Recursion

Corporate Deck May 2025

MAY 2025

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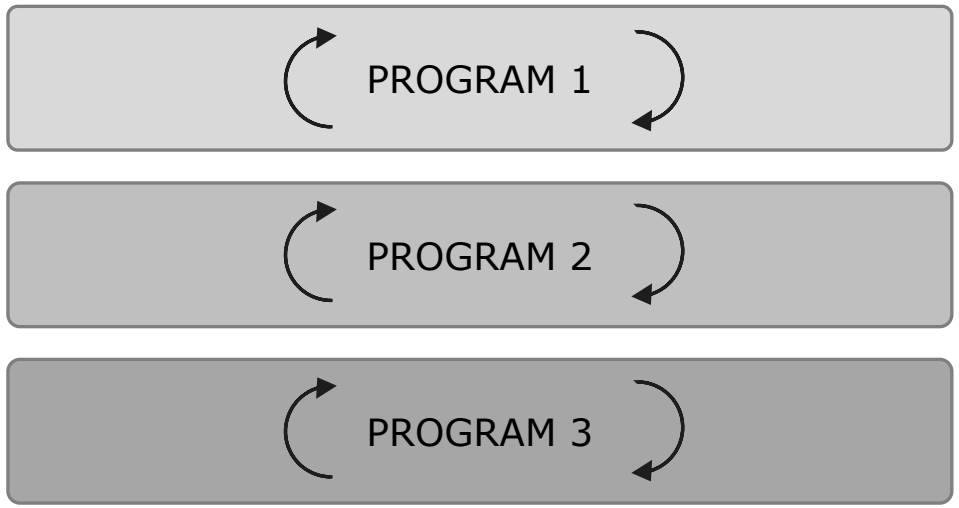
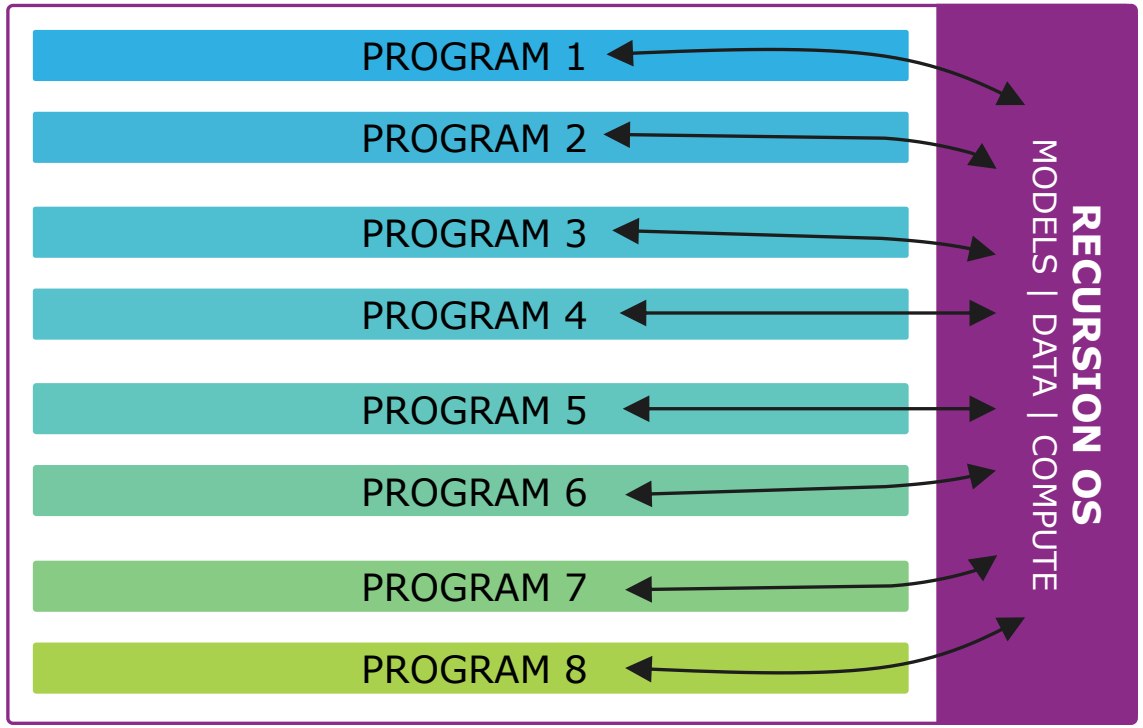
Recursion's Mission: Decode Biology to Radically Improve Lives

Biotech



vs.

Recursion



Recursion OS delivers a decisive, data-led portfolio strategy

Recursion

Evolution of our platform reflects the evolution of our portfolio

Recursion 0.1


Phenomics


SiRNA


Repurposing

e.g.: FAP

Recursion 1.0


Phenomics


Transcriptomics

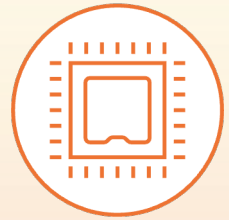

NCEs


CRISPR

e.g.: RBM39

Recursion 2.0


Multimodal data


Compute

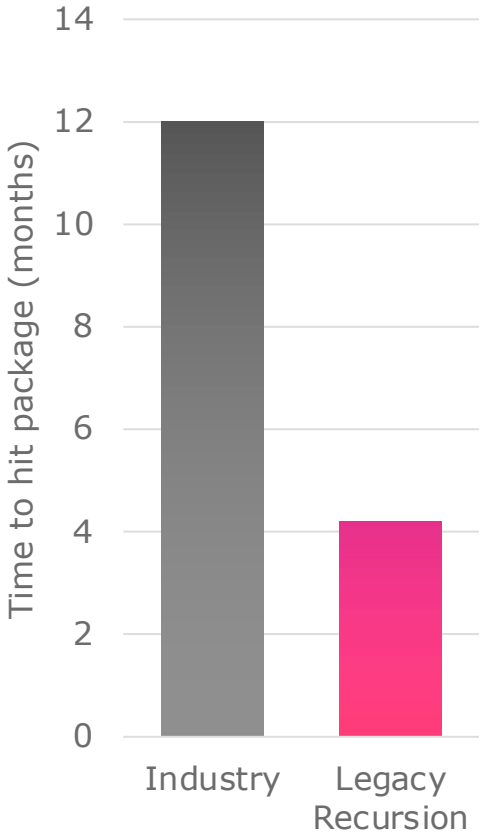

Active design


Combination of RXRX & EXAI

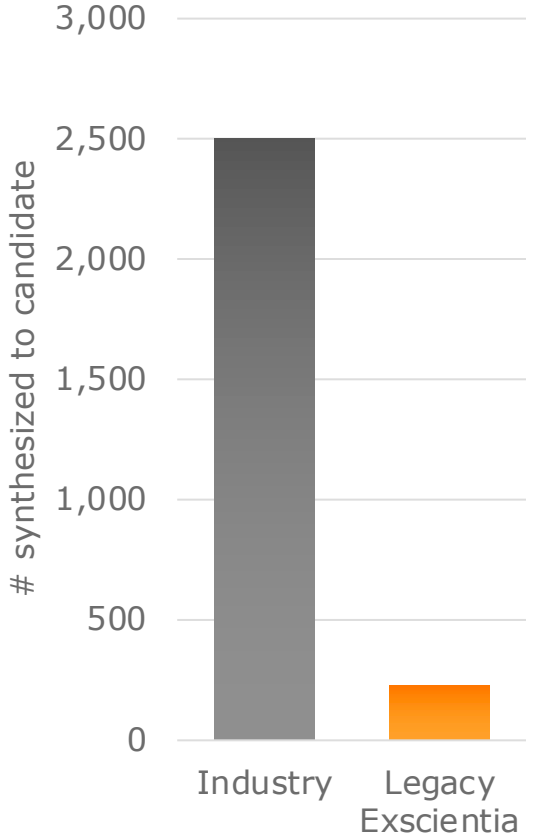
Early-stage discovery programs

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

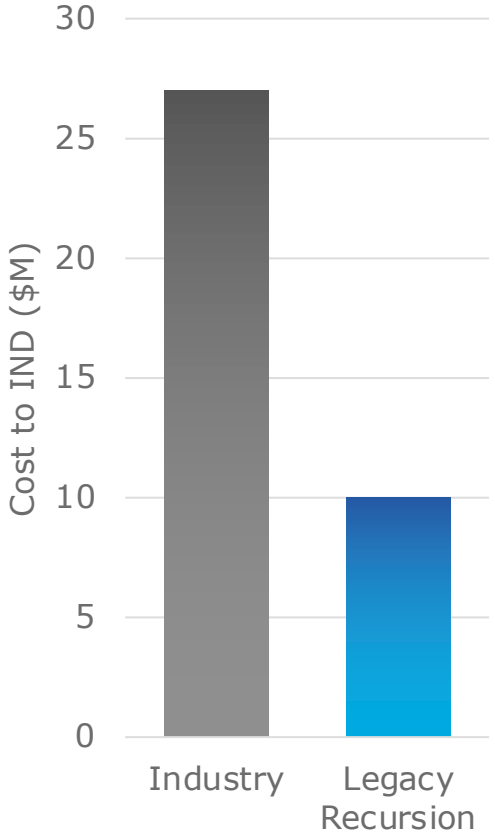
Quickly validated hypotheses and >



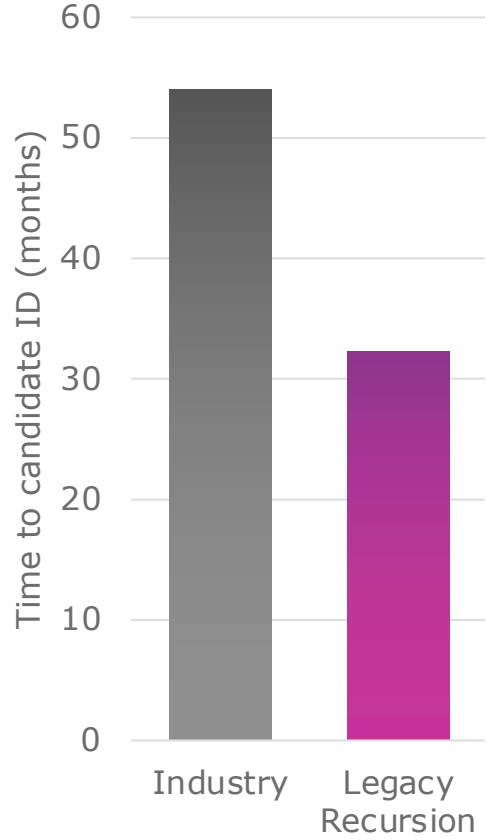
> highly productive compound design to >



> spend less >



> and go faster



(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

Recursion

Sharpening our focus: Why Now

Commit to streamlined integrated portfolio

Deliver on our commitment to a streamlined post-integration pipeline and overall operations by 1Q25, enabling a disciplined R&D strategy built for near and long-term impact

Prioritize with integrated Recursion OS 2.0 platform

Rapidly validate emerging opportunities, double down on winners, and decisively exit efforts that do not meet our bar

Continue to invest in transformational value creation

Focused go-forward strategy, allows for targeted investment in platform and high impact programs, and to fuel innovation with financial discipline amid an uncertain macro environment

Go-Forward Pipeline: Advancing 5+ High-Potential Programs Across Oncology & Rare Disease

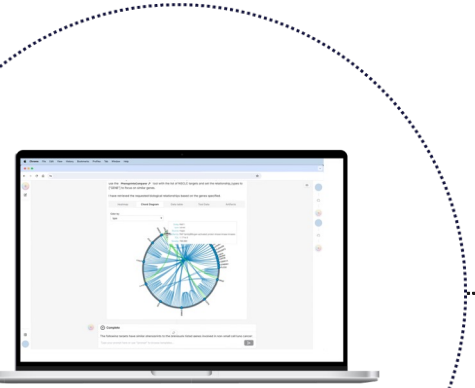
	Candidate	Target	BIC/FIC	Indication	Preclinical	IND-Enabling	Phase 1 / 2	Phase 3
ONCOLOGY	REC-617	**CDK7**	BIC	Advanced solid tumors[1]	*ELUCIDATE*			
ONCOLOGY	REC-1245	**RBM39**	FIC	Biomarker-enriched solid tumors & lymphoma	*DAHLIA*			
ONCOLOGY	REC-3565	**MALT1**	BIC	B-cell malignancies	*EXCELERIZE*			
ONCOLOGY	REC-7735	**PI3Kα H1047R**	BIC	Breast Cancer				
RARE	REC-4881	**MEK1/2**	FIC	Familial adenomatous polyposis (FAP)	*TUPELO*			
RARE	REV102[2]	**ENPP1**	FIC	Hypophosphatasia (HPP)				
	REC-4539 – strategic pause[3]	**LSD1**	BIC	Solid tumors (e.g., SCLC)	*ENLYGHT*			

3 clinical programs deprioritized: REC-994 for CCM, REC-2282 for NF2, REC-3964 for prevention of recurrent *C. difficile* infection

1 preclinical program (REC-4209 for IPF) has also been deprioritized as part of a disciplined, strategic portfolio prioritization, following the integration

1. Includes non-small cell lung cancer (NSCLC), colorectal cancer, breast cancer, pancreatic cancer, ovarian cancer, head and neck cancer
2. Joint Venture with Rallybio
3. Strategic pause to ensure a competitive Target Product Profile

Pharma partnerships with approximately $455M[1] earned to date and potential to receive more than $20B in additional milestones



