



Decoding Biology To Radically Improve Lives

FEBRUARY 2025



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

10

Clinical and pre-clinical programs¹

Oncology, rare diseases, and other high unmet need diseases

~10

Additional advanced discovery programs

10+

Partnered programs

Oncology, immunology, and other high unmet need diseases

4

Large pharma collaborations

~10

Clinical program milestones over the next 18 months²

1

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

~\$450M

Upfront and milestone payments earned to-date

~\$20B potential milestone payments

Roche Genentech



sanofi

Merck KGaA
Darmstadt, Germany

2024 Year in Review: A Scaled Pipeline of Best- & First-In-Class Opportunities

Clinical data read-outs:

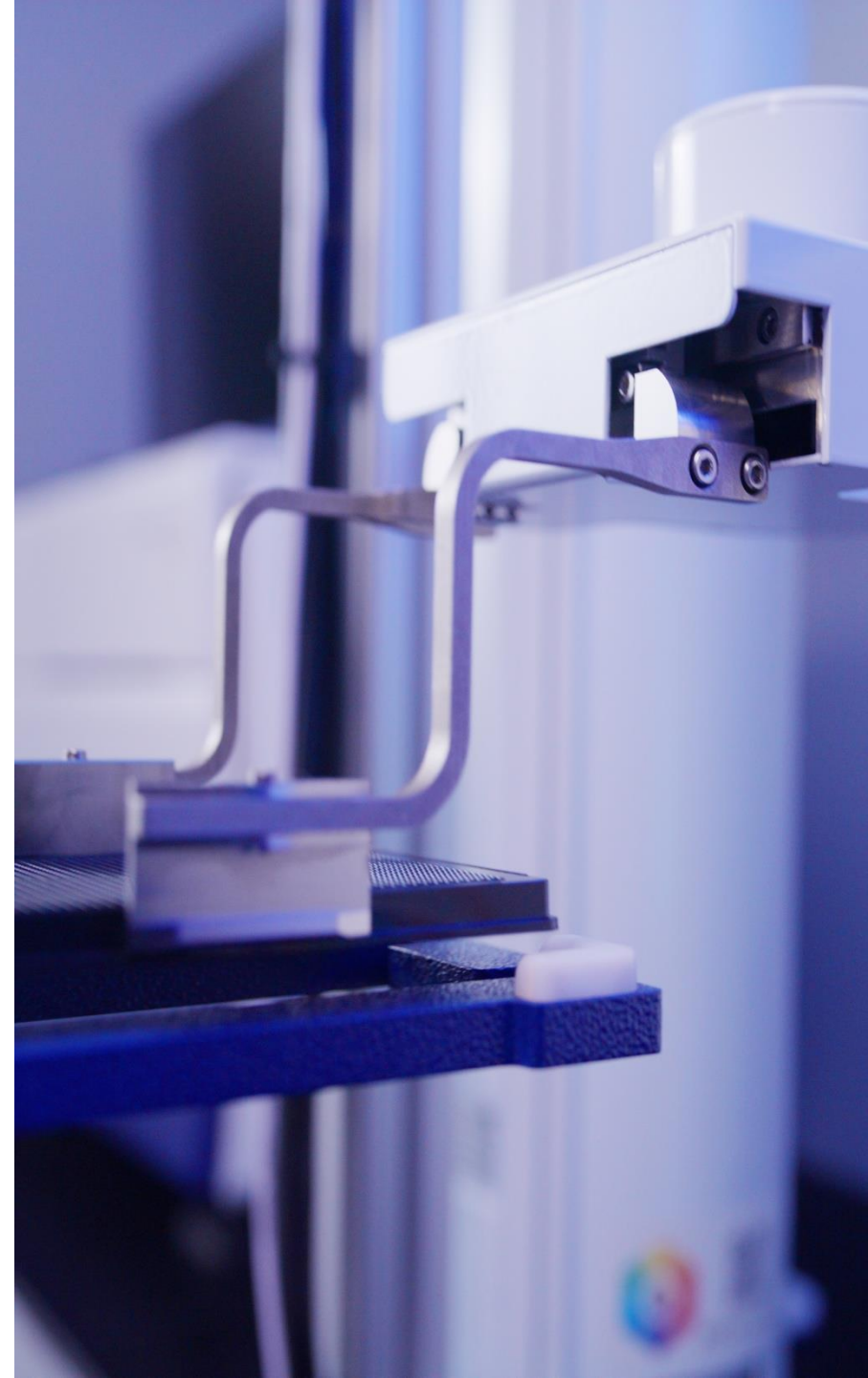
- **REC-617:** Potential best-in-class CDK7 inhibitor for advanced solid tumors showed promising safety and preliminary efficacy in Phase 1 dose escalation
- **REC-994:** Oral superoxide scavenger for potential first-in-disease symptomatic CCM demonstrated safety and encouraging trends in MRI-based lesion reduction and functional improvement in Phase 2

New clinical trials launched:

- **REC-1245:** DAHLIA (RBM39 degrader for solid tumors) – Phase 1/2
- **REC-4881:** TUPELO (FAP) – Phase 1b/2
- **REC-3964:** ALDER (*C. difficile*) – Phase 2

CTA/IND updates for additional programs:

- **REC-4539:** IND clearance for LSD1 (SCLC)
- **REC-3565:** CTA for MALT1 (B-cell malignancies)
- **REC-4209:** Initiated IND-enabling studies (IPF)
- **REV102:** Initiated IND-enabling studies (HPP)



Pipeline of ~10 clinical and preclinical technology-enabled programs

	Candidate	Target	Indication	Preclinical	IND-Enabling	Phase 1/2	Pivotal / Phase 3	Status & Anticipated Milestones
ONCOLOGY	REC-617	CDK7	Advanced solid tumors ¹	ELUCIDATE				✓ Ph1/2 interim data presented ² in 4Q24 • Combination study initiation – 1H25
	REC-1245	RBM39	Biomarker-enriched solid tumors & lymphoma	DAHLIA				✓ FPD – 4Q24 • Ph 1 dose-escalation update – 1H26
	REC-3565	MALT1	B-cell malignancies	EXCELERIZE				✓ CTA Clearance – 4Q24 • Ph 1 FPD – 1H25
	REC-4539	LSD1	Small-cell lung cancer (SCLC)	ENLYGHT				✓ IND Clearance – 1Q25 • Ph 1 FPD – 1H25
RARE	REC-994	Superoxide	Cerebral cavernous malformations (CCM)	SYCAMORE				✓ Ph 2 data presented ³ in 1Q25 • Program update – 2H 2025
	REC-4881	MEK1/2	Familial adenomatous polyposis (FAP)	TUPELO				• Ph 1b/2 safety & early efficacy – 1H25
	REC-2282	HDAC	Neurofibromatosis type 2 (NF2)	POPLAR				✓ Ph 2 fully enrolled – 3Q24 • PFS6 futility – 1H25
	REV102 ⁴	ENPP1	Hypophosphatasia (HPP)					✓ IND-enabling studies initiated
OTHER	REC-3964	TcdB	Prevention of recurrent <i>C. difficile</i> (rCDI)	ALDER				✓ FPD – 4Q24 • Ph 2 update – 1Q26
	REC-4209	Undisclosed	Idiopathic pulmonary fibrosis (IPF)					• IND-enabling studies ongoing
	~10 advanced discovery programs including a PI3Ka H1047Ri							

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

2. AACR Special Conference in Cancer Research: Optimizing Therapeutic Efficacy and Tolerability through Cancer Chemistry; plenary session presentation

3. International Stroke Conference; late breaking oral abstract

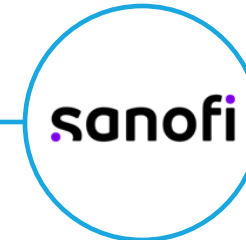
4. Joint venture with Rallybio

2024 Year in Review: Partnerships Delivering Meaningful Milestones



Roche-Genentech

Generated multiple whole-genome phenomaps in oncology and neuro to uncover new targets and potential therapies; \$30M in milestones received



Sanofi

Advanced two programs through initial milestones, generating \$15 million in aggregate payments



Bayer

Delivered 25 multimodal oncology data packages and LOWE software to enhance research capabilities



Merck KGaA

Advanced alliance in oncology and immunology targets

2024 Year in Review: Our Platform Leads the Industry in Data, Models, and Compute

Compute:

- Built Biohive-2 with NVIDIA, believed to be the most powerful wholly-owned and operated supercomputer in biopharma

Data Capabilities:

- Mapped 1.4 million active ligands to binding pockets for structure-based drug discovery and target deconvolution
- Generating up to 16.2M multi-timepoint brightfield images per week
- Produced just under 1 million transcriptomes this year (>1.6 million since launch in 2023), including the first genome-scale CRISPR knockout map in primary human cells

Foundation Models:

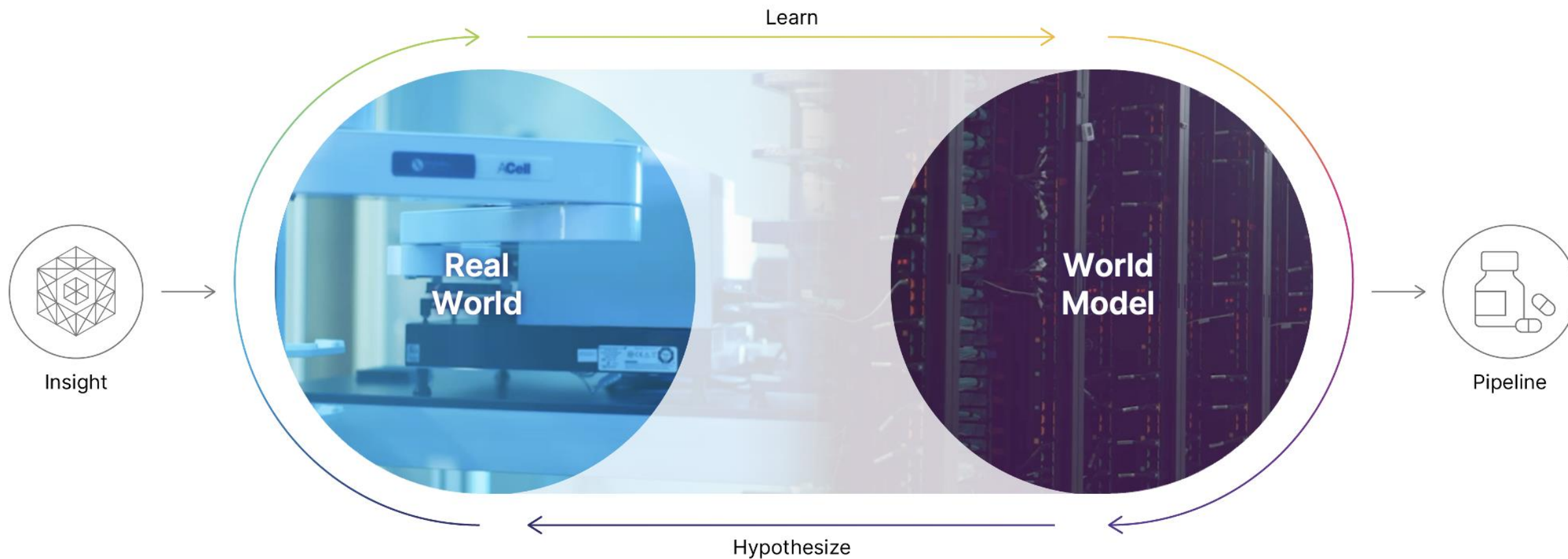
- Phenom-2: improves genetic perturbation separation by 60%
- MolPhenix: 10X improvement in predicting molecule effects on cell assays compared to previous models
- MolGPS: Outperforms state-of-the-art models on 12 of 22 ADMET tasks¹

Causal AI models and ClinTech:

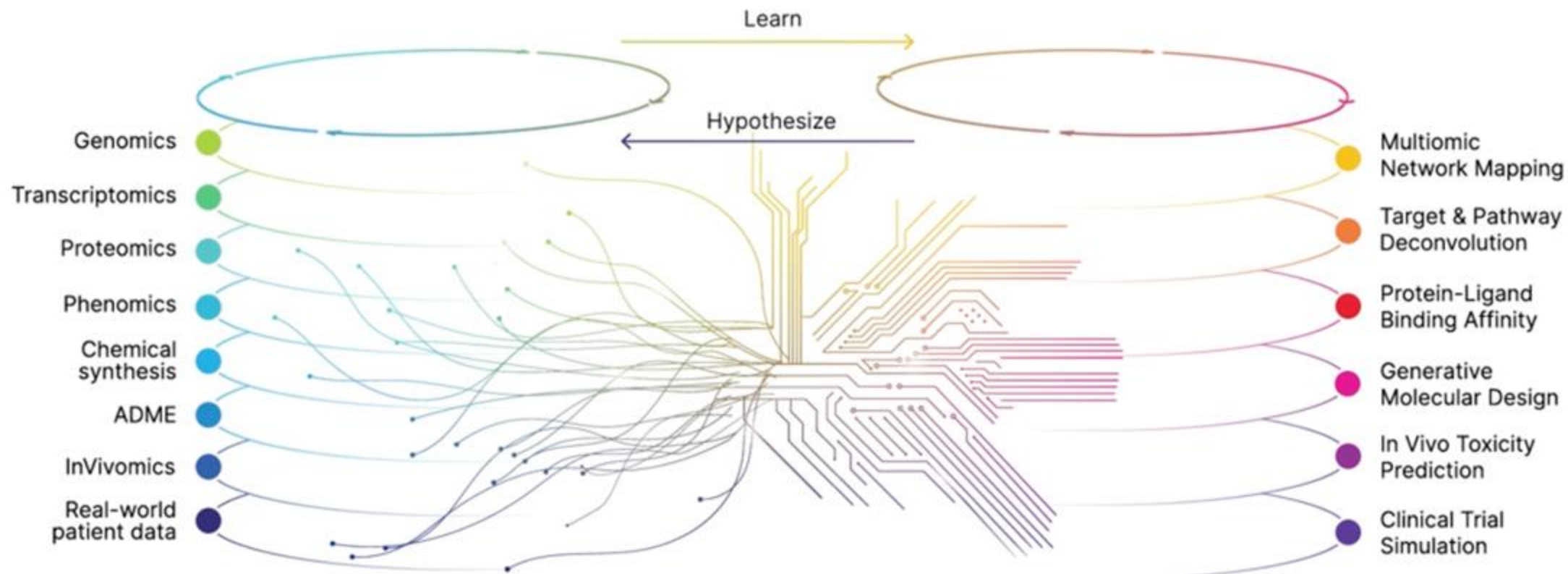
- Utilizing AI models and Tempus data to build a patient stratification framework in SCLC
- Automating site engagement and enrollment to accelerate patient matching



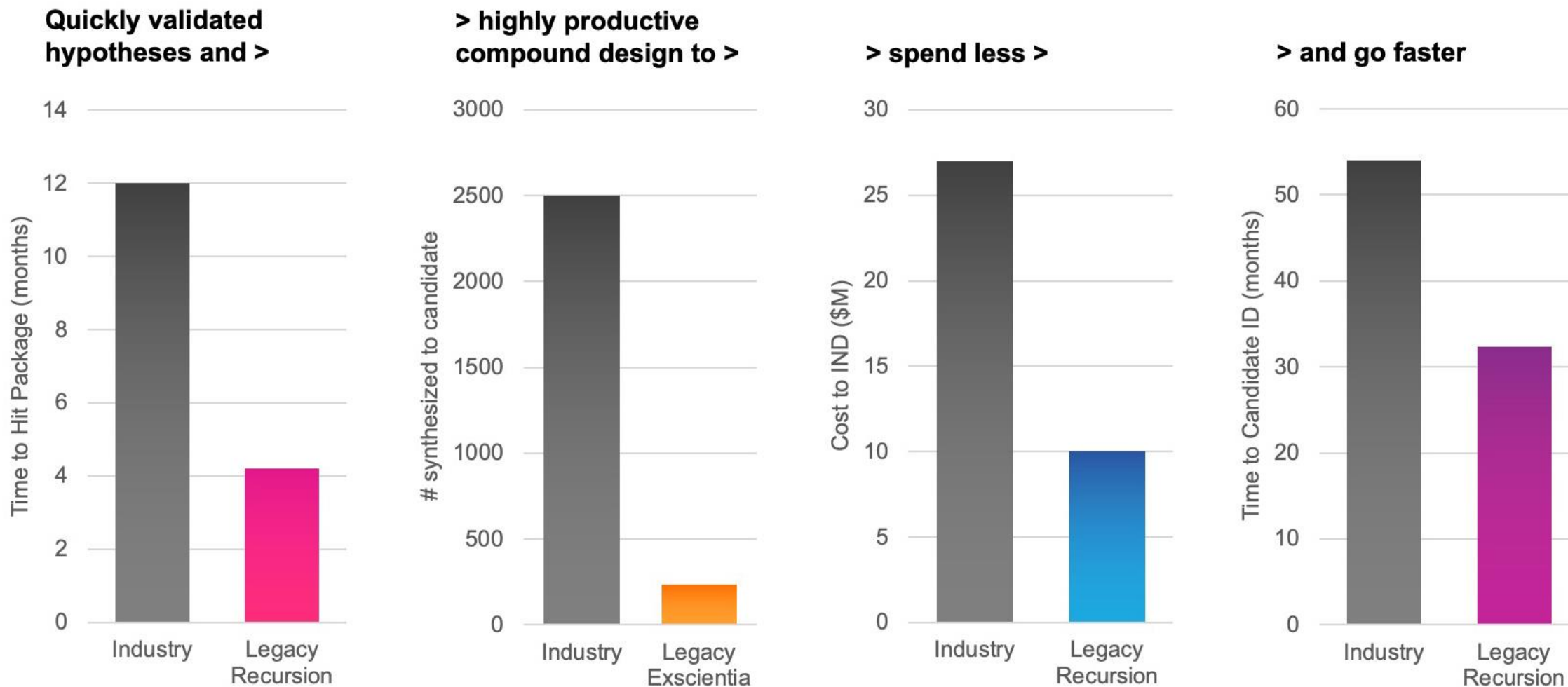
Unified Recursion OS with First-in-Class & Best-in-Class capabilities



Unified Recursion OS with First-in-Class & Best-in-Class capabilities



Recursion OS moves medicines to clinic faster and at a lower cost



(Far Left): Time from hypothesis screening to validated hit package for legacy Recursion programs. (Center Left): Legacy Exscientia compounds synthesized from hit to candidate ID. (Center Right): Total spend from hypothesis screening to the completion of IND-enabling studies for legacy Recursion novel chemical entity (NCE) programs that advanced to clinical trials. The cost to IND has been inflation-adjusted using the US Consumer Price Index (CPI) (Far Right). Time to validated lead is the average of >280 legacy Recursion programs since late 2017 through 2024. Industry data adapted from Paul, et al., Nature Reviews Drug Discovery (2010) 9, 203-214

On-track to deliver at least \$100M in synergies from the business-combination

FY2024 REVENUE

FY24 pro forma revenue
\$82.6 million¹

CASH

\$603 million
in cash, cash equivalents
and restricted cash²

Expected cash runway into 2027

Operating Expense Update

- At least \$100 million in synergies with majority of the run rate amount achieved in 2025
- Carved out Vienna operations with 49% ownership in a newly formed company focused on precision oncology, Alpha Biotechnology GmbH
- On-track to sub-lease multiple legacy sites
- The company will provide a comprehensive update in May 2025

1. Unaudited pro forma consolidated revenue is presented as if the Exscientia business combination had occurred on January 1, 2023. U.S GAAP revenue was \$58.8 million in the Recursion consolidated financial statements
2. Recursion noted that the change in Exscientia's cash and cash equivalents and short term bank deposits from December 31, 2023 to November 20, 2024, the date of the close of the acquisition was \$184 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 5th, 2025
□	REC-3565 (MALT-1i) in B-cell malignancies	Phase 1 first patient dosed	1H25
□	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 PI3K α H1047Ri		FY25

