Recursion

Decoding Biology To Radically Improve Lives

NOVEMBER 2024

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Other important factors and information are contained in 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 and 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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Post-Combination portfolio poised for value creation from a unified, AI-powered Operating System

~10
Clinical and pre-clinical programs[1]

Oncology, Rare diseases, and other High Unmet Need Diseases

~10
Clinical program milestones over the next 18 months[2]

~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

Roche Genentech
sanofi
BAYER
Merck KGaA
Darmstadt, Germany

1. Includes preclinical programs (programs expected to enter the clinic within the next 18 months).
2. Program milestones includes data readouts, preliminary data updates, regulatory submissions, trial initiation, etc.

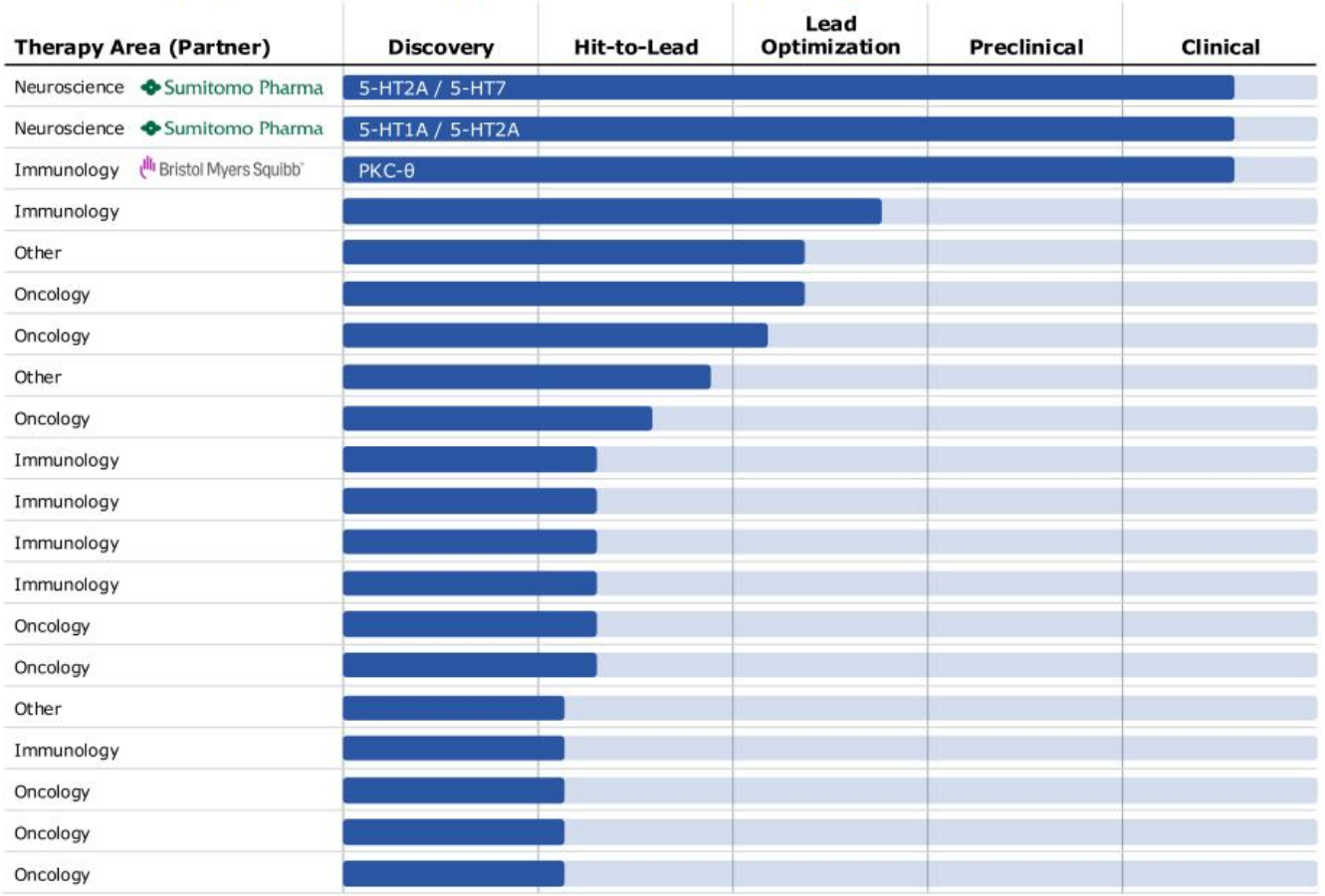
Pipeline of ~**10 clinical and preclinical** technology-enabled programs

	Candidate	Target	Indication	Preclinical	IND-Enabling	Phase 1/2	Pivotal / Phase 3	Next Anticipated Milestone
ONCOLOGY	REC-617[1]	**CDK7**	Advanced Solid Tumors[2]	*ELUCIDATE*				• Initial Ph1 monotherapy safety & PK / PD data expected on Dec 9th, 2024
ONCOLOGY	REC-1245	**RBM39**	Biomarker-Enriched Solid Tumors & Lymphoma	*DAHLIA*				• Ph 1 update on dose-escalation expected in H1, 2026
ONCOLOGY	REC-3565[3]	**MALT1**	B-Cell Malignancies	*EXCELERIZE*				• Ph 1 FPD expected in Q1, 2025
ONCOLOGY	REC-4539[4]	**LSD1**	Small-Cell Lung Cancer (SCLC)					• Ph 1 FPD expected in H1, 2025
RARE	REC-994	**Superoxide**	Cerebral Cavernous Malformations (CCM)	*SYCAMORE*				• Ph 2 data to be shared via a congress / publication / webinar in H1, 2025 • Regulatory update by H2, 2025
RARE	REC-4881	**MEK1/2**	Familial Adenomatous Polyposis (FAP)	*TUPELO*				• Ph 2 safety / early efficacy data expected in H1, 2025
RARE	REC-2282	**HDAC**	Neurofibromatosis Type 2 (NF2)	*POPLAR*				• PFS maturing – PFS6 futility analysis anticipated in H1, 2025
RARE	REV102[5]	**ENPP1**	Hypophosphatasia (HPP)					• Development candidate nomination expected in Q4, 2024
OTHER	REC-3964	**TcdB**	Prevention of Recurrent *C. difficile* (rCDI)	*ALDER*				• Ph 2 update expected in Q1, 2026
OTHER	REC-4209	**Undisclosed**	Idiopathic Pulmonary Fibrosis (IPF)					• IND-enabling studies ongoing
	~10 advanced discovery programs							

REC-4881 in APC/AXIN1 indications has been deprioritized as part of a disciplined, strategic portfolio prioritization as part of the integration

1. Formerly GTAEXS617 2. Includes non-small cell lung cancer (NSCLC), colorectal cancer, breast cancer, pancreatic cancer, ovarian cancer, head and neck cancer
3. Formerly EXS73565 4. Formerly EXS74539 5. Joint venture with Rallybio

Robust pipeline of partnered programs



Partners

Roche Genentech

sanofi

BAYER

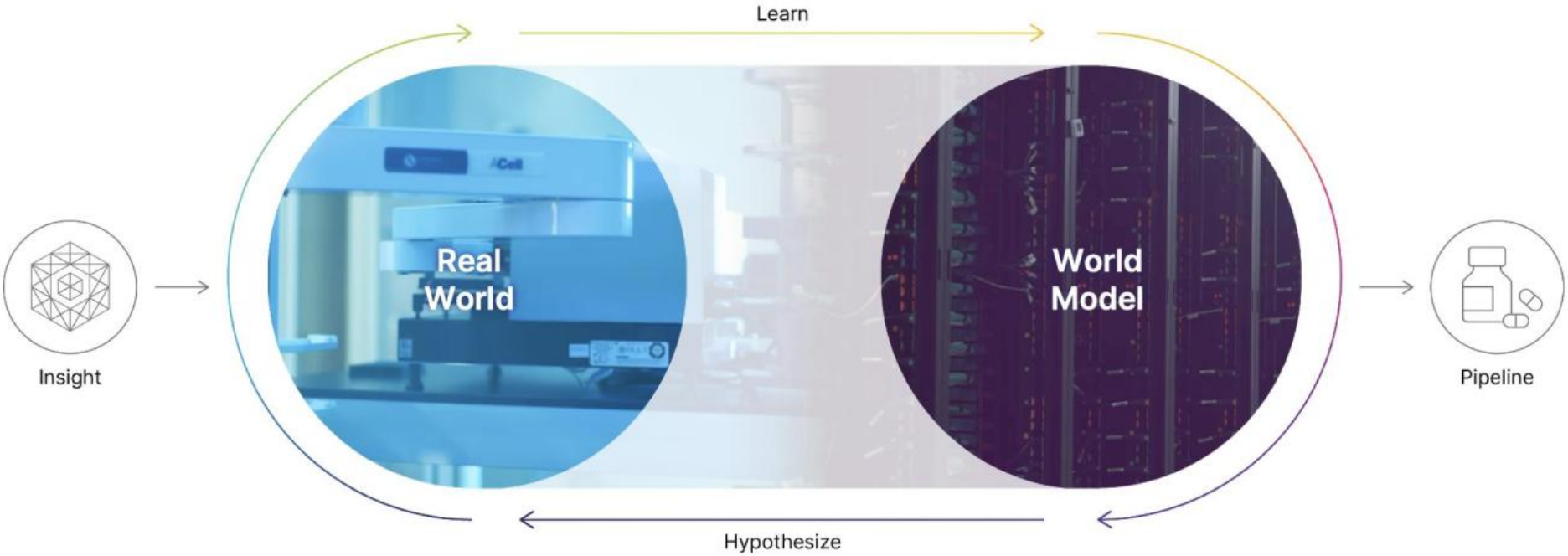
Merck KGaA
Darmstadt, Germany

Bill & Melinda Gates foundation

Note: Respective partner and disclosed target are noted for each clinical program
*Bill & Melinda Gates Foundation (BMGF) is a funder of anti-infective programs