Recursion OS

NEUROSCIENCE & ONCOLOGY

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

ONCOLOGY & IMMUNOLOGY

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

ONCOLOGY

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

ONCOLOGY & IMMUNOLOGY

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

1. Upfront and milestone payments from these therapeutic partnerships
Note: Total deal value may include program milestones (for development, commercialization and net sales) and tiered royalties on net sales

Portfolio Update

Recursion

Go-Forward Pipeline: Advancing 5+ High-Potential Programs Across Oncology & Rare Disease

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ONCOLOGY	REC-7735	**PI3Kα H1047R**	BIC	Breast Cancer				
RARE	REC-4881	**MEK1/2**	FIC	Familial adenomatous polyposis (FAP)	*TUPELO*			
RARE	REV102[2]	**ENPP1**	FIC	Hypophosphatasia (HPP)				
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1. Includes non-small cell lung cancer (NSCLC), colorectal cancer, breast cancer, pancreatic cancer, ovarian cancer, head and neck cancer
2. Joint Venture with Rallybio
3. Strategic pause to ensure a competitive Target Product Profile

Sharpening our focus: Go-forward programs powered by Recursion OS

REC-617 | CDK7
Ph 1/2 Mono; Ph 1 Combo 1H25
Solid tumors[1]

Optimized PK/PD with potential for improved efficacy and safety via a wider therapeutic index

~185,000 addressable patients

REC-1245 | RBM39
Ph 1
Solid tumors[2], Lymphoma

Phenotypic insight to identify novel MOA to modulate DDR biology

~100,000 addressable patients

REC-4881 | MEK1/2
Ph 1b/2
Familial adenomatous polyposis (FAP)

Phenotypic insight on MEK1/2 inhibition for APC-mutant FAP

~50,000 addressable patients

REC-3565 | MALT1
Ph 1
B cell malignancies

Potential for less UGT1A1 inhibition and off-target AEs

~41,000 addressable patients

REC-7735 | PI3Kα H1047R
IND-enabling 2H25
Breast Cancer

Highly selective with potential for improved efficacy and safety via a wider therapeutic index

~11,000 addressable patients

REV102[3] | ENPP1i
IND-enabling
Hypophosphatasia (HPP)

Oral, highly selective & potent, suitable for lifetime dosing

~7,800[4] addressable patients

REC-4539 | LSD1: Precision designed for reversibility and CNS penetration

Strategic pause to ensure a competitive Target Product Profile

Note: Addressable patient populations estimate based on annual US+EU5 and currently identified indications

1. Includes ovarian cancer, breast cancer, non-small cell lung cancer (NSCLC), colorectal cancer, pancreatic cancer, head and neck cancer
2. Biomarker-enriched
3. Joint Venture with Rallybio
4. Diagnosed patients

Sharpening our focus: Disciplined deprioritization – focus where we can win

REC-2282
NF2
	• New findings:
	  • Phase 2 for NF2-related meningioma passed the futility threshold, primarily driven by the 40 mg cohort; however, the 60 mg and combined dose arms did not pass the futility criteria
	  • Limited tumor shrinkage and clinical activity across all arms
	• Totality of data supports the discontinuation of study

REC-994 CCM	• Early data suggested potential promising trends in exploratory efficacy endpoints at 400mg (mean volume reduction, mRS), negative trends in efficacy at 200mg (data were not statistically significant) • New findings: Long-term extension results showed no promising trends in MRI or functional outcomes in the placebo-to-400mg crossover, and the 400mg-to-400mg arm did not continue prior trends and was indistinguishable from natural history • Totality of data supports the discontinuation of study
REC-3964 *C. difficile*	• Evolved treatment options result in lower recurrence rates (~5%); thus limiting unmet need • Strategic decision to focus on areas with greater unmet need • Will consider out license opportunities

Targeted, Differentiated Go-Forward Portfolio Strategy

Powered by the Recursion OS 2.0 platform

Differentiated, product first mindset, powered by Recursion OS 2.0

Integrated, end-to-end tech stack

Multimodal Biology · Design · ClinTech

Explore the uncharted by going after **novel targets** to develop differentiated medicines

First-in-class molecules

Provide **significantly meaningful advancements** with differentiated medicines

Best-in-class molecules

Double down on areas of **expertise and efficiency**

Oncology · I&I · Neuroscience · Rare

Develop **innovative molecules in-house, powered by our platform**

Focus on **quick and clear go/no-go, differentiated TPP that enable rapid POC**

Note: Leverage platform insights to strategically in-license

Internal pipeline momentum: Near Term Catalysts Through 2025 and 2026

2025

H1

REC-617 (CDK7i) in advanced solid tumors
Initiation of combination study

H2

REC-4881 (MEK1/2i) in FAP
Additional safety and efficacy data from TUPELO

REC-617 (CDK7i) in advanced solid tumors
Additional Phase 1 data from ELUCIDATE

REC-7735 (PI3Kα H1047Ri)
DC nomination

2026

H1

REC-1245 (RBM39)
Early safety and PK from monotherapy trial

H2

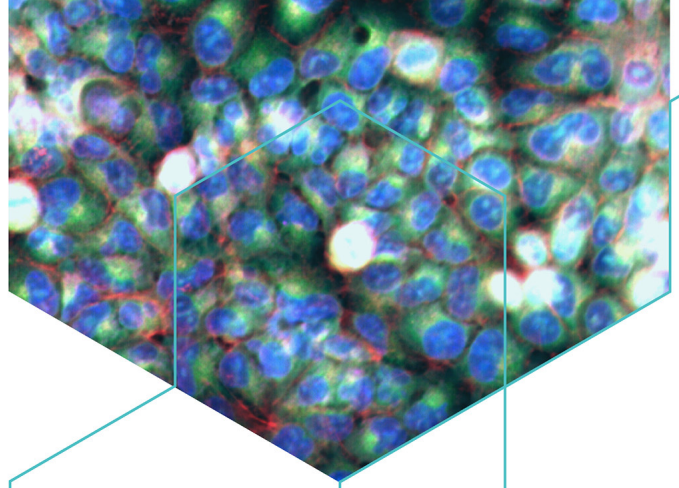
REC-3565 (MALT1)
Early safety and PK from monotherapy trial

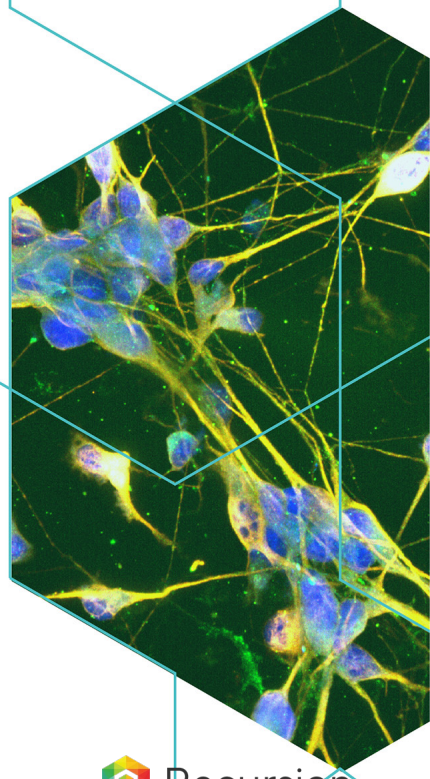
REV102 (ENPP1i)
Initiation of Phase 1

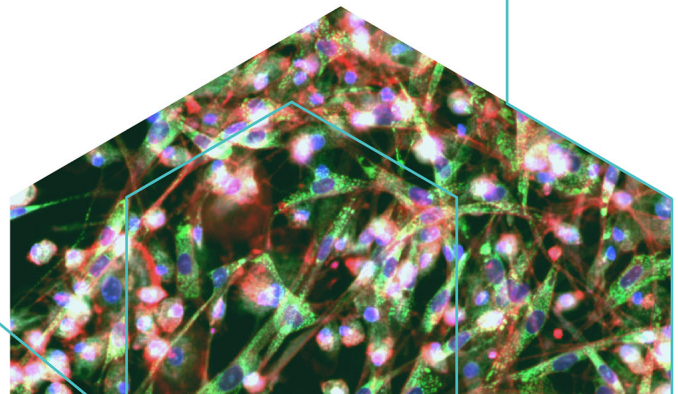
Note: REC-4539 (LSD-1i), Phase 1 TBD

Rare Disease

MEK 1/2







Recursion

REC-4881: Allosteric MEK1/2 Inhibitor

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

Unmet need[1]

- **No systemic therapies** for ~50,000 FAP patients beyond colectomy
- **~15,000 patients** post colectomy, >55 years old

Mechanism of Action

- **Potent, non-competitive, allosteric** MEK1/2 inhibitor

Thesis & Differentiation

- **First oral therapy,** targeting underlying genetic mechanisms
- **Preferential distribution to GI tissues ->** greater activity at lower doses

Recursion Approach

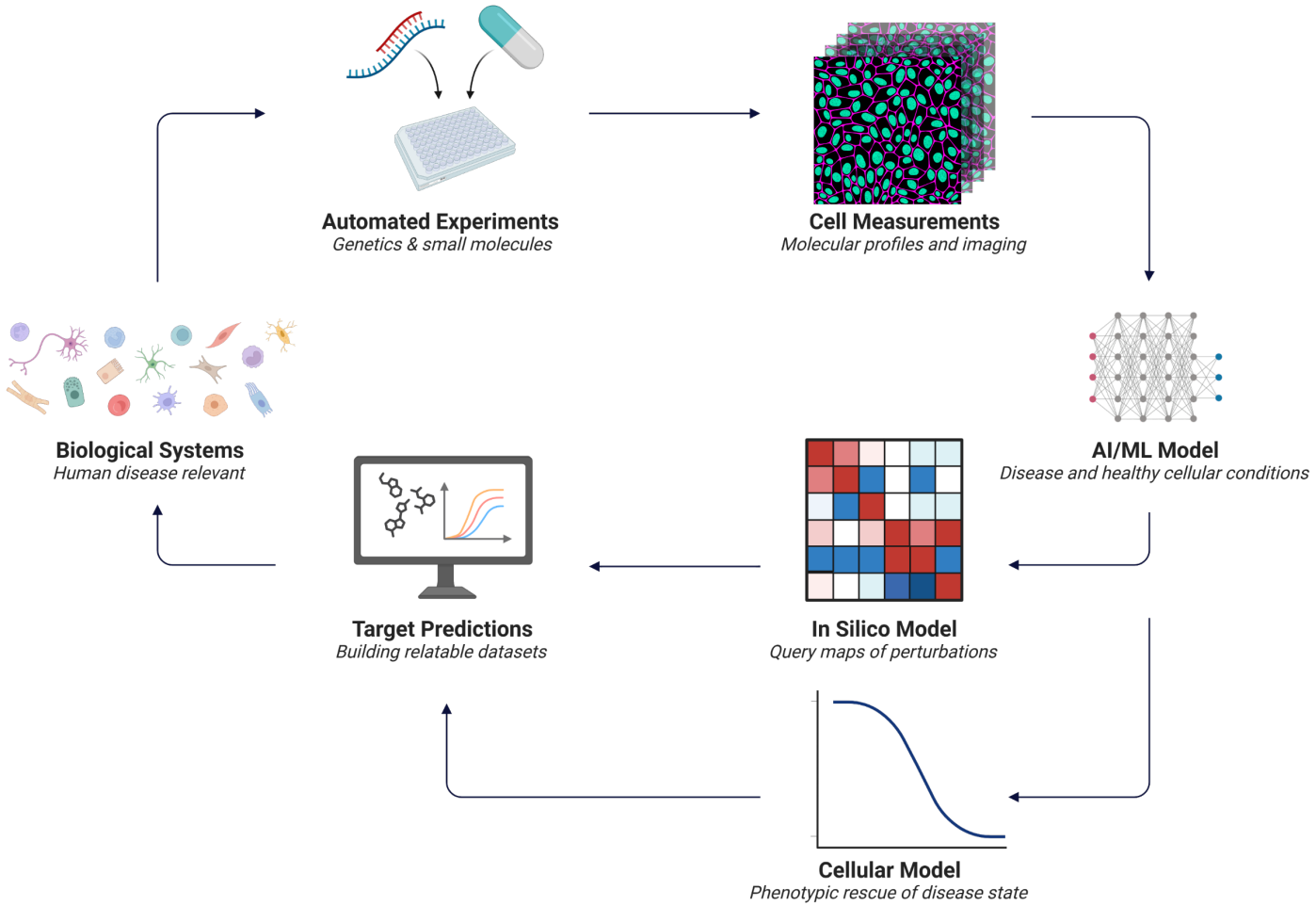
- **Unbiased ML-aided phenotypic** drug screen in **human cancer cells**
- **Validated findings** in vivo demonstrating significant reductions in polyps and adenomas

Program Status

- **Enrollment ongoing**; additional efficacy and safety data, **2H25**

1. US + EU5 diagnosed prevalence of FAP (adult and pediatric), Internal company estimates.

Platform Insight: The AI-powered Recursion OS was leveraged to uncover novel therapeutic mechanisms relevant for FAP



