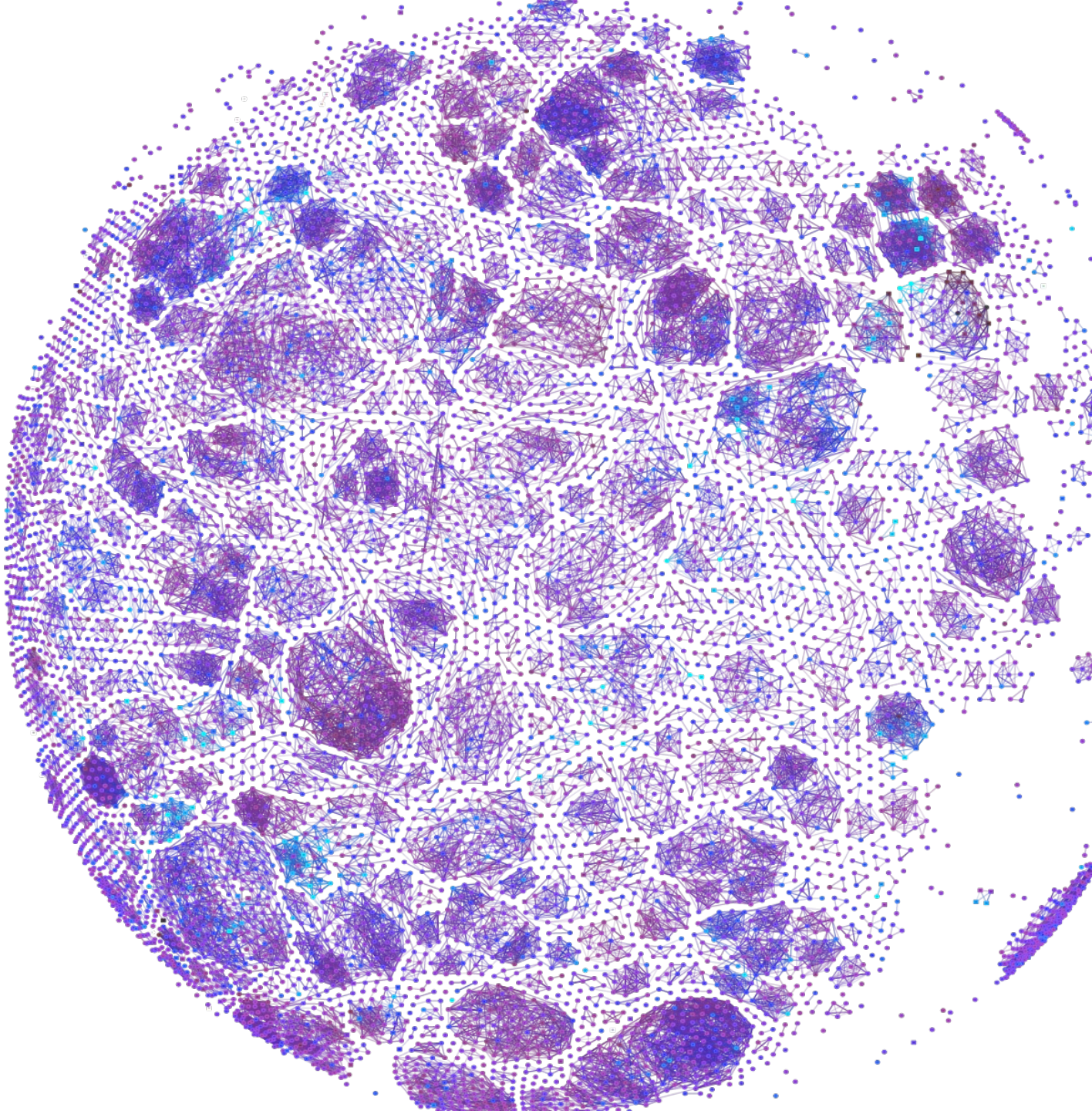
Recursion®

Decoding Biology To Radically Improve Lives

February 2024



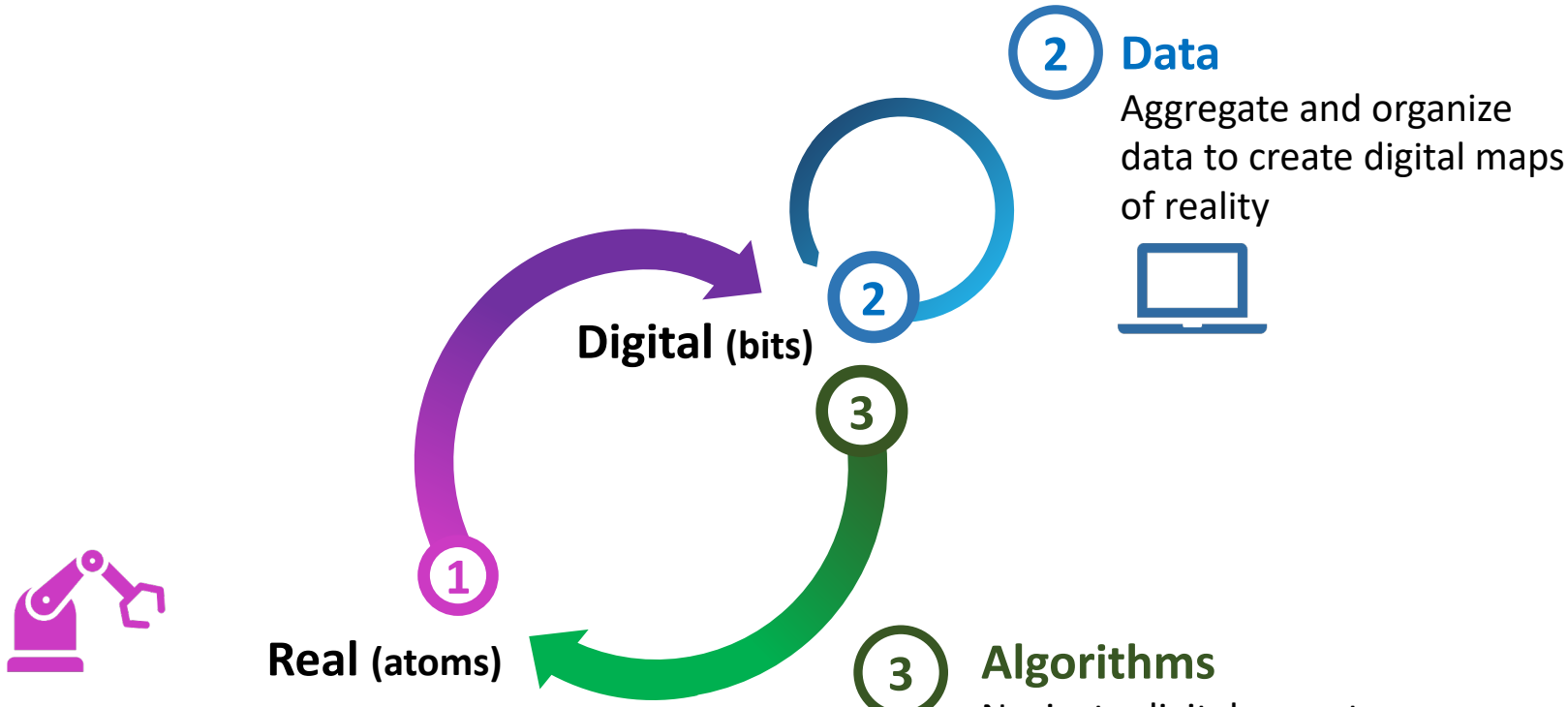
Disclaimers

This presentation and any accompanying discussion and documents contain information that includes or is based upon "forward-looking statements" within the meaning of the Securities Litigation Reform Act of 1995. These forward-looking statements are based on our current expectations, estimates and projections about our industry and our company, management's beliefs and certain assumptions we have made. The words “plan,” “anticipate,” “believe,” “continue,” “estimate,” “expect,” “intend,” “may,” “will” and similar expressions are intended to identify forward-looking statements. Forward-looking statements made in this presentation include outcomes and benefits expected from the Tempus partnership, including our ability to leverage the datasets acquired through the license agreement into increased machine learning capabilities and accelerate clinical trial enrollment; outcomes and benefits expected from the Enamine partnership, including the generating and co-branding of new chemical libraries; our planned expansion of the BioHive supercomputer capabilities; outcomes and benefits from licenses, partnerships and collaborations, including option exercises by partners and additional partnerships and ability to house tools on the BioNeMo Marketplace; the potential for additional partnerships and making data and tools available to third parties; advancements of our Recursion OS, including augmentation of our dataset; outcomes and benefits expected from the Large Language Model-Orchestrated Workflow Engine (LOWE); the occurrence or realization of any near- or medium-term potential milestones; the initiation, timing, progress, results, and cost of our research and development programs and our current and future preclinical and clinical studies, including timelines for data readouts, the potential size of the market opportunity for our drug candidates; our ability to identify viable new drug