Recursion

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



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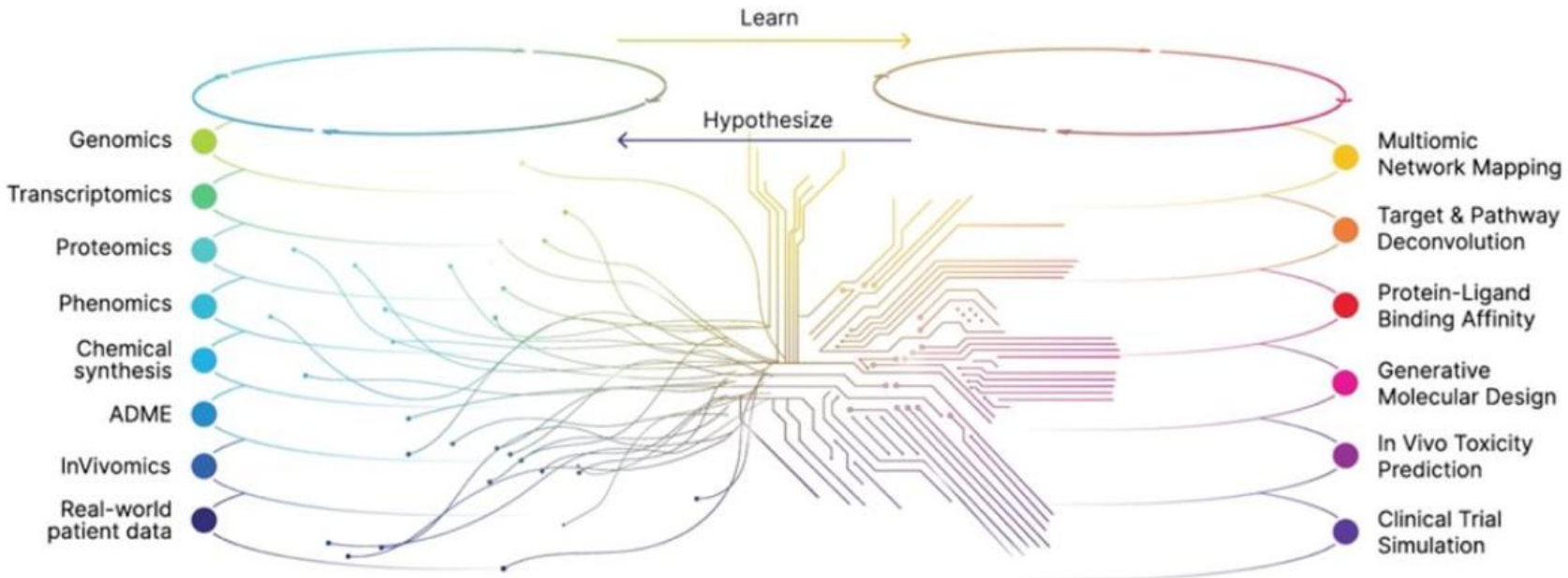

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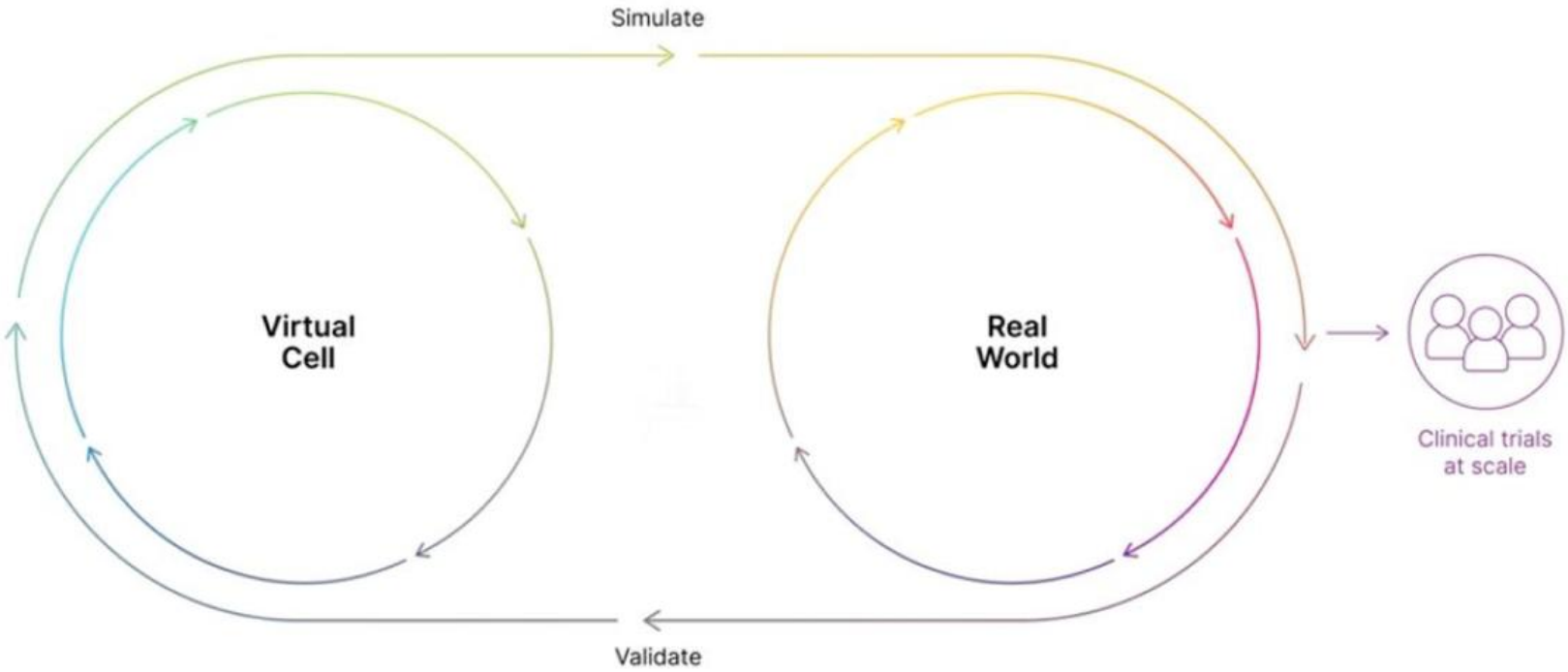


Recursion

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

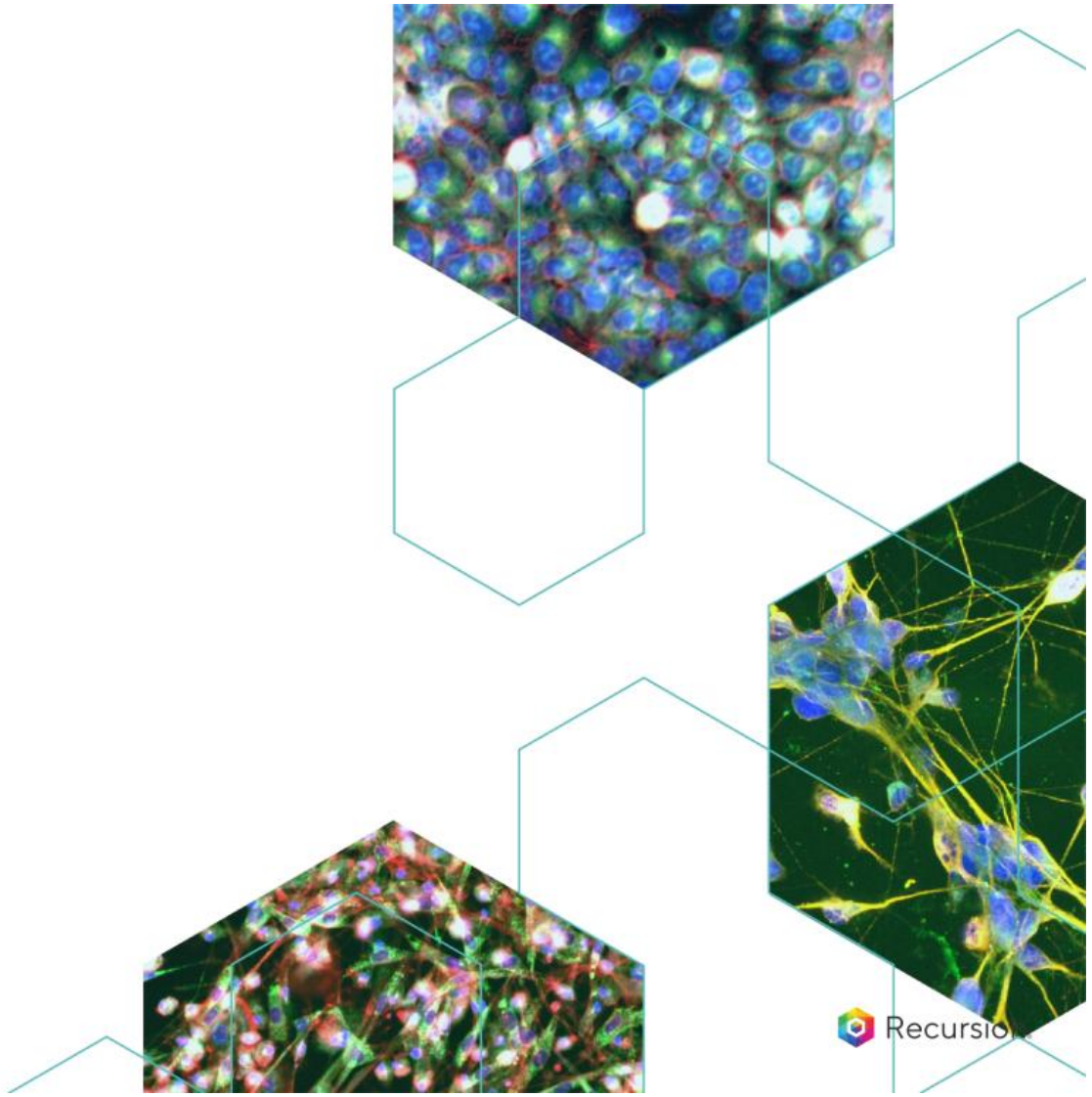
Direct generation of new data internally **to maximize pipeline and partnership value-drivers**

- Licensing
- Augment Recursion OS
- LOWE

Recursion

VALUE CREATION

Pipeline



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Recursion

PIPELINE

Oncology

Recursion

ONCOLOGY

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

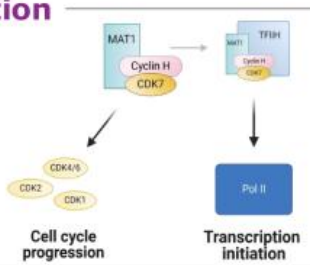
Unmet Need

- **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 + EU[1]

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	H2 2025
Phase 1 *Combo (SERD)* Dose Escalation	**Phase 2** *Combination* Dose Expansion

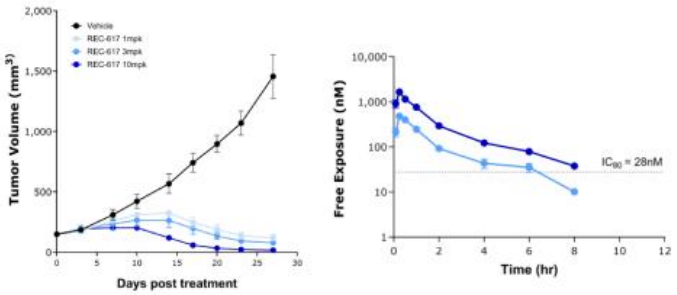
Differentiation

- 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 Preclinical Data

- REC-617 demonstrates **potent tumor regression** with <10 hours of exposure above IC_{80} **to optimize benefit-risk**

CDX Model: OVCAR3[2] | **Mouse PK**[3]



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

- Initial Phase 1 monotherapy safety, PK/PD update expected at **AACR Special Conference in Cancer Research on December 9th**

* Formerly GTAEXS617

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 IC_{80} results in significant body weight loss