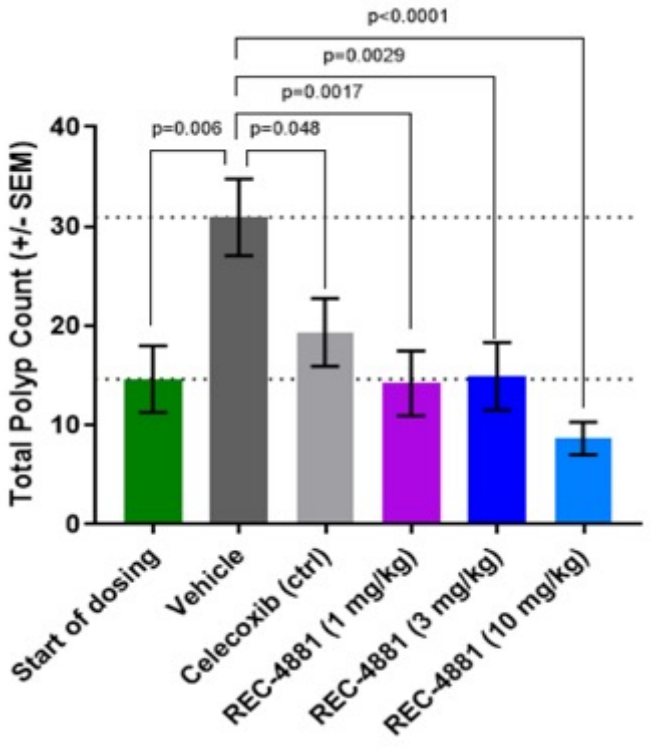
REC-4881 Discovery

- The platform analyzed cellular models of APC gene loss—the root cause of FAP
- **AI/ML** extracts morphological features to **distinguish** "**diseased**" vs. "**healthy**" states
- Numerous compounds screened to identify therapeutic mechanisms that **reverse disease state back to healthy** in a concentration-dependent manner
- **REC-4881** (an **allosteric MEK 1/2 inhibitor**) demonstrated **potent** and **concentration dependent rescue**

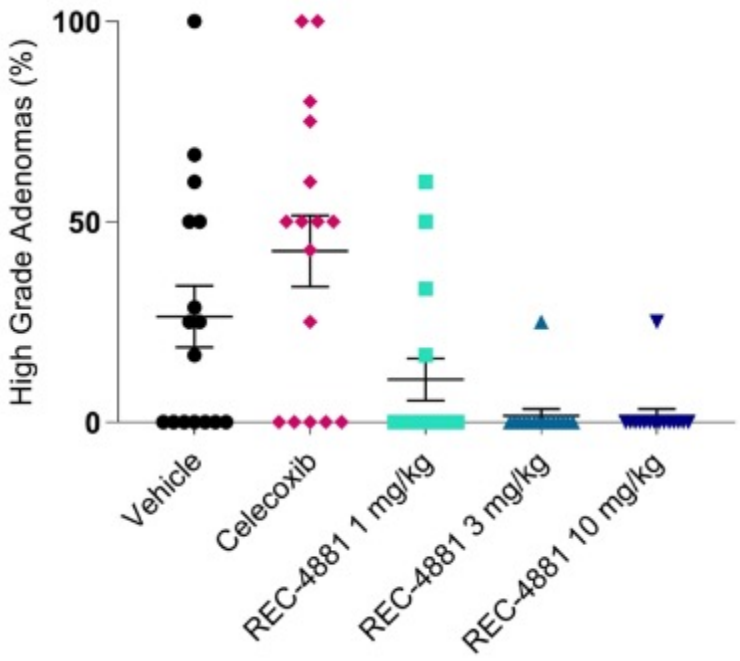
1. Data on File

Preclinical Data: REC-4881 significantly decreased polyps and high-grade adenomas in FAP mouse models

A) Mean Polyps Per Group



B) % Pre-Cancerous Polyps



Preclinical Summary[1]

REC-4881:

- **Reduces polyp count more effectively** than **celecoxib** in $APC^{min/-}$ mice
- **Decreases** both **polyp number and high-grade adenoma** percentage, unlike celecoxib

1. REC-4881 reduces polyp count and eliminates high grade adenomas in Apc^{min} mouse model of FAP.
   A) Mean GI polyp count after oral administration of indicated dose of REC-4881, celecoxib or vehicle control for 8 weeks. Polyp count at start of dosing reflects animals sacrificed at the start of study (15 weeks of age). $P < 0.001$ for all REC-4881 treatment groups versus vehicle control.
   B) Same data displayed in A shown for individual animals on study suggests that at lowest dose tested (1 mg/kg) REC-4881 demonstrates maximum efficacy

Recursion

Phase 1b and Phase 2: REC-4881-201 study design & objectives

- Two stage study designed to assess safety, tolerability, PK/PD and **preliminary efficacy of REC-4881 in FAP**
- Phase 1b (safety run-in) followed by Phase 2 (open-label) evaluating once-daily **REC-4881 for <u>12 weeks</u>**

Phase 1b

Completed:

Single and Multiple Dose Safety

Randomized, double-blind, placebo-controlled (N=7)

5 patients dosed 4 mg, 2 on placebo

Study Objectives:

Safety/tolerability
Pharmacokinetics
Pharmacodynamics

Key Eligibility: FAP ≥ 18 years old

Phase 2: Open-label Signal Seeking

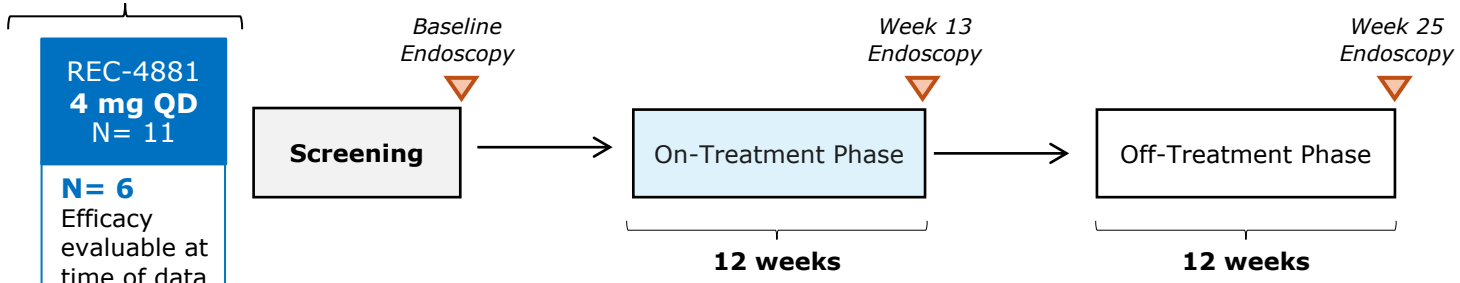
Dose Finding
N= 14

REC-4881
4 mg QD
N= 11

N= 6
Efficacy evaluable at time of data cutoff[1]

REC-4881
8 mg QD
N= 3[1]

Not efficacy evaluable



Key Eligibility:

- Confirmed *APC* mutation
- ≥ 55 years old[2]
- Post-colectomy/proctocolectomy
- No cancer present
- Polyps in upper and/or lower GI

Primary Objectives :

- Safety/tolerability
- Percent change from baseline in polyp burden after 12 weeks of treatment and off-treatment

1. Efficacy Evaluable Population: Defined as all participants who have measurable disease (non-zero polyp burden) at end of baseline endoscopy, received at least 75% of study drug, and have at least one post-baseline on study endoscopic assessment
2. After analysis in Phase 1b the eligibility criteria was shifted to enroll only patients 55+ years of age to minimize TRAEs associated with MEK1/2 inhibition
3. Participants from RP2D in Dose Finding will Contribute to the sample size in Cohort Expansion

Phase 1b and Phase 2: REC-4881 summary of adverse events

Event, n (%)	Placebo (N=2)	REC-4881 4 mg (N=16)[3]	REC-4881 8 mg (N=3)	REC-4881 Total (N=19)
Any Treatment Emergent Adverse Event (TEAE)	**2** (100)	**13** (81.2)	**3** (100)	**16** (84.2)
TEAEs Grade ≥3	**0**	**5** (31.2)	**0**	**5** (26.3)
Any TEAE related to study drug (TRAE)	**1** (50.0)	**13** (81.2)	**2** (66.7)	**15** (78.9)
Grade ≥3 TRAE	**0**	**3** (18.8)	**0**	**3** (15.8)
Discontinuation due to Related-TEAE	**0**	**3** (18.8)	**0**	**3** (15.8)
Dose interruption due to Related-TEAE	**0**	**1** (6.20)	**0**	**1** (5.3)
Dose modification due to Related-TEAE	**NA**	**0**[2]	**1** (33.3)	**1** (5.3)

REC-4881 Preliminary Safety

4 mg dose:

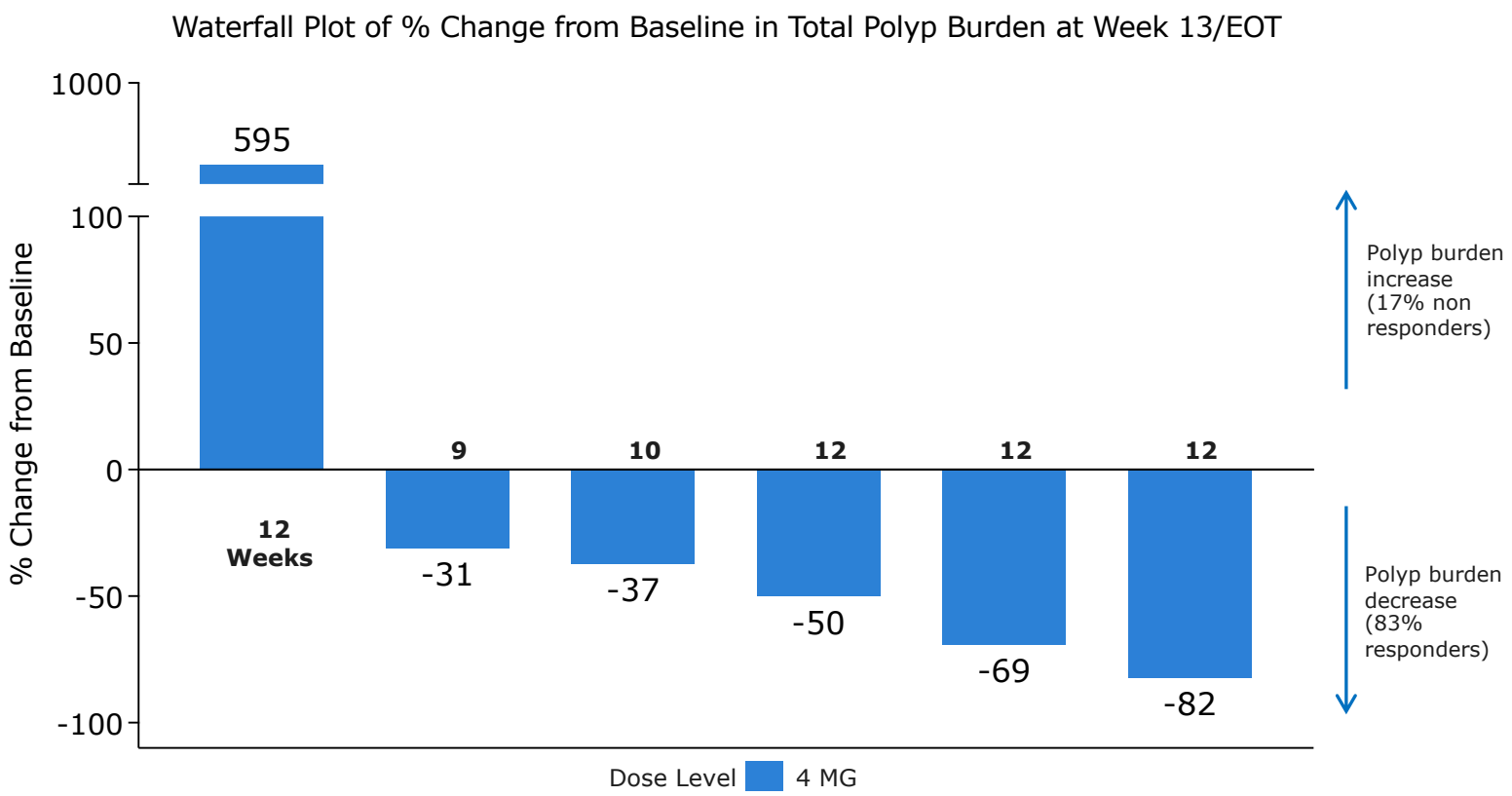
- **Most common TRAEs**: dermatitis acneiform (50%; All G1/2), rash (31.2%; 25% G1/2 and 6% G3), diarrhea (31.2%; All G1/2), blood CPK increase (25%; All G1/2), and LVEF decrease (25%; 19% G1/2, 6% G3)
- **Grade 3 TRAEs:** Rash (6%, n=1), CRP increase (6%, n=1), LVEF Decrease (6%, n=1)[1]

8 mg dose:

- **No Grade 3 TRAEs**
- **Grade 2 TRAEs:** Rash (33%, n=1)

1. LVEF decrease was transient, and patient recovered following drug withdrawal
2. 4 mg is the lowest dose available in this Study
3. For 4mg, N=5 patients in Phase 1b and N=11 patients in Phase 2 were dosed

Preliminary Results: 43% median reduction in total polyp burden on 4 mg REC-4881



Data excludes one 4mg patient who received only 3 weeks of REC-4881 dosing and WK13 endoscopy was performed 10 weeks post last dose. Percent (%) change from baseline calculates the change between post-resection value from screening visit to the pre-resection value at Week 13/EOT visit. Subjects with absolute value of 0 at baseline are not displayed.
Data Snapshot Date: 2025-04-02; Data Cut-off Date: 2025-03-17; Report generated on: 2025-04-28