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



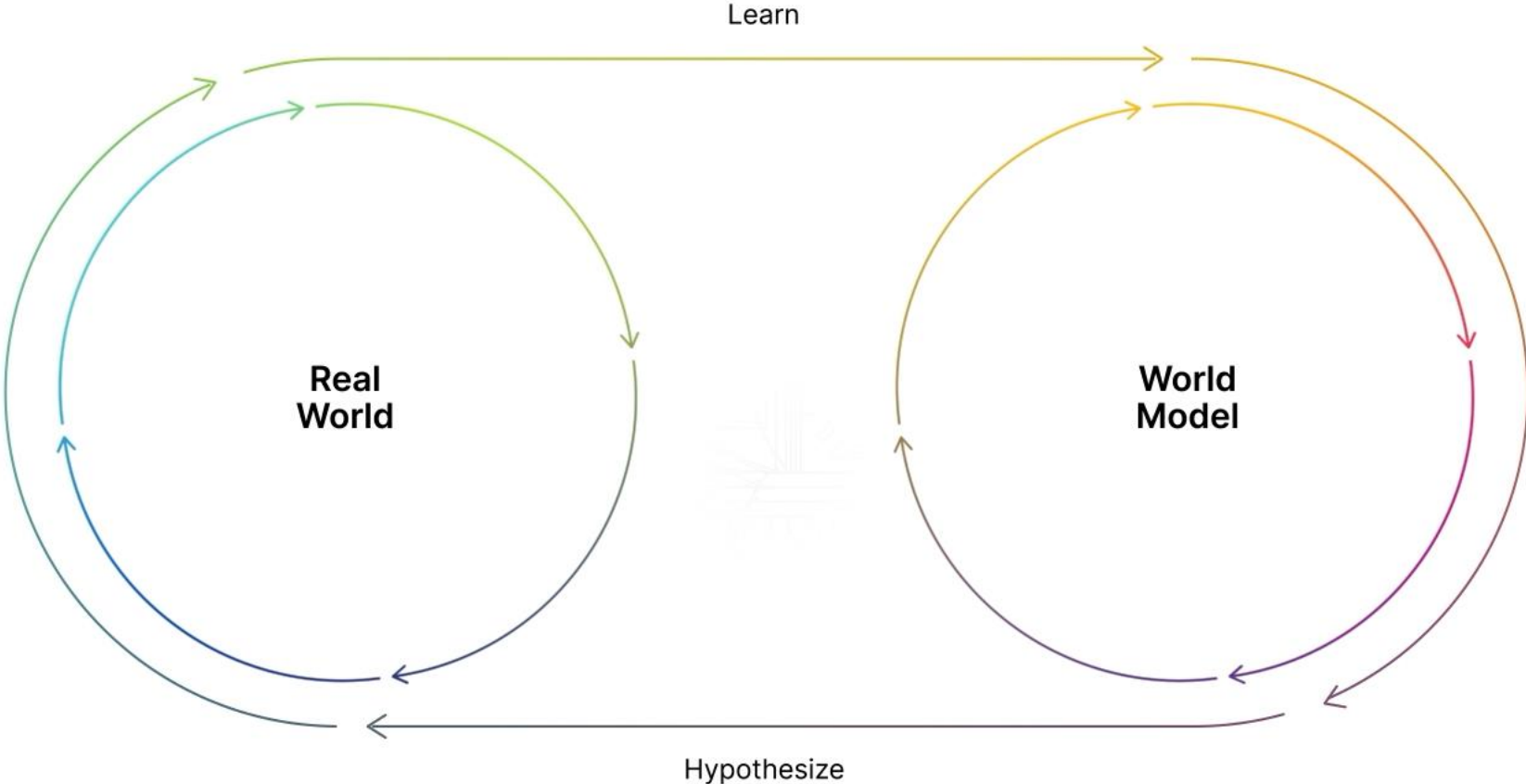
Real World



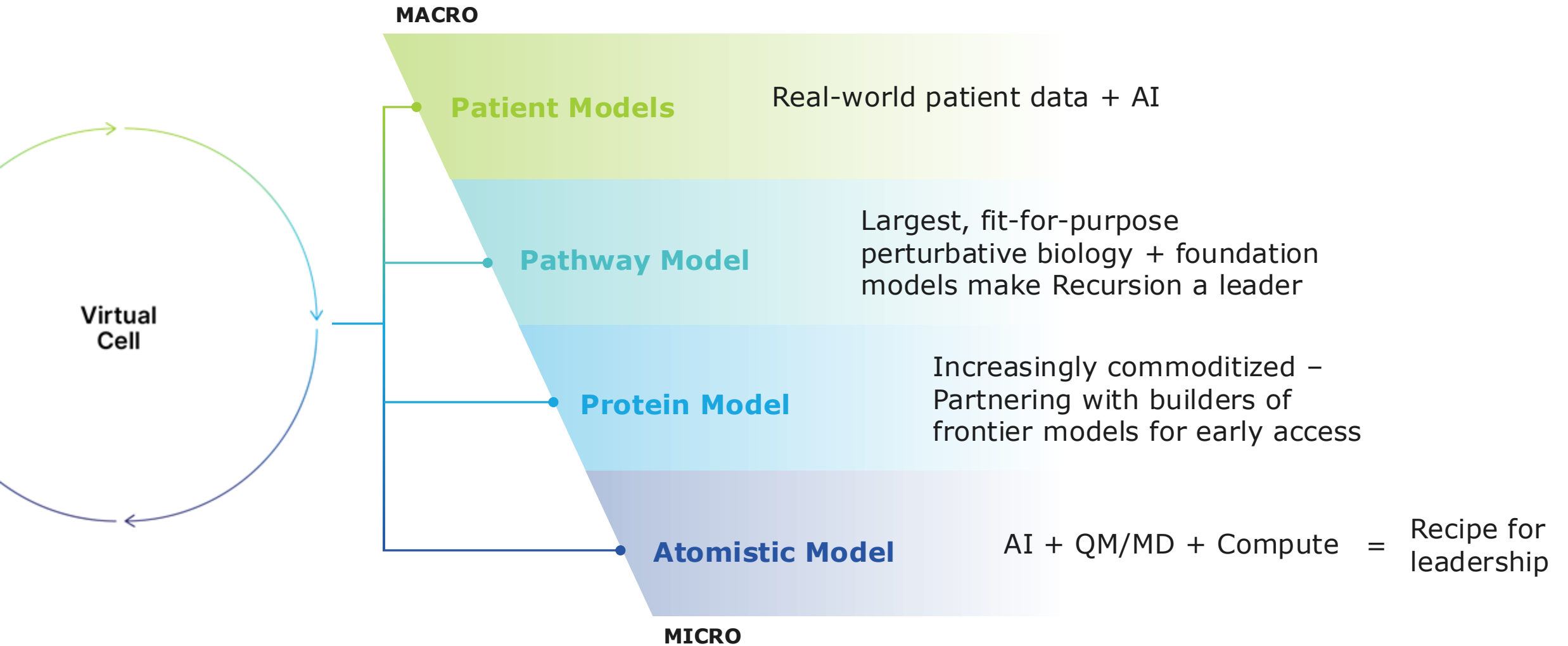
World Model



The Race to Accurately Simulate Biology at Scale



The Race to Accurately Simulate Biology at Scale



We harness value from the Recursion OS with a multi-pronged capital efficient business strategy



PIPELINE

Pipeline strategy

Build internal pipeline in indications with potential for **advance transformational medicines for patients**

- Oncology
- Rare disease
- Other areas of high unmet need

PARTNERSHIP

Partnership strategy

Partner in **complex therapeutic areas** requiring large financial commitment or competitive arbitrage
Leverage partner knowledge and clinical development capabilities

- Neuroscience
- Oncology
- Immunology
- Other large, intractable areas of biology

DATA

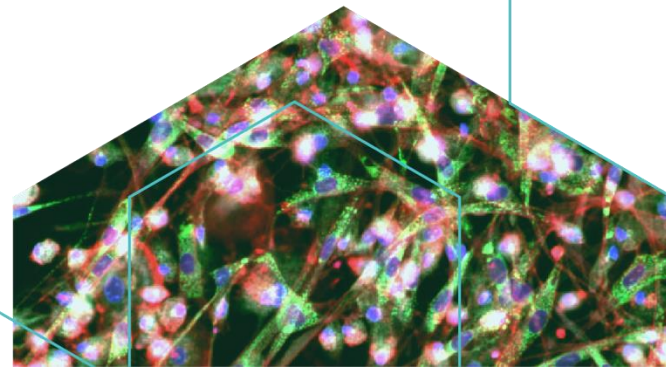
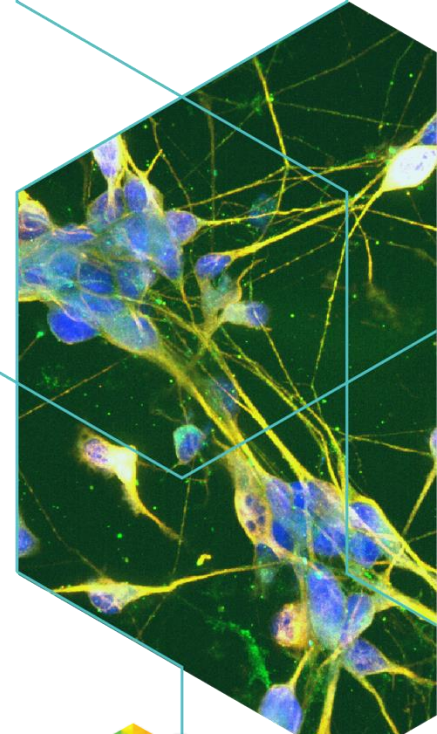
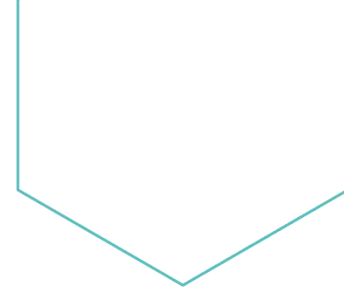
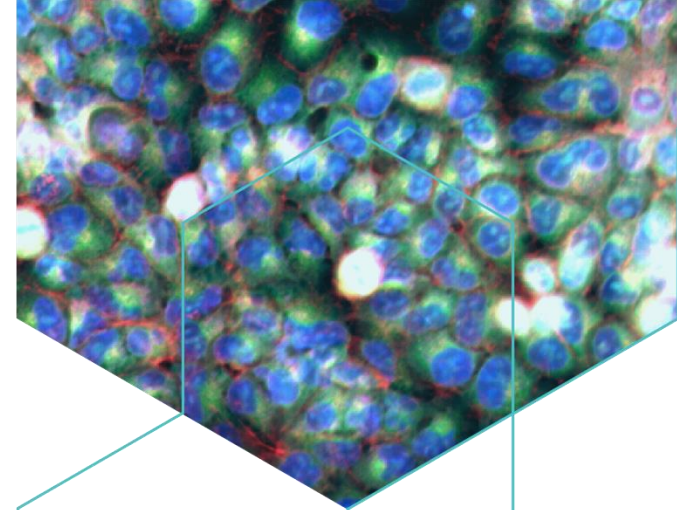
Data strategy

License subsets of data and key tools
Direct generation of new data internally **to maximize pipeline and partnership value-drivers**

- Licensing
- Augment Recursion OS
- LOWE

VALUE CREATION

Pipeline



PIPELINE

Oncology

Advanced Solid Tumors (CDK7 Inhibitor): REC-617*

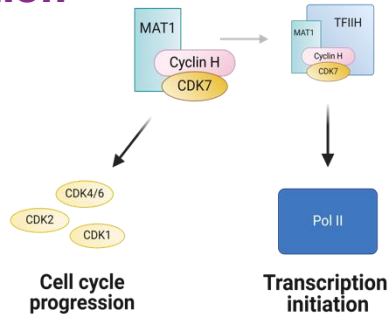
Unmet Need

- **Aberrant CDK7** overexpression common in advanced **transcriptionally-addicted** solid tumors
- Potential to address **multiple indications**, including post CDK4/6 population patients

~185,000
Treatable US + EU¹

Mechanism of Action

- **Reversible** CDK7 inhibitor
- **Dual function** that targets both cell cycle progression and transcriptional regulation



Development Strategy

CURRENT STAGE

ELUCIDATE

Phase 1
Monotherapy
Dose Escalation

EXPECTED STAGES

Q1 2025

Phase 1
Combo (SERD)
Dose Escalation

H2 2025

Phase 2
Combination
Dose Expansion

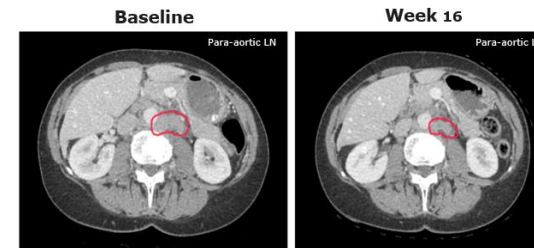
Differentiation

- Potential **Best-in-Class** and **First-in-Class** CDK7 Inhibitor
- Designed with **reduced transporter interactions** to **minimize GI adverse events** seen with competitor molecules



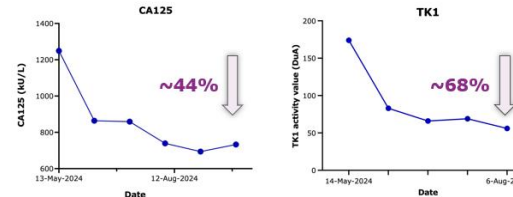
Key Clinical Data

- REC-617 safe and well-tolerated in Phase 1 dose escalation with **no treatment-related discontinuations**



1 cPR

At week 16 (-34%) in late-line ovarian cancer patient



~44%

Reduction in CA125 in late-line ovarian cancer patient

Recursion Approach

- **AI-powered precision design** to optimize PK/PD and **maximize potential therapeutic index**

136

Novel compounds synthesized to candidate ID

What's Next

- Combination studies initiation expected in **1H25**
- Additional Phase 1 data expected in **2H25**

1. Advanced solid tumors including breast, NSCLC, ovarian, pancreatic, colorectal and head & neck. US and EU5 treatable incidence, 2022.
 2. Besnard et al, AACR (2022).
 3. PK studies conducted in CD1 mice, single-dose administration. >10 hr IC₅₀ results in significant body weight loss.

Solid Tumors & Lymphoma (RBM39 Degradar): REC-1245

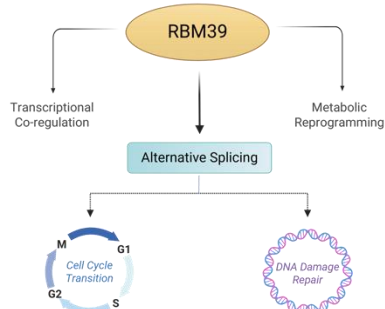
Unmet Need

- Solid tumor and lymphoma patients experience disease progression while on frontline therapies
- Potential as a single agent or in combination with chemo/IO

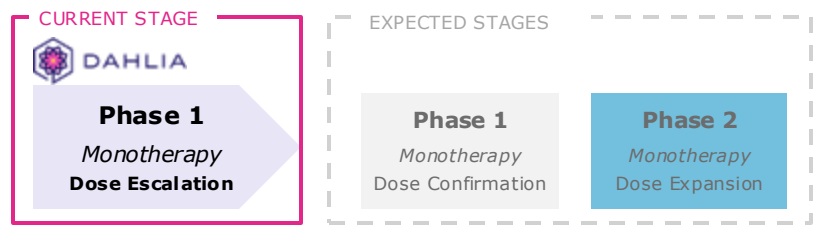
>100,000
Treatable US + EU¹

Mechanism of Action

- **Molecular glue** RBM39 degrader via E3 ligase adaptor DCAF15
- **Disrupts RNA splicing** to downregulate cell cycle checkpoints, DDR networks, triggering cell stress, apoptosis

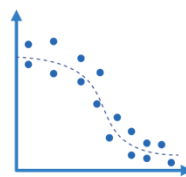


Development Strategy



Differentiation

- Potential **First-in-Class** RBM39 Degradar
- **No significant** in vitro safety concerns (hERG, CEREP)



Highly Potent & Selective



Minimal Off-Target Liabilities

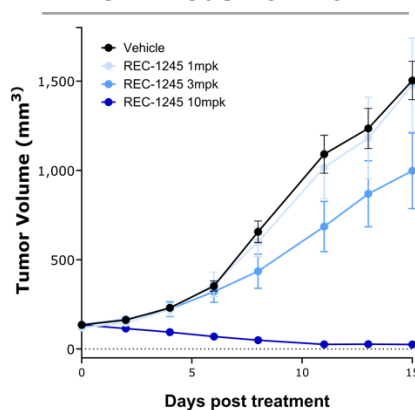


Biomarker Defined Population

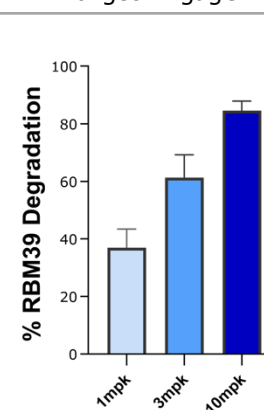
Key Preclinical Data

- REC-1245 shows significant **monotherapy regressions**
- **Dose-dependent** anti-tumor activity correlates with PD

CDX Model: OVK18²



PD: Target Engagement³



Recursion Approach

- Unbiased **ML-powered phenomap insight** to identify **novel DDR signature** and relate cellular phenotypes

204

Novel compounds synthesized to candidate ID

18 months

From Target ID to IND-Enabling studies

What's Next

- Phase 1 update in dose-escalation expected in **1H26**

1. Internal company estimates. Assumes US+EU5 addressable incidence with biomarker-enriched solid tumors and other select histologies.
 2. N=8 mice per group REC-1245 administered BID PO at doses noted.
 3. PD evaluated after 5 days BID oral administration of REC-1245 at doses noted; N=3 mice per group in PD portion.

B-Cell Malignancies (MALT1 Inhibitor): REC-3565*

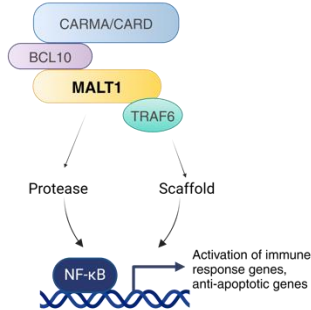
Unmet Need

- Mutations causing **constitutive MALT1 protease activity** and MALT1-cIAP fusions are aggressive with **limited treatment options**
- Potential to enhance **NF-κB inhibition** with BTK inhibitors

~41,000
Treatable US + EU¹

Mechanism of Action

- **Reversible** allosteric MALT1 inhibitor
- **Dampens NF-κB signaling** which drives survival and proliferation of B-cell tumors including ABC-DLBCL, MCL, FL, and CLL



Development Strategy

Q1 2025

EXCELERIZE

Phase 1
Monotherapy
Dose Escalation

EXPECTED STAGES

Phase 1
Combination
Dose Escalation

Phase 1
Combination
Dose Expansion

Differentiation

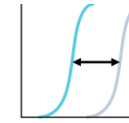
- Potential **Best-in-Class** MALT1 Inhibitor
- **Low UGT1A1** anticipated liability versus competitors
- **No significant off-target** safety concerns (CEREP, Kinome)



Lower Predicted
Jaundice Risk



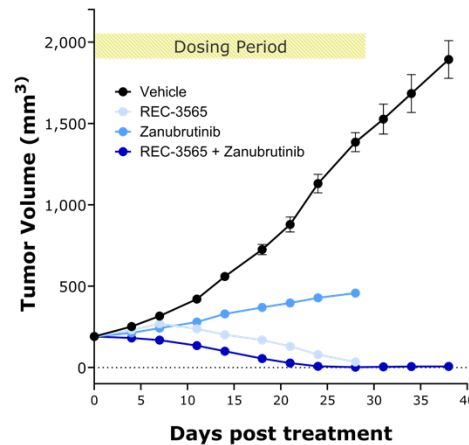
High Oral
Bioavailability



Wider
Therapeutic Index

Key Preclinical Data

- REC-3565 monotherapy shows significant **tumor regression**
- **Sustained** anti-tumor activity **in combo** with zanubrutinib



CDX Model: OCI-Ly10²

70%

Of mice in combination arm (REC-3565 + zanu) had no palpable tumors 10-days post last dose

Recursion Approach

- **AI-powered** precision designed **novel molecule** using molecular dynamics and hotspot analysis

344

Novel compounds synthesized to candidate ID

What's Next

- Phase 1 First Patient Dosed in B-Cell Malignancies (e.g., chronic lymphocytic leukemia) expected in **1H25**

Small-Cell Lung Cancer (LSD1 Inhibitor): REC-4539*

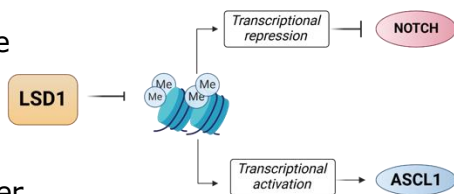
Unmet Need

- **SCLC** is a highly progressive disease with **5-year OS ~3%** in the extensive stage
- Clinical trial enrollment **remains NCCN-recommended** after 1L chemo/IO, despite advancements with DLL3-targeting BiTEs²

>45,000
Treatable US + EU¹

Mechanism of Action

- **Reversible** LSD1 inhibitor that can selectively upregulate NOTCH signaling
- **Promotes differentiation** of neuroendocrine cancer cells



Development Strategy

H1 2025

Phase 1
Monotherapy
Dose Escalation

EXPECTED STAGES

Phase 1
Combination
Dose Escalation

Phase 1
Combination
Dose Expansion

Differentiation

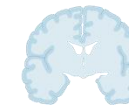
- Potential **Best-in-Class** LSD1 Inhibitor
- **Shorter-predicted half-life** plus **reversible MOA** to manage **on-target AEs**



Lower Predicted
Thrombocytopenia



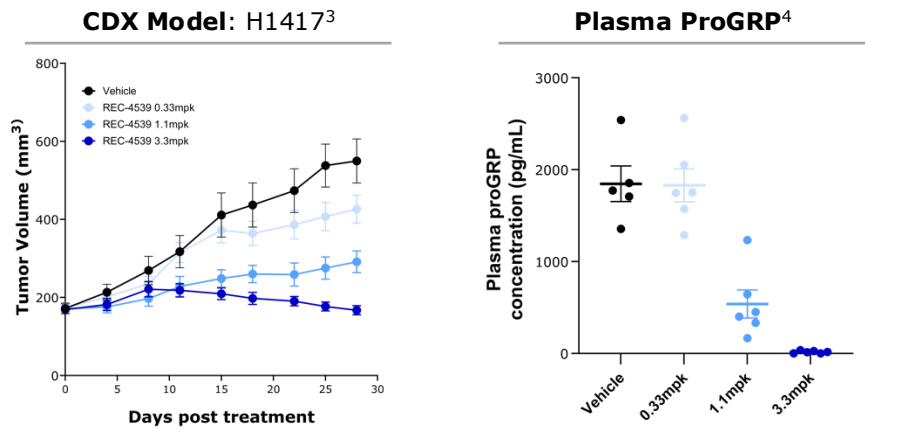
Shorter
Half-Life



Optimal
CNS Exposures

Key Preclinical Data

- **Dose-dependent** efficacy in SCLC human xenograft model
- Well tolerated with limited impact on platelet levels



Recursion Approach

- Precision design using **Active Learning**, combining reversibility with **CNS penetration**

414

Novel compounds
synthesized to candidate ID

What's Next

- Phase 1 First Patient Dosed in SCLC expected in **1H25**

1. EvaluatePharma Epidemiology 2023 (US and EU5).
 2. Referenced with permission from the NCCN Clinical Practice Guidelines in Oncology (NCCN Guidelines®) for Small Cell Lung Cancer V.3.2025.
 3. Payne et al. AACR, (2023).
 4. Data on File.