candidates for clinical development and the accelerating rate at which we expect to identify such candidates; our expectation that the assets that will drive the most value for us are those that we will identify in the future using our datasets and tools, and many others. Forward-looking statements made in this presentation are neither historical facts nor assurances of future performance, are subject to significant risks and uncertainties, and may not occur as actual results could differ materially and adversely from those anticipated or implied in the forward-looking statements. For a discussion of factors that could affect our business, please refer to the "Risk Factors" sections in our filings with the U.S. Securities and Exchange Commission, including our Annual Report on Form 10-K for the Fiscal Year ended December 31, 2023. This presentation does not purport to contain all the information that may be required to make a full analysis of the subject matter. We undertake no obligation to correct or update any forward-looking statements, whether as a result of new information, future events or otherwise.

Certain information contained in this presentation relates to or is based on studies, publications, surveys and other data obtained from third-party sources and the company’s own internal estimates and research. While the company believes these third-party sources to be reliable as of the date of this presentation, it has not independently verified, and makes no representation as to the adequacy, fairness, accuracy or completeness of, any information obtained from third-party sources. In addition, all of the market data included in this presentation involves a number of assumptions and limitations, and there can be no guarantee as to the accuracy or reliability of such assumptions. Finally, while the company believes its own internal research is reliable, such research has not been verified by any independent source.

Any non-Recursion logos or trademarks included herein are the property of the owners thereof and are used for reference purposes only.