REC-4881 Preliminary Efficacy

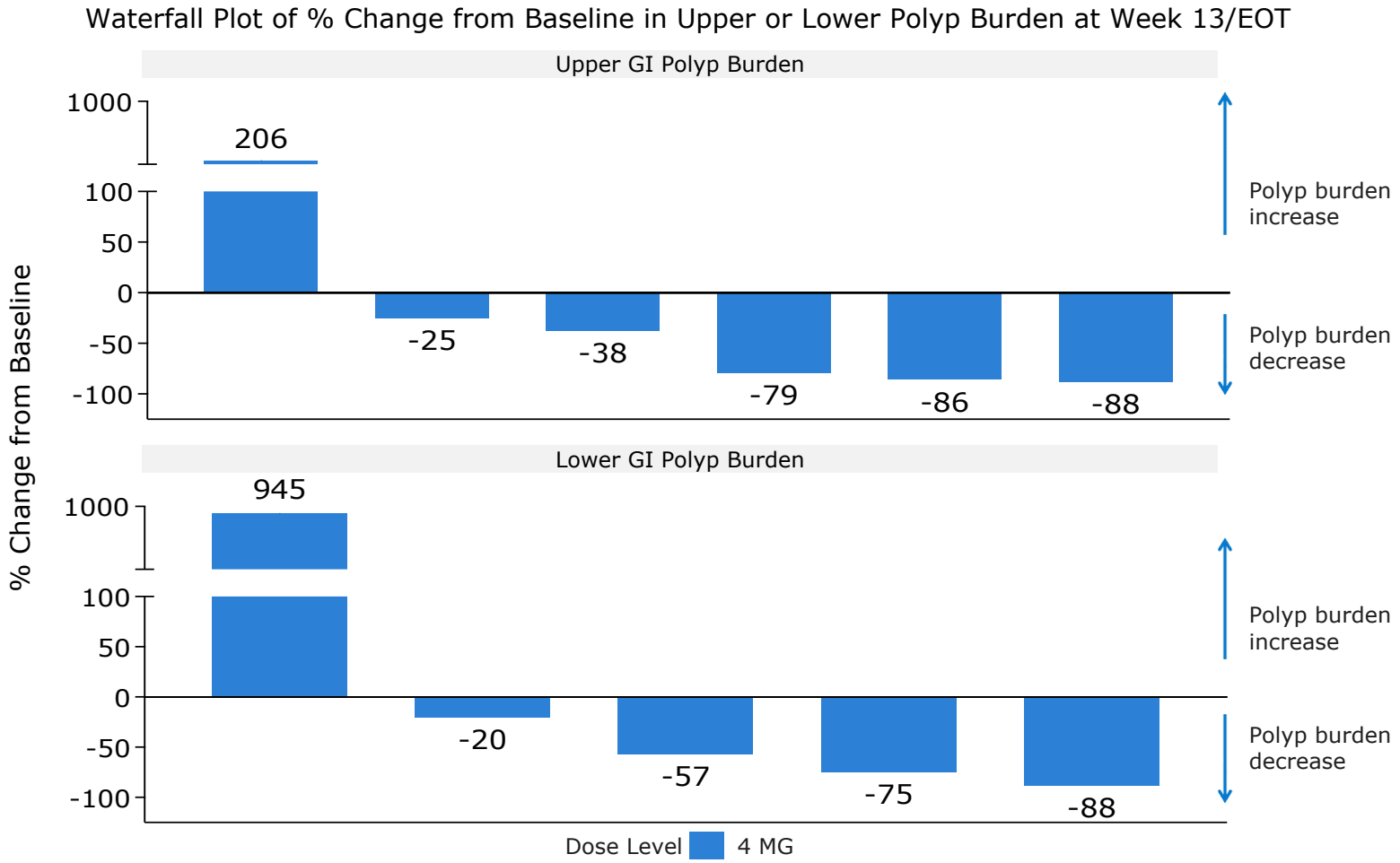
- **6 patients** on 4 mg efficacy evaluable[1]
  - 100% (n=6) on therapy for at least 9 weeks
- **43% median reduction** in total polyp burden
- **> 30% reduction** in total polyp burden (sum of polyp diameters) at week 13 assessment
- **At week 25, 2 out of 2 patients** on the 12-week on/12-week off regimen **maintained a durable >30% reduction[2]**

TUPELO

1. Efficacy Evaluable Population: Defined as all participants who have measurable disease (non-zero polyp burden) at end of baseline endoscopy, received at least 75% of study drug, and have at least one post-baseline on study endoscopic assessment.
2. A third patient, Patient 001-2001, who reached W25, did not perform W25 Assessment

Recursion

Preliminary Results: Reductions in polyp burden seen across upper and lower GI tract



REC-4881 Preliminary Efficacy

- **Upper GI Burden**
  - **6 patients** efficacy evaluable[1]
  - **58% median reduction** in UGI polyp burden
- **Lower GI Burden**
  - **5 patients** efficacy evaluable[1]
  - **57% median reduction** in LGI polyp burden

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1. Efficacy Evaluable Population: Defined as all participants who have measurable disease (non-zero polyp burden) at end of baseline endoscopy, received at least 75% of study drug, and have at least one post-baseline on study endoscopic assessment.

Preliminary Results: 50% of patients on 4 mg REC-4881 demonstrated a reduction in Spigelman stage

Subject ID	Polyp Burden Screening -> W13 (CfB%)	Polyp Count Screening -> W13 (CfB%)	Spigelman Stage Screening –>W13
001-2001	-31%	-22%	NA-> II
016-2001	-50%	-56%	III -> I
016-2002	-69%	-78%	IV -> II
003-2001	-82%	-79%	III -> II
003-2002	-37%	-35%	II -> II
001-2003	+595%	+454%	II -> IV

>20% decrease (PR)	±20% increase/decrease (SD)	>20% increase (PD)

*Spigelman Stage can be confounded by sampling errors

REC-4881 Preliminary Efficacy

- **Two patients** with a 2-point change in Spigelman Stage
- **Effects in upper GI include** polyp burden reduction, polyp count, and Spigelman downstaging

TUPELO

Recursion

REC-4881: MEK1/2 – Summary and Next Steps

Biological Insight

MEK1/2 inhibition identified as a unique mechanism for FAP through an unbiased ML-aided phenotypic screen

In Vivo Data

Significant reduction in **both polyp count** and **precancerous adenomas** at lowest dose tested

Clinical

43% median reduction in total polyp burden at week 13 and **50%** of patients demonstrated **reduction in Spiegelman Stage**

REC-4881 Target Profile

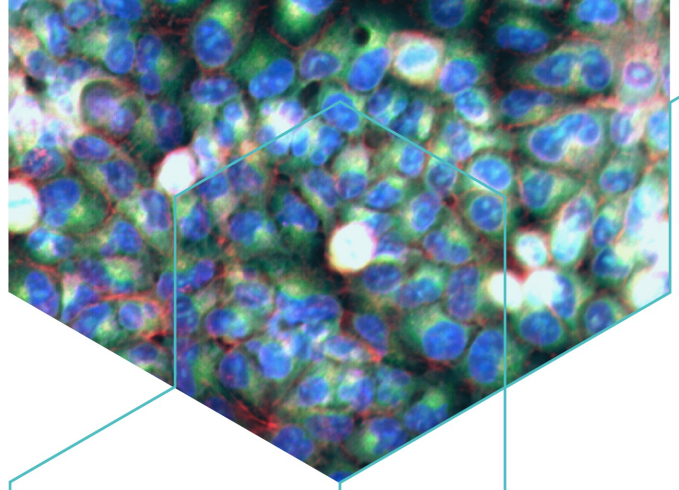
- **Orally bioavailable**, highly potent and selective MEK1/2 inhibitor
- **Differentiated ADME** profile may enhance exposures at the site of GI adenomas
- 4mg dose **well-tolerated** with a safety profile consistent with MEK inhibitors
- **ODD** in US and EU; **FTD** in US

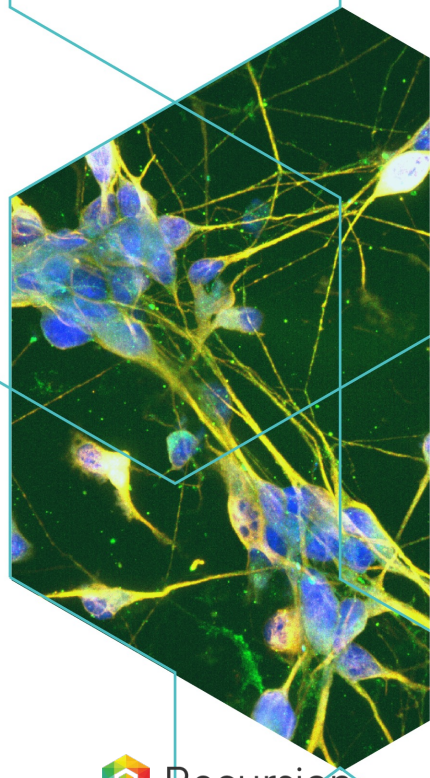
What's Next

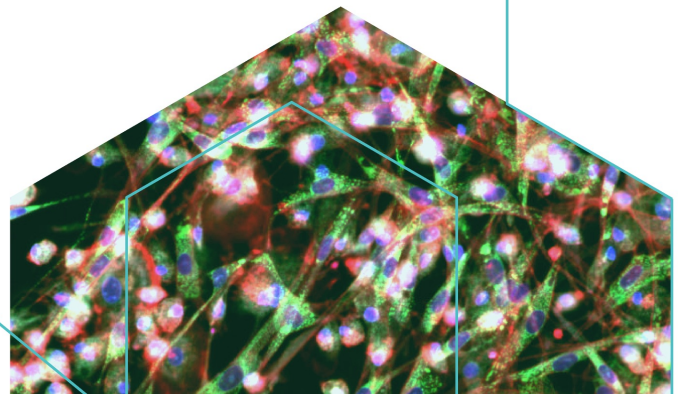
- **Enrollment ongoing;** additional efficacy and safety data, **2H25**

Oncology

PI3Kα H1047R







Recursion

REC-7735: PI3Kα H1047R

A precision designed highly selective for PI3Kα H1047R for PI3K mutant-selective cancers

Unmet need

- **14%[1] of all HR+/HER2- Breast cancer** with PI3Kα-H1047R mutation
- **~11,000[2]** advanced or metastatic patients

Mechanism of Action

- **PI3Kα-H1047R mutant selective inhibitor**

Thesis & Differentiation[3]

- **CNS penetrant** molecule, **>100x selectivity** vs WT PI3Kα
- **Low risk** of AEs
- **Superior in vivo efficacy vs Alpelisib & Capivasertib,** comparable to STX-478
- **Synergy with SERD ± CDK4/6i** combo at **low doses**

Recursion Approach

- AI-powered precision design to **optimize selectivity** and **limit** metabolic liabilities such as **hyperglycemia**
- 242 novel compounds synthesized to candidate ID

Program Status

- Candidate Profiling
- **DC nomination 2H25**

1. Mutation prevalence: Millis et al. (2016); Martinez-Saez et al. (2020)
2. First Line treated incidence for HR+ HER2- BC in US and EU5 from 2024 EvaluatePharma Epi multiplied by mutation prevalence
3. Based on preclinical data

Platform Insight: Recursion OS leveraged to maximize selectivity, developing a structurally differentiated molecule with superior ADME

Biology → Design → Prioritize

Novel insights

- Generated **proprietary structural biology** insights

Molecular Dynamics (MD) simulations

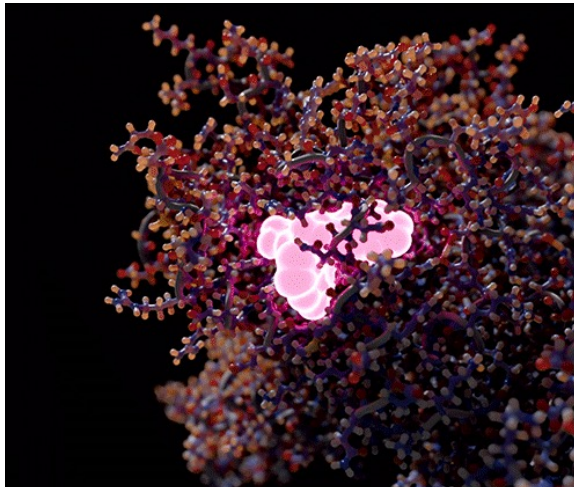
- Utilized to explore binding pocket of interest and ligand flexibility

MD-identified "hotspots"

- To inform efficient ML-guided optimisation

Novel molecules with optimized potency

- Designed using generative 3D evolution and scored with ML and physics-based methods