PIPELINE

Rare disease

Cerebral Cavernous Malformation (Superoxide Scavenger): REC-994

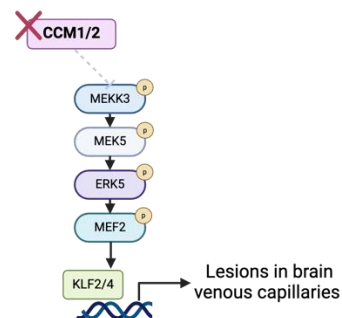
Unmet Need

- **No approved therapy**
- **Surgical resection** or stereotactic radiosurgery is non curative and **not always feasible** because of location

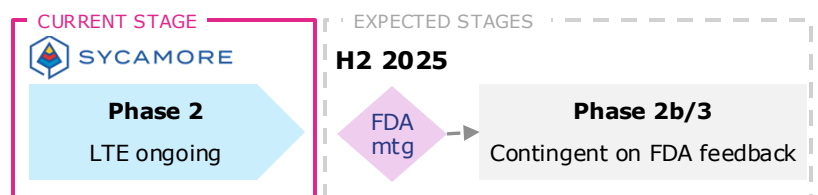
~360,000
Symptomatic
US + EU¹

Mechanism of Action

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



Development Strategy



Differentiation

- Potential **First-in-Disease** oral therapeutic for CCM
- **No TEAEs** leading to discontinuation up to **800 mg** in Ph 1³



Safe and well-tolerated MOA



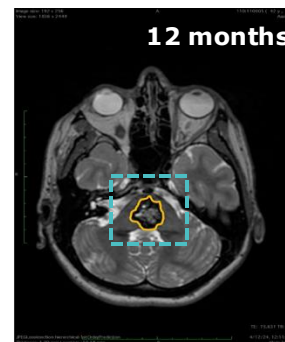
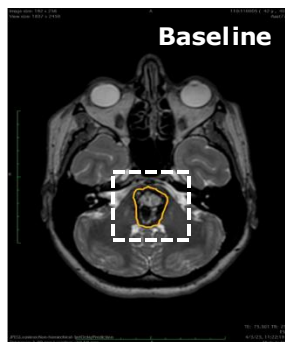
High oral bioavailability



Encouraging Ph 2 efficacy trends

Key Data

- **Reduces lesion number & size** in preclinical models
- Phase 2 **primary endpoint** of safety and tolerability met
- **REC-994 400 mg improved clinical function** (mRS) in in CCM patients, including those with **brainstem lesions**



~38%

Reduction in baseline lesion volume in 39 yr old female diagnosed with sporadic CCM brainstem lesion

Recursion Approach

- **Unbiased ML-aided** phenotypic drug screen to identify effective therapeutics driving CCM

80%
Of Ph2 patients continued to LTE

ODD
In US + EU

What's Next

- LTE ongoing
- Program updates expected in **2H25**

1. Prevalence for hereditary and sporadic symptomatic population; Internal company estimates.
 2. Gibson et al, Circulation (2015) and Data on File.
 3. Alfa et al, Pharmacol Res Perspect (2024).
 LTE: long-term extension; ODD: Orphan Drug Designation

Familial Adenomatous Polyposis (MEK1/2 inhibitor): REC-4881

Unmet Need

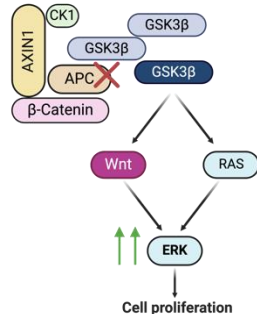
- **No approved therapy**
- **Colectomy** during adolescence is standard of care
- Patients at **significant risk of GI** cancer and suffer substantial decrease in **quality-of-life**

~50,000

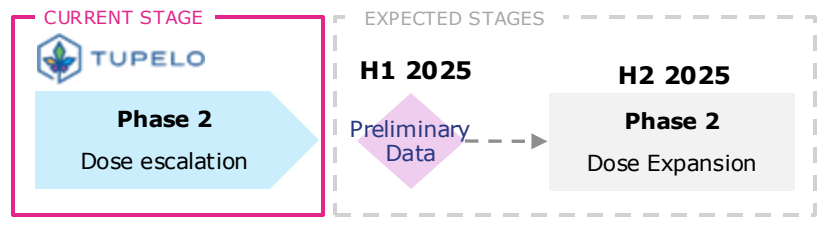
Diagnosed
US + EU¹

Mechanism of Action

- **Loss of APC** drives FAP disease progression through aberrant pathway signaling (e.g., Wnt/B-catenin, MAPK signaling)
- REC-4881 **selectively blocks** the activation of ERK (MAPK pathway)

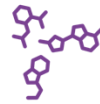


Development Strategy



Differentiation

- Potential **First-in-Disease** and **Best-in-Class** for FAP
- **Potent, non-competitive, allosteric** MEK1/2 inhibitor
- Oral 4 mg dose is **pharmacologically active**



Proof-of-mechanism
in Phase 1b



Validated
target

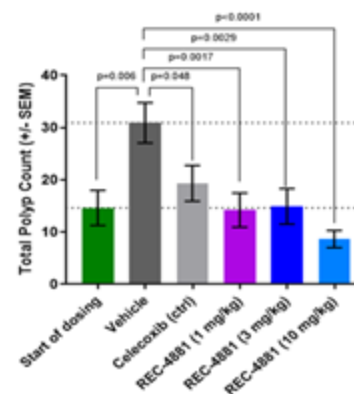


Preferential GI
exposure

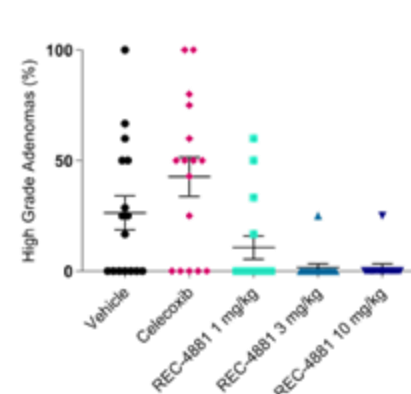
Key Preclinical Data²

- **APC^{min/-} mouse model:** Significantly reduces **polyp count** and **pre-cancerous adenoma**, outperforming celecoxib

Mean Polyps Per Group²



% Pre-Cancerous Polyps²



Recursion Approach

- Unbiased **ML-aided phenomap insight** in human cancer cells

FTD
In US

ODD
In US + EU

What's Next

- Phase 1b/2 safety & early efficacy data expected in **1H25**

1. Prevalence for adult and pediatric population, Internal company estimates.

2. Data on file.

FTD: Fast Track Designation; ODD: Orphan Drug Designation

Hypophosphatasia (ENPP1 Inhibitor): REV102

Unmet Need

- Opportunity to significantly **reduce costs & treatment burden**
- Many patients, particularly adults, may have difficulty accessing ERT
- Those who can access ERT face high treatment burden and tolerability hurdles

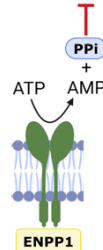
>7,800

Diagnosed prevalence
US + EU¹

Mechanism of Action

- ENPP1 inhibition is a **genetically validated** target in HPP models
- Potent ENPP1 inhibitor that **restores Ppi balance** and enables bone mineralization

Pathologic soft tissue calcification



Development Strategy

EXPECTED STAGES

2025

IND-Enabling Studies

2026

Phase 1

Healthy Volunteers

Differentiation

- Potential **First-in-Class** and **Best-in-Class** ENPP1 Inhibitor
- **Non-immunogenic small molecule** offering potentially safer solution than ERT (3-6 injections per week)



No significant in vitro safety concerns



Affordable oral treatment option

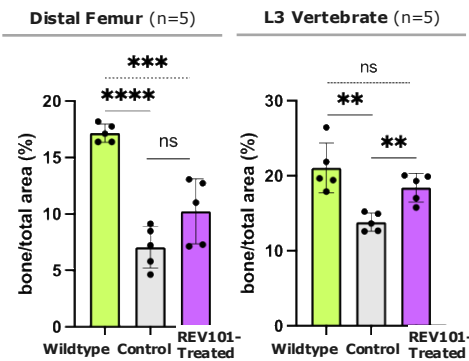


Potential as mono or combo therapy

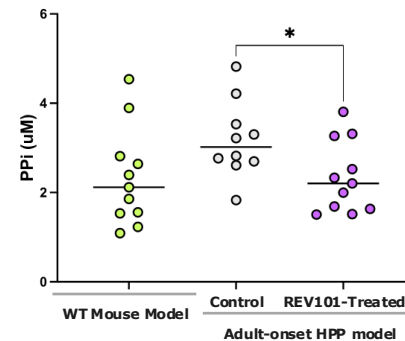
Key Preclinical Data²

- Improvement in mineralization in mouse models of HPP
- Significantly reduced Ppi levels to that of wild-type mice

Bone Morphometric Analysis



Plasma Levels of Ppi



Recursion Approach³

- **Precision designed for** both **high potency** and a lifetime of **chronic dosing**
- **Structurally distinct** differences vs competitor ENPP1 inhibitors
- **Maintain selectivity** and deliver a candidate with **high oral bioavailability** in the clinic

What's Next

- IND-enabling studies ongoing

1. HPP prevalence at birth. Mornet et al, 2020.
 2. Narisawa et al. ASBMR (2024).
 3. Joint venture with Rallybio
 ERT= Enzyme Replacement Therapy

Neurofibromatosis Type 2 (HDAC Inhibitor): REC-2282

Unmet Need

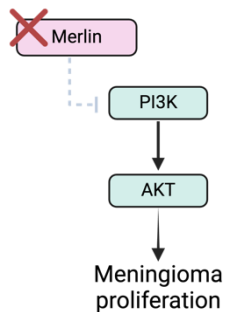
- **No approved therapy**
- Surgery/RT is standard of care (when feasible)²
- **Location** may make **complete resection untenable**, leading to hearing loss, facial paralysis, poor balance and visual difficulty

~33,000

Treatable
US + EU¹

Mechanism of Action

- **Loss of Merlin (NF2)** leads to PI3K signaling and meningioma proliferation
- **REC-2282** indirectly facilitates **AKT dephosphorylation** by disrupting the PP1-HDAC interaction



Differentiation

- Potential **First-in-Disease** and **Best-in-Class** for NF2
- Potential to **rescue disease-inducing effects** of NF2 loss



High oral
bioavailability



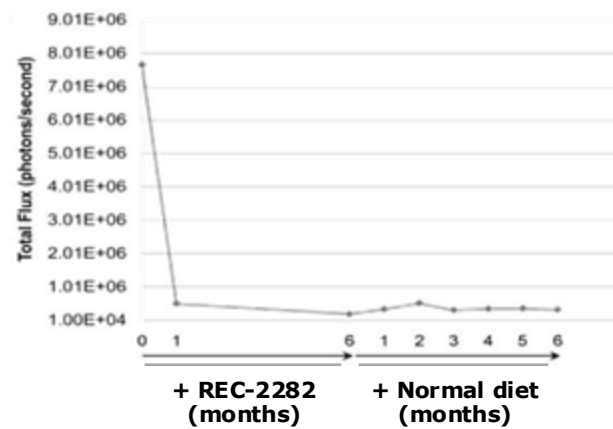
Improved CNS
penetration



Reduced
off-target effects

Key Preclinical Data

- **Prevents growth & regrowth** of NF2-deficient meningioma model in mice³



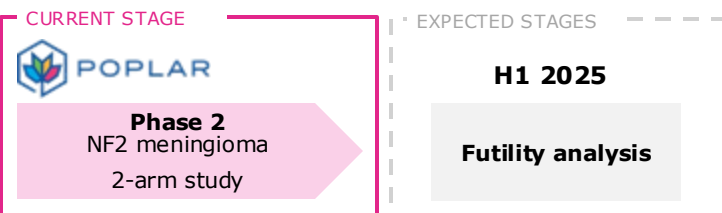
Recursion Approach

- Unbiased **ML-aided phenomap insight and drug screen** in human cells

FTD
In US

ODD
In US + EU

Development Strategy



1. Annual US and EU5 incidence for all NF2-driven meningiomas.
 2. Rogers et al. J Neurosurg, (2015).
 3. Data on File.
 FTD: Fast Track Designation; ODD: Orphan Drug Designation

PIPELINE

Other areas of high unmet need

C. difficile (*C. diff* Toxin B Selective Inhibitor): REC-3964

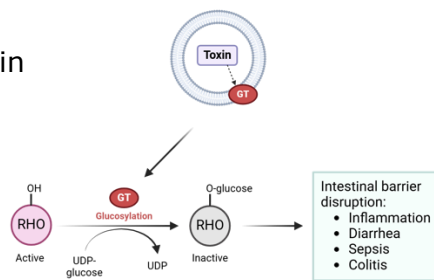
Unmet Need

- **Limited treatment options** for high-risk population with recurrent CDI cases
- Ability to address populations not eligible for FMT or microbiome-based therapies

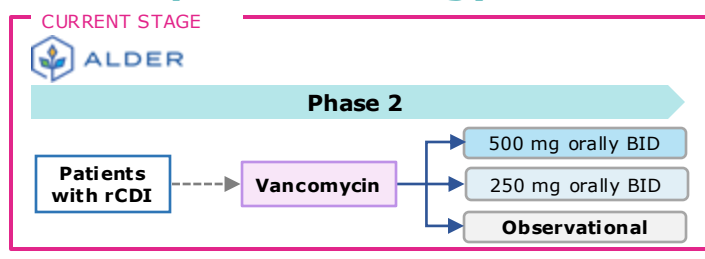
~175,000
Recurrent *C. diff*
cases US¹

Mechanism of Action

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



Development Strategy



Differentiation

- Potential **First-in-Class** as non-antibiotic oral for rCDI
- **Highly potent** and **well-tolerated** with no reported DLTs, SAEs or treatment-related discontinuations in Phase 1



Safe and well-tolerated MOA



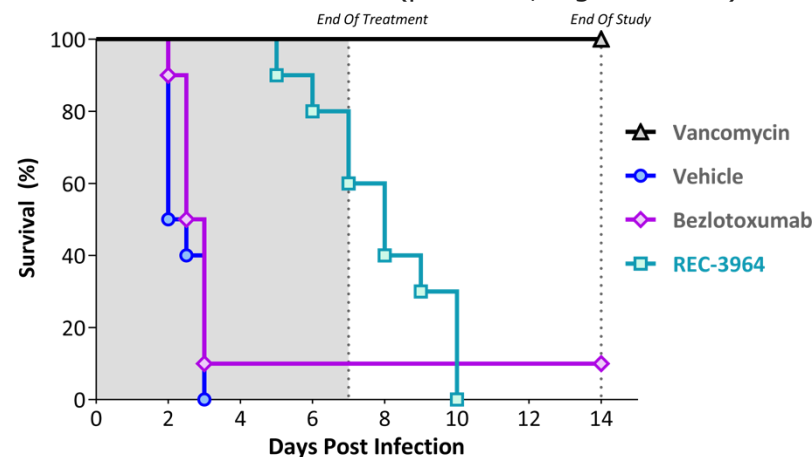
High oral bioavailability



Bacterial toxin selective

Key Preclinical Data

- REC-3964 significantly extended survival vs bezlotoxumab alone at the end of treatment ($p < 0.001$, log rank test)²



Recursion Approach

- Unbiased **ML-aided conditional phenotypic drug screen** in human cells

123

Novel compounds synthesized to candidate ID

What's Next

- Phase 2 update expected in **1Q26**

Idiopathic Pulmonary Fibrosis (Target Epsilon - Undisclosed): REC-4209

Unmet Need

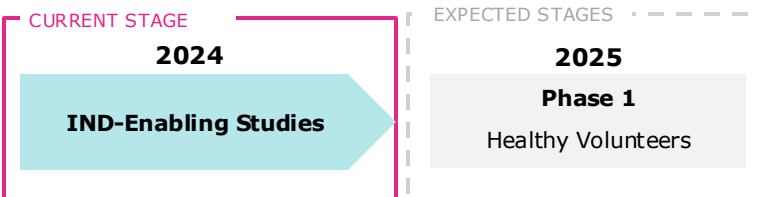
- **Approved therapies show modest slowing** of IPF progression
- **No improvement in survival (mOS 3-5 years) or quality of life** with current treatments

~130,000
Diagnosed prevalence
US¹

Mechanism of Action

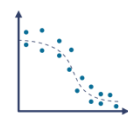
- **Reversible**, orally bioavailable, and potent Target Epsilon inhibitor
- Promotes **tissue repair and has potential to reverse fibrosis** likely by modulating TGF- β
- **Modulator of immuno-mesenchymal** populations in fibrosis, which **reduces fibrotic markers** in vivo and in vitro models of fibrotic disease

Development Strategy



Differentiation

- Potential **First-in-Class** treatment for IPF
- Potential for **safe** and **well-tolerated** novel treatment
- **In vitro models suggest** capability of reversing the fibrotic process driving IPF progression



Highly potent and selective



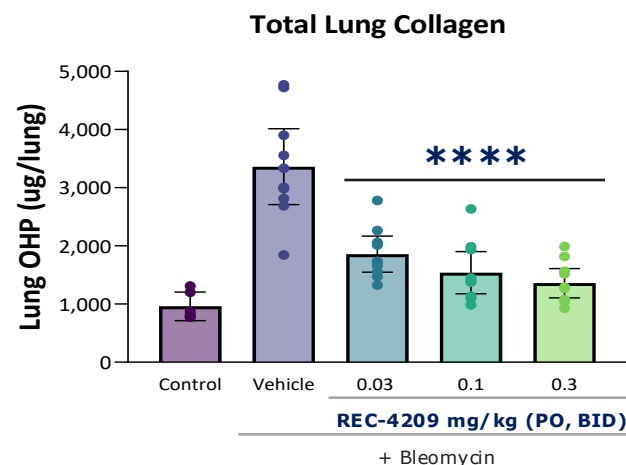
Novel mechanism of action



Potential fibrotic disease reversal

Key Preclinical Data

- REC-4209 at low doses reduces total lung collagen by 45% to 60% versus vehicle mice²



Recursion Approach

- Unbiased **ML-powered phenomap drug screen** in human cells

204

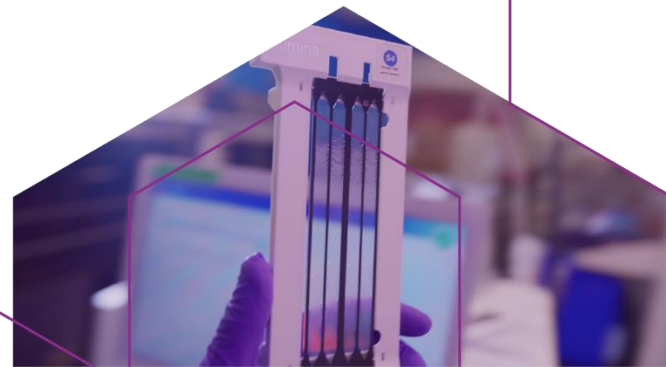
Novel compounds synthesized to candidate ID

What's Next

- IND-enabling studies ongoing

VALUE CREATION

Partnerships & Data Strategy



We harness value from the Recursion OS with a multi-pronged capital efficient business strategy



PIPELINE

Pipeline strategy

Build internal pipeline in indications with potential for **advance transformational medicines for patients**

- Oncology
- Rare disease
- Other areas of high unmet need

PARTNERSHIP

Partnership strategy

Partner in **complex therapeutic areas** requiring large financial commitment or competitive arbitrage
Leverage partner knowledge and clinical development capabilities

- Neuroscience
- Oncology
- Immunology
- Other large, intractable areas of biology