Recursion

ONCOLOGY

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

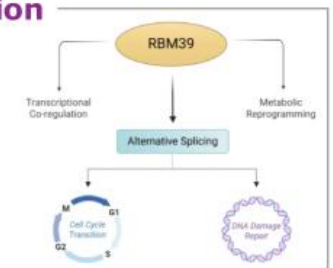
Unmet Need

- 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 + EU[1]

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

CURRENT STAGE

DAHLIA

Phase 1
Monotherapy
Dose Escalation

EXPECTED STAGES

Phase 1
Monotherapy
Dose Confirmation

Phase 2
Monotherapy
Dose Expansion

Differentiation

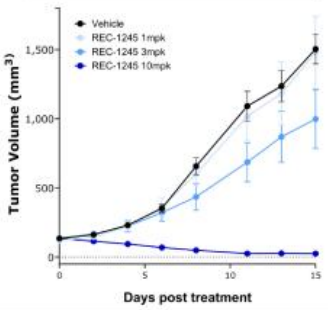
- Potential **First-in-Class** RBM39 Degrader
- **No significant** in vitro safety concerns (hERG, CEREP)

Highly Potent & Selective | Minimal Off-Target Liabilities | Biomarker Defined Population

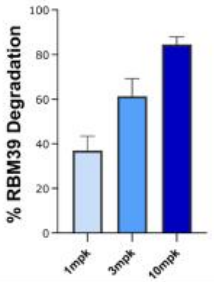
Key Preclinical Data

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

CDX Model: OVK18[2]



PD: Target Engagement[3]



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

- Ph 1 initiation expected in **Q4 2024**
- Ph 1 update in dose-escalation expected in **H1 2026**

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

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ONCOLOGY

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

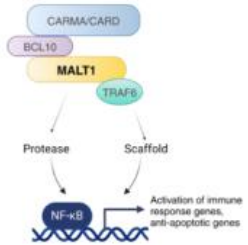
Unmet Need

- 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 + EU5[1]

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

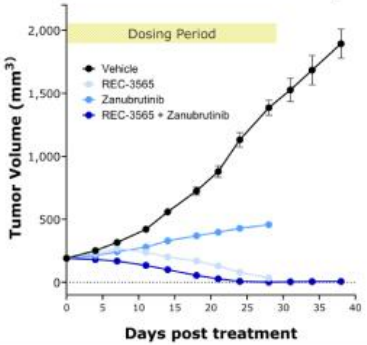
Differentiation

- 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-Ly10[2]

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 **Q1 2025**

* Formerly EXS73565.
1. Cerner Enviza Treatment Architecture Reports 2023, rounded to nearest 1,000 patients per year. 2. Payne et al. ENA, (2024)

Recursion

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

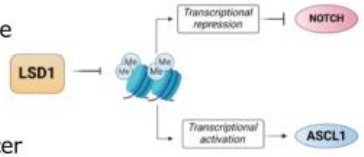
Unmet Need

- **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 BiTEs[2]

>45,000
Treatable US + EU5[1]

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

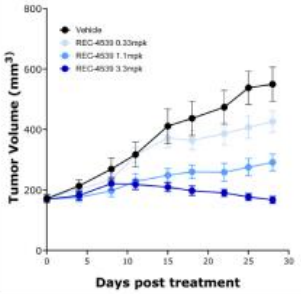
Differentiation

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

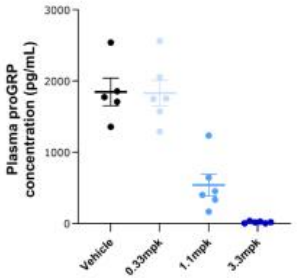
Key Preclinical Data

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

CDX Model: H1417[3]



Plasma ProGRP[4]



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 **H1 2025**

* Formerly EXS74539.

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

Recursion

PIPELINE

Rare disease

Recursion

RARE DISEASE

Cerebral Cavernous Malformation (Superoxide Scavenger): REC-994

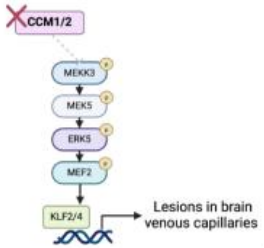
Unmet Need

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

~360,000
Symptomatic US + EU5[1]

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

CURRENT STAGE

SYCAMORE

Phase 2
LTE ongoing

EXPECTED STAGES

H2 2025

FDA mtg → **Phase 2b/3**
Contingent on FDA feedback

Differentiation

- Potential **First-in-Disease** oral therapeutic for CCM
- **No TEAEs** leading to discontinuation up to **800 mg** in Ph 1[3]

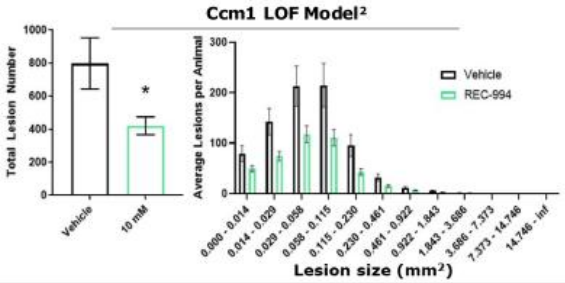
Safe and well-tolerated MOA

High oral bioavailability

Encouraging Ph 2 efficacy trends

Key Preclinical Data

- **Reduces lesion number & size** in LOF mouse models
- Phase 2 **primary endpoint** of safety and tolerability met
- **Phase 2 encouraging trends in lesion volume reduction** consistent with *in vivo* POC



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

- **Phase 2** data expected to be shared at an upcoming medical congress / publication/webinar in **H1 2025**
- **FDA guidance** expected in **H2 2025**

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

Recursion

RARE DISEASE

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

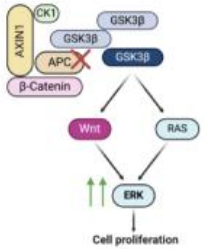
Unmet Need

- **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 + EU5[1]

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

CURRENT STAGE

TUPELO

Phase 2
Dose escalation

EXPECTED STAGES

H1 2025
Futility analysis

H2 2025
Phase 2
Dose Expansion

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

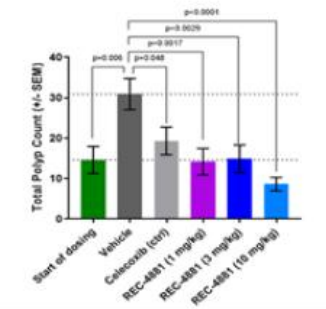
Validated target

Preferential GI exposure

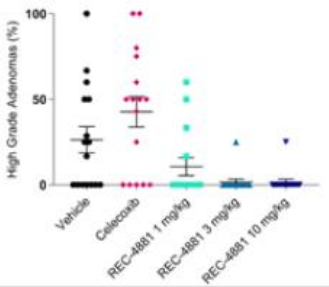
Key Preclinical Data[2]

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

Mean Polyps Per Group[2]



% Pre-Cancerous Polyps[2]



Recursion Approach

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

FTD	ODD
In US	In US + EU

What's Next

- **Futility analysis** for reduction in polyp burden expected **in H1 2025**

1. Prevalence for adult and pediatric population, Internal company estimates. 2. Data on file
FTD: Fast Track Designation; ODD: Orphan Drug Designation

Recursion

RARE DISEASE

Hypophosphatasia (ENPP1 Inhibitor): REV102

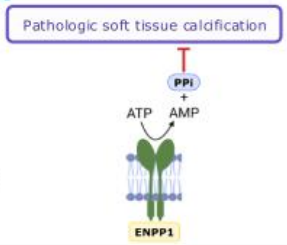
Unmet Need

- 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 + EU5[1]

Mechanism of Action

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



Development Strategy

EXPECTED STAGES

2025	2026
IND-Enabling Studies	**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)

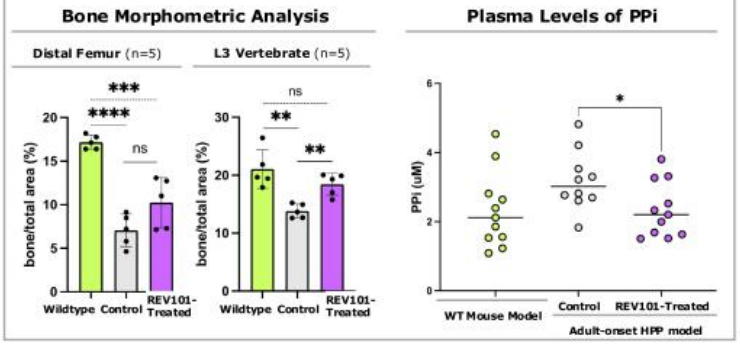
No significant in vitro safety concerns

Affordable oral treatment option

Potential as mono or combo therapy

Key Preclinical Data[2]

- Improved in mineralization in mouse models of HPP
- Significantly reduced PPi levels to that of wild-type mice



Recursion Approach[3]

- **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

- **Development candidate nomination** expected in Q4 2024

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

Recursion

RARE DISEASE

Neurofibromatosis Type 2 (HDAC Inhibitor): REC-2282

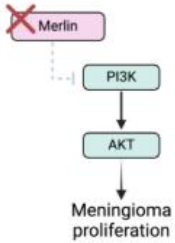
Unmet Need

- **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 + EU5[1]

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



Development Strategy

CURRENT STAGE

POPLAR

Phase 2
NF2 meningioma
2-arm study

EXPECTED STAGES

H1 2025

Futility analysis

Differentiation

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

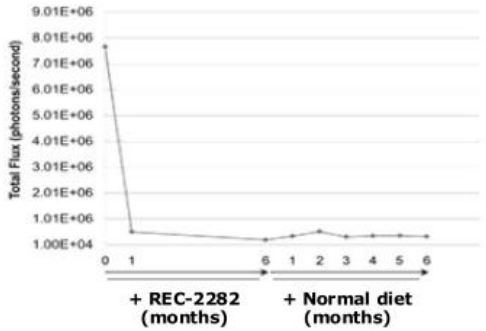
High oral bioavailability

Improved CNS penetration

Reduced off-target effects

Key Preclinical Data

- **Prevents growth & regrowth** of NF2-deficient meningioma model in mice[3]



Recursion Approach

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

FTD	ODD
In US	In US + EU

What's Next

- **Phase 2 PFS data maturing**
- Futility analysis (PFS6) expected in **H1 2025**

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

Recursion

PIPELINE

Other areas of high unmet need

Recursion

OTHER

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

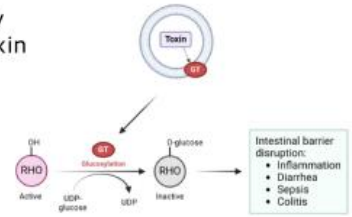
Unmet Need

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

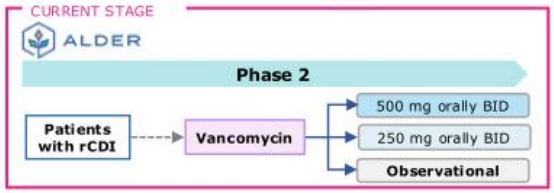
~175,000
Recurrent *C. diff* cases US[1]