There is a formula for mapping and navigating complex systems using technology

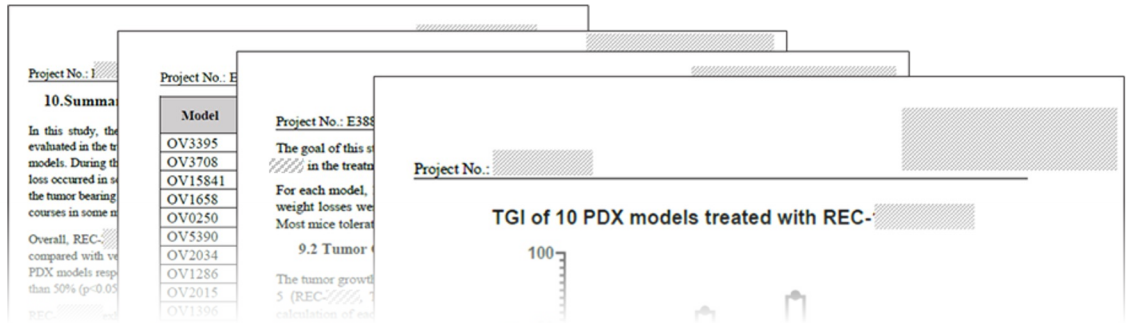


Recursion

Data roadblocks make mapping and navigating biology difficult

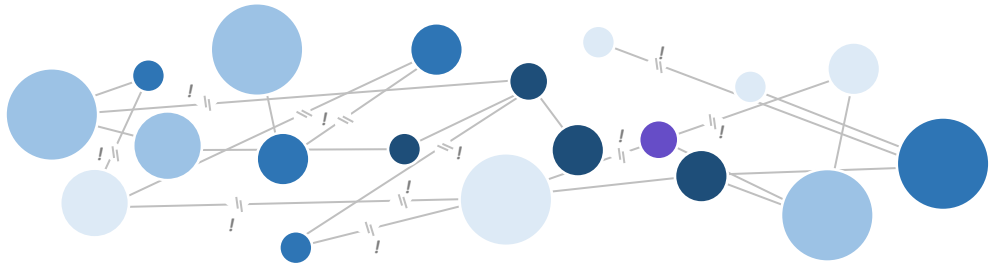
Analog Standard

The fax machine is alive and well in medicine, while in biopharma, study results from CROs are still often reported as PDFs or scanned printouts



Siloed Data in Pharma

Biopharma has 100s of petabytes of scientific data stored on a project-by-project basis without the meta-data or annotation needed to relate it to other projects or questions in biology



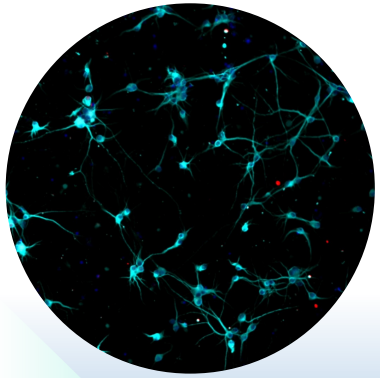
Reproducibility Crisis

Multiple studies have shown that the vast majority of published academic literature cannot be recapitulated

Baker, M. Irreproducible biology research costs put at $28 billion per year. Nature (2015). https://doi.org/10.1038/nature.2015.17711

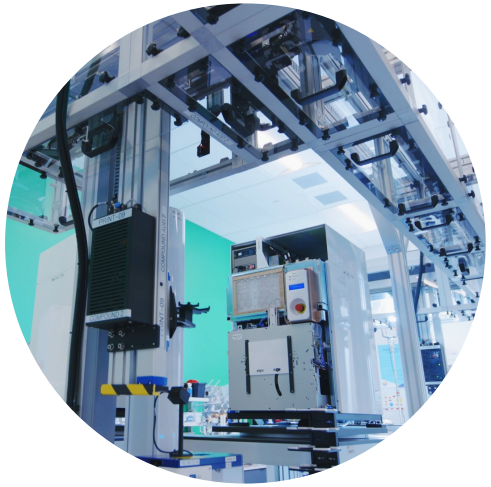
Recursion

We are building and aggregating the right datasets to map and navigate biology



Data
Each week we digitize millions of our own experiments across multiple layers of biology from cell to animal

Profile Systems
We have built and continue to scale among the world's most prolific automated wet labs