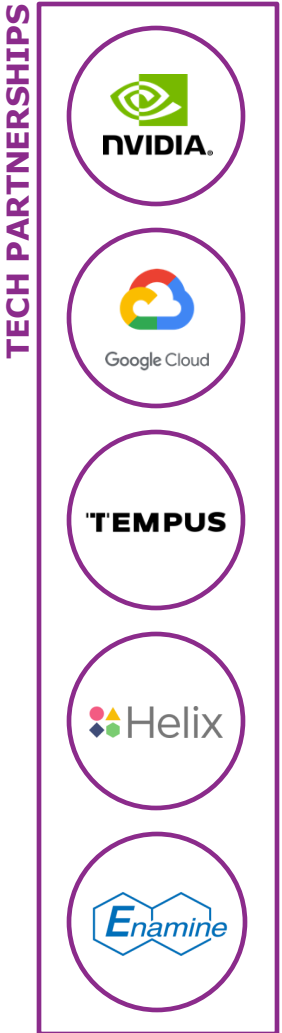
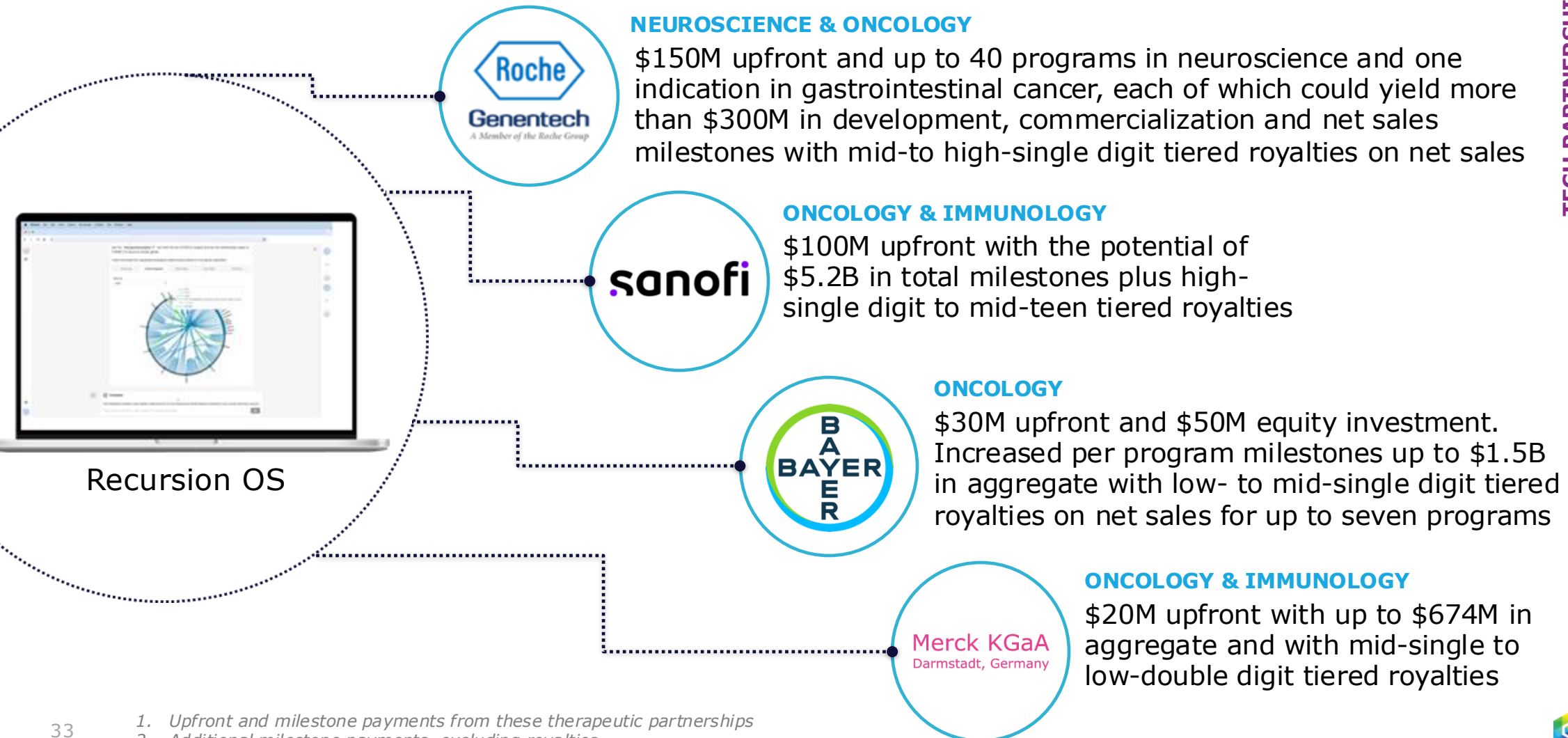
DATA

Data strategy

License subsets of data and key tools
Direct generation of new data internally **to maximize pipeline and partnership value-drivers**

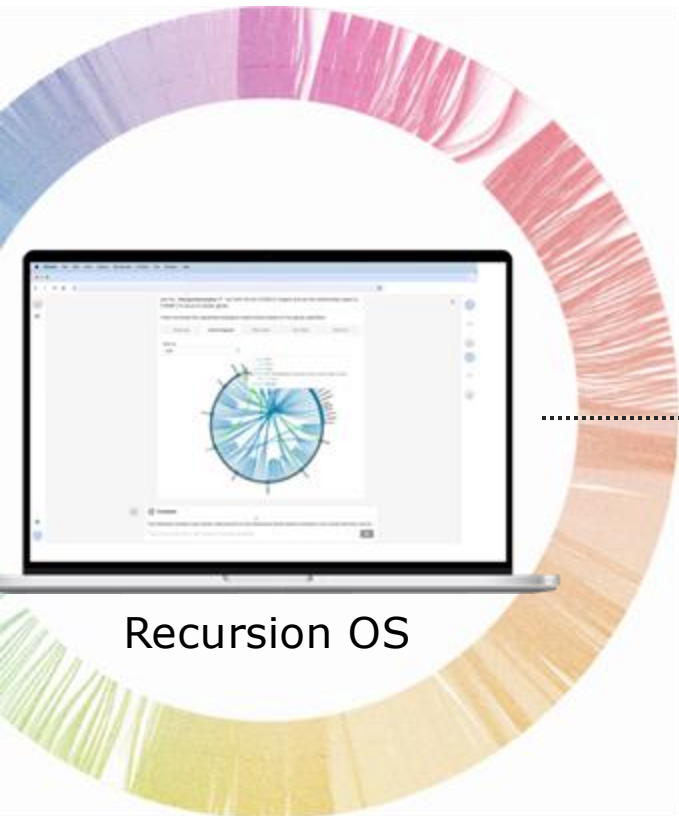
- Licensing
- Augment Recursion OS
- LOWE

Partnerships with approximately \$450M¹ earned to date and potential to receive more than \$20B² in additional milestones



33
1. Upfront and milestone payments from these therapeutic partnerships
2. Additional milestone payments, excluding royalties

We harness value from the Recursion OS with a multi-pronged capital efficient business strategy



PIPELINE

Pipeline strategy

Build internal pipeline in indications with potential for **advance transformational medicines for patients**

- Oncology
- Rare disease
- Other areas of high unmet need

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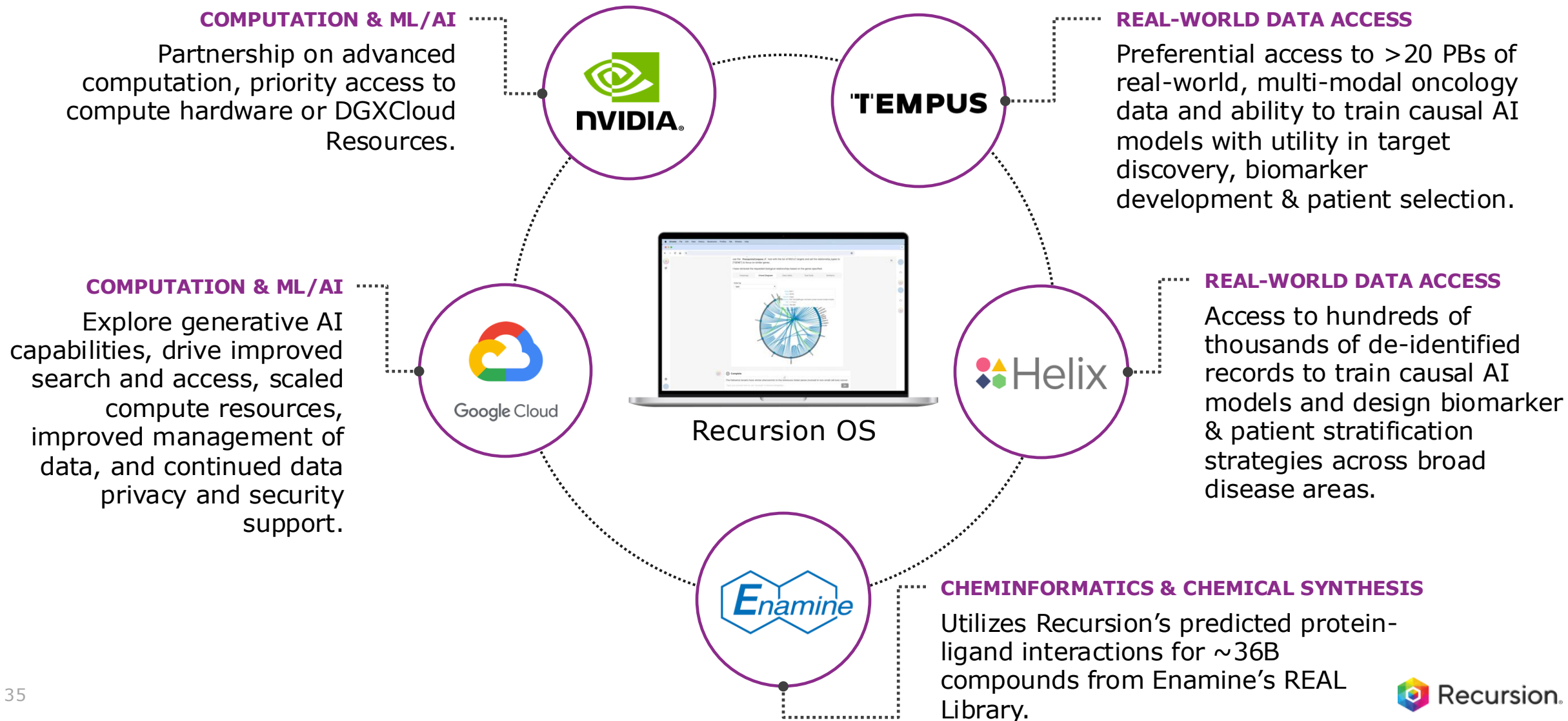
DATA

Data strategy


License subsets of data and key tools
Direct generation of new data internally **to maximize pipeline and partnership value-drivers**

- Licensing
- Augment Recursion OS
- LOWE

We license subsets of data and key tools to generate new data to maximize pipeline and partnership value-drivers



LOWE puts the Recursion OS at your fingertips via natural language without any coding expertise required



LOWE helps you orchestrate complicated workflows using natural language. Try one of the templates below or enter a prompt manually.

- Listing Known Targets Associated with a Specific Phenotype
- Identifying Top Compounds with Similar Phenoprints to a Given ...
- Request for Novel Compounds with Same Scaffold as a Given M...

[Browse templates](#)

Type your prompt here or use "/prompt" to browse templates...

D Give me a list of targets involved in non small cell lung cancer

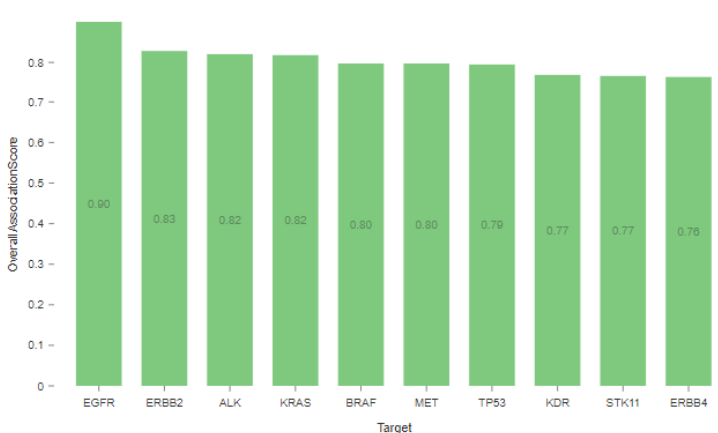
Thought Complete

The user has asked for a list of targets involved in non-small cell lung cancer (NSCLC). To provide this information, I will use the **PhenotypeToTarget** tool to retrieve a list of known biological entities (gene symbols) associated with NSCLC. The input will be the phenotype name "non small cell lung cancer."

I have retrieved the following targets for non small cell lung cancer.

Visualisation Data table Tool Data Artifacts

Select Y Value: overall_score



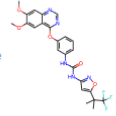
Target	Overall Association Score
EGFR	0.90
ERBB2	0.83
ALK	0.82
KRAS	0.82
BRAF	0.80
MET	0.80
TP53	0.79
KDR	0.77
STK11	0.77
ERBB4	0.76

D Find the top 50 commercially available compounds that have a similar structure to COC1=CC2=NC=NC(OC3=CC(NC(=O)NC4=NO(C(C)(C)(F)(F)F)=C4)=CC=C3)=C2=C1OCC and place an order for all 50

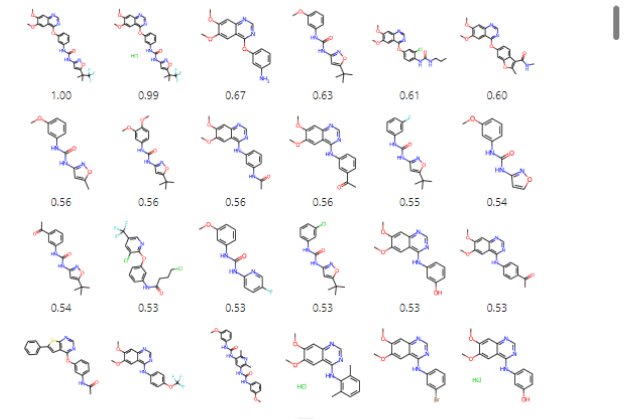
Thought Complete

The user has requested to find the top 50 commercially available compounds similar to a given SMILES string and then place an order for all 50. I will first use the **MolSimilaritySearch** tool to find the top 50 similar compounds and then use the **OrderCompounds** tool to place an order for them.

I have retrieved the most similar compounds starting from the following structure



Molecule grid Data table Tool Data Artifacts



Score
1.00
0.99
0.67
0.63
0.61
0.60
0.56
0.56
0.56
0.56
0.55
0.54
0.54
0.53
0.53
0.53
0.53
0.53
0.53

Type your prompt here or use "/prompt" to browse templates...

Culture and Team



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
Johnson&Johnson



Ben Taylor
Chief Financial Officer & President Recursion UK
Goldman Sachs **AETION**



David Mauro, MD PHD
Chief Medical Officer



David Hallett, PHD
Chief Scientific Officer



Ben Mabey
Chief Technology Officer



Kristen Rushton
Chief Operations Officer
Myriad genetics



Nathan Hatfield
Chief Legal Officer
WILSON SONSINI



Matt Kinn
Chief Business Officer



Erica Fox
Chief People & Impact Officer



Lina Nilsson, PHD
SVP, Head of Platform



Board of Directors



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



Zachary Bogue
Co-Founder & Partner of Data Collective



Blake Borgeson, PHD
Co-Founder of RXRX



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



Chris Gibson, PHD
Co-Founder & Chief Executive Officer



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



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



Zavain Dar
Co-Founder & Partner of Dimension



Our people are the most important ingredient for our mission



~800 employees

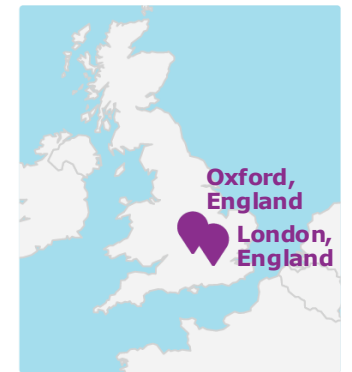
- Technology – data science, software engineering, automation, etc.
- Life Sciences – biology, chemistry, development, etc.
- Strategic Operations

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

Corporate ESG Performance

RATED BY ISS ESG

Prime

Rated

MORNINGSTAR | SUSTAINALYTICS

MSCI ESG RATINGS

A

CCC B BB BBB A AA AAA

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

Community Impact

altitude ▲ lab

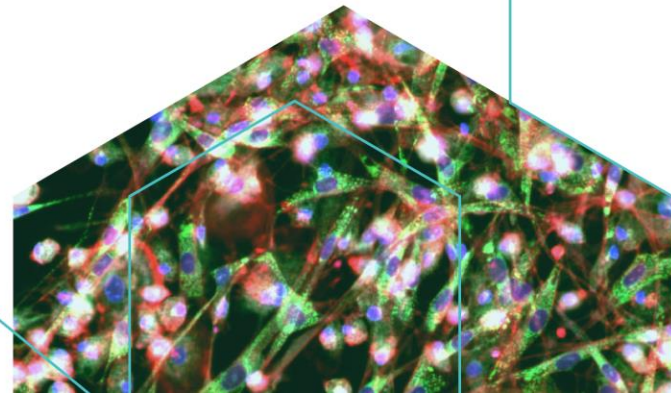
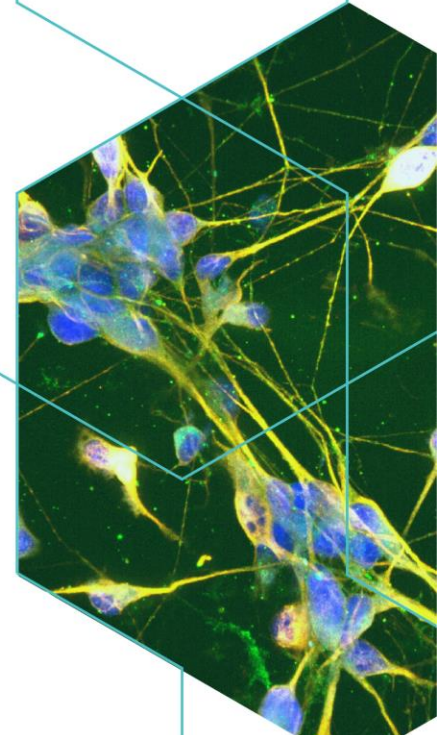
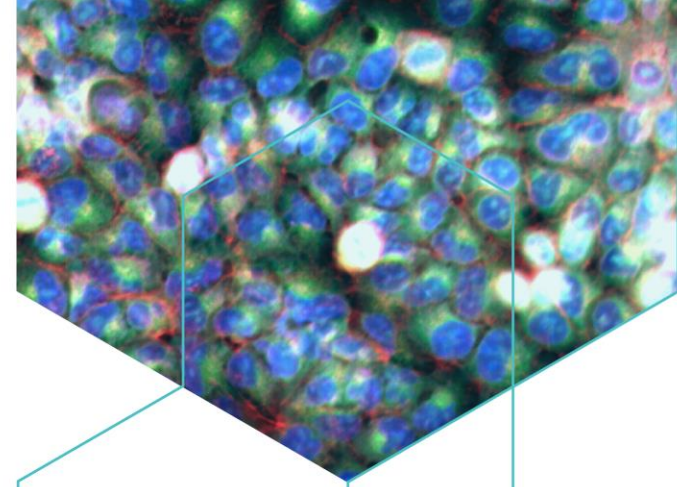
Founding Partner,
Life Science Accelerator

biohive™

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 both cell cycle progression and transcriptional regulation

Thesis & Differentiation

- **Non-covalent binding and improved selectivity** to decrease off-target 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

Recursion Approach

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

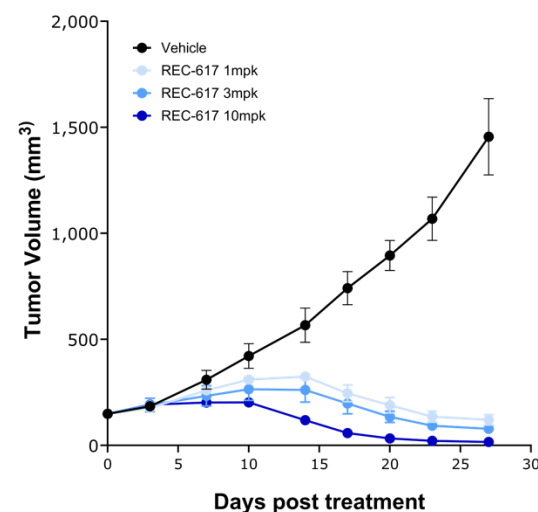
Meets or exceeds criteria Minor deviation Major deviation

Development Candidate (DC) Criteria:

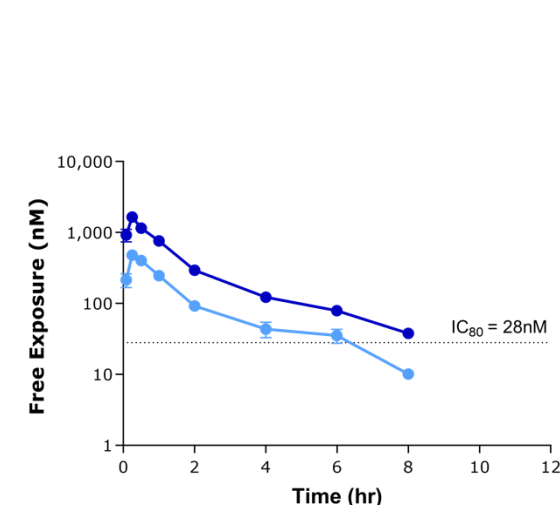
- **CDK7 IC50:** green <10nM; yellow 10-30nM; red >30nM
- **CDK7 selectivity:** green >100-fold; yellow 30-100-fold; red <30-fold
- **HCC70 IC50:** green <100nM; yellow 100-500nM; red >500 nM
- **Caco-2 A2B (efflux):** green >5(<3); yellow >1.5 (<10); red <1.5 (>30)
- **Half-life:** green <15, yellow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

Potent tumor regression with minimal IC₈₀ exposure

CDX Model: OVCAR3²



Mouse PK³



- 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

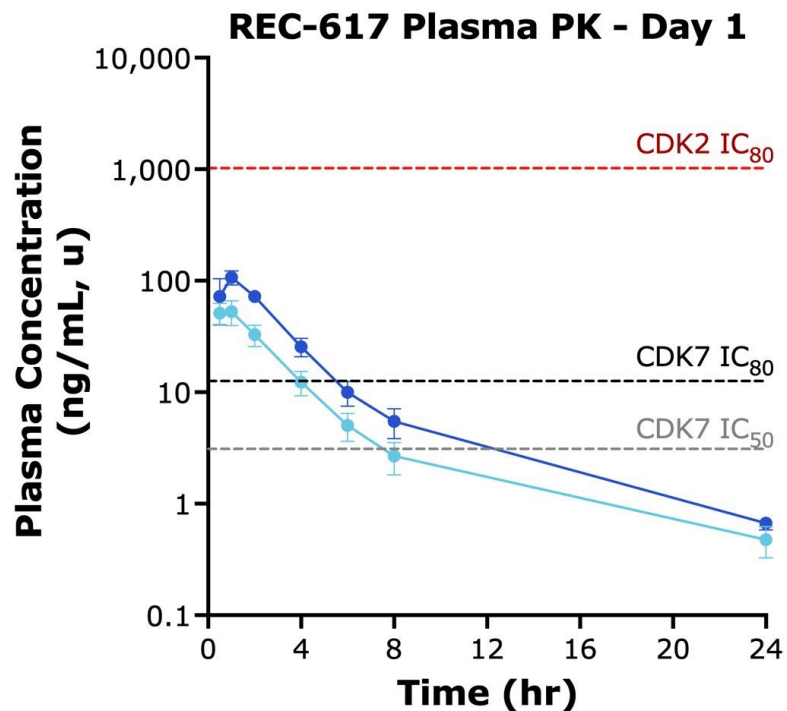
REC-617 Competitive Profile

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

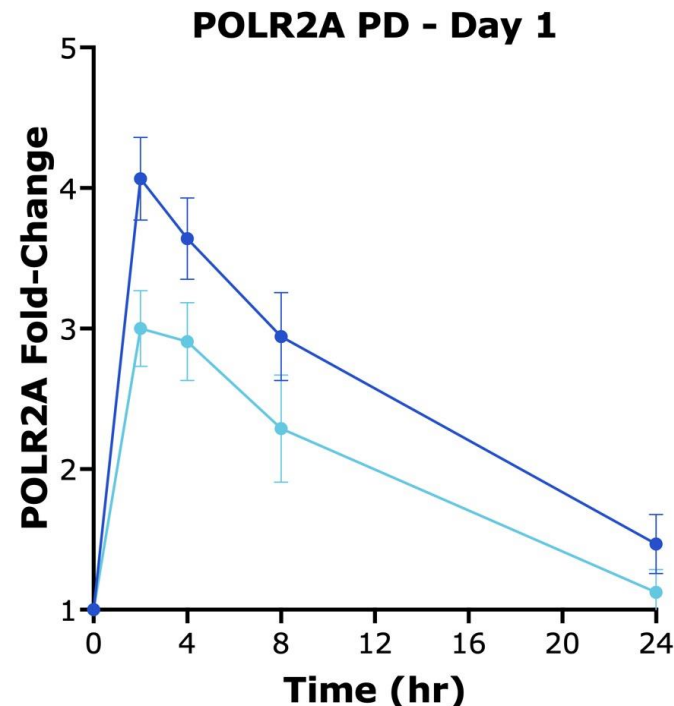
Trial Update

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



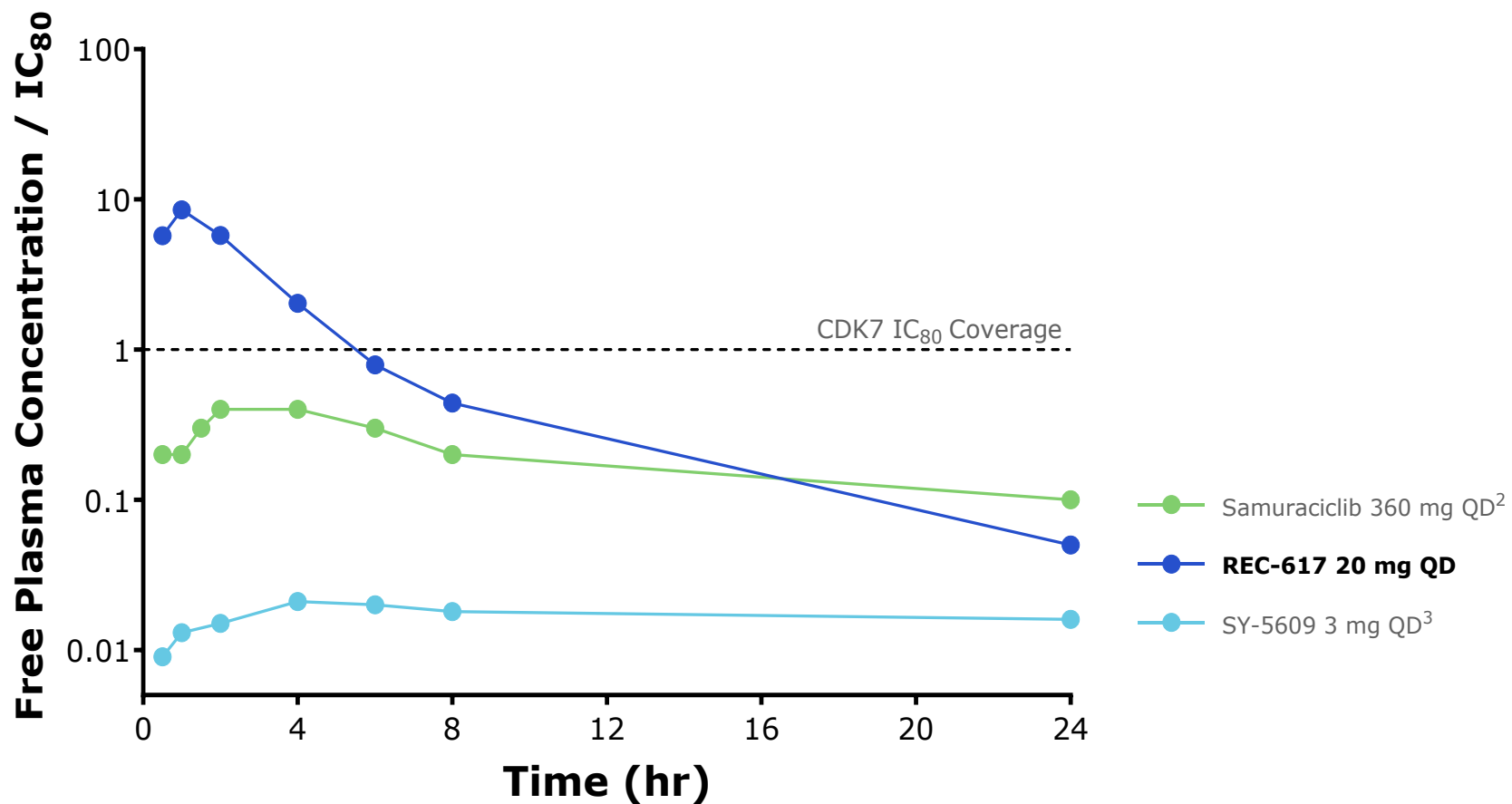
● REC-617 20 mg QD
● REC-617 10 mg QD



PK/PD Summary

- **Dose-Linear PK:** REC-617 exceeds CDK7 IC_{80} with rapid absorption (T_{max} 0.5–2h) and short $t_{1/2}$ (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-617**¹ compared to two clinical CDK7 inhibitors
- REC-617 is **more rapidly absorbed** (earlier T_{max}) 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

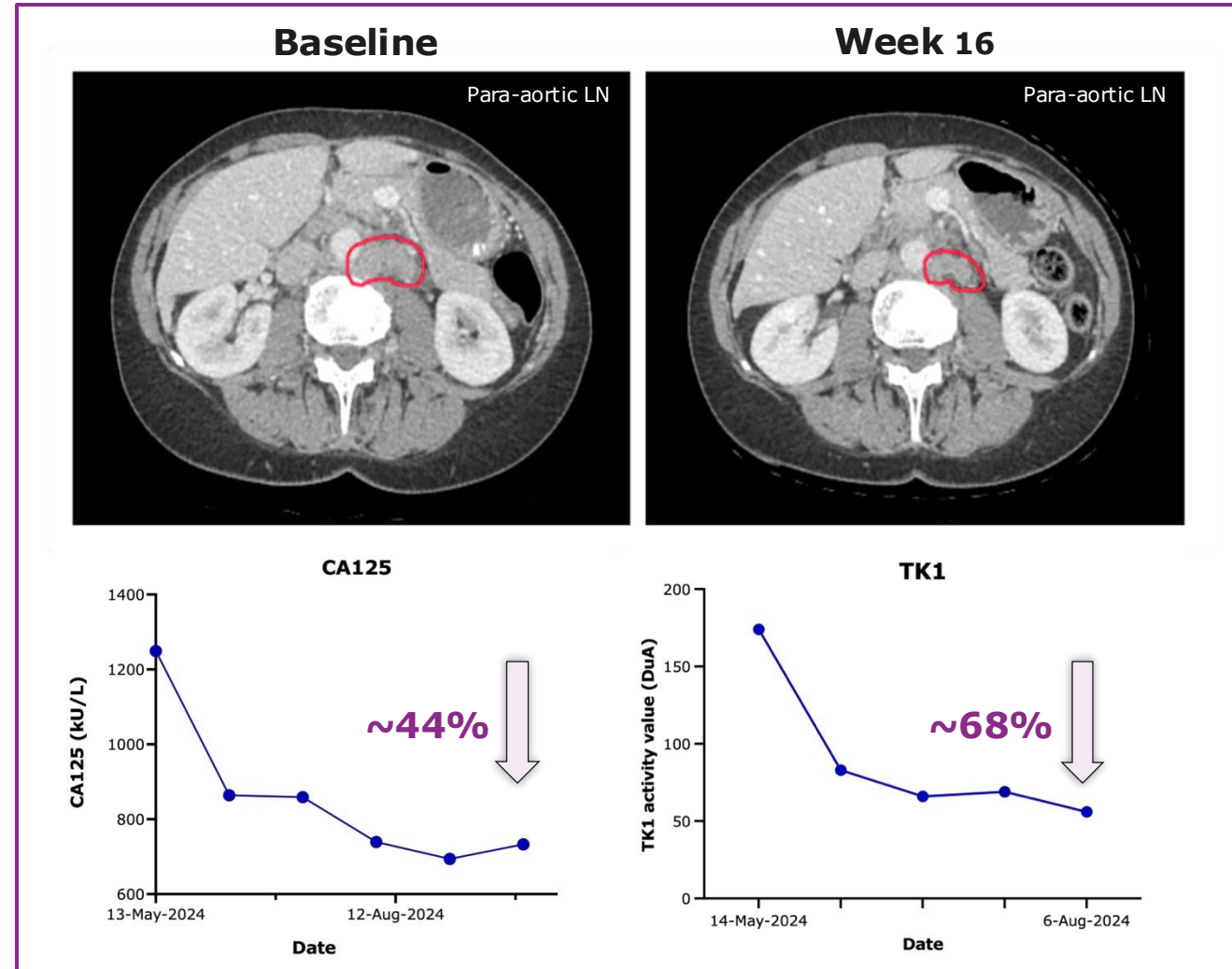
- **Partial response (-34%)** achieved at Week 16
- Meaningful reduction of tumor markers
- Response ongoing after 6+ months treatment

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



REC-1245: RBM39 Degradator

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

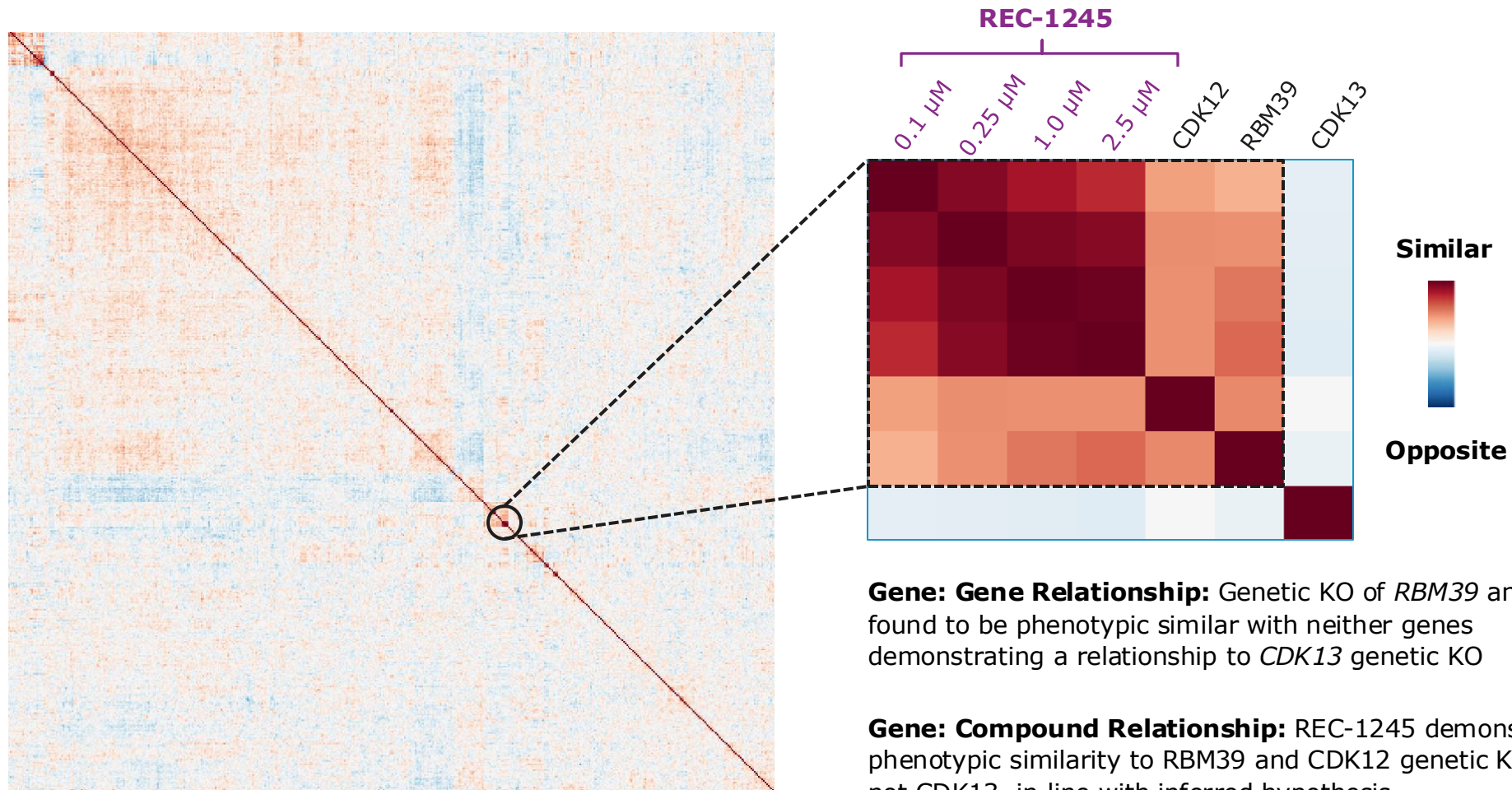
- **>100,000 patients** with solid tumor or lymphoma experience disease progression while on frontline therapies
- Potential to be used as a **single agent or in combination** with chemo/IO

Recursion Approach

- **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

Recursion OS Novel Insight



Gene: Gene Relationship: Genetic KO of *RBM39* and *CDK12* found to be phenotypic similar with neither genes demonstrating a relationship to *CDK13* genetic KO

Gene: Compound Relationship: REC-1245 demonstrates phenotypic similarity to RBM39 and CDK12 genetic KO but not CDK13, in line with inferred hypothesis

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

Key Preclinical Data¹

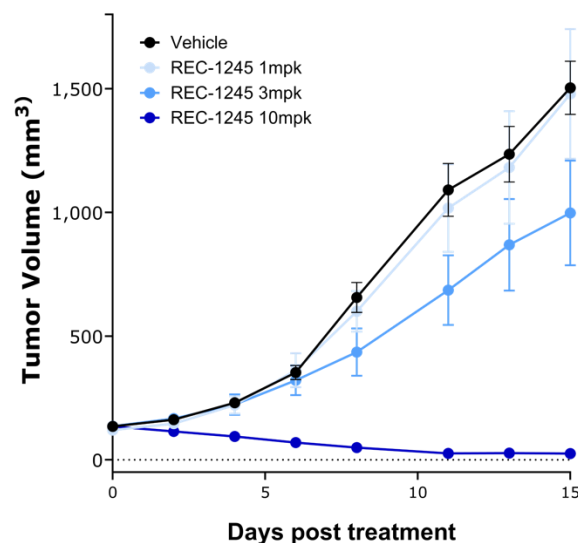
REC-1245 is highly selective and potent¹

Assay	DC Criteria	REC-1245
RBM39 Degradation DC ₅₀	<100 nM	Meets or exceeds criteria
CDK12 Kinase	No sig. activity	Meets or exceeds criteria
CEREP Safety Panel	No sig. activity	Meets or exceeds criteria
hERG IC ₅₀ (μM)	>30	Meets or exceeds criteria
Oral Bioavailability (%F)	>30	Meets or exceeds criteria

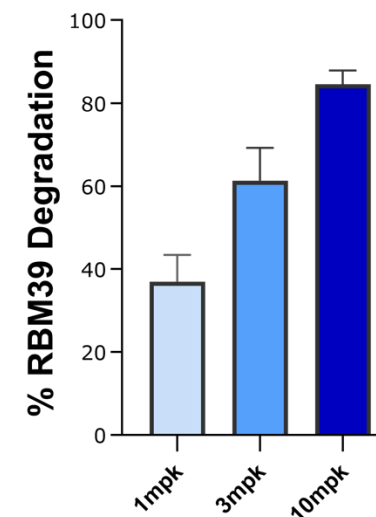
■ Meets or exceeds criteria
 ■ Minor deviation
 ■ Major deviation

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

1. Data on File.

2. N=8 mice per group in TV portion. REC-1245 administered BID PO.

3. PD evaluated after 5 days BID oral of REC-1245 at doses noted; N=3 mice per group in PD portion.

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 dose-confirmation

RP2D

Phase 2
Dose Expansion

Key Study Characteristics

Study objectives:

- Safety/Tolerability
- RP2D
- ORR

Eligibility:

- 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
- Phase 1 update in dose-escalation in **1H26**

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 **1H25**

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

Recursion Approach

- **AI powered** precision-designed 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

Assay	DC Criteria	Ph 1 large pharma	Ph1 biotech	REC-3565
MALT1 IC ₅₀ (nM)	<100	Yellow	Green	Green
OCI-Ly3 proliferation IC ₅₀ (nM)	<400	Yellow	Green	Green
UGT1A1 IC₅₀ (μM)	>10	Red	Red	Green
Caco-2 A2B (efflux) 10 ⁻⁶ cm/s	>5 (<3)	Green	Yellow	Green

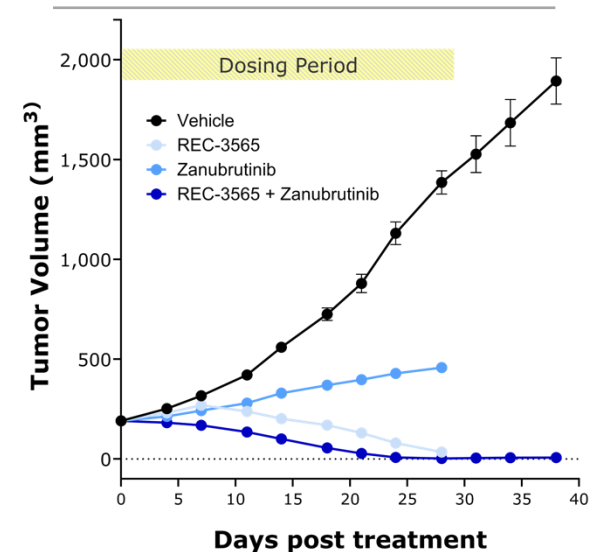
■ Meets or exceeds criteria
 ■ Minor deviation
 ■ Major deviation