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

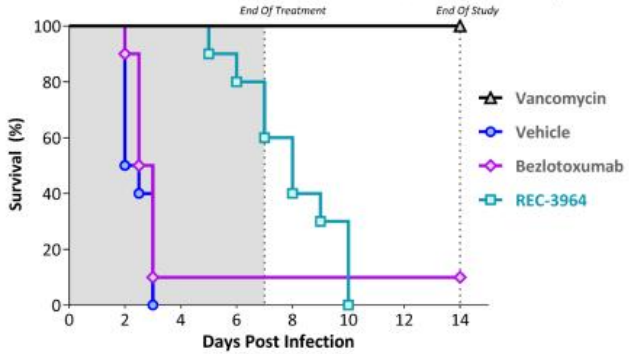
Safe and well-tolerated MOA

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

- **First Patient Dosed in the Phase 2** ALDER trial expected in Q4 2024
- Phase 2 update expected in **Q1 2026**

1. Incidence of addressable US cases of recurrent CDI, Shields et al., Anaerobe (2016). 2. N=10 hamsters per group. *C. difficile* strain 630, Data on File

Recursion

OTHER

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 US[1]

Mechanism of Action

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

Development Strategy

CURRENT STAGE

2024

IND-Enabling Studies

EXPECTED STAGES

2025

Phase 1

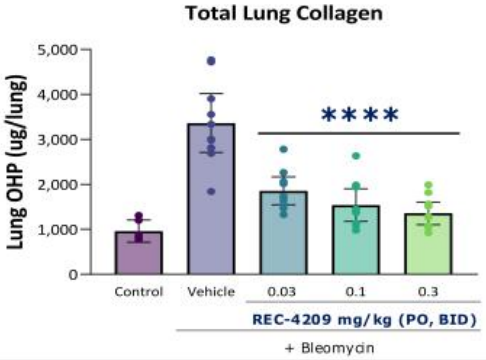
Healthy Volunteers

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

Key Preclinical Data

- REC-4209 at low doses reduces total lung collagen by 45% to 60% versus vehicle mice[2]



Recursion Approach

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

204

Novel compounds synthesized to candidate ID

What's Next

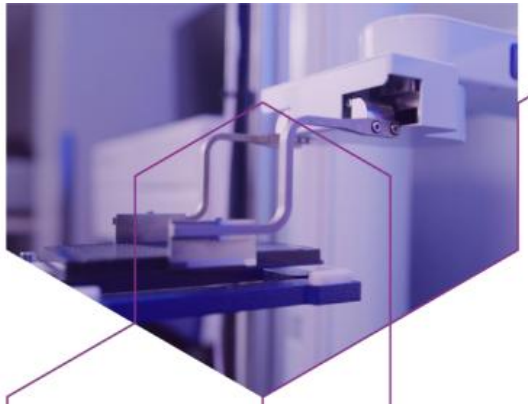
- **IND-enabling studies ongoing**

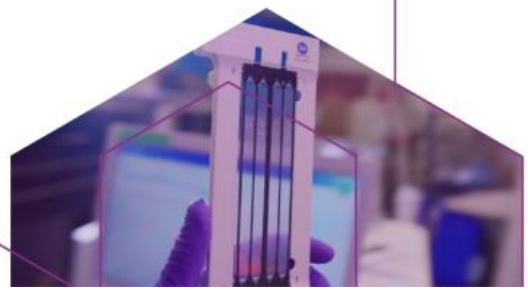
1. Global Data, Internal company estimates on IPF prevalence, Collard et al., Chest (2014).
2. Groups compared against Vehicle. ****p<0.0001; one-way ANOVA with Tukey's multiple comparison test. Data reflects mean ± 95% CI

Recursion

VALUE CREATION

Partnerships & Data Strategy





Recursion

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

Recursion

Partnerships with approximately $450M[1] earned to date and potential to receive more than $20B[2] in additional milestones

Recursion OS

Roche / Genentech (A Member of the Roche Group)

NEUROSCIENCE & ONCOLOGY

$150M upfront and up to 40 programs in neuroscience and one indication in gastrointestinal cancer, each of which could yield more than $300M in development, commercialization and net sales milestones with mid-to high-single digit tiered royalties on net sales

sanofi

ONCOLOGY & IMMUNOLOGY

$100M upfront with the potential of $5.2B in total milestones plus high-single digit to mid-teen tiered royalties

BAYER

ONCOLOGY

$30M upfront and $50M equity investment. Increased per program milestones up to $1.5B in aggregate with low- to mid-single digit tiered royalties on net sales for up to seven programs

Merck KGaA Darmstadt, Germany

ONCOLOGY & IMMUNOLOGY

$20M upfront with up to $674M in aggregate and with mid-single to low-double digit tiered royalties

TECH PARTNERSHIPS

NVIDIA

Google Cloud

TEMPUS

Helix

Enamine

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

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DATA

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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

Recursion

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

COMPUTATION & ML/AI

Partnership on advanced computation, priority access to compute hardware or DGXCloud Resources.

REAL-WORLD DATA ACCESS

Preferential access to >20 PBs of real-world, multi-modal oncology data and ability to train causal AI models with utility in target discovery, biomarker development & patient selection.

COMPUTATION & ML/AI

Explore generative AI capabilities, drive improved search and access, scaled compute resources, improved management of data, and continued data privacy and security support.

Recursion OS

REAL-WORLD DATA ACCESS

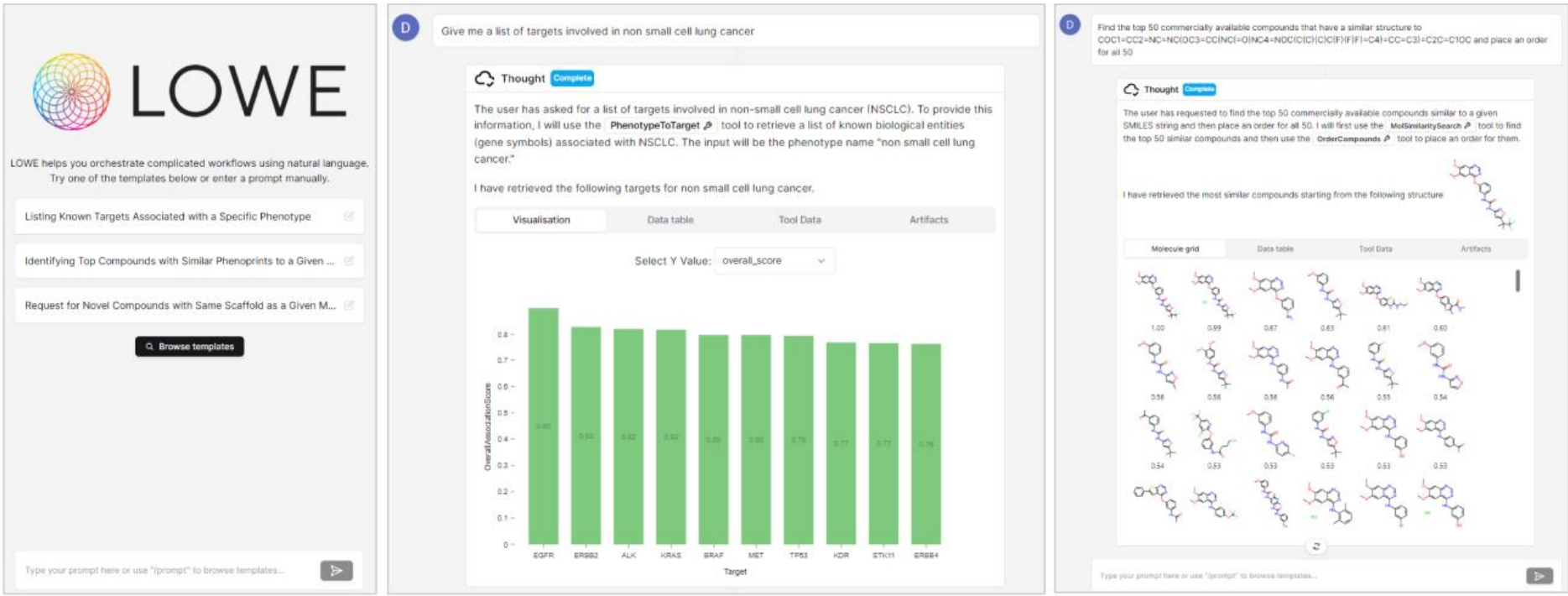
Access to hundreds of thousands of de-identified records to train causal AI models and design biomarker & patient stratification strategies across broad disease areas.

CHEMINFORMATICS & CHEMICAL SYNTHESIS

Utilizes Recursion's predicted protein-ligand interactions for ~36B compounds from Enamine's REAL Library.

Recursion

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



 Note: Large Language Model-Orchestrated Workflow Engine (LOWE) is Recursion's LLM-based software that can perform complex drug discovery tasks and orchestrate both wet-lab and dry-lab components of the Recursion OS using a natural language interface

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
CODIAK CHECKMATE PHARMACEUTICALS

David Hallett, PHD
Chief Scientific Officer
evotec MERCK

Ben Mabey
Chief Technology Officer

Kristen Rushton
Chief Operations Officer
Myriad genetics

Nathan Hatfield
Chief Legal Officer
WILSON SONSINI

Matt Kinn
Chief Business Officer
BCG UBS

Erica Fox
Chief People & Impact Officer
Google PRIMER

Lina Nilsson, PHD
SVP, Head of Platform
ENLITIC UC Berkeley

Board of Directors

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

Zachary Bogue
Co-Founder & Partner of Data Collective
DC

Blake Borgeson, PHD
Co-Founder of RXRX
MIRI

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

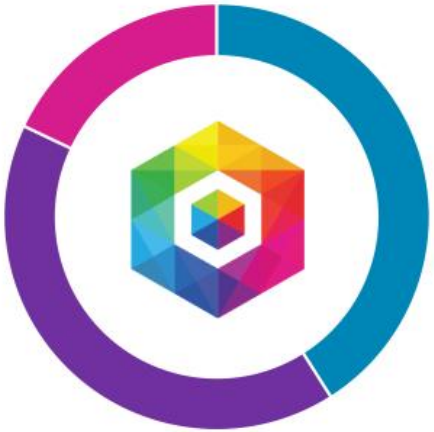
Chris Gibson, PHD
Co-Founder & Chief Executive Officer

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
MERCK THE UNIVERSITY OF UTAH