Class leading selectivity

- Over WT enzyme to maximize efficacy and therapeutic index

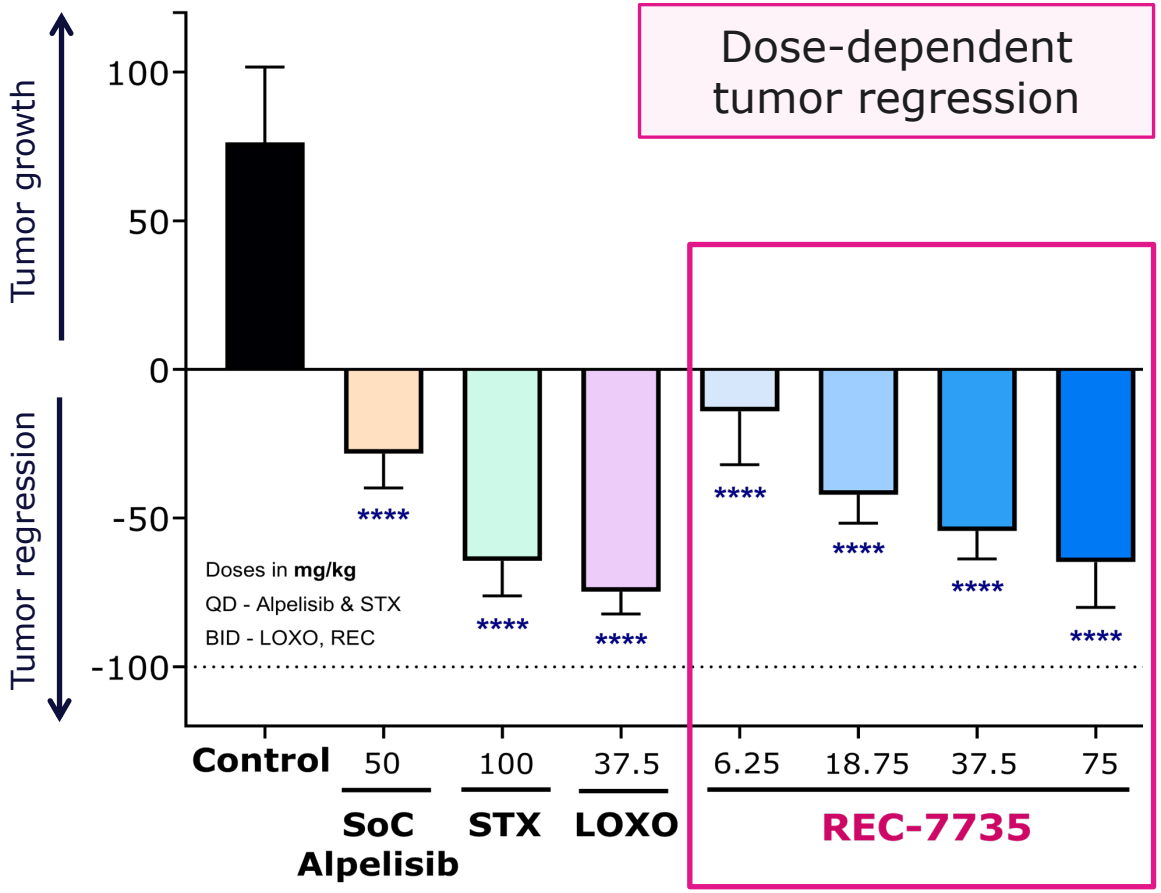
Novel structures

- 2D ML methods guided optimization into distinct and preferable physico-chemical space

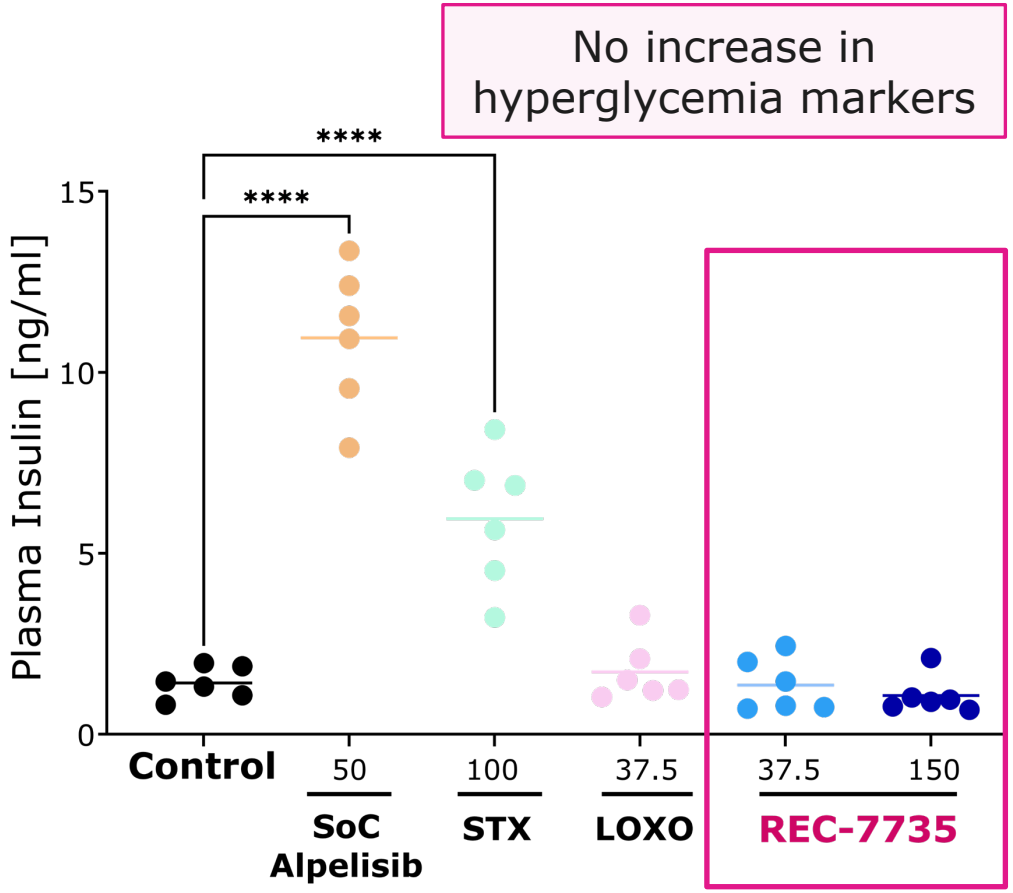
Recursion OS built fit-for-purpose assay cascade and
delivered a differentiated Candidate in **18 months and fewer than 250 compounds**

Demonstrated preliminary competitive efficacy with no hyperglycemia versus SoC in preclinical models

CDX Model: PI3Ka H1047R[1]



Naïve Wildtype Mice[2]



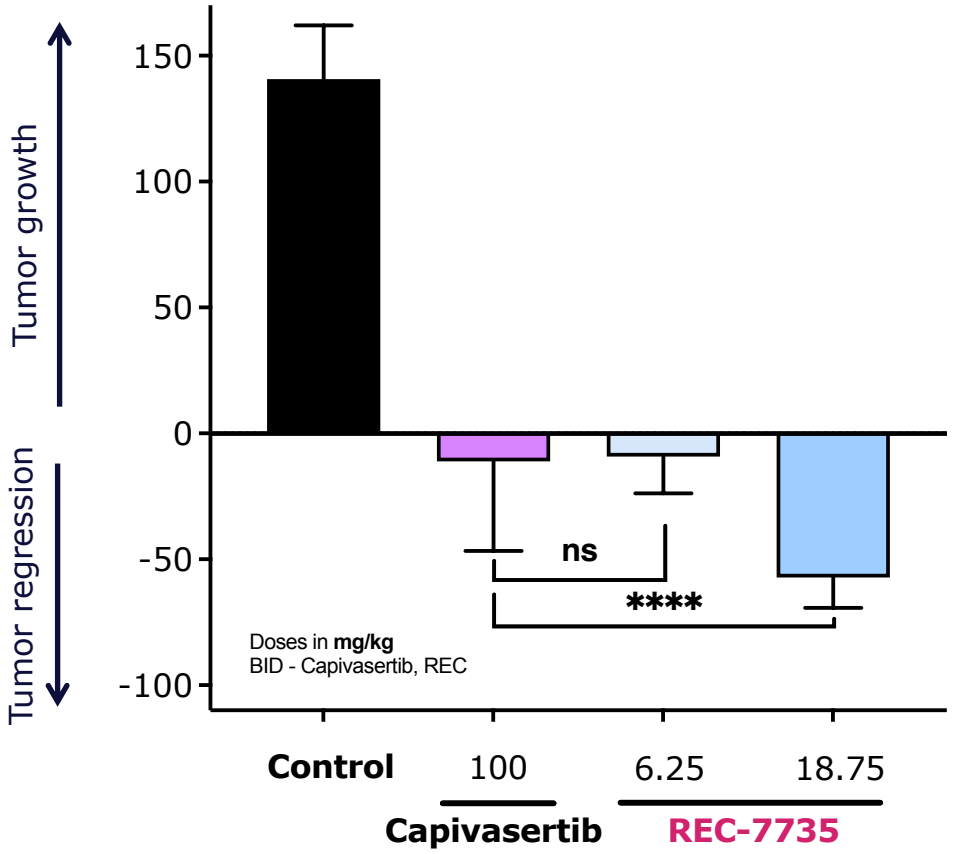
Note: Alpelisib and STX (STX-478) were dosed QD; LOXO (LOXO-783) and REC-7735 were dosed BID. Doses are represented as mg/kg. LOXO has been discontinued

1. 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.
2. In vivo naive 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.

Recursion

Low dose outperforms high dose SoC Capivasertib (AKTi)[1]

CDX Model: PI3Ka H1047R[2]



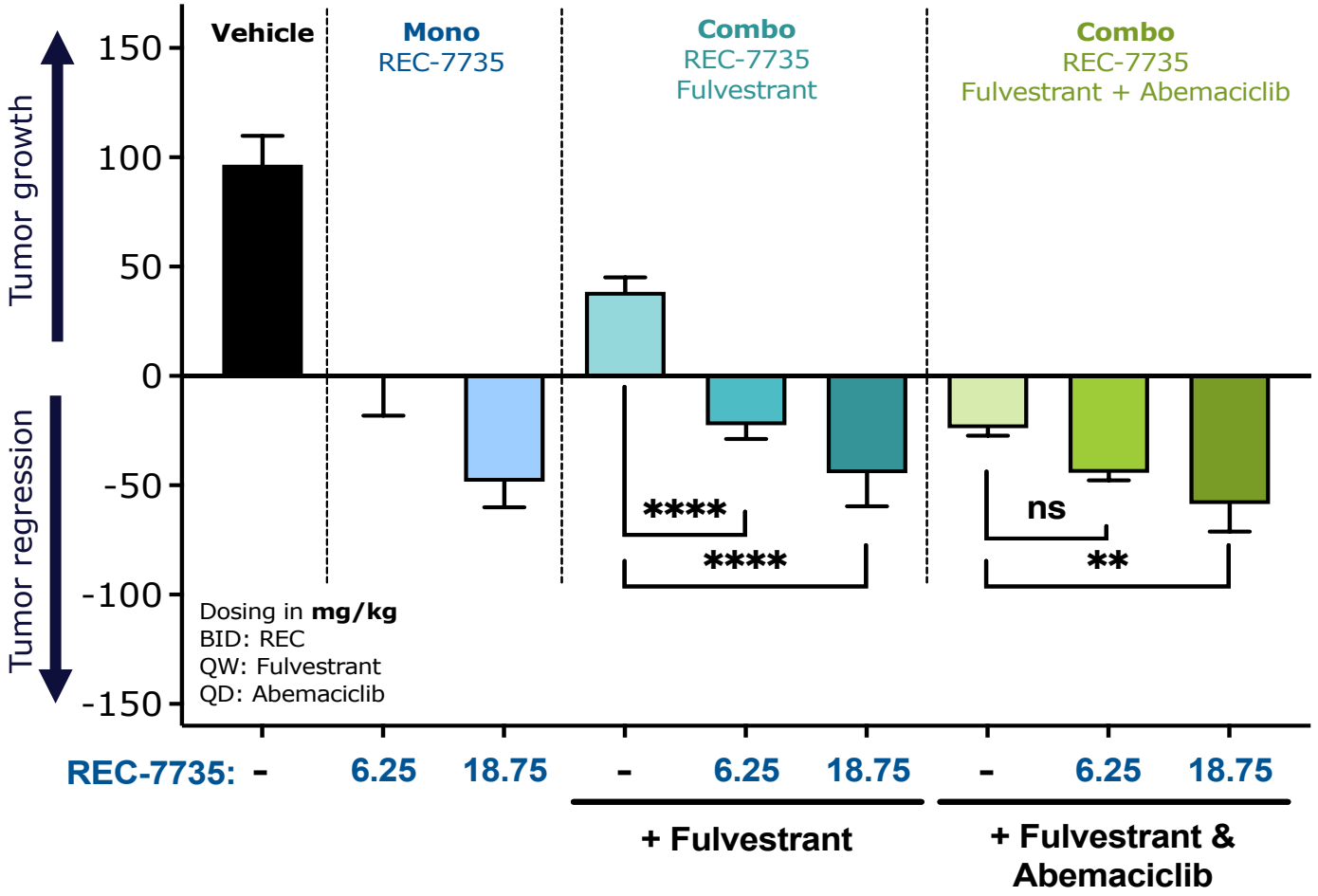
- REC-7735 (6.25mg/kg BID) shows similar anti-tumor activity to Capivasertib (100mg/kg BID)
  - AKT inhibitor, shown promising clinical efficacy with endocrine therapy
- **REC-7735 (18.75mg/kg BID) significantly outperforms high-dose Capivasertib** (100mg/kg BID)
- Capivasertib caused significant body weight loss and animal deaths, while **REC-7735 was well-tolerated**

1. Capivasertib (AKTi) in combination with fulvestrant (SERD) is used in breast cancer (HR+/HER2-) for patients with PIK3CA, AKT1, or PTEN alterationin combination with fulvestrant (SERD).
2. In vivo CDX Model using T47D (PI3Ka H1047R mutant) cell line. n=8 mice per group. Data represents tumor growth inhibition and regression after 21 days of dosing.

Recursion

Low dose outperforms & synergizes SoC Fulvestrant (SERD) ± Abemaciclib (CDK4/6i) combo[1]

CDX Model: PI3Ka H1047R[2]



- REC-7735 shows strong, significant tumor regression at low doses (≥6.25mg/kg BID)
- REC-7735 alone outperforms Fulvestrant/Abemaciclib combination therapy
- Even low-dose REC-7735 significantly enhances the efficacy of Fulvestrant ± Abemaciclib

1. Fulvestrant (SERD) alone and or in combination with Abemaciclib (CDK4/6i) is used in HR+/HER2- advanced or metastatic breast cancer.
2. In vivo CDX Model using T47D (PI3Ka H1047R mutant) cell line. n=8 mice per group. Data represents tumor growth inhibition and regression after 14 days of dosing.