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 μM:** green >10 μM; yellow <10->1 μM; red <1 μM
- **Caco-2 A2B (efflux):** green >5(<3); yellow >1-<5(>3-<10); red <1(>10)

Single-agent and synergistic activity in vivo²

CDX Model: OCI-Ly10²



70%

Of mice in combination arm (REC-3565 + zanu) had no palpable tumors 10-days post last dose

- OCI-Ly10 and Rec-1 cells are sensitive to both MALT1i and zanutrutinib *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 zanutrutinib

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

Development Strategy

EXCELERIZE Phase 1 study design

Phase 1 Dose Escalation

Q1 2025

Part A Monotherapy

- N ~30
- R/R B-Cell Malignancies
- REC-3565 PO QD or BID

RD

Part B Combination

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 no-observed-adverse-effect level (NOAEL)** enabling clinical trials

Trial Update

- Trial initiation expected in **1H25**

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

Recursion Approach

- **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

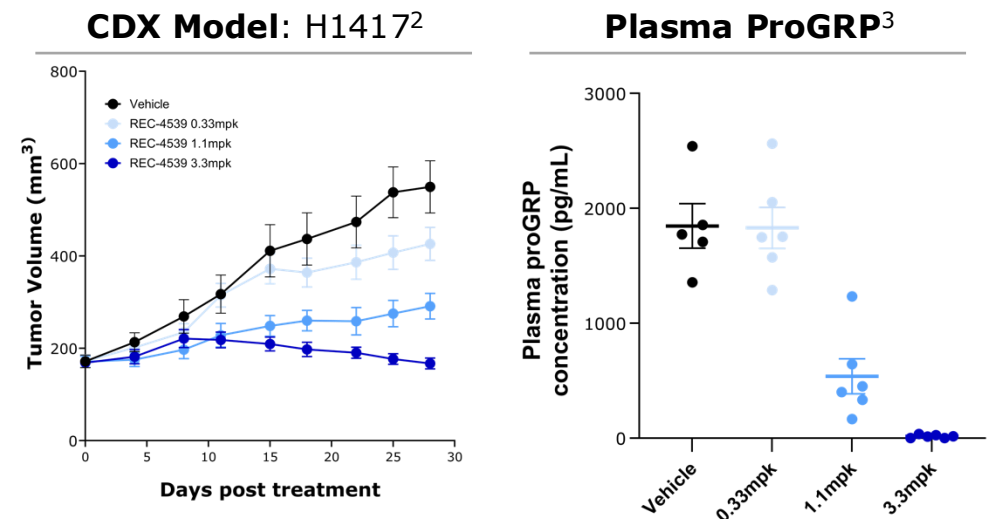
Assay	DC Criteria	Competitor 1	Competitor 2	REC-4539
Brain: Plasma Ratio	>0.5	Major deviation	Major deviation	Meets or exceeds criteria
MDCK-MDR1 Efflux Ratio (Pgp)	<2	Minor deviation	Minor deviation	Meets or exceeds criteria
Predicted Human Half-life	QD dosing	Major deviation	Major deviation	Meets or exceeds criteria

■ Meets or exceeds criteria
 ■ Minor deviation
 ■ Major deviation

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



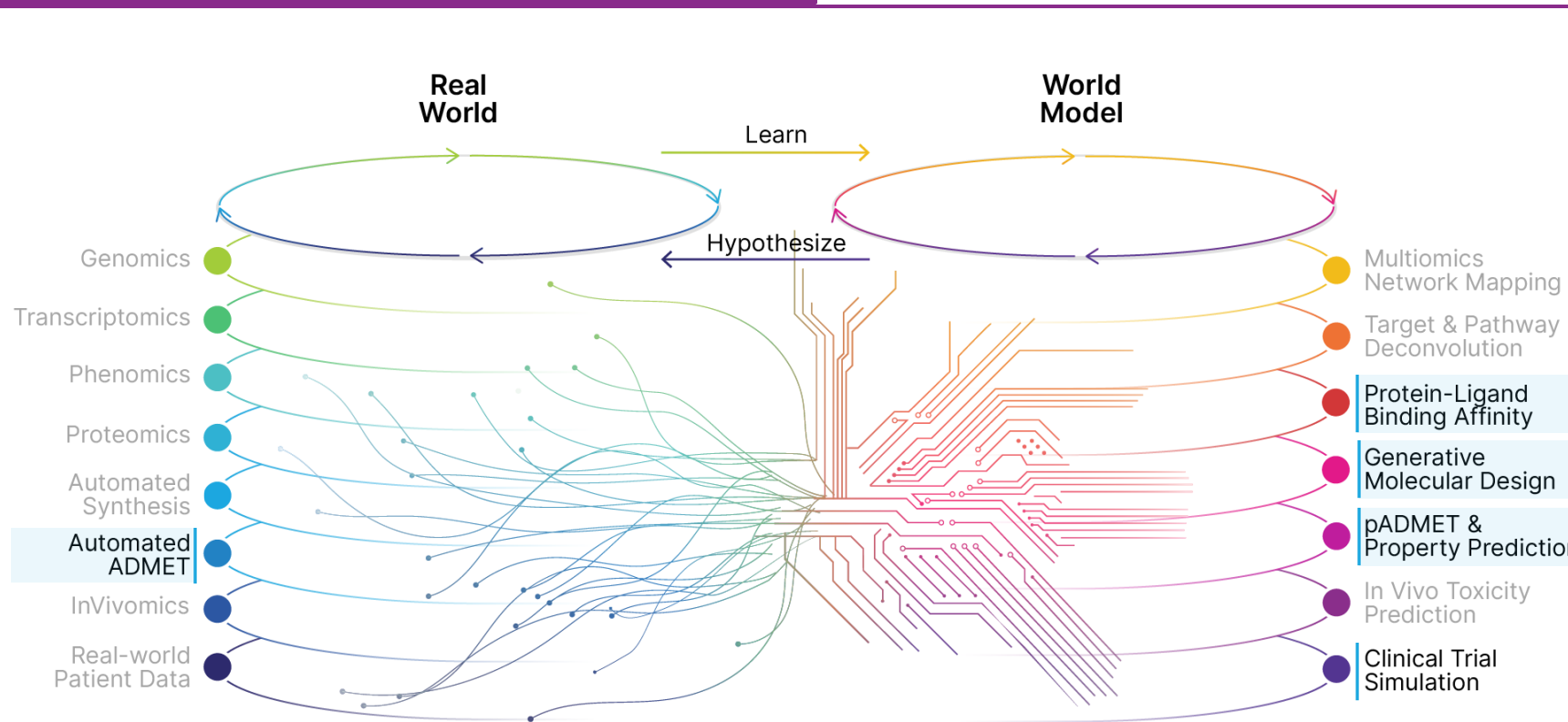
- Dose-dependent regression
- Well-tolerated with limited impact on platelet levels

Trial Update

- Phase 1 **First Patient Dosed** in SCLC expected in **1H25**

REC-7735 (PI3K α H1047R): An AI-designed, highly selective H1047R-targeting PI3K inhibitor designed to enhance therapeutic index*

Recursion OS: REC-7735 Discovery



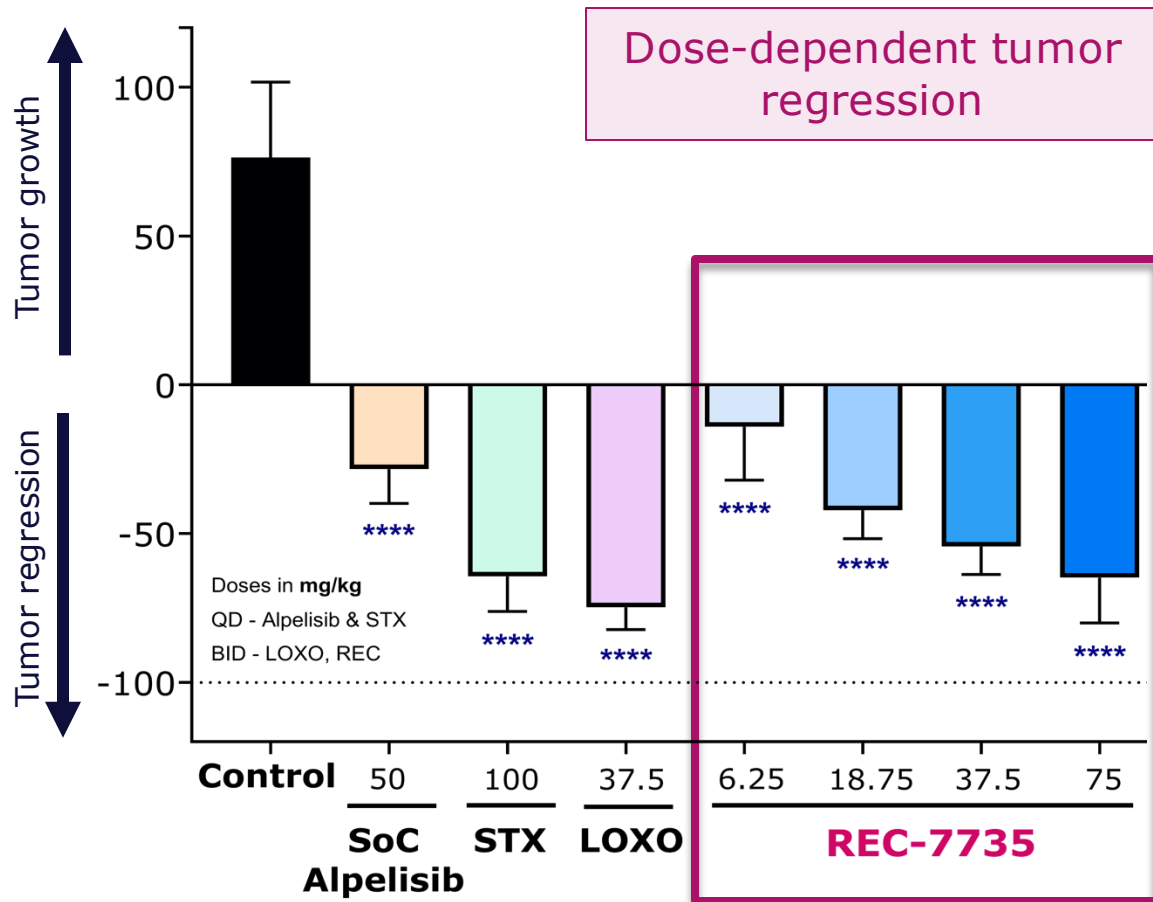
Our Approach: AI-powered precision design using automated synthesis and generative molecular design intended to mitigate adverse events seen in class

PI3K α H1047R

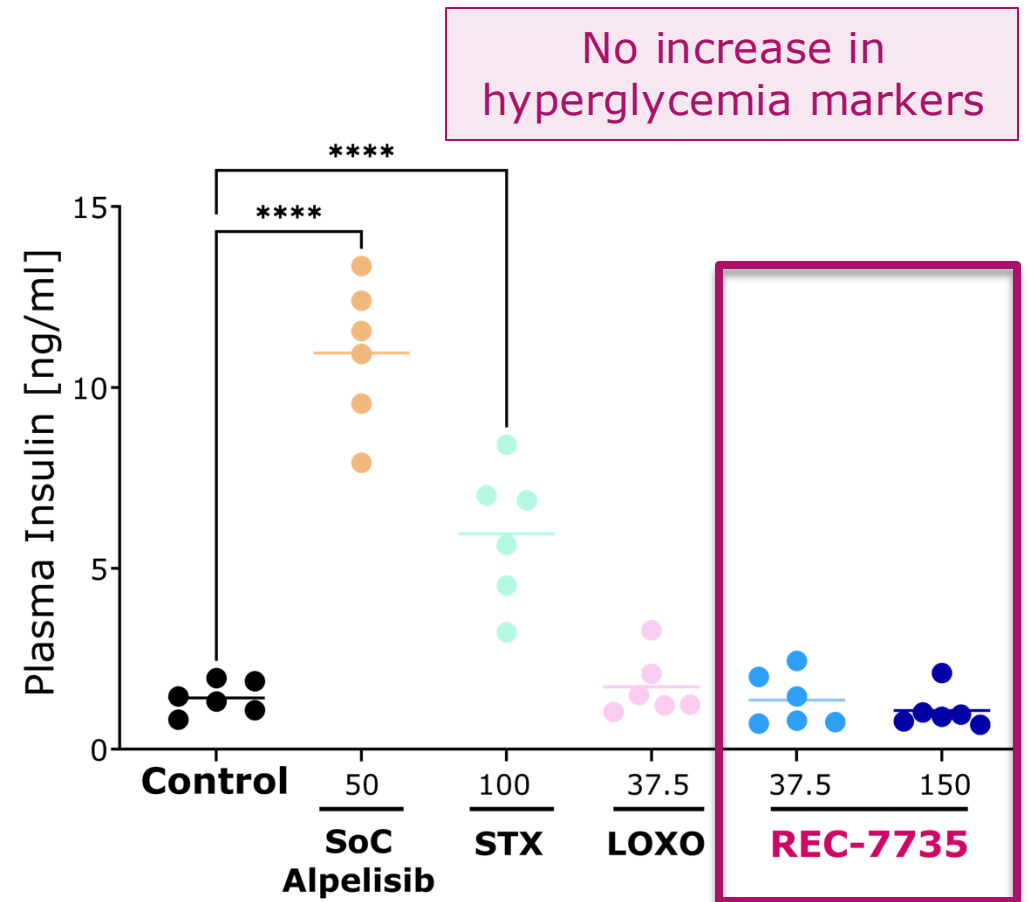
- **>100-fold better selectivity** against WT PI3K α , 10x-fold better selectivity vs. other WT sparing inhibitors
- **Limited hyperglycemia observed** in vivo at highest doses (vs. STX-478)
- In vivo efficacy **superior to SoC (alpelisib)** and comparable to STX-478
- **CNS penetrant** with low-risk of dose-limiting AEs

In vivo: REC-7735* demonstrates preliminary competitive efficacy with limited hyperglycemia versus SoC in preclinical models

CDX Model: PI3Ka H1047R¹



Naïve Wildtype Mice²



*Program is currently in candidate profiling / late lead optimization

Note: Alpelisib and STX (STX-478) were dosed QD; LOXO (LOXO-783) and REC-7735 were dosed BID. Doses are represented as mg/kg.

- In vivo CDX Model using T47D (PI3Ka H1047R mutant) cell line. n=10 mice per group. Data represents tumor growth inhibition and regression after 14 days of dosing. Pharmacokinetic (plasma and tumor) and pharmacodynamic (tumor pAKT) data were consistent with the observed tumor growth inhibition and regression.
- In vivo naïve wild-type, non-tumor bearing mice. n=6 mice per group. Data represents plasma insulin after 5 days of dosing. To note, plasma glucose and serum C-peptide, an inflammation marker, showed similar trends.

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
- Program update in 2H 2025

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}

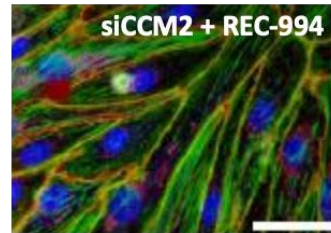
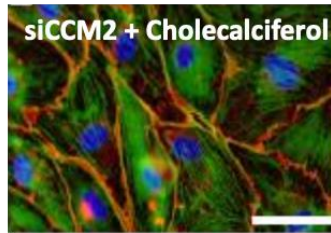
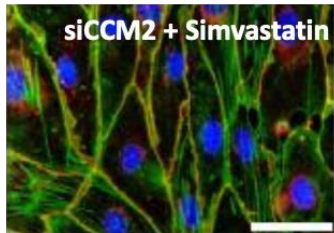
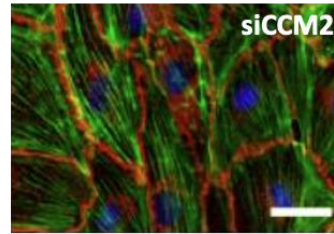
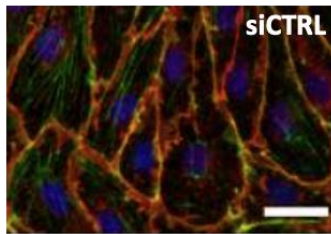
Recursion Approach

- **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

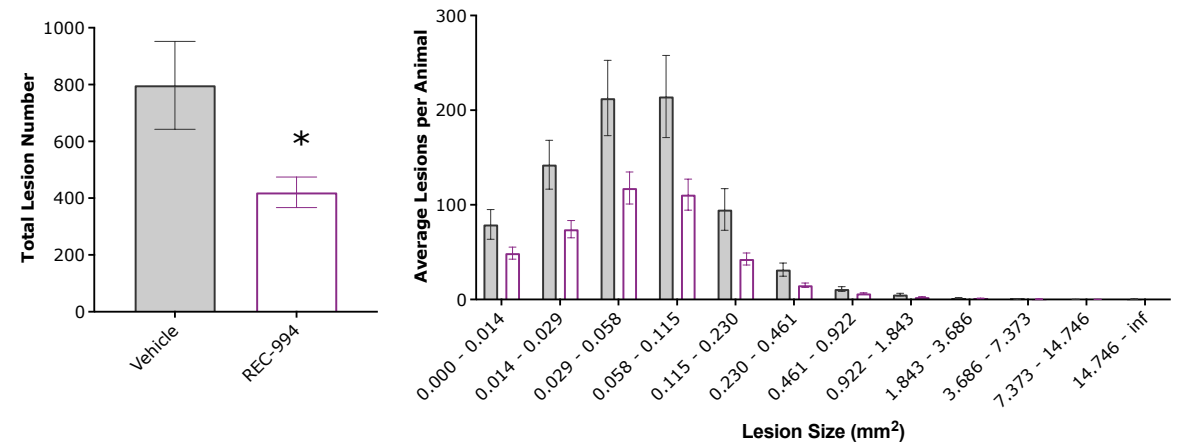
Identified REC-994 as potential rescue molecule in phenotype associated with CCM2 loss of function



Key Preclinical Data¹

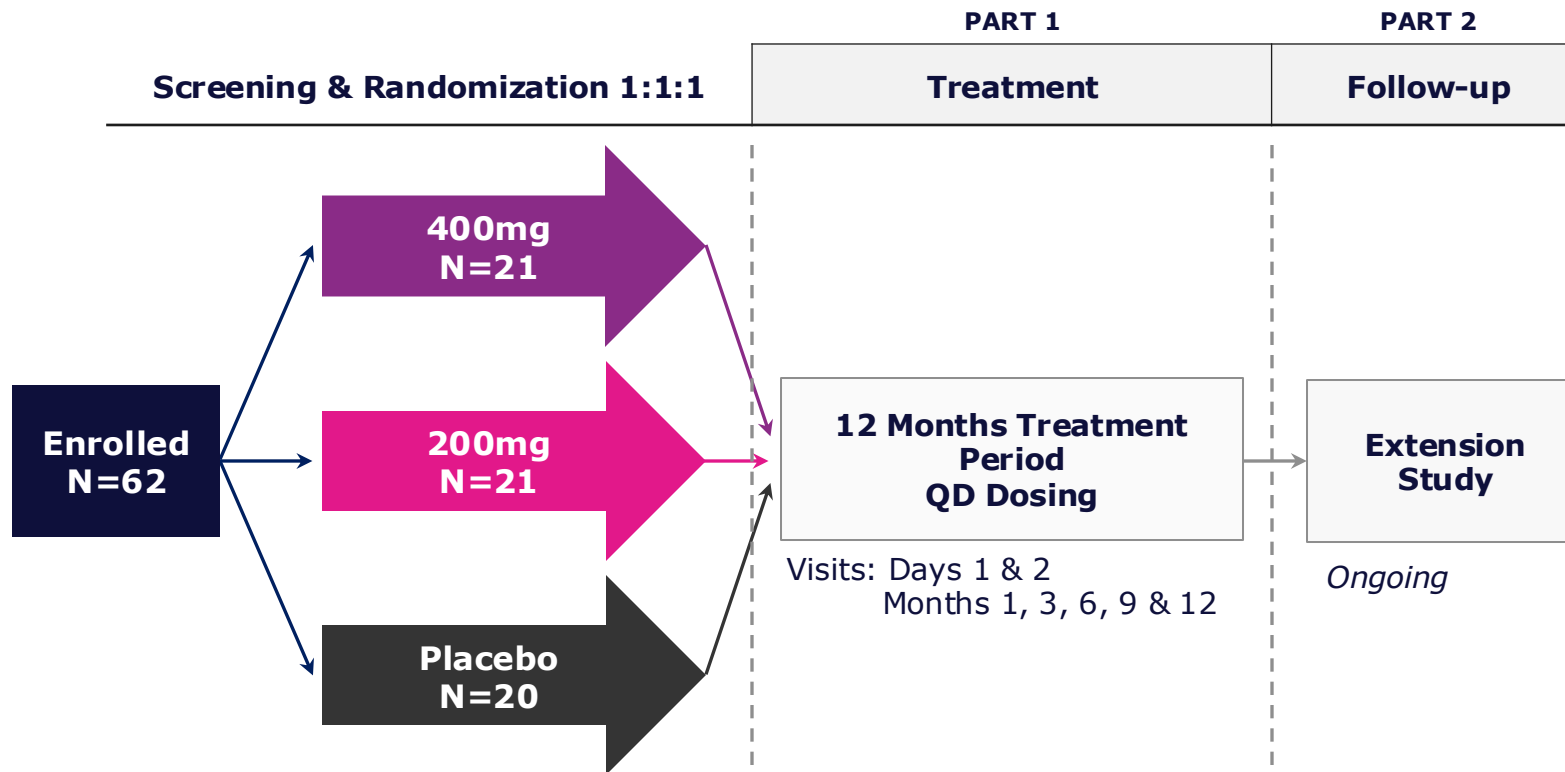
Reduces lesion number & size in *Ccm1* and *Ccm2*² loss of function (LOF) mouse models