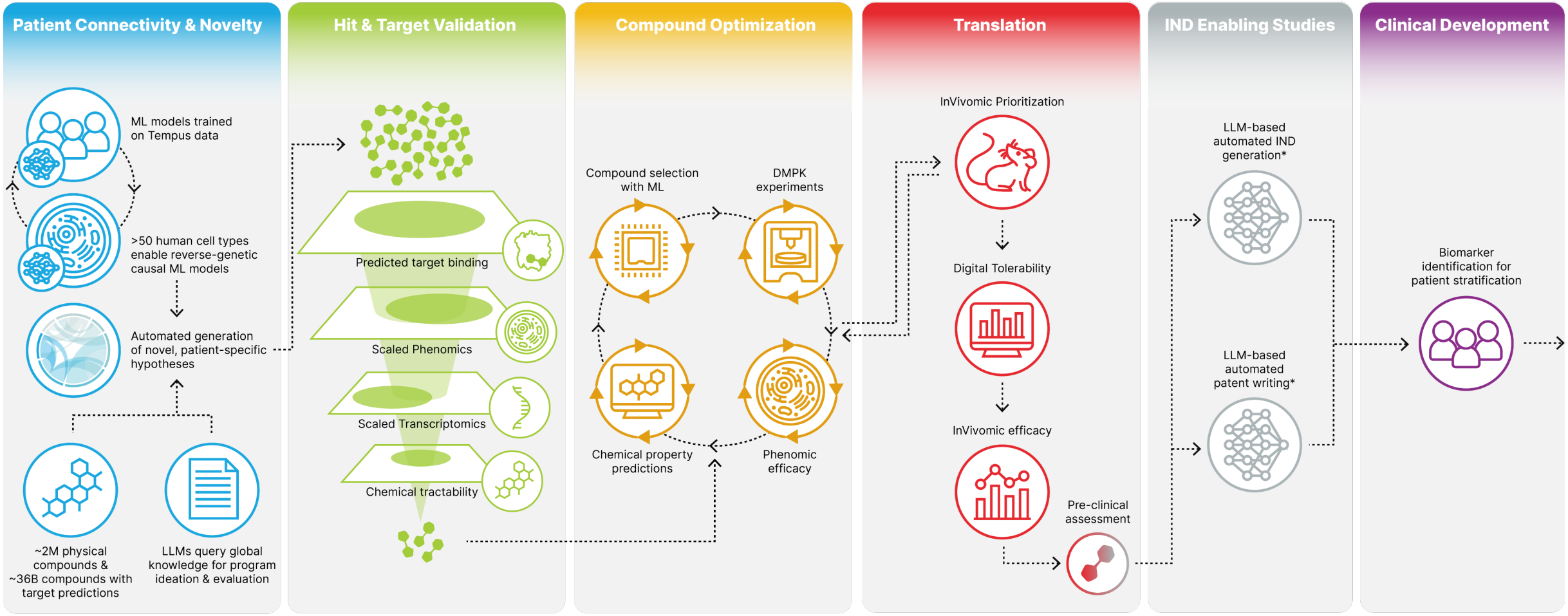


Algorithms
We own and operate one of the fastest supercomputers on earth, allowing us to train LLMs & FMs fit for the purpose of drug discovery

Improved and scaled clinical pipeline

Recursion

The Recursion OS combines many tools to industrialize drug discovery



*Currently being explored

Recursion

In Brief:
The Recursion Value Proposition

New programs are initiated automatically by LLMs tuned to act on Recursion data arbitrage