Recursion

REC-7735: PI3Kα H1047R – Summary & Next Steps

Biological Insight

High selectivity for **H1047R mutant PI3Kα** over WT to reduce dose-limiting hyperglycemia

Design

AI-driven generative design via **hotspot molecular dynamics** to discover a **unique chemical series**

In Vivo Data

Significant tumor regressions at low doses **outperforms** clinically approved agents

Clinical

Data supports targeting **H1047R mutant breast cancer** as a **monotherapy** or in **combination** with standard of care treatments

REC-7735 Target Profile

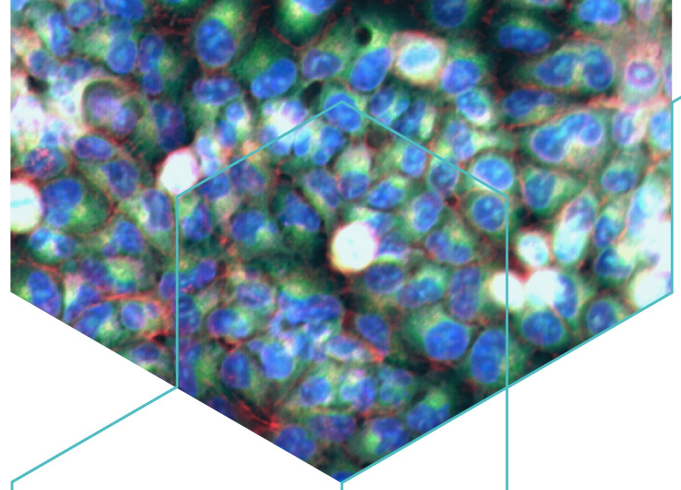
- Potential **best-in-class** PI3Kα H1047R inhibitor
- **>100-fold selective** against WT PI3Kα
- **No significant** in-vitro safety concerns, superior BSEP, off-target & liver spheroid profile **versus competitors**
- **Highly CNS penetrant** with **low-risk** of dose-limiting AEs

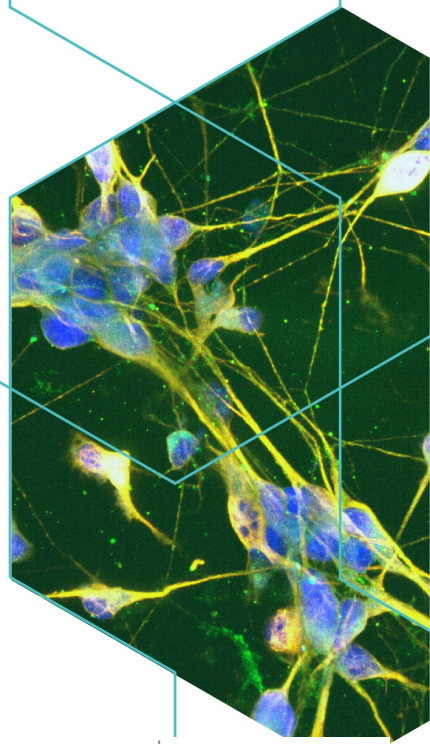
What's Next

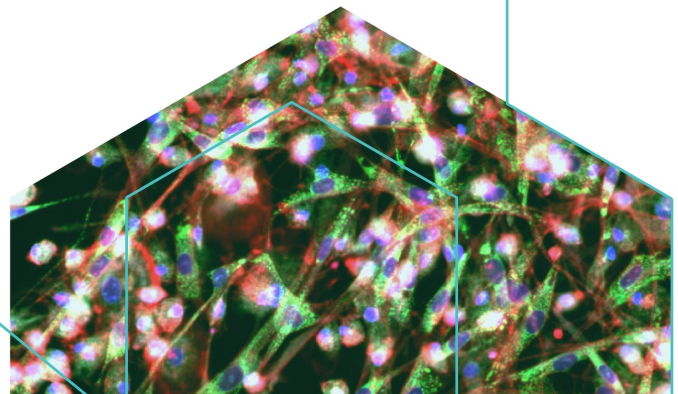
- Development candidate nomination **2H25**

Rare Disease

ENPP1







Recursion | Rallybio

REV102: ENPP1 Inhibitor

A preliminarily non-immunogenic, potent, highly selective ENPP1 inhibitor for hypophosphatasia (HPP) in development with Rallybio

Unmet need[1]

- **~7,800 diagnosed** HPP patients, mostly with limited ERT access
- Opportunity to **significantly reduce costs**

Mechanism of Action

- **Potent, highly selective ENPP1** inhibitor

Thesis & Differentiation

- **First oral disease-modifying therapy**
- **ENPP1** inhibition **a genetically validated** target in HPP models
- **Non-immunogenic** small molecule, potentially safer than ERT
- **More tolerable and affordable** vs ERTs

Recursion Approach[2]

- **Precision designed for** both **high potency, chronic dosing**
- **Maintained selectivity** for candidate with **high oral bioavailability** in clinic

Program Status

- **IND-enabling studies ongoing**
- **Phase 1 initiation expected 2H26**

Note: ERT = Enzyme Replacement Therapy

1. HPP prevalence at birth across US+EU5. Mornet et al, 2020
2. Joint Venture with Rallybio

Recursion | Rallybio

Platform Insight: Precision designed for both high potency and a lifetime of chronic dosing using Recursion OS

Biology | Design | Prioritize

Novel insights

- **ENPP1 inhibition** helps correct phenotypes caused by **LOF mutations in *ALPL* gene**
- **Oral drug** reduces cost and improves convenience and access over ERT

Generative Approach

ML models

Selection Coverage Score

Novel scaffolds

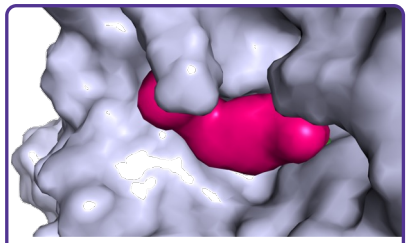
- Structurally distinct molecules to prevent selectivity issues

High bar for safety

- Optimized pharmacophore against closely-related metalloenzymes through structural enablement and 3D / ML model platform

Low (predicted) human oral dose

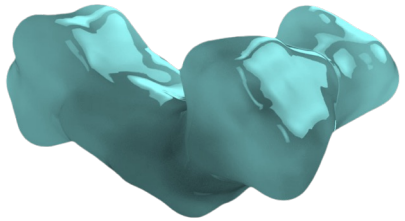
- Highly optimized ADME using robust 2D ML methods



3D interaction filters

Efficient unbiased testing

- Modern use of information theory to select fewest compounds for testing but with maximal information gain



Recursion OS Platform:

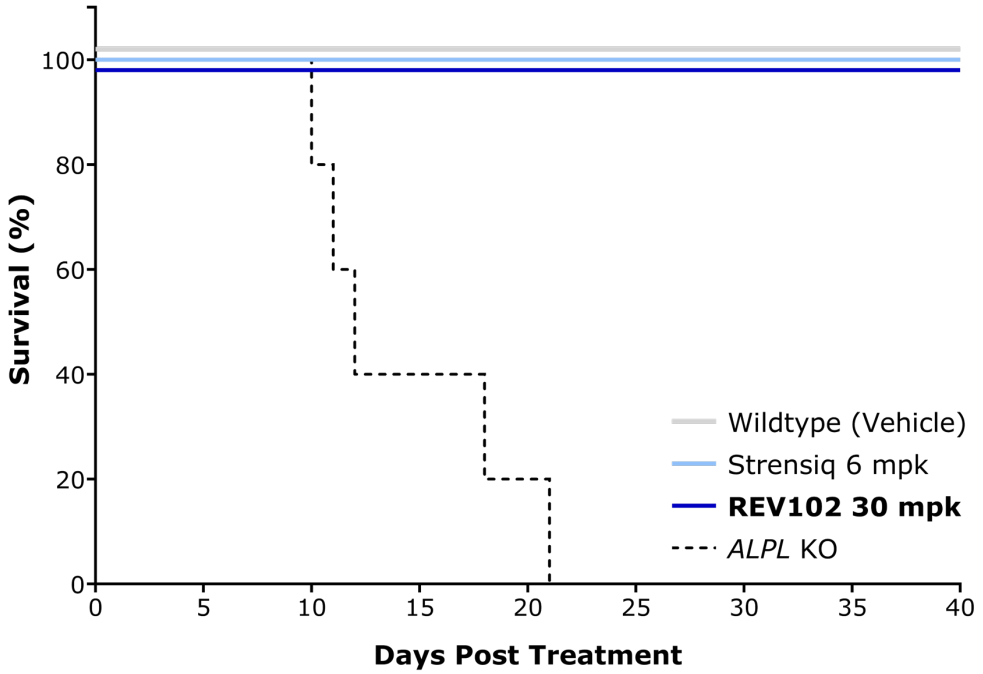
A highly potent, selective, orally bioavailable compound developed through our multi-parameter optimization engine

Recursion | Rallybio

Preclinical: REV102 restores bone health and survival in an unpublished *ALPL* early-onset knockout mouse model of HPP

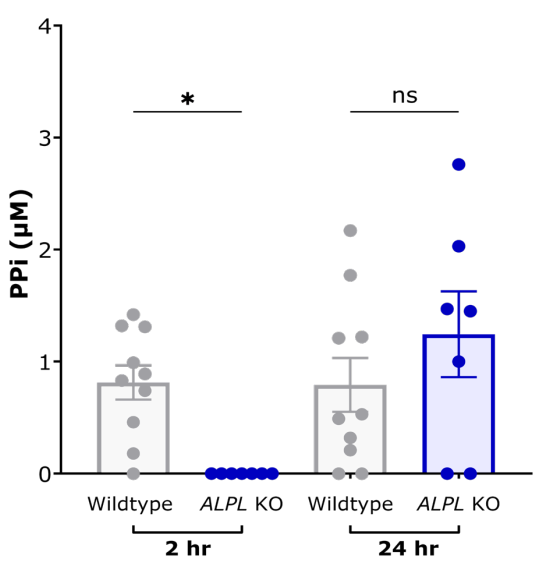
In Vivo Efficacy

Early-Onset HPP Model[1]

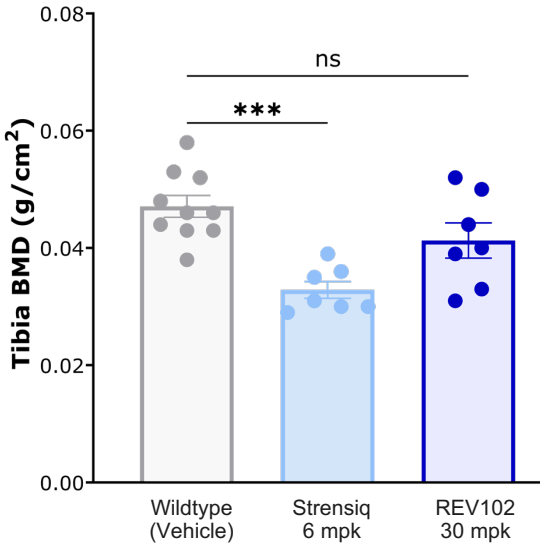


- REV102 **extends survival** in an early-onset *ALPL* KO mice model
- REV102 **transiently reduces PPi levels** to enhance mineralization
- **Tibia BMD** is **fully restored** to **wildtype** levels in treated mice

Plasma PPi[2]



Bone Density[3]



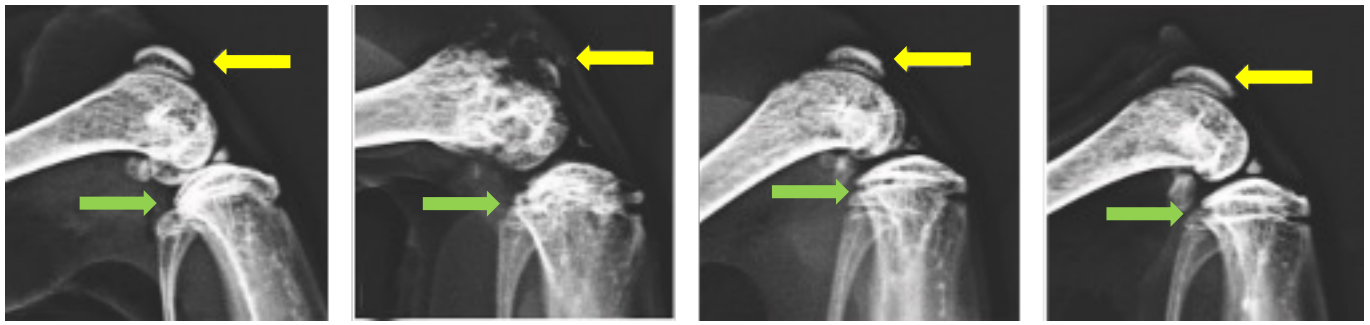
1. N=10 mice in wildtype group, N=5 mice in ALPL KO group, N=7 in Strensiq and REV102 groups. REV102 and Strensiq administered QD SC. Vehicle treated ALPL KO mice stopped growing at day 7-10 and all died at day 22
2. PPi concentrations at 2h or 24h after the last dose of 30 mg/kg REV102. <LLOQ data points are shown on the graph as 0 values.
3. ALPL KO group not shown given all mice die around weaning age

Recursion | Rallybio

Preclinical: Chronic dosing of REV102 improves bone phenotype in a late-onset knockout mouse model of HPP

In Vivo Efficacy[1]

