)

Thesis & Differentiation

- Develop the first oral therapy for the treatment of FAP
- Target underlying genetic mechanisms that drive the FAP disease progression
- Preferential distribution to GI tissues vs competitors which may enable greater activity at lower doses

Unmet Need¹

- No approved systemic therapies and significant unmet need for ~50,000 FAP patients beyond colectomy
 - Includes \sim 7,000² advanced duodenal polyposis patients in the US at high-risk of developing cancer

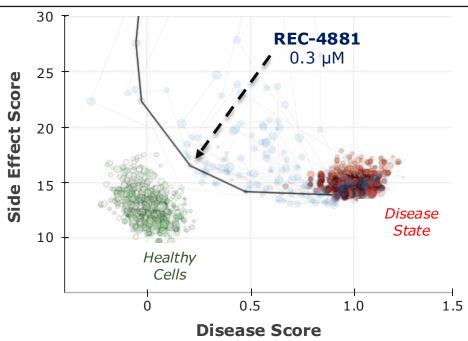
- Unbiased ML-aided phenotypic drug screen in human cancer cells
- Validated findings in vivo demonstrating significant reductions in polyps and adenomas



REC-4881 (MEK1/2 Inhibitor): Highly selective and potent molecule demonstrated superior in vivo efficacy versus celecoxib

Recursion OS Insight

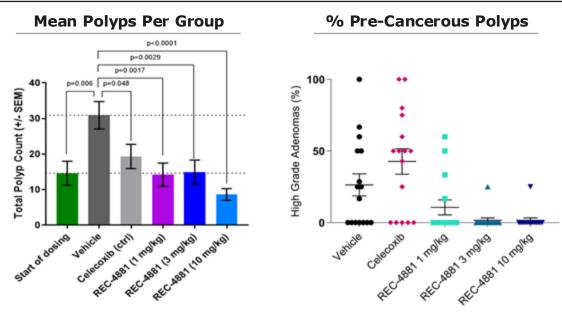
REC-4881 suppresses disease-inducing effects of APC mutations



- AI/ML extracts morphological features to distinguish "diseased" vs. "healthy" states
- Compounds co-treated with APC siRNA for 24 hours to find hits that reverse disease state back to healthy in a concentration-dependent manner

Key Preclinical Data¹

REC-4881 decreases polyp count and pre-cancerous adenomas



- Significantly reduces polyp count at all dose levels, outperforming celecoxib in APC^{min/-} mouse
- Unlike celecoxib, REC-4881 reduces both polyp numbers and % of adenomas
- Meaningful efficacy seen at lowest dose tested (1mg/kg) suggests potential for therapeutic activity at reduced systemic exposures

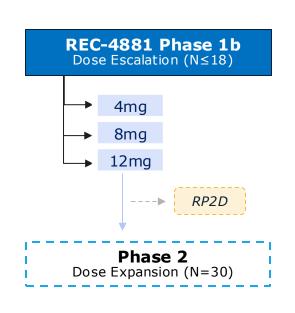


REC-4881 (MEK1/2 Inhibitor): Study Design and Next Steps

Development Strategy



UPELO Phase 1b/2 study design



Key Eligibility

- Confirmed APC mutation
- ≥ 55 years old
- Post-colectomy/proctocolectomy
- No cancer present
- Polyps in upper and lower GI

Study Objectives:

- Identify RP2D
- Safety/tolerability
- Reduction in polyp burden at week 12

REC-4881 Competitive Profile

- Early PD data indicates 4 mg dose is pharmacologically active and well-tolerated
- Fast Track Designation in FAP granted by FDA in 2022
- ODD in US and EU

Trial Update

- Futility reduction in polyp burden; assessed after 10 evaluable patients at the RP2D
- Ph 1b/2 safety & early efficacy data 1H25



REV102: ENPP1 Inhibitor

A safe and highly selective ENPP1 inhibitor for hypophosphatasia (HPP)

IND enabling studies expected to initiate in 2025

Program Status

Potential first-in-class and best-in-class ENPP1 inhibitor for the treatment of patients with HPP