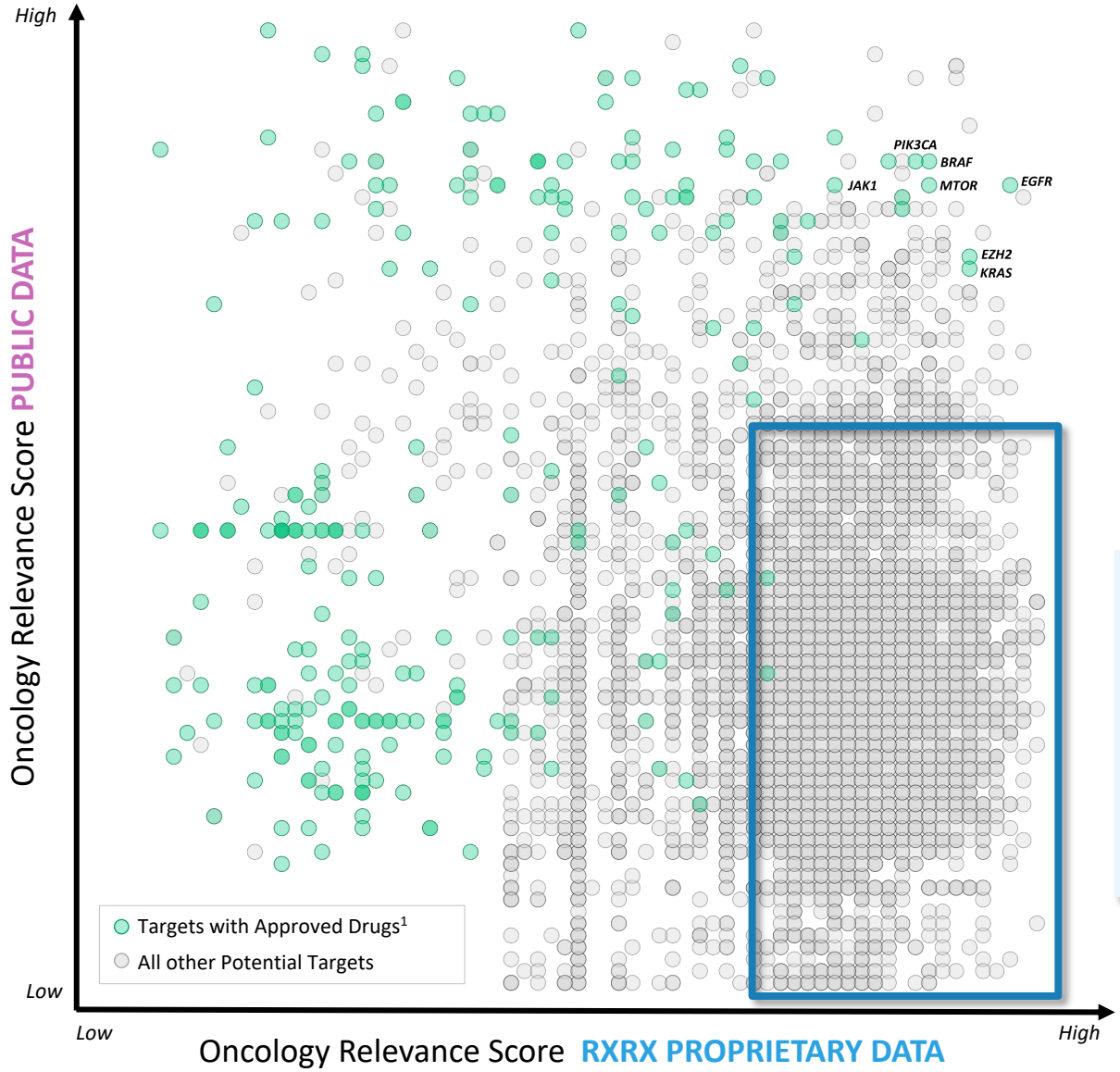
Our method uncovers innovative targets that we believe provide a **differentiated therapeutic potential for oncology R&D**

LLMs harness Public Datasets such as:

- Cancer Dependency Map
- Open Targets
- TCGA
- CCLE
- COSMIC

LLMs harness RXRX Proprietary Datasets such as:

- Phenomap inferences
- Matchmaker assessments
- Invivomics experiments
- ADME predictions