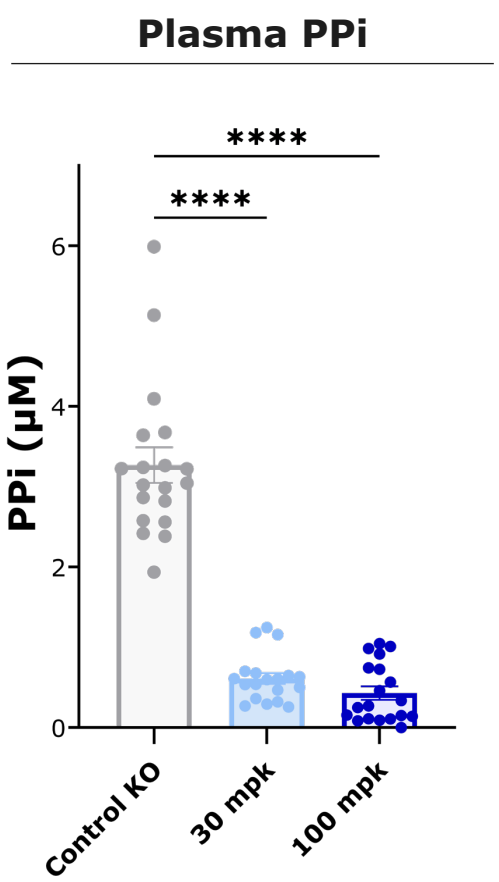
Late-Onset HPP Model[2]



Wildtype | Control KO | REV102 30mpk | **REV102 100mpk**

Patella (yellow arrow)
Growth plate (green arrow)

- Untreated mice present **underdeveloped patellofemoral** joints
- REV102 treatment corrects bone defect and **improves patella structure**
- REV102 treatment **significantly reduces plasma PPi concentrations**



1. Unpublished, data in collaboration with Professor Jose Luis Millán
2. Foster et al. 2017.

Recursion | Rallybio

REV102: ENPP1 – Next Steps

Biological Insight

Reduction of PPi production via controlled ENPP1 inhibition to restore bone hypomineralization

Design

AI-driven generative design via **fragment screening** to enhance **metalloenzyme selectivity**

In Vivo Data

Significant survival benefit in HPP mice through transient PPi reduction validates mechanistic rationale

Clinical

Opportunity to address significant unmet needs in **juvenile** and **adult-onset HPP patients**

REV102 Target Profile

- Potential **first-in-class** ENPP1 inhibitor
- **High oral bioavailability** supports QD or BID dosing
- **No kinases** inhibited >70% at 10 μM
- No significant in-vitro safety liabilities identified

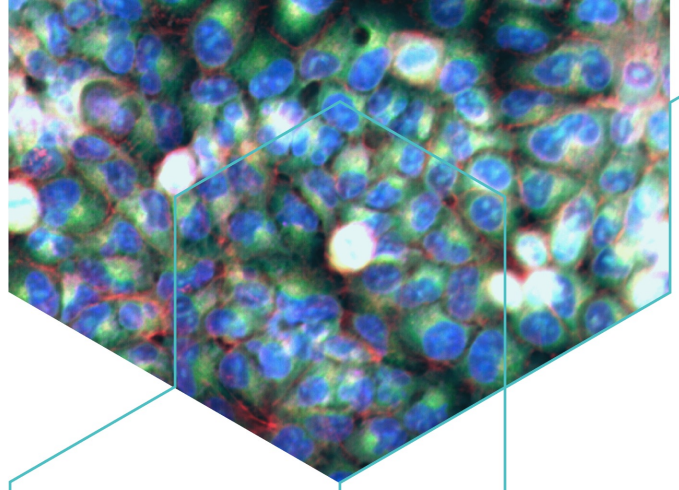
What's Next

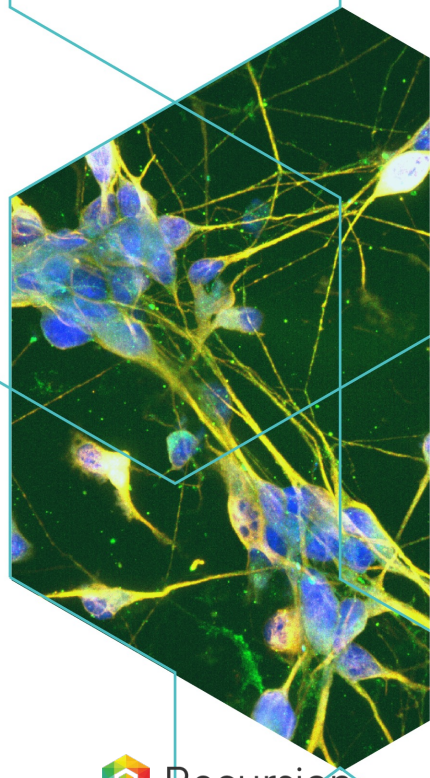
- IND-enabling studies **ongoing**
- **Phase 1 initiation 2H26**

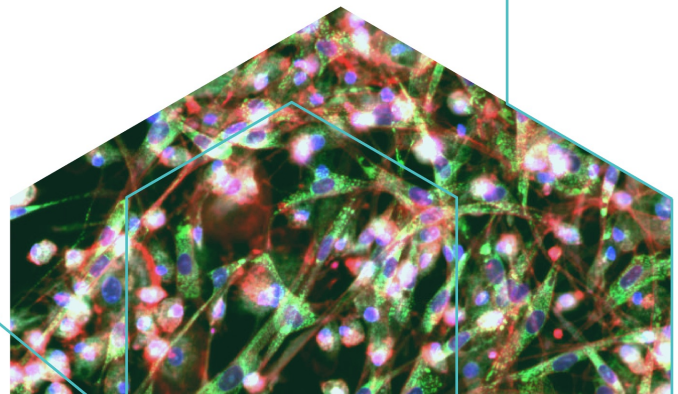
Clinical Portfolio

Oncology

RBM39 · CDK7 · MALT1





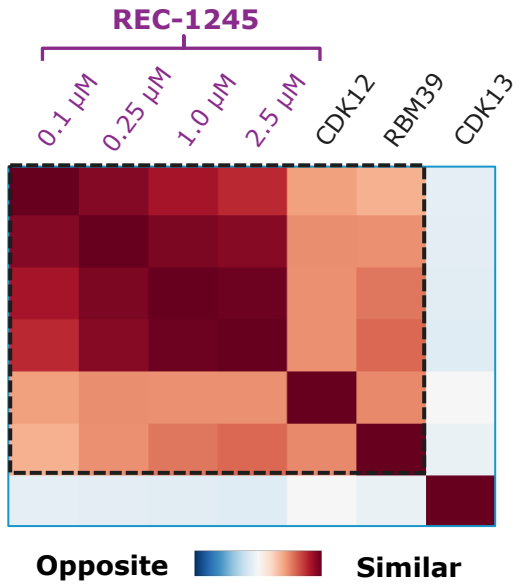


Recursion

REC-1245 (RBM39 degrader): Summary & next steps

Monotherapy dose escalation ongoing with preliminary update 1H26

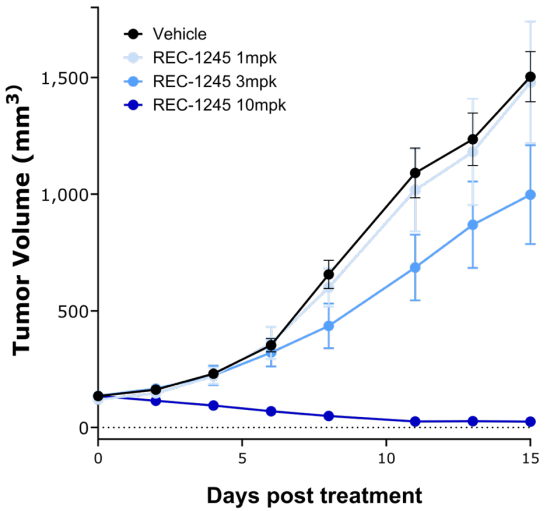
Recursion OS Insight | Preclinical Validation | Clinical Development



Identified through phenotypic discovery platform

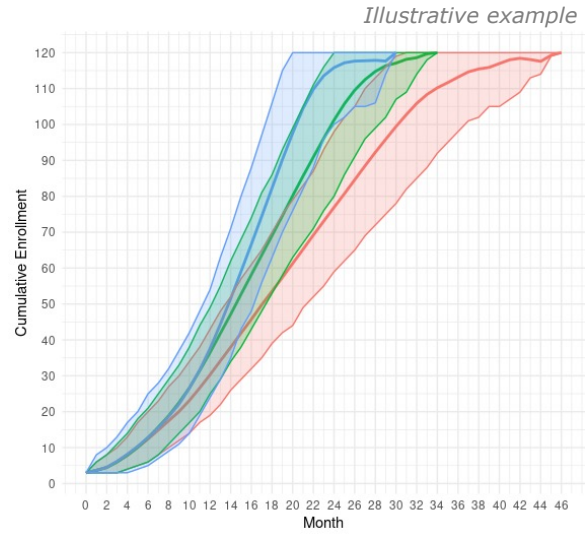
- Novel target mimicking CDK12 loss
- Hypothesized to modulate DDR

CDX Model: OVK18[1]



Compelling efficacy and PK/PD with REC-1245 treatment

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



Ongoing ClinTech work to accelerate enrollment and identify new indications

- Using RWD to identify quality sites for faster enrollment
- Ongoing causal AI modeling to explore potential additional indications

1. N=8 mice per group in TV portion. REC-1245 administered BID PO.
2. PD evaluated after 5 days BID oral of REC-1245 at doses noted; N=3 mice per group in PD portion.

Recursion

REC-617 (CDK inhibitor): Summary & next steps

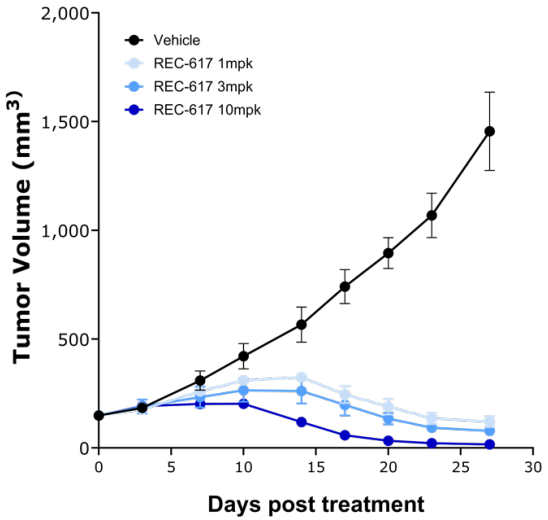
Monotherapy dose escalation ongoing with update 1H26; Combination study to initiate 1H25

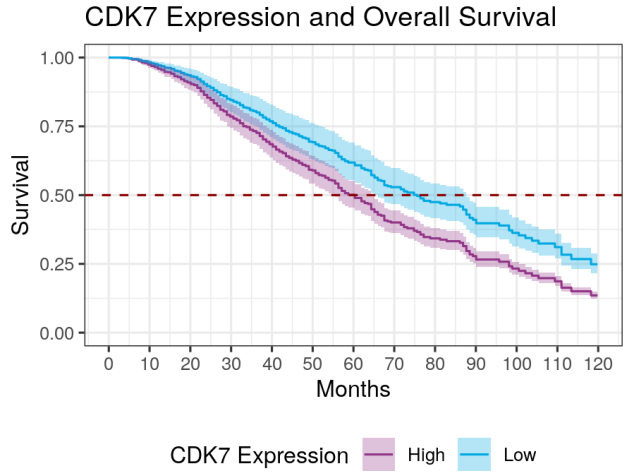
Recursion OS Insight → Preclinical Validation → Clinical Development

Target affinity and selectivity	CDKC IC_{50} (nM)	
	CDK family selectivity	
Cell potency	HCC70 (breast cancer) IC_{50} (nM)	
	OVCAR-3 (ovarian cancer) IC_{50} (nM)	
Safety and metabolism	hERG IC_{50} (μM)	
	Human microsome Clint μL/min/mg	
	Human hep Clint μL/min/10^6 cells	
ADME	Caco-2 A2B (efflux) 10^6 cm/s	
	pH 7.4 μg/mL	
	F % (p.o.)	

136 novel compounds
to Candidate ID in **<12 months**

CDX Model: OVCAR[1]





Designed to optimize PK/PD and maximize potential therapeutic index

- 2D ML to optimize ADME for high permeability and low efflux
- 3D ML maximized key pocket interactions & selectivity against related kinases

Potent tumor regression with REC-617 treatment

- <10 hours of exposure above IC80 to optimize benefit-risk
- No body weight loss

Human Genetics Data and Cell Line screens to validate new indications

- Causal AI approach to determine impact of CDK7 inhibition on survival
- 2 new potential indications identified

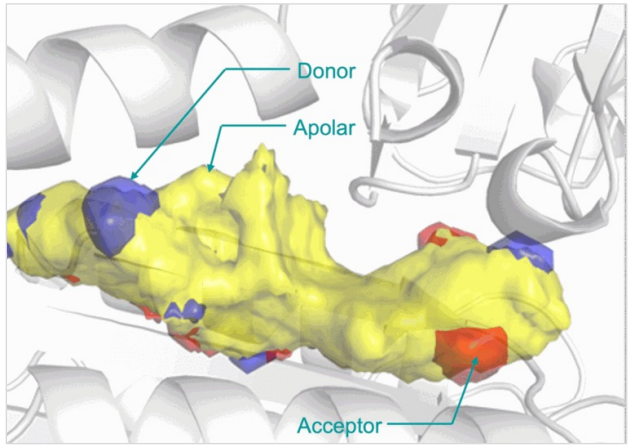
1. Besnard et al, AACR (2022)
2. By RECIST 1.1. Response evaluation criteria in solid tumors, PR: decrease of more than 30% in the sum of the longest diameters of target lesions + no new lesions + no progression of non target lesions.