Zavain Dar
Co-Founder & Partner of Dimension
DIMENSION LUX+

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 – Prime – RATED BY ISS ESG

Rated – 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

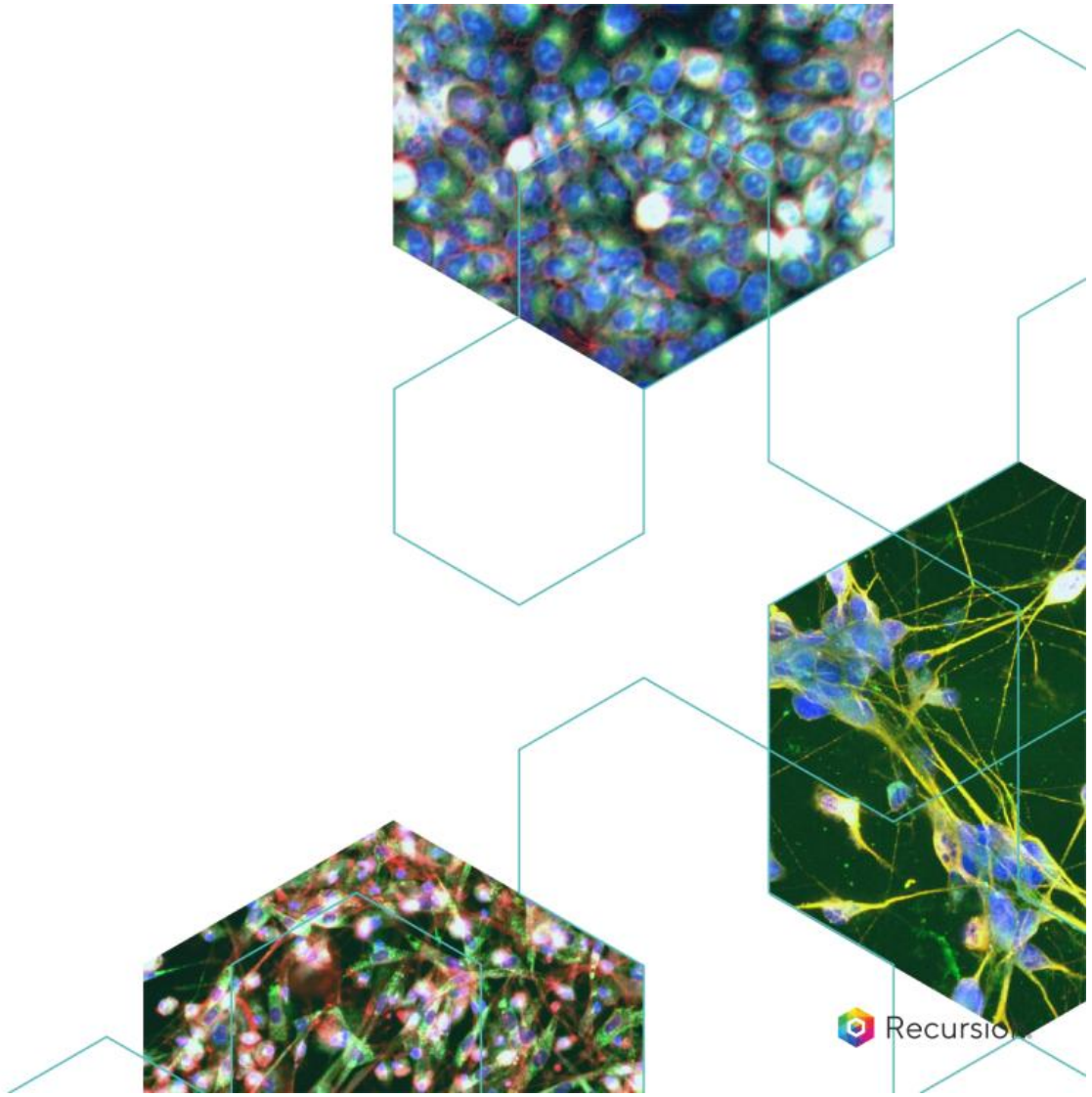
altitude lab
Founding Partner,
Life Science Accelerator

biohive
Founding Member,
Life Science Collective

Recursion

APPENDIX

Pipeline Details



PIPELINE

Oncology

Recursion

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 expected at **AACR Special Conference in Cancer Research 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 IC_{80} with a **short half-life** to reduce on-target toxicity • **Rapid absorption and permeability** at lowest possible dose
Unmet Need[1]	• **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

* Formerly GTAEXS617.

1. Advanced solid tumors including breast, NSCLC, ovarian, pancreatic, colorectal and head & neck. US and EU5 treatable incidence, 2022.

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

Initial clinical safety and PK/PD update on track for Q4 2024

Key Preclinical Data

REC-617 has Best-in-Class potential[1]

Designed to avoid efflux transporter substrate to minimize GI adverse events

Category	Assay	DC Criteria	Ph 1 Competitor	Ph 1/2 Competitor	**REC-617**
Potency & Selectivity	CDK7 IC50 (nM)	<10			
	CDK family selectivity	>100-fold			
	HCC70 (breast cancer) IC50 (nM)	<100			
ADME	**Caco-2 A2B (efflux) 10^{-6} cm/s**	>5 (<3)			
	Predicted human half-life (hr)	<15			

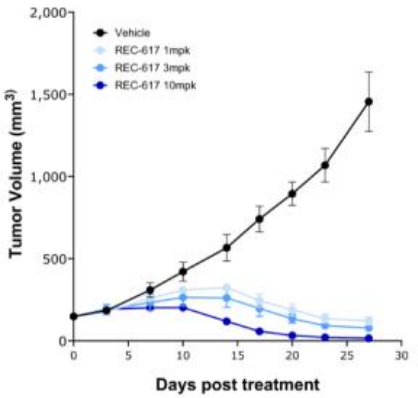
Meets or exceeds criteria | Minor deviation | Major deviation

Development Candidate (DC) Criteria:

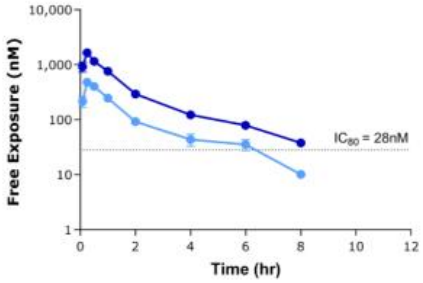
- **CDK7 IC50:** green <10nM; yellow 10-30nM; red >30nM
- **CDK7 selectivity:** green >100-fold; yellow 30-100-fold; red <30-fold
- **HCC70 IC50:** green <100nM; yellow 100-500nM; red >500 nM
- **Caco-2 A2B (efflux):** green >5(<3); yellow >1.5 (<10); red <1.5 (>30)
- **Half-life**: green <15, yellow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

Potent tumor regression with minimal IC_{80} exposure

CDX Model: OVCAR3[2]



Mouse PK[3]



- REC-617 demonstrates potent tumor regression with less than 10 hours of exposure above IC_{80} to optimize benefit-risk

1. Data on File. 2. Besnard et al, AACR (2022). 3. PK studies conducted in CD1 mice, single-dose administration. >10 hr IC_{80} results in significant body weight loss

Recursion

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 expected **Dec 9, 2024 (AACR Special Conference in Cancer Research)**

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 • Phase 1/2 study **initiation** expected in **Q4 2024** • **Phase 1 monotherapy** update on dose-escalation expected in **H1 2026**
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 Need[1]	• **>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

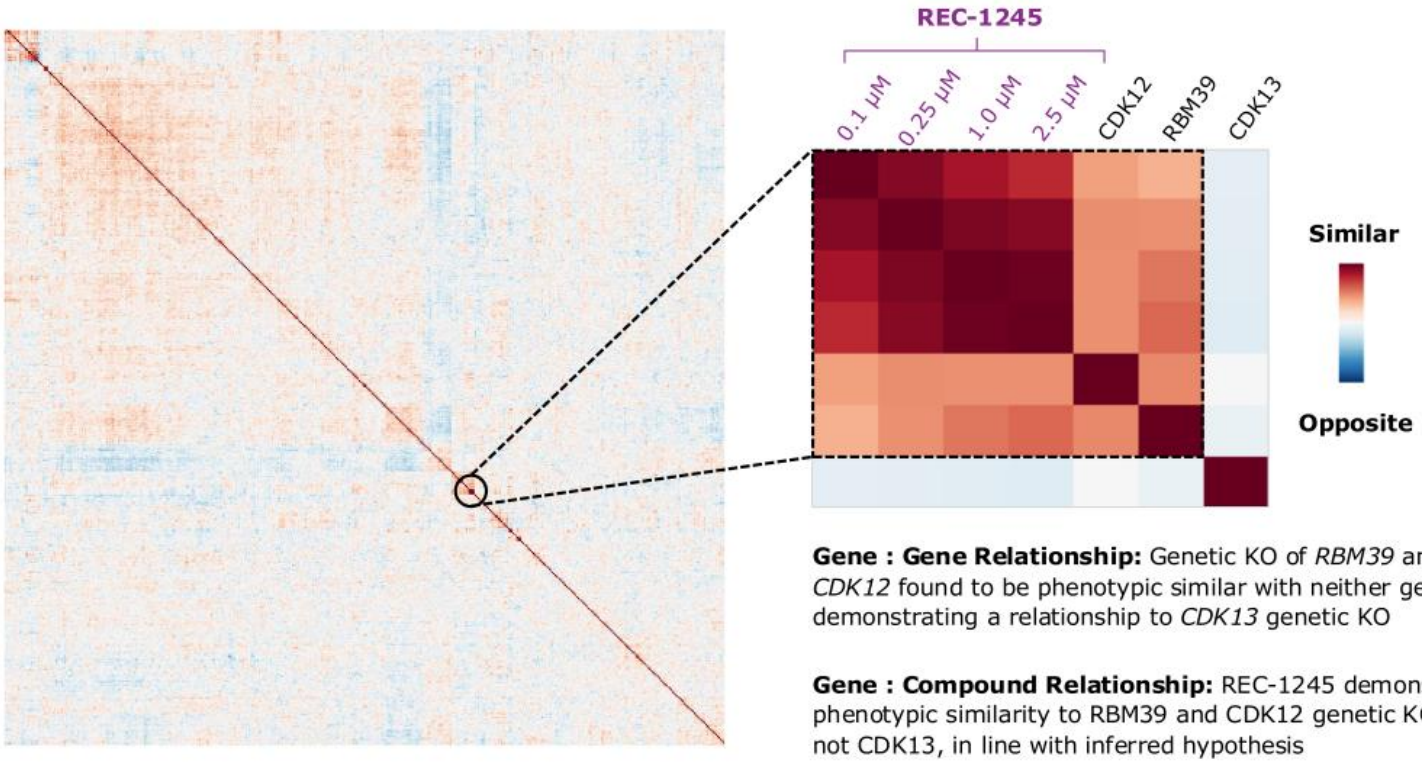
Recursion Approach

- **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 industry[2])**

1. Internal company estimates. Assumes US+EU5 addressable incidence with biomarker-enriched solid tumors and other select histologies.
2. Paul et al, Nat Rev Drug Discov (2010)

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

 1. Data on File.

REC-1245 (RBM39 degrader): Robust efficacy/PK/PD in biomarker-positive disease relevant preclinical tumor models with Phase 1 initiation expected Q4 2024

Key Preclinical Data[1]