Recursion Approach²

chronic dosing

Mechanism of Action

- Potent ENPP1 inhibitor is a non-immunogenic small molecule that
- restores PPi balance
 Highly selective ENPP1 inhibitor with low nM potency

• **Structurally distinct** differences vs competitor ENPP1 inhibitors

high potency and a lifetime of

Precision designed for both

Thesis & Differentiation

- ENPP1 inhibition is a genetically validated target in HPP models
- Potential for first oral disease-modifying therapy (compared to multiple weekly injections) without dose-limiting adverse events
- **Non-immunogenic** small molecule approach offering potentially safer solution than enzyme replacement therapy (ERT)
- REV102 offers a more tolerable and affordable option to ERTs

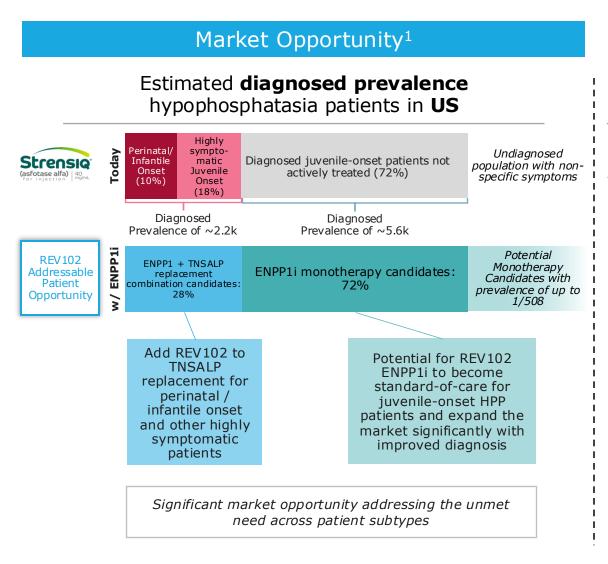
Unmet Need¹

- ~7,800 diagnosed prevalence of HPP across US and EU5
- · Many patients, particularly adults, may have difficulty accessing ERT
- Those who can access ERT face high treatment burden and tolerability hurdles
- Opportunity to significantly reduce costs and treatment burden

Maintain selectivity and deliver a candidate with high oral bioavailability in the clinic



REV102 (ENPP1 Inhibitor): OS insights validated using in vivo mouse model showing significant difference in restoring HPP biomarker that promotes bone mineralization



Key Preclinical Data²

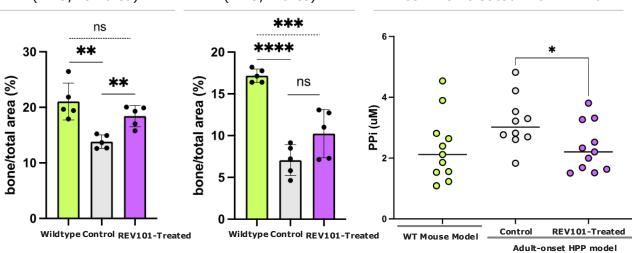
Bone morphometry

2D analysis of trabecular bones

Plasma levels of PPi After 100-day dosing

L3 Vertebrate Distal Femur (n=5, females) (n=5, males)

PPi lowered by ~30% in adult HPP mice when treated with REV101



Data is for REV101 (1st gen tool compound); compound being developed is REV102

What's Next

Initiate IND-enabling studies in 2025



REC-2282: Pan-HDAC Inhibitor

CNS-penetrating pan-HDAC inhibitor for the first oral therapeutic to treat Neurofibromatosis Type 2 (NF2)

Program Status

- Potential first-in-disease and best-in-class therapy for NF2 mutant meningioma
- Data maturing with PFS6 results expected 1H25

Mechanism of Action

- Orally bioavailable, CNS penetrant, and potent pan-HDAC inhibitor
- Loss of Merlin (NF2) leads to PI3K signaling and meningioma proliferation REC-2282 indirectly facilitates AKT dephosphorylation by disrupting the PP1-HDAC interaction

Thesis & Differentiation

- Develop the first therapeutic for NF2 meningioma
- Highly selective molecule with favorable brain exposure and reduced risk of cardiac toxicity