Ccm1 LOF Model



REC-994 (Superoxide Scavenger): A randomized, double-blind, placebo-controlled phase 2 trial in symptomatic CCM patients

Sycamore 994-201



Enrollment Criteria

- MRI-confirmed CCM lesion(s)
- Familial or sporadic
- Symptoms directly related to CCM
- Modified Rankin score ≥ 1

Study Objectives

- **Primary:** Safety and tolerability
- **Secondary:** neurologic disability (mRS), Patient-reported outcomes, disease-associated symptoms, MRI-or CT-related hemorrhagic events, pharmacokinetics
- **Exploratory:** Lesion volume change, Hemosiderin ring changes, and Biomarkers

REC-994 (Superoxide Scavenger): Baseline characteristics were generally balanced between treatment arms

Characteristic	Placebo (N=20)	REC-994 200 mg (N=21)	REC-994 400 mg (N=21)	Total (N=62)
Age, years	47.1 (15.8)	46.6 (14.5)	48.9 (10.8)	47.5 (13.6)
At time of diagnosis, years	37.1 (15.2)	36.7 (20)	40.6 (14.6)	38.1 (16.6)
Sex, n (%)				
Female	14 (70.0)	9 (42.9)	16 (76.2)	39 (62.9)
Male	6 (30.0)	12 (57.1)	5 (23.8)	23 (37.1)
White race, n (%)	17 (85.0)	18 (85.7)	17 (81.0)	52 (83.9)
BMI, kg/m ²	28.1 (7.8)	27.6 (5.7)	27.1 (5.7)	27.6 (6.3)
CCM Disease Etiology, n (%)				
Familial	12 (60.0)	13 (61.9)	9 (42.9)	34 (54.8)
Sporadic	8 (40.0)	7 (33.3)	12 (57.1)	27 (43.5)
Unknown	-	-	-	1 (1.61)
Previous symptom related to CCM, n (%)				
Focal neurological deficit	15 (75.0)	15 (71.4)	13 (61.9)	43 (69.4)
Intracerebral hemorrhage	11 (55.0)	14 (66.7)	16 (76.2)	41 (66.1)
Headache	12 (60.0)	16 (76.2)	12 (57.1)	40 (64.5)
Epileptic seizure	5 (25.0)	4 (19.0)	6 (28.6)	15 (24.2)
Genetic mutations, n (%)				
CCM1 (KRIT1)	6 (30)	7 (33.3)	3 (14.3)	16 (25.8)
CCM2	3 (15)	5 (23.8)	5 (23.8)	13 (21)
CCM3 (PDCD10)	0	0	2 (9.5)	2 (3.2)

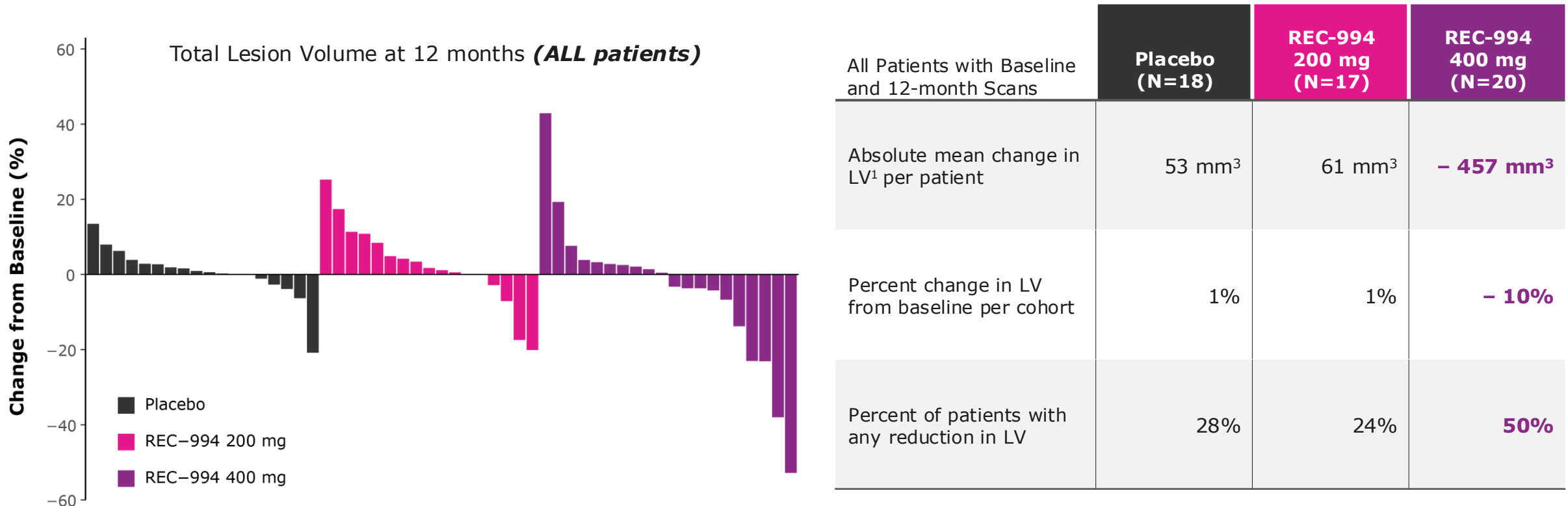
REC-994 (Superoxide Scavenger): No new safety signals observed, with incidence of adverse events comparable across arms

Event, n (%)	Placebo (N=20)	REC-994 200 mg (N=21)	REC-994 400 mg (N=21)	Total (N=62)
Any Treatment Emergent Adverse Event (TEAE)	17 (85.0)	18 (85.7)	15 (71.4)	50 (80.6)
TEAEs Grade ≥3	4 (20.0)	7 (33.3)	3 (14.3)	14 (22.6)
Any TEAE related to study drug¹	2 (10.0)	0	5 (23.8)	7 (11.3)
Grade ≥3 TEAE	0	0	0	0
Discontinuation due to TEAE	0	0	0	0
Dose interruption due to TEAE	0	0	0	0

- **Most common adverse** events reported in at least 10% of participants included:
 - Covid-19
 - Dizziness
 - Headache
 - Back pain
 - Constipation
- **No SAEs related** to study drug
- Majority of TEAEs were **Grade 1-2**
- **No treatment-related** adverse events that led to discontinuations

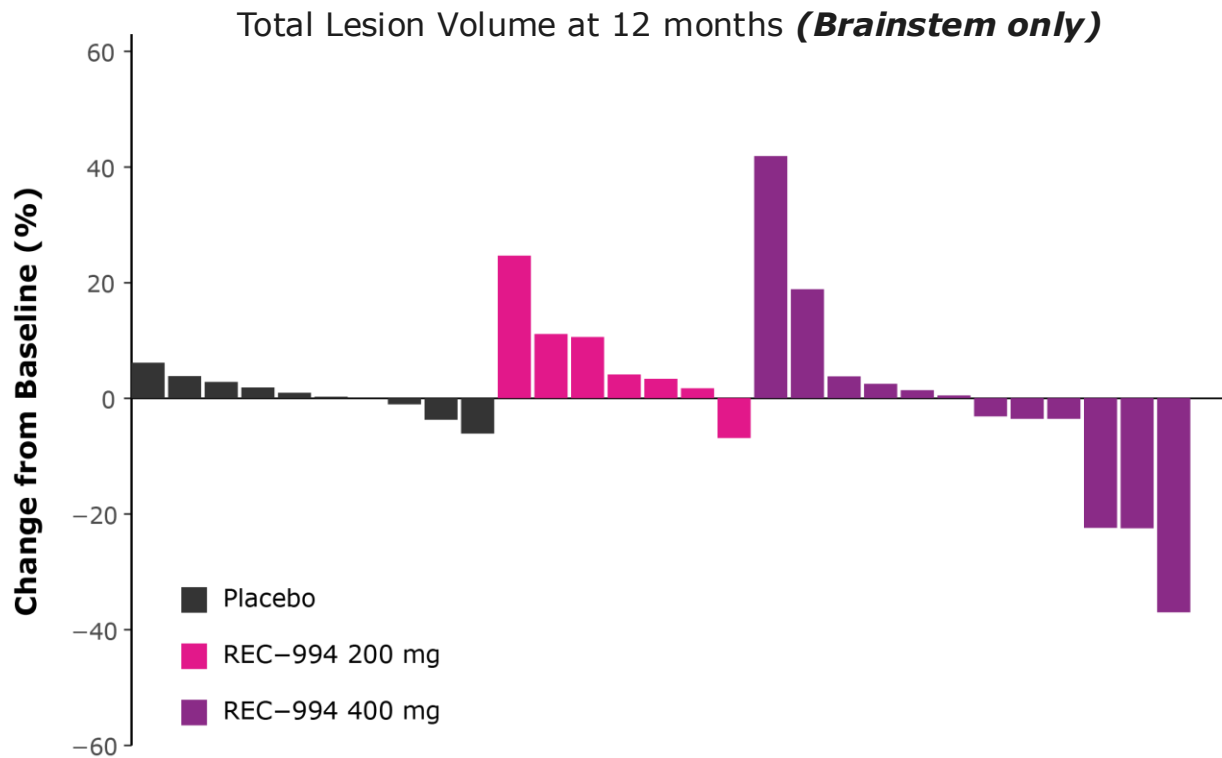
1. In the REC-400 mg arm these consisted of dizziness, rash, anemia, nausea and peripheral edema. In the placebo arm these consisted of dizziness and erythema multiforme. Across both arms, TEAEs related to study drug were Grade 1 or 2.

REC-994 (Superoxide Scavenger): 50% of patients on 400 mg achieved a reduction in total lesion volume (LV)



1. Exploratory analysis of change from baseline between treatment and placebo for change in lesion volume (LV) at month 12 for REC-994 200 mg (p=0.912) and REC-994 400 mg (p=0.089) assessed by mixed model for repeated measures (MMRM) analysis.

REC-994 (Superoxide Scavenger): 50% of patients on 400 mg achieved a reduction in total lesion volume (LV)



Brainstem only patients with Baseline and 12-month Scans	Placebo (N=10)	REC-994 200 mg (N=7)	REC-994 400 mg (N=12)
Absolute mean change in LV ¹ per patient	-23 mm ³	123 mm ³	- 256 mm³
Percent change in LV from baseline per cohort	-0.4%	7.5%	-5.6%
Percent of patients with any reduction in LV	27%	13%	50%

1. Exploratory analysis of change from baseline between treatment and placebo for change in lesion volume (LV) at month 12 for REC-994 200 mg ($p=0.987$) and REC-994 400 mg ($p=0.449$) assessed by mixed model for repeated measures (MMRM) analysis.

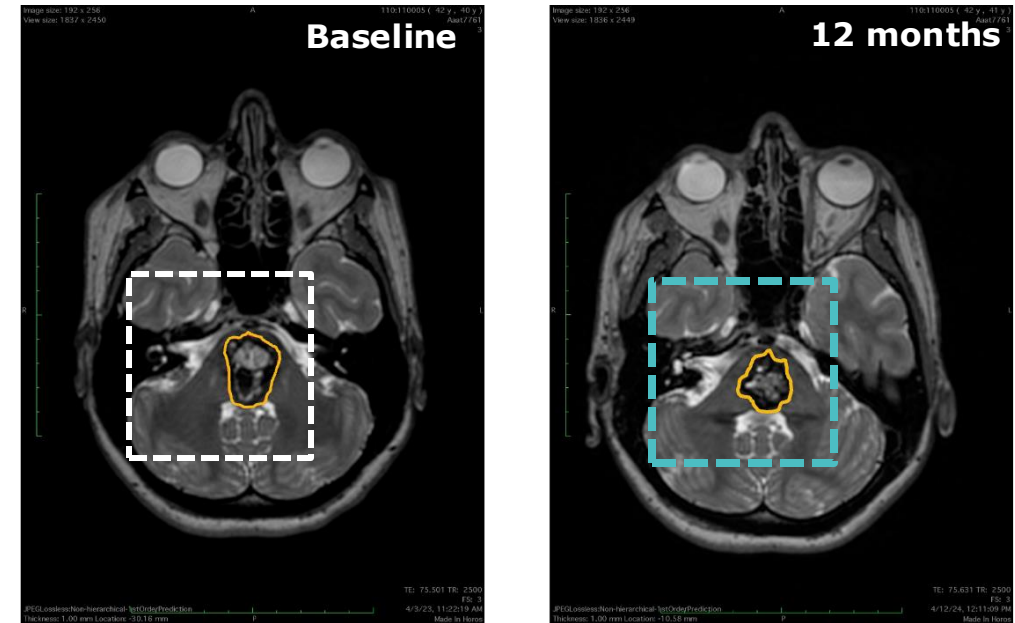
REC-994 (Superoxide Scavenger): Reductions in brainstem lesion volume and resolution in CCM-associated symptoms

- REC-994 was observed to be **safe and well tolerated** with **no treatment related > Grade 3** adverse events
- **REC-994 400 mg** treatment reduced lesion size and volume in CCM patients, including those with brainstem lesions
- REC-994 400 mg **improved clinical function** as seen by **modified Rankin score (mRS)**, in CCM patients, including those with brainstem lesions

Clinical Updates

- Long-term Extension (Part 2) **ongoing**
- All placebo patients in LTE have transitioned to **400 mg QD**
- Program update expected in **2H25**

39-year-old female diagnosed with a **sporadic CCM brainstem lesion**



- **~38% reduction** in baseline lesion volume (-2,630 mm³ absolute lesion volume change)
- **Complete resolution** of moderate/severe CCM symptoms including impaired mobility, headaches, hearing, and blurry vision symptoms

REC-4881: MEK1/2 Inhibitor

A highly selective and potent MEK1/2 inhibitor as chemoprevention for 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 ~7,000² **advanced duodenal polyposis** patients in the US at high-risk of developing cancer

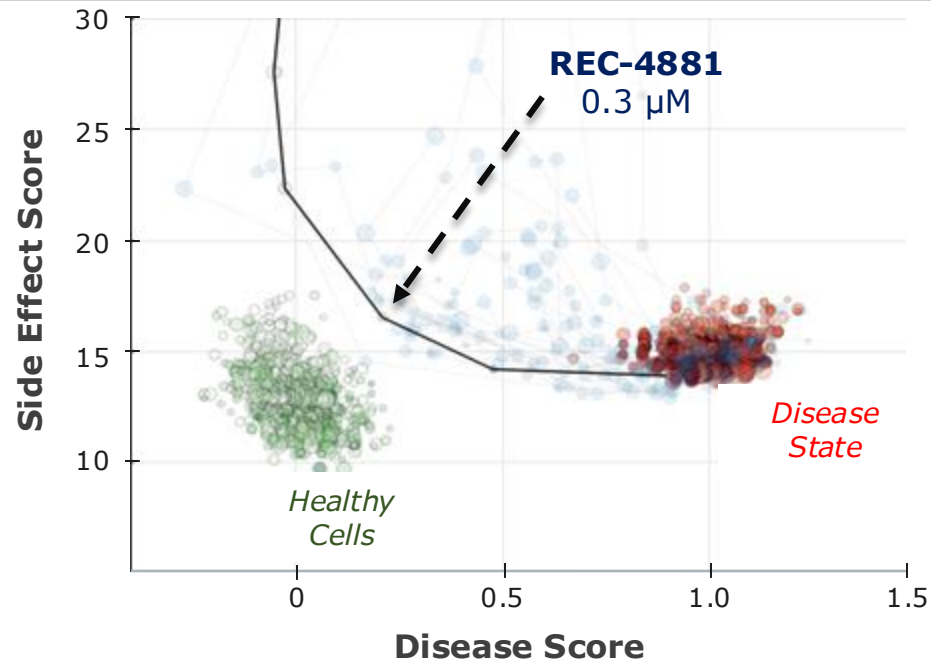
Recursion Approach

- **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

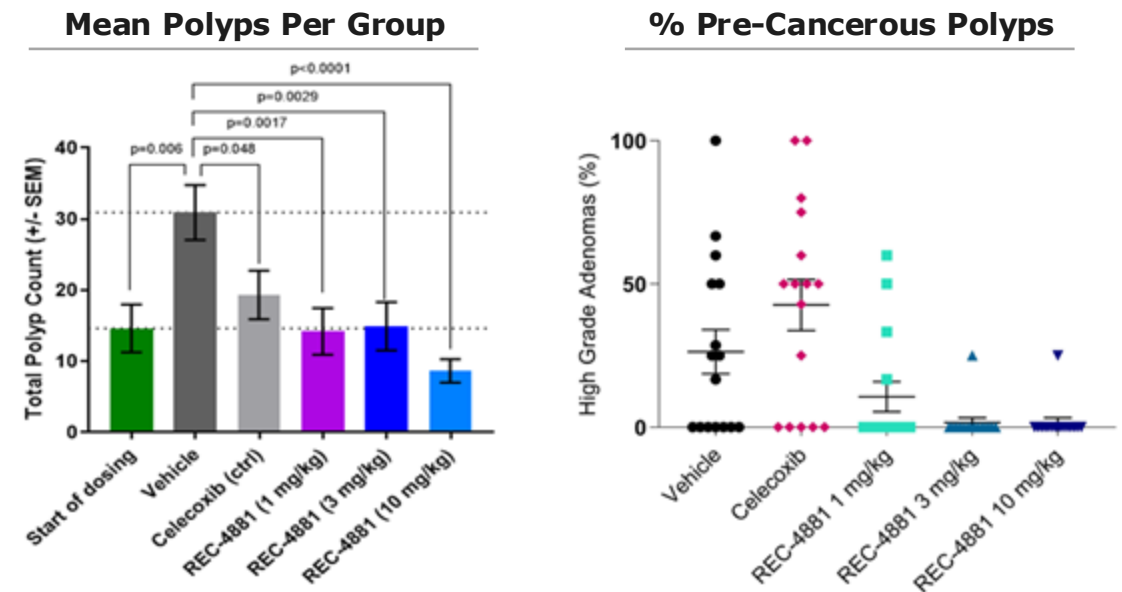
REC-4881 suppresses disease-inducing effects of APC mutations



- 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



TUPELO Phase 1b/2 study design

REC-4881 Phase 1b Dose Escalation (N≤12)

4mg

8mg

RP2D

Phase 2

Dose Expansion (N=30)

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
- Phase 1b/2 safety & early efficacy data expected in **1H25**

REV102: ENPP1 Inhibitor

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

Program Status

- Potential **first-in-class** and **best-in-class** ENPP1 inhibitor for the treatment of patients with HPP
- IND enabling studies expected to initiate in **2025**

Mechanism of Action

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

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**

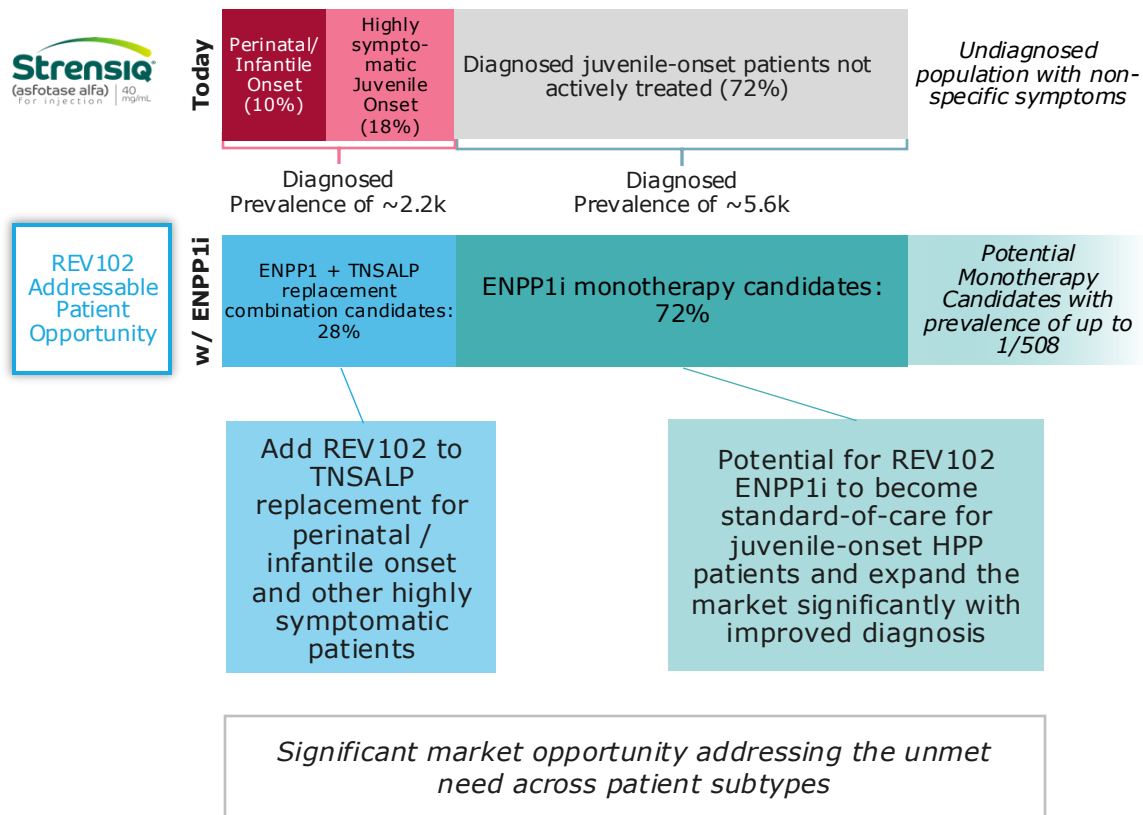
Recursion Approach²

- **Precision designed for both high potency** and a lifetime of **chronic dosing**
- **Structurally distinct** differences vs competitor ENPP1 inhibitors
- **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