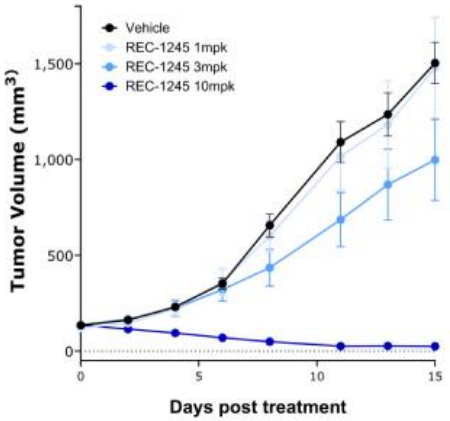
REC-1245 is highly selective and potent

Category	Assay	DC Criteria	REC-1245
Potency / Selectivity	RBM39 Degradation DC_{50}	<100 nM	
	CDK12 Kinase	No sig. activity	
In Vitro Safety	CEREP Safety Panel	No sig. activity	
	hERG IC_{50} (μM)	>30	
Pharmacokinetics	Oral Bioavailability (%F)	>30	

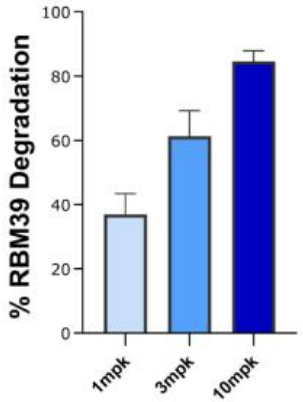
Meets or exceeds criteria | Minor deviation | Major deviation

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

CDX Model: OVK18[2]



PD: Target Engagement[3]



- 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

Recursion

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

- Monotherapy dose escalation trial initiation expected **Q4 2024**
- 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 **Q1 2025**
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 Need[1]	• **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

*Formerly EXS73565.

1. Cerner Enviza Treatment Architecture Reports 2023, rounded to nearest 1,000 patients per year.

REC-3565 (MALT1 inhibitor): Minimal UGT1A1 liability vs competitors and significant tumor regression observed in vivo with Phase 1 initiation anticipated on Q1 2025

Key Preclinical Data

REC-3565 has Best-in-Class potential[1]

Category	Assay	DC Criteria	Ph 1 large pharma	Ph1 biotech	**REC-3565**
Potency & Selectivity	MALT1 IC_{50} (nM)	<100			
	OCI-Ly3 proliferation IC50 (nM)	<400			
ADME	**UGT1A1 IC50 (µM)**	>10			
	Caco-2 A2B (efflux) 10^{-6} cm/s	>5 (<3)			

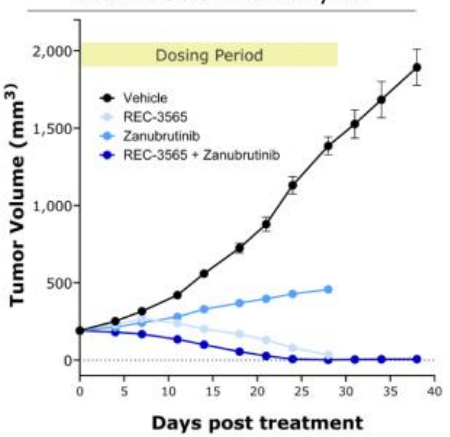
Meets or exceeds criteria | Minor deviation | Major deviation

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 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 vivo[2]

CDX Model : OCI-Ly10[2]



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 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

 1. Data on File. 2. Payne et al. ENA, (2024)

Recursion

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 **Q1 2025**

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 **1H 2025**
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 Need[1]	• **>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

*Formerly EXS74539.

1. EvaluatePharma Epidemiology 2023 (US and EU5)

REC-4539 (LSD1 inhibitor): Sufficient CNS exposures vs competitors and compelling dose-response demonstrated in vivo with Phase 1 initiation anticipated in H1 2025

Key Preclinical Data

REC-4539 has Best-in-Class potential[1]

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

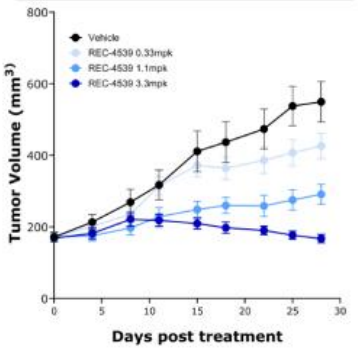
Meets or exceeds criteria | Minor deviation | Major deviation

Development Candidate (DC) Criteria:

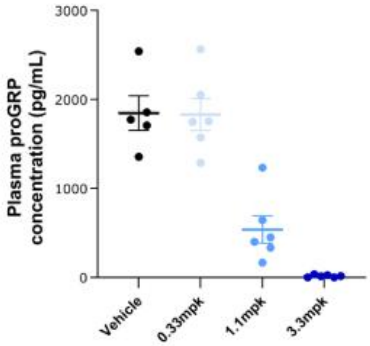
- **Brain:plasma ratio:** green >0.5; red <0.5
- **MDCK-MDR1 efflux ratio (Pgp):** green <2; yellow >2-<10; red >10
- **Predicted half-life:** green <24 hours; yellow 24-48h hours; red >48 hours

REC-4539 highly efficacious in SCLC xenograft model[2]

CDX Model: H1417[2]



Plasma ProGRP[3]



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

Trial Update

- Phase 1 **First Patient Dosed** in SCLC expected **H1 2025**

1. Data on File. 2. Payne et al. AACR (2023). 3. Data on File

Recursion

PIPELINE

Rare disease

Recursion

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 **H2 2025** 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 Need[1]	• ~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

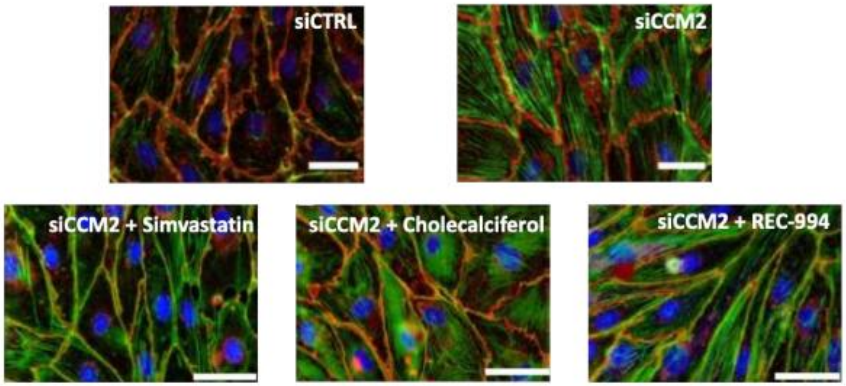
- **Unbiased ML-aided** phenotypic drug screen to identify effective therapeutics driving CCM
- In vivo POC demonstrated lesion reductions that were also observed in the Ph2 trial

1. Prevalence for hereditary and sporadic symptomatic population, Internal company estimates. 2. Smith ER. N Engl J Med (2024). 3. Horne MA, et al. Lancet Neuro, (2016).

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

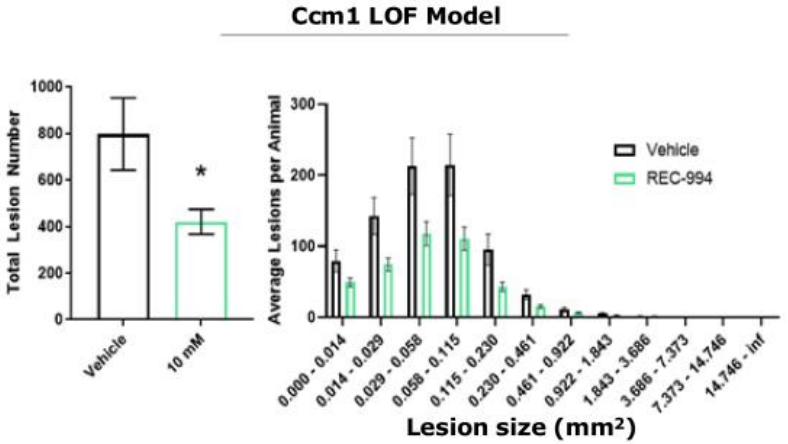
Recursion OS Insight

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



Key Preclinical Data[1]

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



 1. Gibson et al, Circulation (2015) and Data on File. 2. Data not shown

Recursion

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

Trial Update

SYCAMORE

- 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 expected to be presented at **forthcoming meeting in 2025**

SYCAMORE – CCM: Part 1: A Phase 2 Randomized, Double-Blind, Placebo-Controlled Clinical Trial to Evaluate the Safety, Efficacy and Pharmacokinetics of Two Doses of REC-994; Part 2: A Long-Term Blinded Extension Clinical Trial to Evaluate Long-Term Safety Tolerability and Efficacy of REC-994

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 **H1 2025**
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 Need[1]	• **No approved systemic therapies and significant unmet need** for ~50,000 FAP patients beyond colectomy – Includes ~7,000[2] **advanced duodenal polyposis** patients in the US at high-risk of developing cancer

Recursion Approach

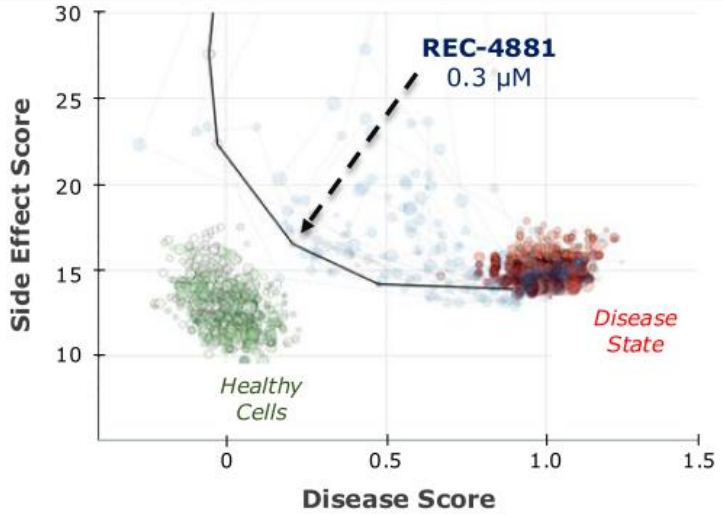
- **Unbiased ML-aided phenotypic** drug screen in **human cancer cells**
- **Validated findings** in vivo demonstrating significant reductions in polyps and adenomas

 1. US + EU5 diagnosed prevalence of FAP (adult and pediatric), Internal company estimates. 2. US addressable patients ≥ 55 years old.