Unmet Need¹

- No approved therapy for ~33,000 NF2 meningioma patients beyond surgery
- Surgery only feasible in a limited number of patients and carries high rate of recurrence²

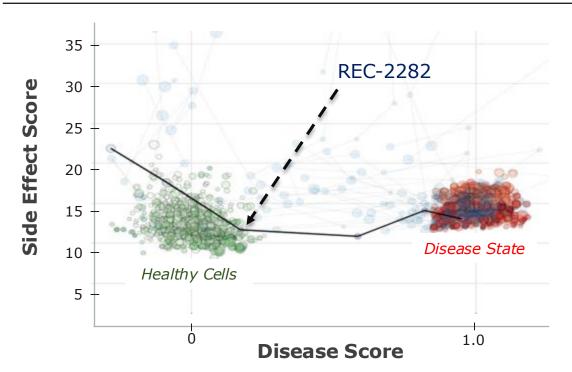
- Unbiased ML-aided
 phenomap insight and drug
 screen in human cells
- Identify effective therapeutics that rescue disease-inducing effects of NF2 loss



REC-2282 (Pan-HDAC Inhibitor): Identified as a unique HDAC inhibitor in Recursion's unbiased screen modeling NF2 loss-of-function

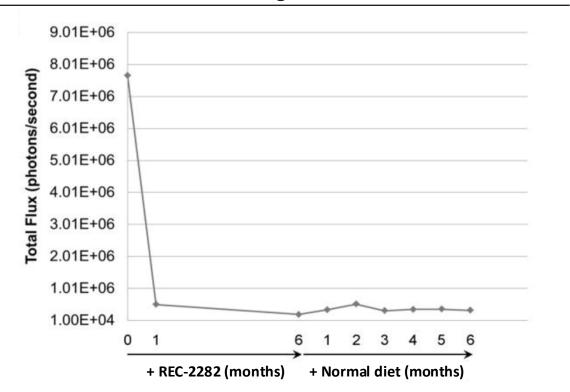
Recursion OS Insight

REC-2282 demonstrates concentration-dependent reversal of NF2 loss



Key Preclinical Data¹

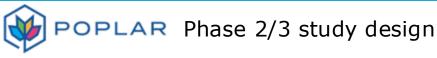
Prevents growth & regrowth of NF2-deficient meningioma model in mice



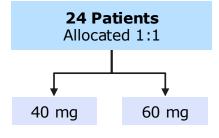


REC-2282 (Pan-HDAC Inhibitor): Study Design and Next Steps

Development Strategy



REC-2282 Phase 2 Portion Open-label, 2-arm study



Primary Endpoint:

PFS6

Secondary Endpoint:

Safety, ORR

Key Eligibility

- MRI-confirmed progressive meningioma
- Sporadic meningioma with confirmed NF2 mutation
- Familial NF2 meningioma
- Have documented progression with past 24 months

REC-2282 Competitive Profile

- Orally bioavailable and CNS penetrant
- Fast Track Designation in NF2 granted by FDA in 2021
- ODD in US and EU

Trial Update

- Phase 2 Data maturing
- Futility analysis (PFS6) expected in 1H25



PIPELINE

Other areas of high unmet need



REC-3964: C. difficile Toxin B Selective Inhibitor

Non-antibiotic selective toxin-inhibitor for the prevention of recurrent *C. difficile* infection (rCDI)

Program Status

- First-in-class therapy for prevention rCDI
- First patient dosed in the Phase 2 ALDER trial in 4Q24
- Phase 2 update expected in 1Q26

Mechanism of Action

- **Highly potent**, **orally bioavailable** *C. diff* toxin B (TcdB) selective inhibitor
- Selectively inhibits catalytic activity of bacterial glucosyltransferase

Thesis & Differentiation

- Develop the **first non-antibiotic oral therapy** that is safe and convenient
- Selectively targets bacterial toxin while sparing the host to minimize adverse events
- Preclinical efficacy demonstrates superiority in survival versus bezlotoxumab

Unmet Need¹

- ~175,000 cases of rCDI with limited treatment options for high-risk population
- Ability to address populations not eligible for FMT or microbiome-based therapies