- Compound promiscuities



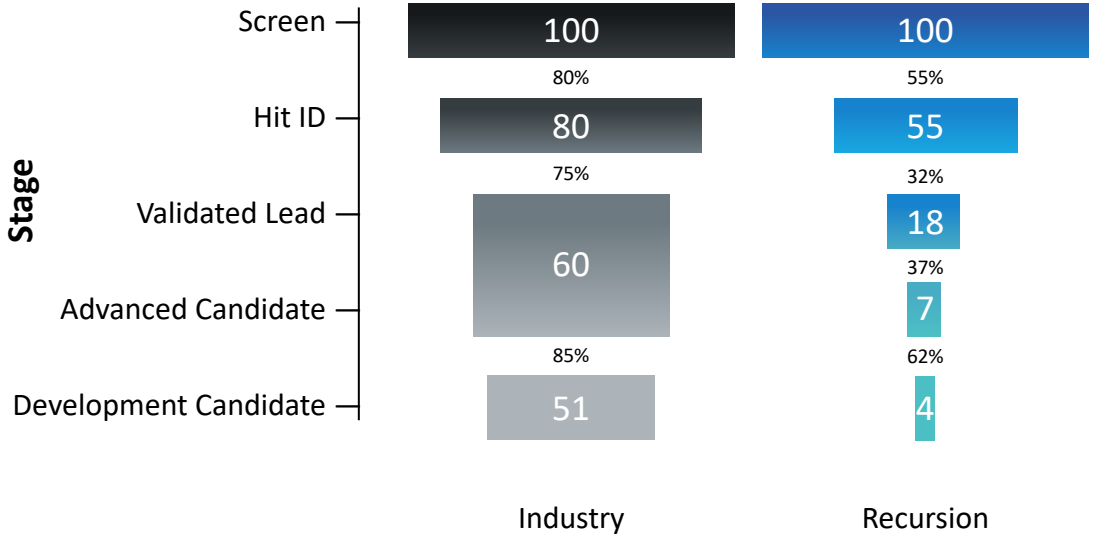
We expect to initiate 300 exploratory programs in 2024 from this space, where our proprietary data provides a distinct arbitrage, with significant human effort reserved for novel relationships that confirmed and validated on our platform. Previously, over 40 FTEs were deployed to explore our maps and public data manually to initiate programs.