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

Recursion OS Insight

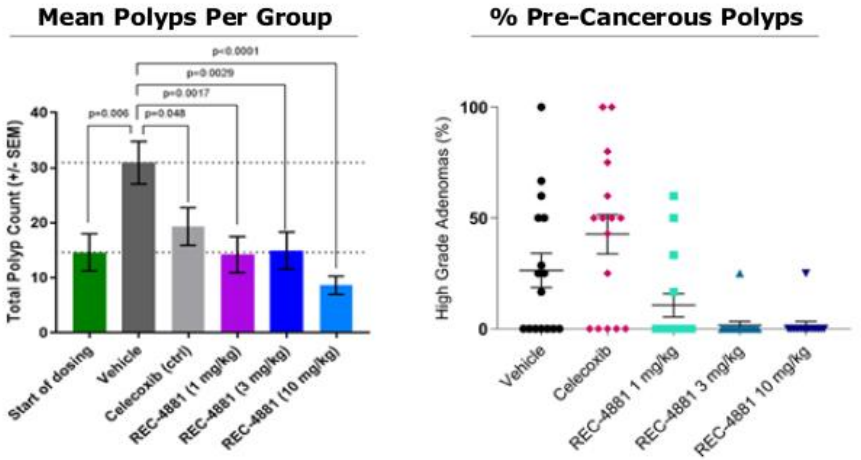
REC-4881 suppresses disease-inducing effects of APC mutations



- 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 Data[1]

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

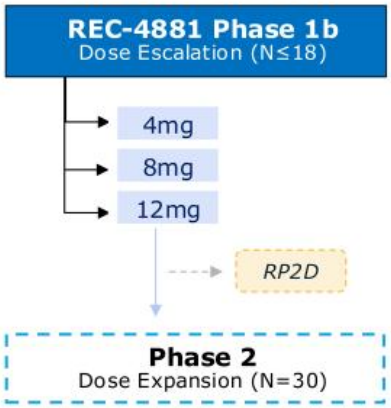
1. Data on File

Recursion

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

Development Strategy

TUPELO 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
- Futility analysis expected in **H1 2025**

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 • Development candidate nomination expected in Q4 2024
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 Need[1]	• **~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 Approach[2]

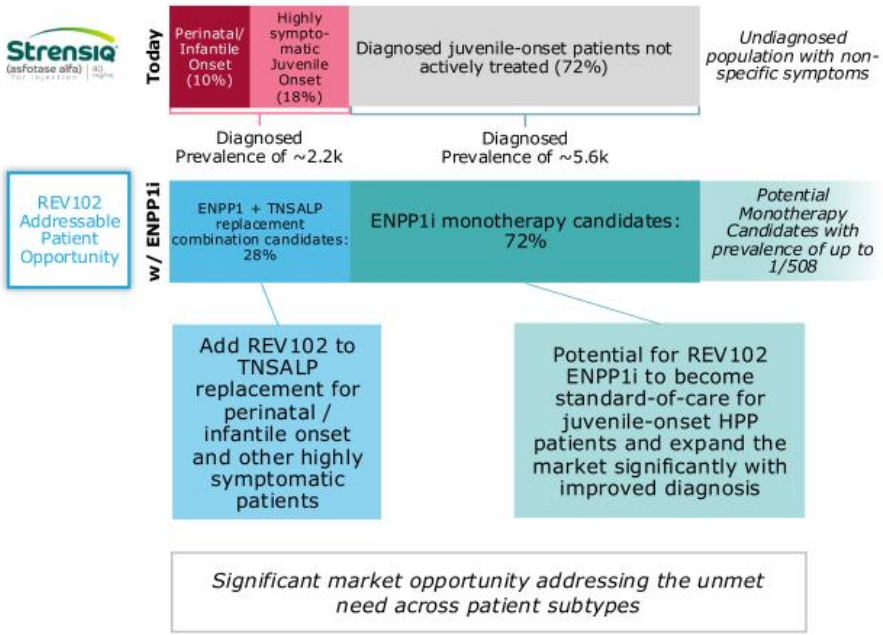
- **Precision designed for** both **high potency** and a lifetime of **chronic dosing**
- **Structurally distinct** differences vs competitor ENPP1 inhibitors
- **Maintain selectivity** and deliver a candidate with **high oral bioavailability** in the clinic

 1. HPP prevalence at birth. Mornet et al, 2020. 2. Joint Venture with Rallybio

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

Market Opportunity[1]

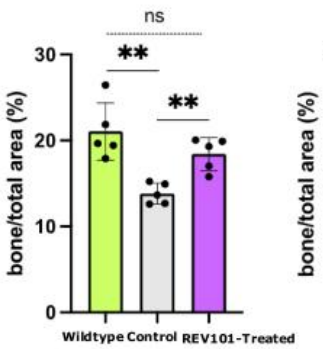
Estimated **Diagnosed Prevalence** Hypophosphatasia Patients in **US**

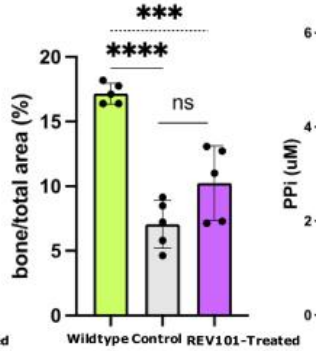


Significant market opportunity addressing the unmet need across patient subtypes

Key Preclinical Data[2]

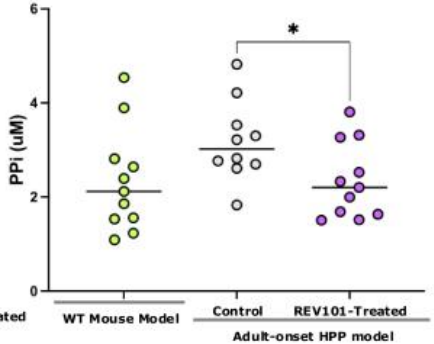
Bone Morphometry
2D Analysis of Trabecular Bones





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

- **Development candidate nomination** expected in Q4 2024

1. EvaluatePharma and https://www.ncbi.nlm.nih.gov/pmc/articles/PMC7868366/, https://bmcmusculoskeletdisord.biomedcentral.com/articles/10.1186/s12891-019-2420-8, Trinity Market Research 2021. 2. Narisawa et al. ASBMR (2024)

Recursion

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 H1 2025
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 Need[1]	• **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 recurrence[2]

Recursion Approach

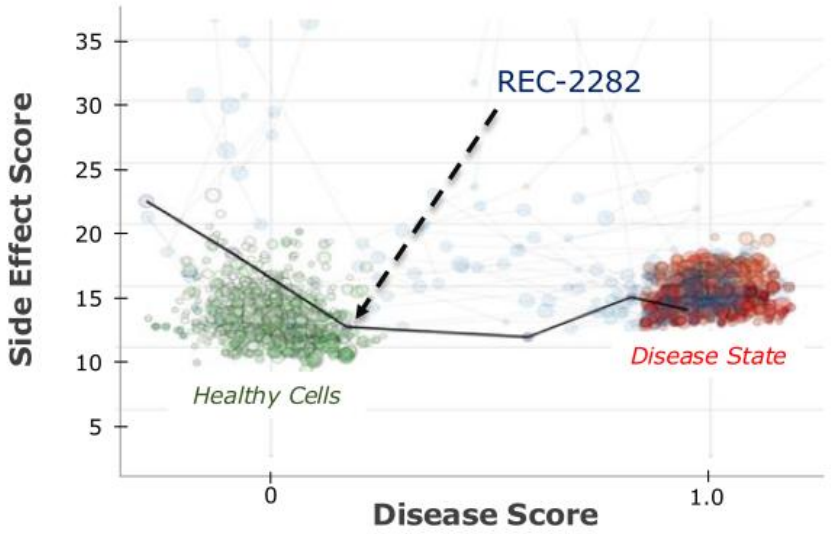
- Unbiased **ML-aided phenomap insight and drug screen** in human cells
- Identify effective therapeutics that **rescue disease-inducing effects of *NF2*** loss

 1. US + EU5 treatable incidence for all NF2-driven meningiomas. 2. Rogers et al. J Neurosurg, (2015)

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

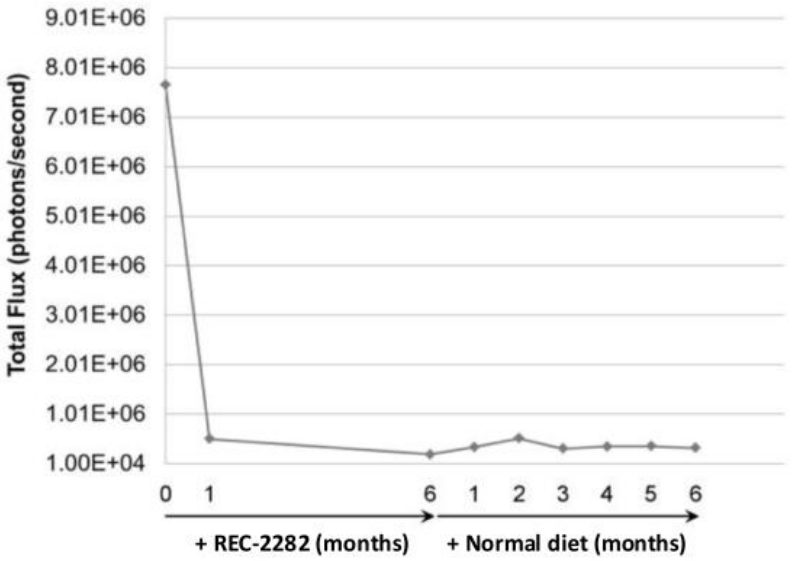
Recursion OS Insight

REC-2282 demonstrates concentration-dependent reversal of NF2 loss



Key Preclinical Data[1]

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



1. Data on File

Recursion

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 **Data maturing**
- Futility analysis (PFS6) expected in **H1 2025**

PIPELINE

Other areas of high unmet need

Recursion

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 expected **in Q4 2024** • Phase 2 update expected **in Q1 2026**
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 Need[1]	• **~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

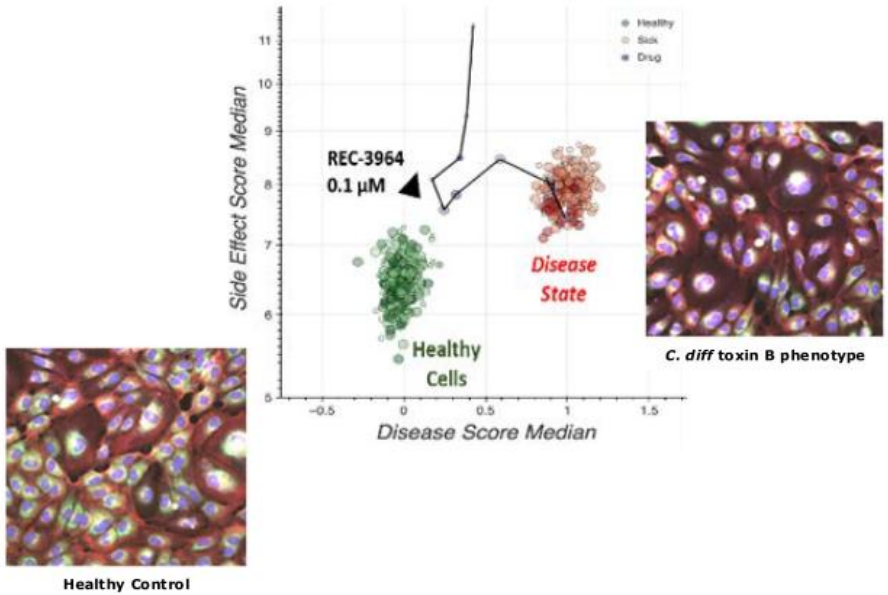
- Unbiased **ML-aided conditional phenotypic** drug screen in **human cells**
- Identified **novel mechanisms** that mitigated the effect of *C. diff.* toxin B treatment