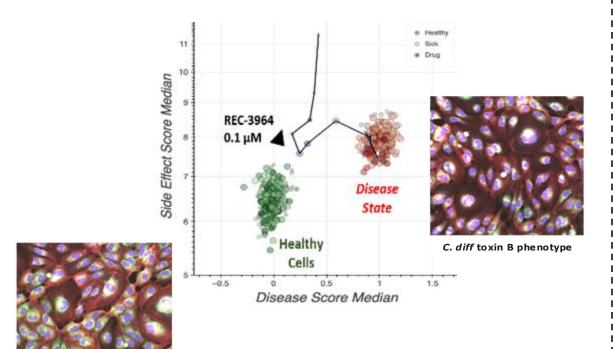
- Unbiased ML-aided conditional phenotypic drug screen in human cells
- Identified novel mechanisms that mitigated the effect of C. diff. toxin B treatment



REC-3964 (CDI TcdB Inhibitor): Identified as potential superior inhibitor compared to SOC in in vitro and in vivo preclinical studies

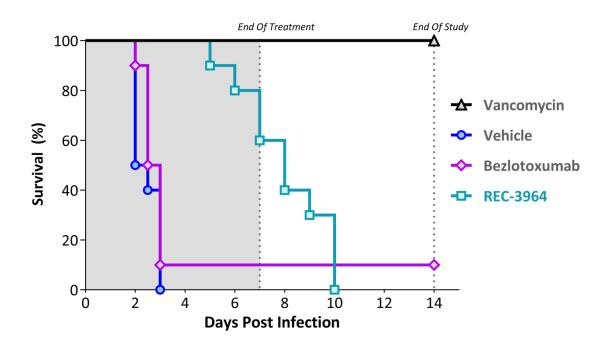
Recursion OS Insight

REC-3964 potently inhibits Toxin B with some activity against Toxin A, while bezlotoxumab is specific to Toxin B



Key Preclinical Data¹

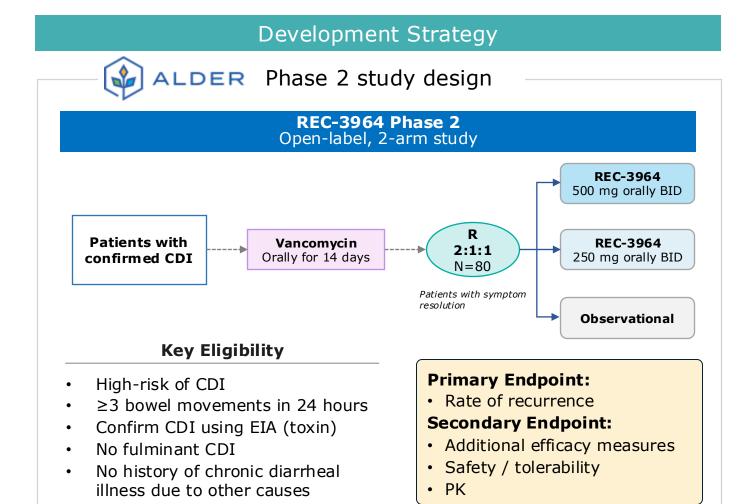
REC-3964 significantly extended survival vs. bezlotoxumab alone at the end of treatment (p<0.001, log rank test)





Healthy Control

REC-3964 (CDI TcdB Inhibitor): Study Design and Next Steps



REC-3964 Competitive Profile

- Highly potent, orally bioavailable
- Potential first-in-class therapy for prevention of rCDI
- First non-antibiotic oral therapy

Trial Update

- First Patient Dosed in 4Q24
- Program update expected 1Q26



REC-4209: Target Epsilon

Highly potent and potential First-in-Class medicine for the treatment of Idiopathic Pulmonary Fibrosis (IPF)

Program Status

- First-in-class therapeutic for treatment of IPF
- IND enabling studies ongoing

Mechanism of Action

- Reversible, orally bioavailable, and potent Target Epsilon inhibitor
- Promotes tissue repair and reverses fibrosis by potentially modulating TGF-B

Thesis & Differentiation

- Develop a novel preferred treatment option that is safe and well-tolerated
- In vitro models suggest capability of reversing the fibrotic process driving IPF progression