Market Opportunity¹

Estimated **diagnosed prevalence** hypophosphatasia patients in **US**



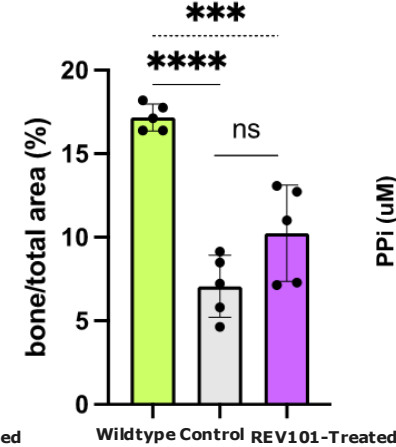
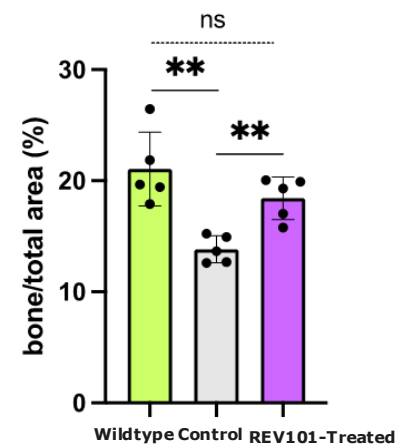
Key Preclinical Data²

Bone morphometry

2D analysis of trabecular bones

L3 Vertebrae
(n=5, females)

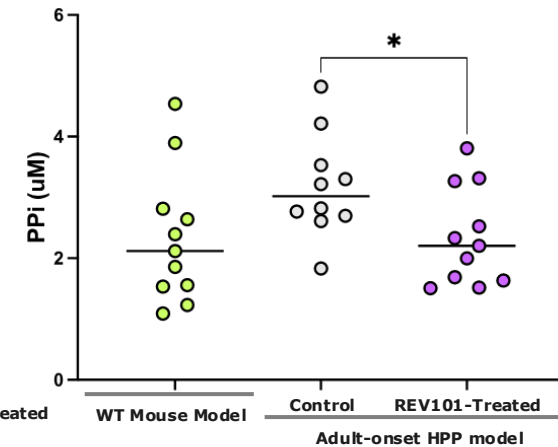
Distal Femur
(n=5, males)



Plasma levels of PPI

After 100-day dosing

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

- **IND-enabling** studies ongoing

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

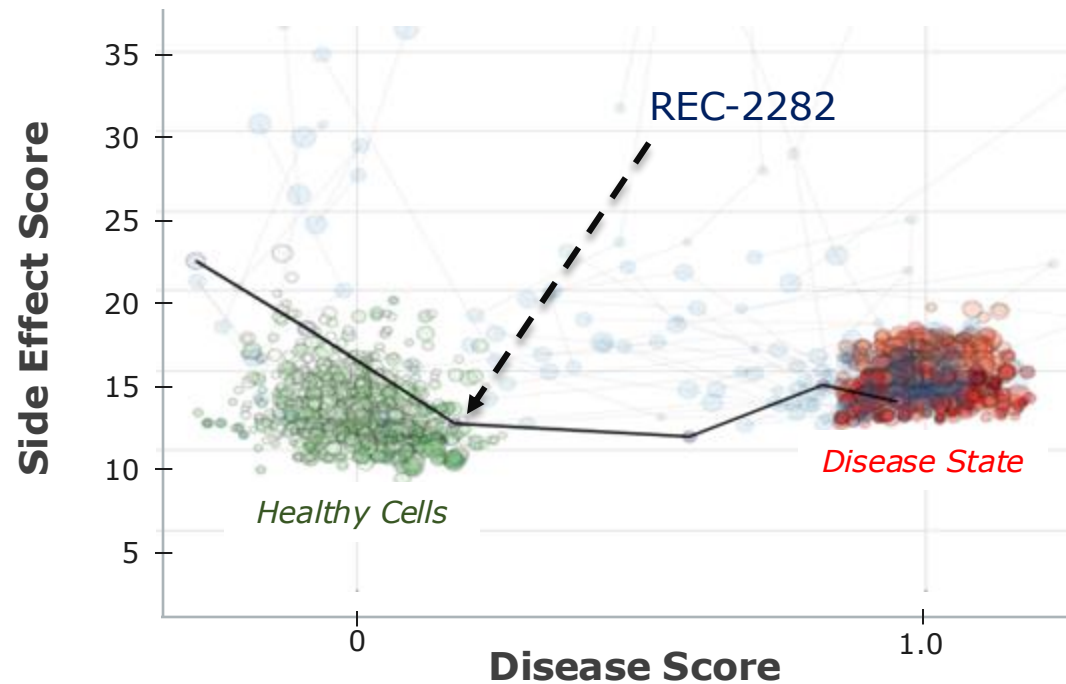
Recursion Approach

- 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

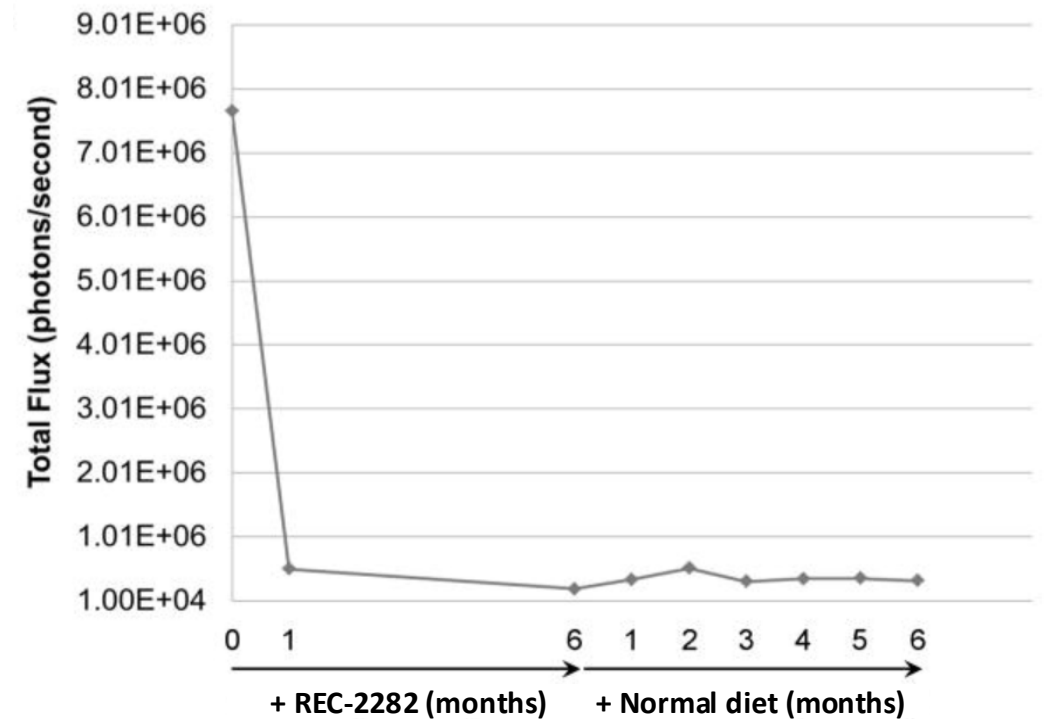
Recursion OS Insight

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



POPLAR Phase 2/3 study design

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

24 Patients
Allocated 1:1

40 mg

60 mg

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 PFS **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**

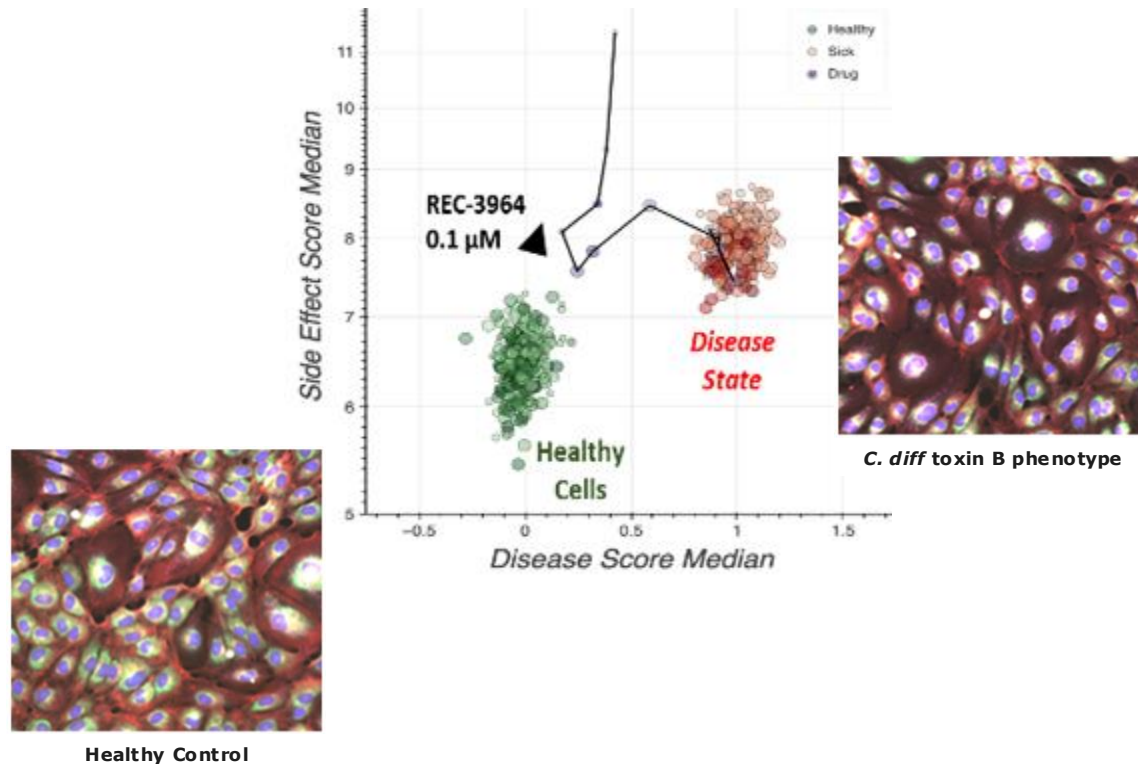
Recursion Approach

- 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

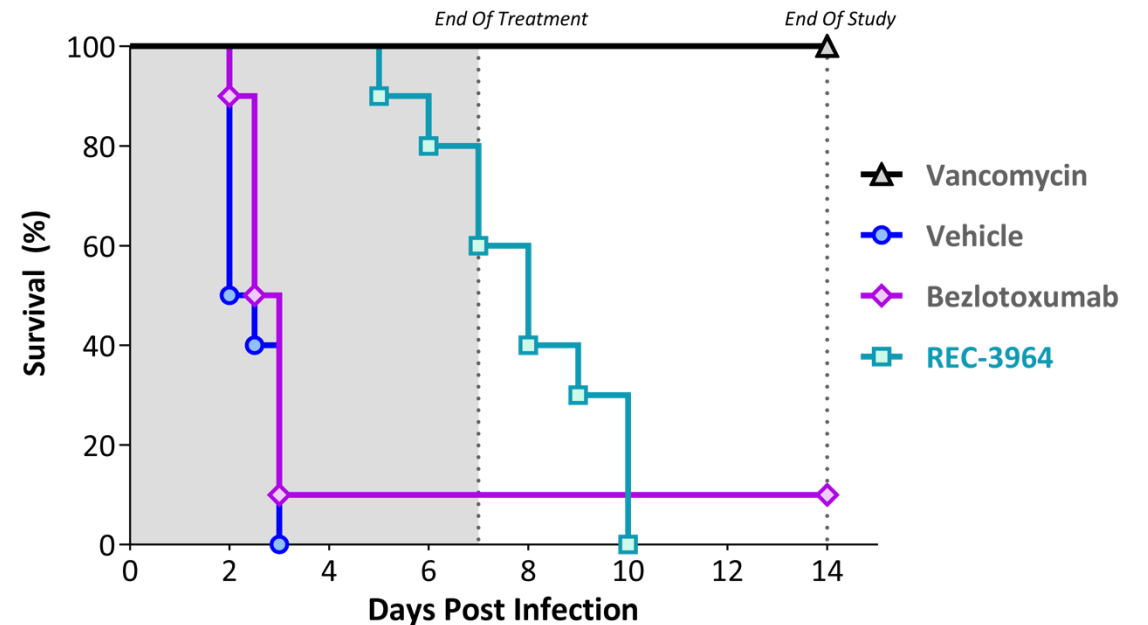
Recursion OS Insight

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)



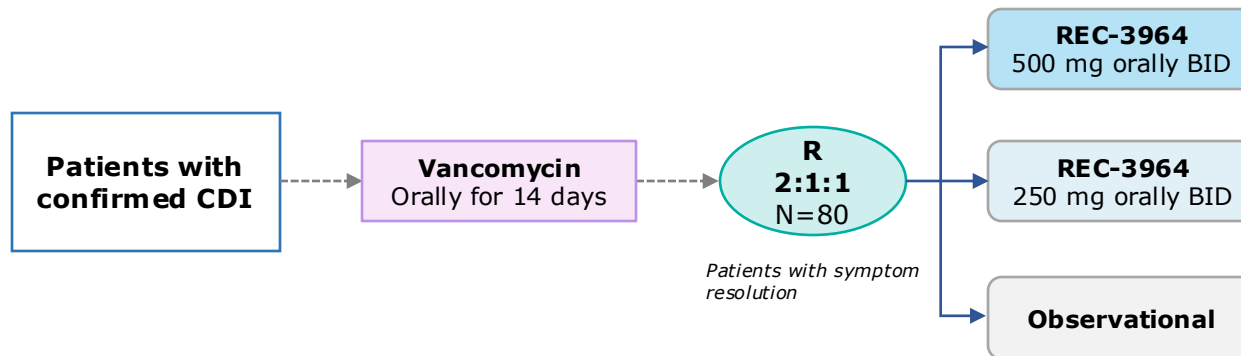
REC-3964 (CDI TcdB Inhibitor): Study Design and Next Steps

Development Strategy



ALDER Phase 2 study design

REC-3964 Phase 2 Open-label, 2-arm study



Key Eligibility

- High-risk of CDI
- ≥ 3 bowel movements in 24 hours
- Confirm CDI using EIA (toxin)
- No fulminant CDI
- No history of chronic diarrheal illness due to other causes

Primary Endpoint:

- Rate of recurrence

Secondary Endpoint:

- Additional efficacy measures
- Safety / tolerability
- PK

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**
- Phase 2 update expected in **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- β

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

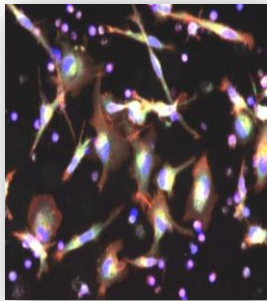
Recursion Approach

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

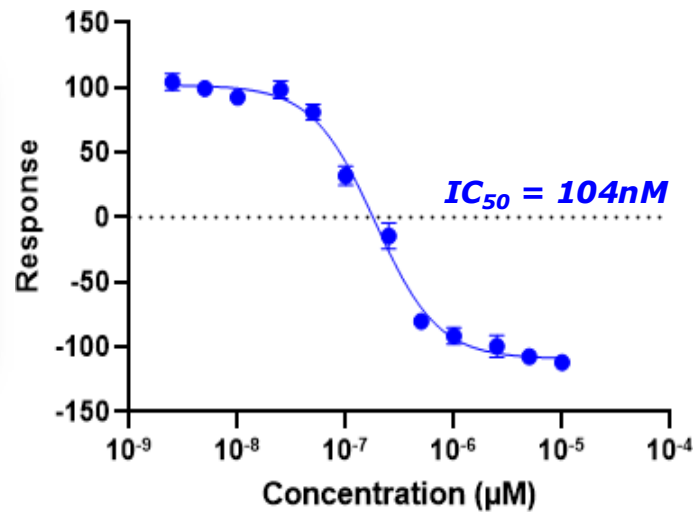
REC-4209 (Target Epsilon): Identified as a novel mechanism in Recursion's screen with compelling preclinical efficacy demonstrated in bleomycin lung fibrosis mouse model

Recursion OS Insights¹

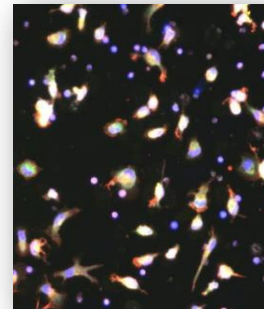
Disease State
(Fibrocytes)



In Vitro Fibrocyte Rescue Assay

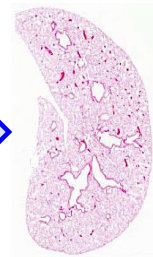


Control State
(Pentraxin-2)



Fibrotic Lung

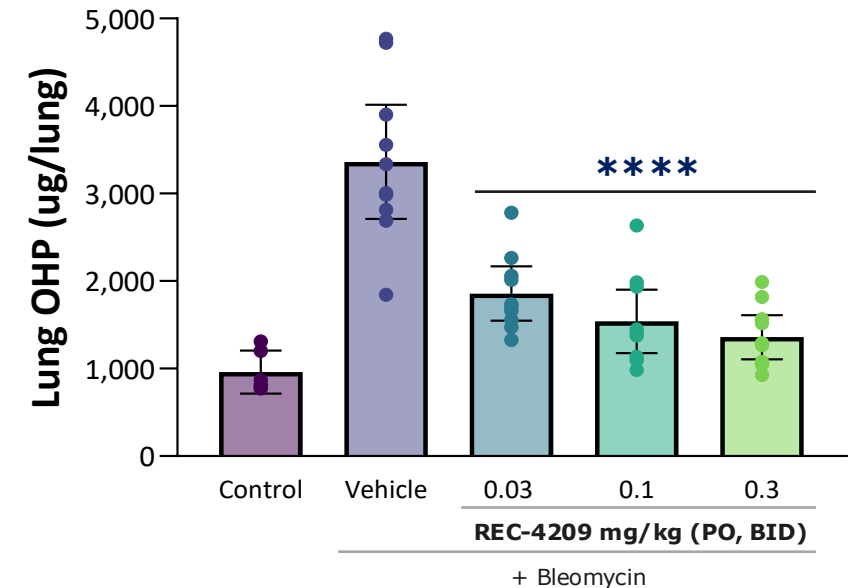
+ REC-4209



Normal Lung

Key Preclinical Data²

Total Lung Collagen



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

What's Next

- IND-enabling studies ongoing**

1. Data on File.

2. Groups (n=10 per group; n=6 in control) compared against Vehicle. ****p<0.0001; one-way ANOVA with Tukey's multiple comparison test. Data reflects mean ± 95% CI.





APPENDIX

Partnerships & Data Strategy Details



Exciting scientific collaborations span biopharma, tech & data


Therapeutic discovery partnerships


 <p>Announced Dec. 2021</p>	<ul style="list-style-type: none">• 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	 <p>Announced Jan. 2022</p>	<ul style="list-style-type: none">• \$100M upfront with the potential of \$5.2B in total milestones plus high-single digit to mid-teen tiered royalties• 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
 <p>Announced Sept. 2020 Updated Nov. 2023</p>	<ul style="list-style-type: none">• \$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	 <p>Announced Sept. 2023</p>	<ul style="list-style-type: none">• \$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

 <p>NVIDIA</p> <p>Announced July 2023</p>	<ul style="list-style-type: none">• \$50M equity investment• Partnership on advanced computation (e.g., foundation model development)• Priority access to compute hardware or DGXCloud Resources• BioHive-2: helped design and build next generation supercomputer
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 <p>Google Cloud</p> <p>Announced Oct. 2024</p>	<ul style="list-style-type: none">• 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
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Real-world data access

<p>TEMPUS</p> <p>Announced Nov. 2023</p>	<ul style="list-style-type: none">• Preferential access to >20 PBs of real-world, multi-modal 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
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 <p>Announced May 2024</p>	<ul style="list-style-type: none">• 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
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Cheminformatics and chemical synthesis

 <p>Announced Dec. 2023</p>	<ul style="list-style-type: none">• Utilizes Recursion's predicted protein-ligand interactions for ~36B compounds from Enamine's REAL Library• Aim to generate enriched screening libraries & co-brand customer offerings
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