1. Incidence of addressable US cases of recurrent CDI, Shields et al., Anaerobe (2016)

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

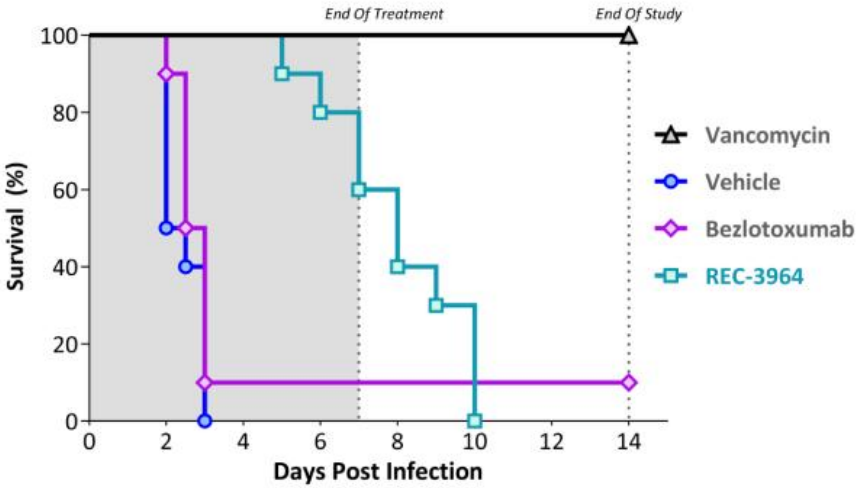
Recursion OS Insight

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



Key Preclinical Data[1]

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



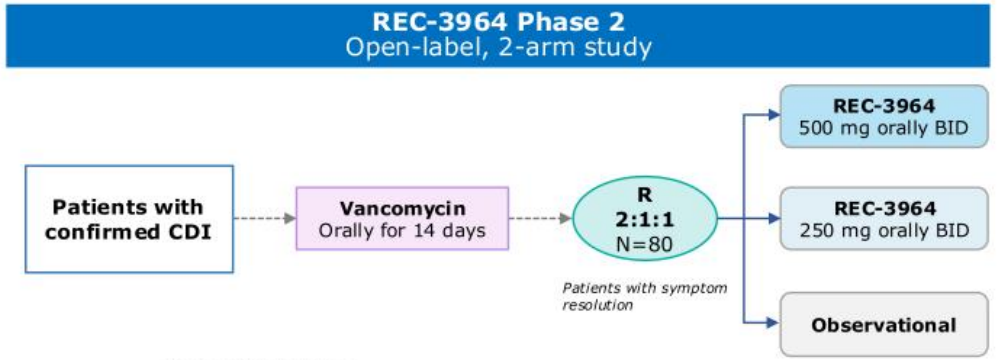
1. N=10 hamsters per group. *C. difficile* strain 630, Data on File Data on File

Recursion

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

Development Strategy

ALDER Phase 2 study design



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 expected in **Q4 2024**
- Program update expected **Q1 2026**

Recursion

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 submission** expected in **2025** • **Phase 1** study in healthy volunteers expected to initiate in **2025**
Mechanism of Action	• Reversible, orally bioavailable, and potent Target Epsilon inhibitor • Promotes tissue repair and reverses fibrosis by potentially modulating TGF-ß
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 Need[1]	• **~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

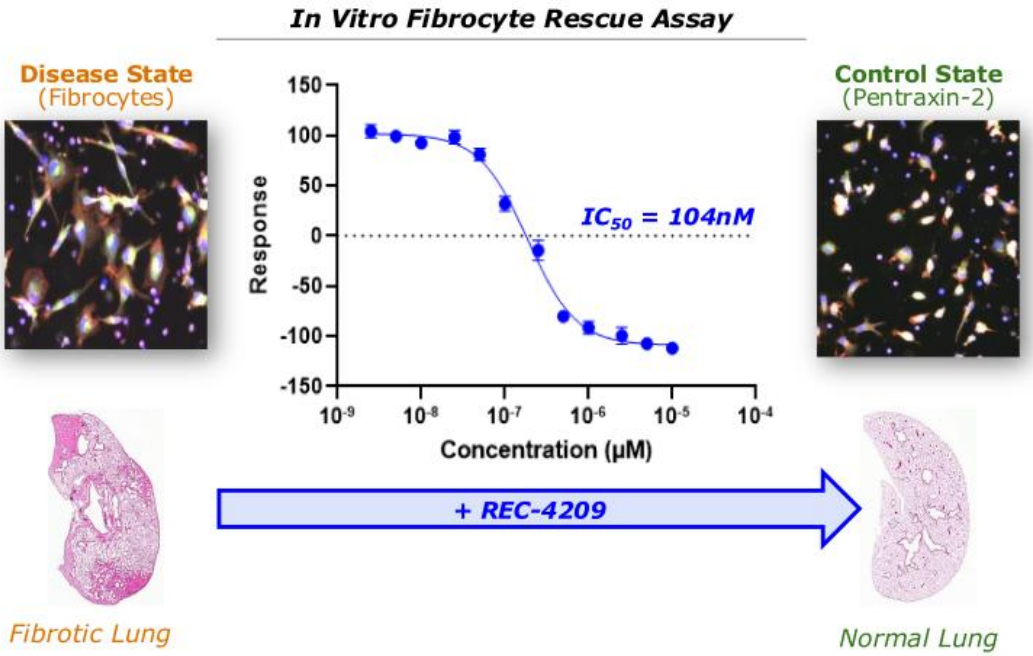
Recursion Approach

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

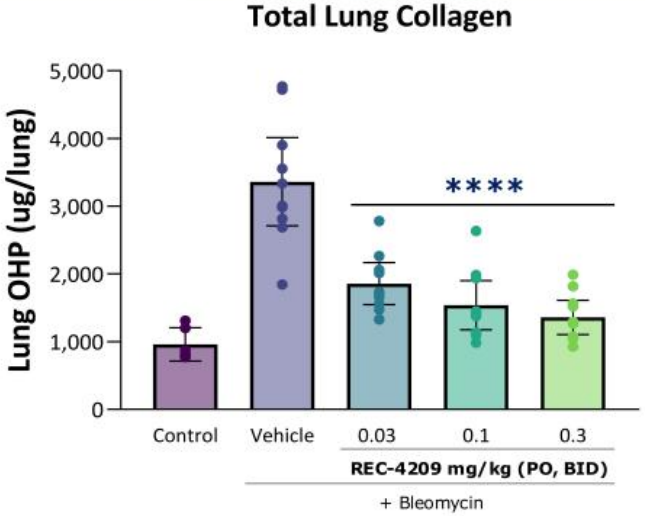
1. Global Data, Internal company estimates on IPF prevalence, Collard et al., Chest (2014)

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 Insights[1]



Key Preclinical Data[2]



- 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

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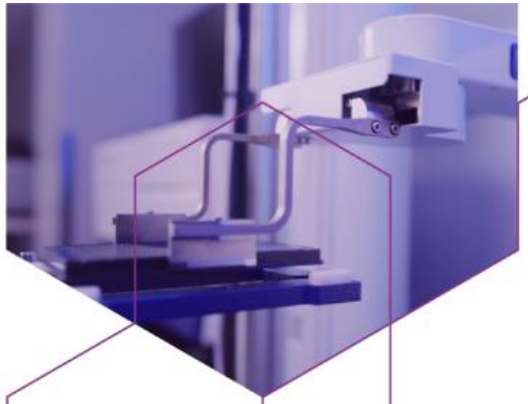
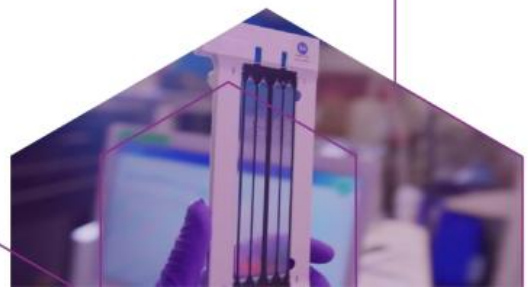
APPENDIX

Partnerships & Data Strategy Details





Recursion

Exciting scientific collaborations span biopharma, tech & data

Therapeutic discovery partnerships

Roche / Genentech

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 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

BAYER

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

NVIDIA Announced July 2023	• **$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**
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 • Recursion will also explore **making some of its AI models available on Google Cloud**

Real-world data access

TEMPUS Announced Nov. 2023	• **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
Helix Announced May 2024	• 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

Enamine Announced Dec. 2023	• 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

Announcing OpenPhenom for non-commercial use

AVAILABLE ON:

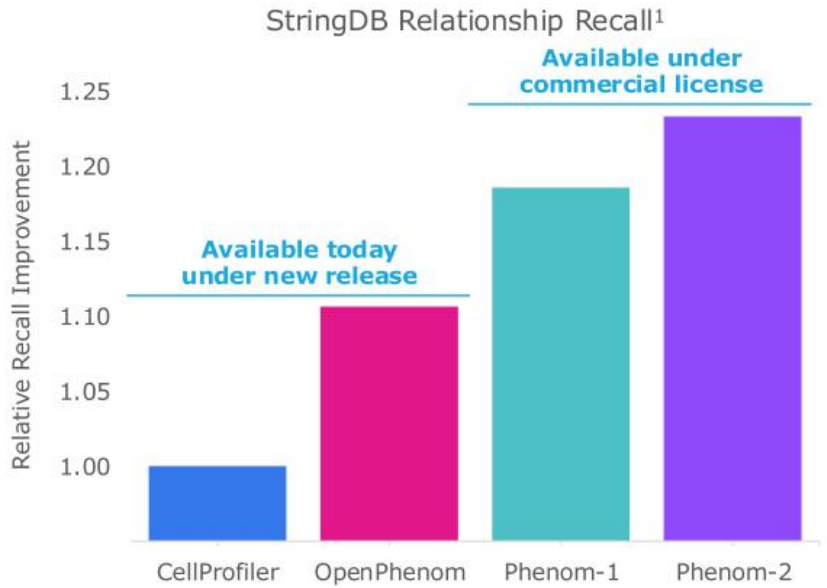
Hugging Face Google Cloud

COMING SOON TO:

NVIDIA

- Publicly accessible Foundation Model for microscopy data workflows
- Replaces legacy image segmentation and feature extraction software packages for non-commercial applications

OpenPhenom outperforms 'gold standard' CellProfiler package is industry benchmarking tasks



1. Recall of known biological relationships (gene-gene) annotated in StringDB using the public JUMP-CP dataset


Recursion®