Unmet Need¹

- ~130,000 patients with IPF in the US
- Approved therapies show modest slowing of IPF progression
- No improvement in survival (mOS 3-5 years) or quality of life with current treatments

- Unbiased ML-powered phenomap drug screen in human cells
- Identify novel mechanisms that reversed the differentiation of fibrocytes

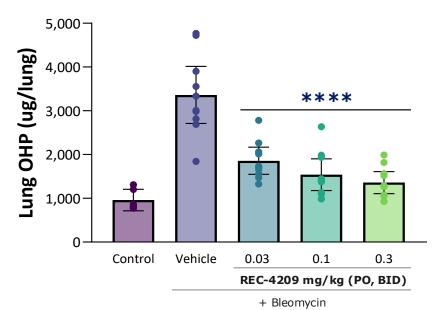


REC-4209 (Target Epsilon): Identified as a novel mechanism in Recursion's screen with compelling preclinical efficacy demonstrated in bleomycin lung fibrosis mouse model

Recursion OS Insights¹ In Vitro Fibrocyte Rescue Assay **Control State Disease State** 150-(Fibrocytes) (Pentraxin-2) 100 50 $IC_{50} = 104nM$ -50 -100--150 -10-9 10-8 10-7 Concentration (µM) + REC-4209 Fibrotic Lung Normal Lung







 REC-4209 at low doses reduces total lung collagen by 45% to 60% versus vehicle mice

What's Next

IND-enabling studies ongoing



APPENDIX

Partnerships & Data Strategy Details



Exciting scientific collaborations span biopharma, tech & data

Therapeutic discovery partnerships



Announced Dec. 2021

- Up to or exceeding \$300M in possible program milestones for up to 40 programs
- One program and one map already optioned
- Mid- to high-single digit tiered royalties on net sales

sanofi

Announced Jan. 2022

- \$100M upfront with the potential of \$5.2B in total milestones plus high-single digit to mid-teen tiered rovalties
- Up to 15 novel small molecule candidates across oncology and immunology
- New discovery stage program added identified and initially advanced by Exscientia in Dec. 2023
- **3 programs advanced** through initial milestones



Announced Sept. 2020

Updated Nov. 2023

- \$30M upfront and \$50M equity investment
- Increased per program milestones which may be up to **\$1.5B** in aggregate for up to 7 oncology programs
- Low- to mid-single digit royalties on net sales
- Recursion owns all algorithmic improvements
- First beta-user of LOWE

Merck KGaA

Darmstadt, Germany

Announced Sept. 2023

- **\$20M upfront** at initiation for three projects with up to \$674M in discovery, development, regulatory and sales-based milestones
- Mid-single to low-double digit tiered royalties



Exciting scientific collaborations span biopharma, tech & data

Platform, technology, and data partnerships

Computation and ML/AI



- \$50M equity investment
- Partnership on **advanced computation** (e.g., foundation model development)

Announced July 2023

- **Priority access** to compute hardware or **DGXCloud** Resources
- BioHive-2: helped design and build next generation supercomputer



Google Cloud

Announced Oct. 2024

- Includes exploring generative AI capabilities (including **Gemini models**) and driving improved search and access with **BigQuery**
- Scaled compute resources, improved management of petabytes of RX data, and continued data privacy and security support
- OpenPhenom S/16 model available on Google Cloud

Real-world data access

TEMPUS

Announced Nov. 2023

- Preferential access to >20 PBs of real-world, multimodal oncology data, including DNA & RNA sequencing and clinical outcome data for >100,000 patients
- Ability to train causal AI models with utility in target discovery, biomarker development & patient selection
- Opportunity to accelerate clinical trial enrollment through broad clinical network



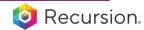
Access to hundreds of thousands of de-identified records. including Helix's Exome+(R) genomics & longitudinal health data, to train causal AI models and design biomarker & patient stratification strategies across broad disease areas

Cheminformatics and chemical synthesis



Announced Dec. 2023

- Utilizes Recursion's predicted protein-ligand interactions for ~36B compounds from Enamine's **REAL Library**
- Aim to generate **enriched screening libraries** & cobrand customer offerings



Recursion