Recursion

REC-3565 (MALT1 inhibitor): Summary & next steps

Monotherapy dose escalation ongoing with preliminary update 1H26

Recursion OS Insight | Preclinical Validation | Clinical Development

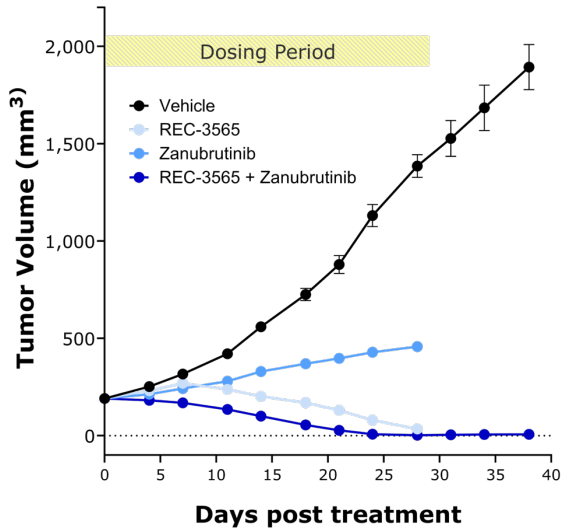


344 novel compounds
to Candidate ID

Designed to deliver balanced compound with improved safety (UGT1A1) and efficacy

- Leveraged molecular dynamics & hotspot analysis

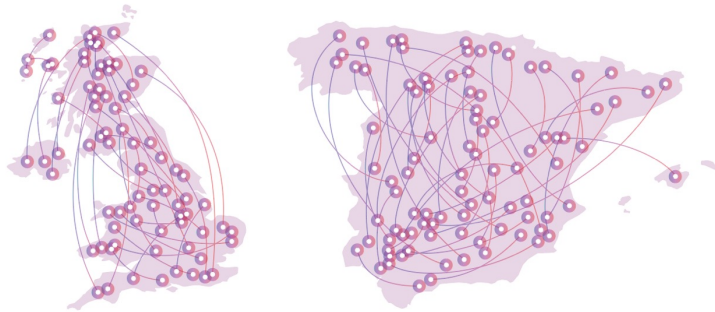
CDX Model: OCI-Ly10[1]



Single-agent and synergistic activity

- Single agent showed tumor growth regression
- 70% of mice in combo arm had no palpable tumors 10-days after last dose

Illustrative example



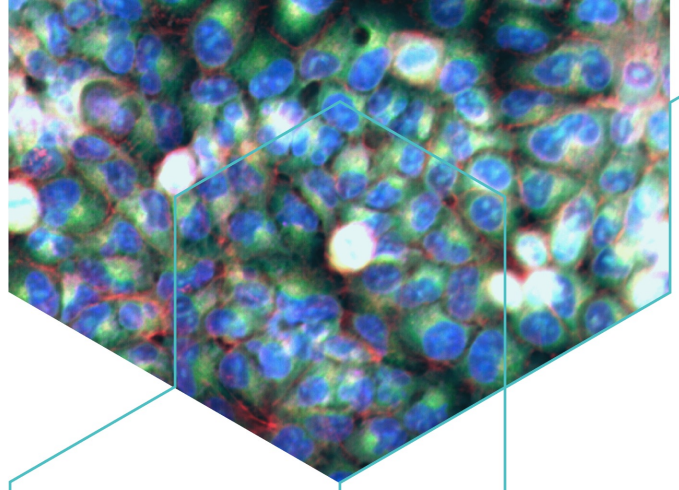
RWD to combat competition for trial enrollment

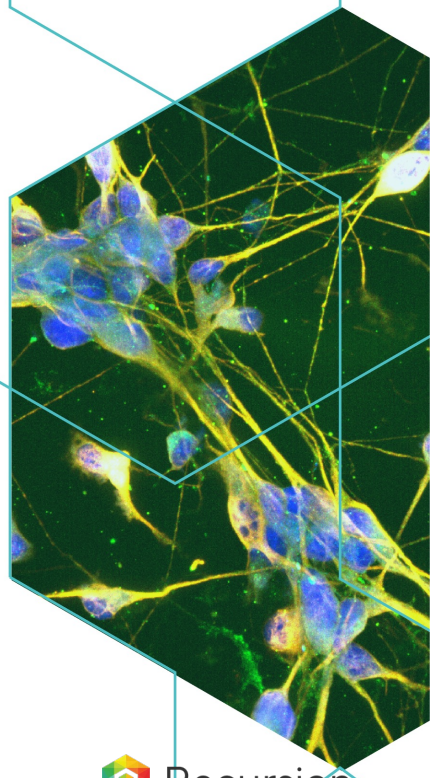
- Advanced analytics for strategic site recommendations and patient targeting
- >50 new potential sites identified in UK and Spain

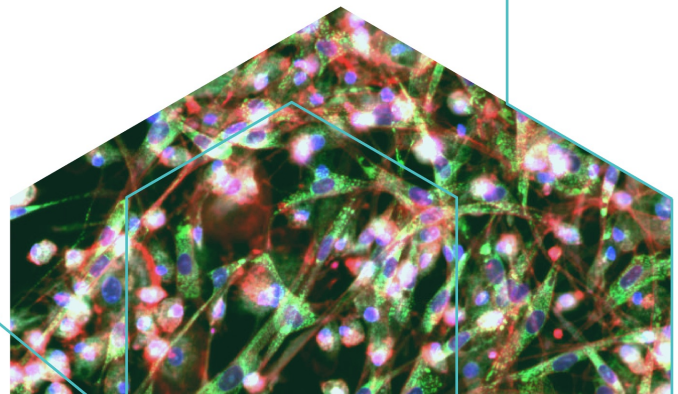
1. Payne et al. ENA, (2024).

Portfolio

Partnered Programs







Recursion

Sanofi collaboration advancing novel targets in I&I and oncology – 4 milestones achieved, multiple additional expected

Biology → Design → Optimize

Leveraging suite of tools tailored for each program to collaboratively identify and drive up to Development Candidate[1]

State of the art **in-vivo facilities & biology labs**

- In-Vivomics
- Digital Toxicology
- Bespoke biological validation

Applying **Recursion OS** cutting-edge generative design platform

- Gambit
- MMPA
- Retrosynthesis
- ML based MPO filter options

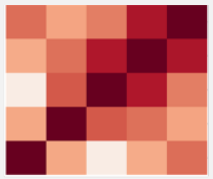
QM/MD simulations to explore protein and ligand flexibility

- ABFE
- RBFE
- Co-folding
- QM based confirmational analysis

Active Learning to maximize optimization and exploration

- GP Learning
- Coverage Score

Future Biological Insights to be identified from Recursion Phenomap



Recursion OS Platform

4 **Program milestones achieved** to date

What's next

- **Complete first development candidates** and advance programs into the clinic
- Continue to **advance broad pipeline** of first-in-class and best-in-class medicines with Recursion OS

1. Sanofi to take Development Candidate through IND enabling studies, clinical trials, regulatory approval & commercialization

Roche and Genentech collaboration within Neuroscience and GI Oncology Indication – unbiased novel biological insights to programs

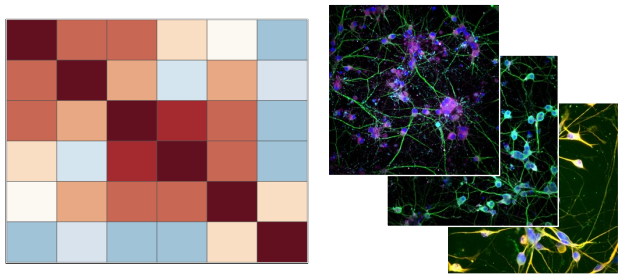
Biology | Design | Optimize

~5 Phenomaps

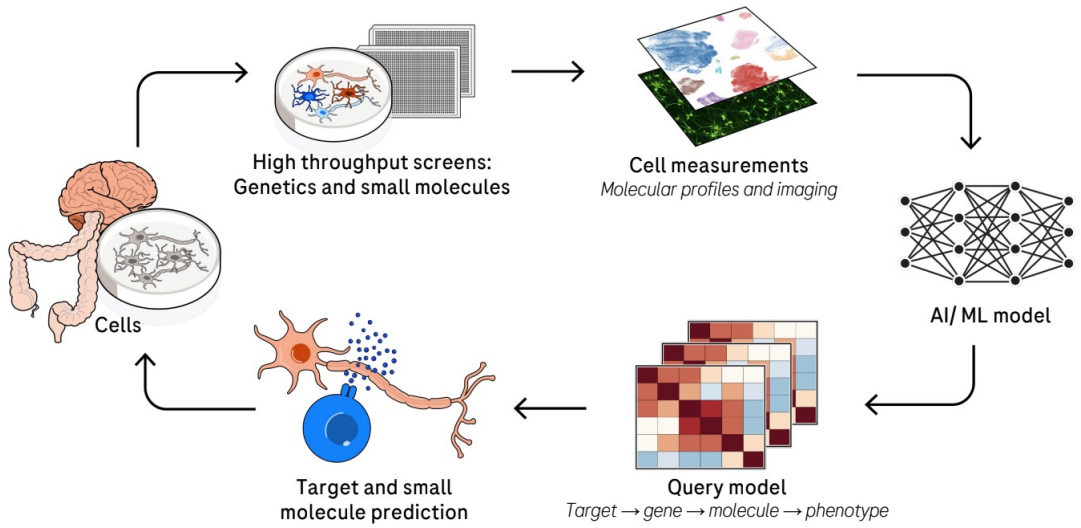
Derived from over
1 Trillion iPSC cells and
100 Billion GI Onc relevant cells

~5,000 transcriptomes

From multiple disease-relevant cell types, subjected to **compound treatments and/or gene KO**, resulting in **~ 171 TB of data**



Lab in the Loop



Collaboratively working to identify novel biological insights from phenomaps for validation

Recursion OS Platform

What's next

- **Additional phenomap** builds ongoing
- Leveraging Recursion OS and collaborating with Roche and Genentech to **identify new programs** in a GI Oncology Indication & Neuroscience

Lab in the Loop image credit: Genentech 2024

Internal and External Pipeline Momentum

FY 2025 and 2026 Upcoming Pipeline Catalysts

2025

H1

REC-617 (CDK7i) in advanced solid tumors
Initiation of combination studies

H2

REC-4881 (MEK1/2i) in FAP
Additional safety and efficacy data from TUPELO

REC-617 (CDK7i) in advanced solid tumors
Additional Phase 1 data from ELUCIDATE

REC-7735 (PI3Kα H1047Ri)
DC nomination

2026

H1

REC-1245 (RBM39)
Early safety and PK from monotherapy trial

H2

REC-3565 (MALT1)
Early safety and PK from monotherapy trial

REV102 (ENPP1i)
Ph1 initiation – 2H26

Partnership Catalysts

Potential for **additional phenomap options**

Potential for **multiple new project initiations**

Potential for **multiple programs optioned** by partners

Roche Genentech
Merck KGaA Darmstadt, Germany
sanofi
BAYER

Recursion

Financial Update

Recursion

Focusing resources on core capabilities to improve probability of success in the industry

- Prioritized pipeline and streamlined operations
- Reduced platform costs associated with capacity, while protecting capability
- Continued investment in key aspects of platform to drive near and long-term value
- Delivering on partnerships and continue to achieve cash-generating milestones
- Limited expected impact to business from macro factors (China & tariffs)

$509.2 million in cash[1] as of March 31, 2025

1Q25 cash burn[2] of $118 million, excluding:
- Partnership inflows
- Financing inflows
- Transaction costs

Expected cash runway until mid-2027

1. Cash, cash equivalents and restricted cash
2. Cash burn, defined as operating cash flow less capital expenditures, excluding partnership and financing inflows and approximately $16m of transaction expenses

On track to achieve substantial cost savings as a combined entity

FY2024

$606 million
combined cash burn, excluding partnership and financing inflows:

Cash flow items[1]	2024 ($m)
RXRX cash flow from operating activities	(359)
RXRX capital expenditures	(14)
RXRX partnership inflow addback	(30)
EXAI pre-combination net cash flows	(184)
EXAI partnership inflow addback	(19)
Total combined	**$(606)**

FY2025E

≤$450 million
expected cash burn, excluding potential partnership and financing inflows:

Primary areas of budget savings:

- Pipeline prioritization
- Duplicated corporate expenses
- Reduction in capacity of platform
- Increasing administrative efficiency
- Rationalization of facilities and office locations
- Greater purchasing power with vendors
- Carve out of Austrian operations

1. RXRX numbers are for FY2024; EXAI pre-combination net cash flows (November 20, 2024 cash minus January 1, 2024 cash for EXAI), EXAI partnership inflows of $19 million for FY24

Recursion 2.0

Recursion

Solidifying Recursion's leadership in TechBio

Our sustainable and continued growth plan

- **Commitment to internal pipeline** – Strong investment behind more focused pipeline
- **Execution on partnerships** – Fully resourced to deliver on programs and achieve milestones
- **Transforming drug discovery and development** – Expanding leadership in AI-based drug discovery and automation by advancing differentiated molecules
- **Increasing efficiency** – Our scale and technologies continue to allow us to do more with less
- **Investment in Recursion OS** – Integrated end-to-end tech stack driving better decision making

Recursion

Corporate Deck May 2025

MAY 2025