[1] *Ochoa, D. et al. Nucleic Acids Research. (2023).*

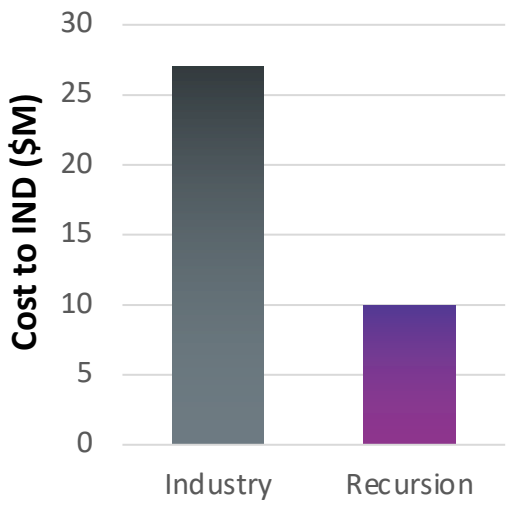
Recursion

The Recursion OS maps and navigates biology to shift drug discovery from bespoke science to scaled engineering

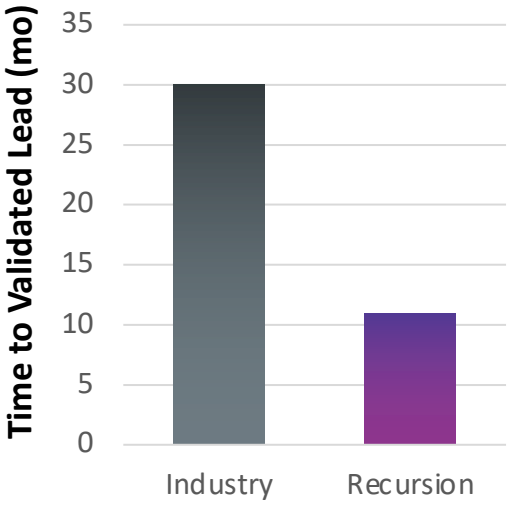
Failing faster and earlier to ›



› spend less ›



› and go faster



All industry data has been adapted from Paul, et al. Nature Reviews Drug Discovery. (2010) 9, 203–214. The cost to IND has been inflation-adjusted using the US Consumer Price Index (CPI).
The Recursion data shown for the transition stages and time to validated lead is the average of all Recursion programs since late 2017 through 2023.
The Recursion data shown for cost to IND pertains to the actual and projected costs for a novel chemical entity to reach IND.

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 **accelerated path** to approval

- **Precision Oncology**
- **Rare Disease**

Partnerships

Partnership Strategy

Partner in **complex therapeutic areas** requiring large financial commitment or competitive arbitrage

Leverage partner knowledge and clinical development capabilities

- **Neuroscience***
- **Undruggable Oncology**
- **Other large, intractable areas of biology (e.g. CV/Met)**

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**

**Includes a single oncology indication from our Roche and Genentech collaboration.*

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 **accelerated path** to approval

- **Precision Oncology**
- **Rare Disease**

Partnerships

Partnership Strategy

Partner in **complex therapeutic areas** requiring large financial commitment or competitive arbitrage

Leverage partner knowledge and clinical development capabilities

- **Neuroscience***
- **Undruggable Oncology**
- **Other large, intractable areas of biology (e.g. CV/Met)**

Data

Data Strategy

License subsets of data and key tools

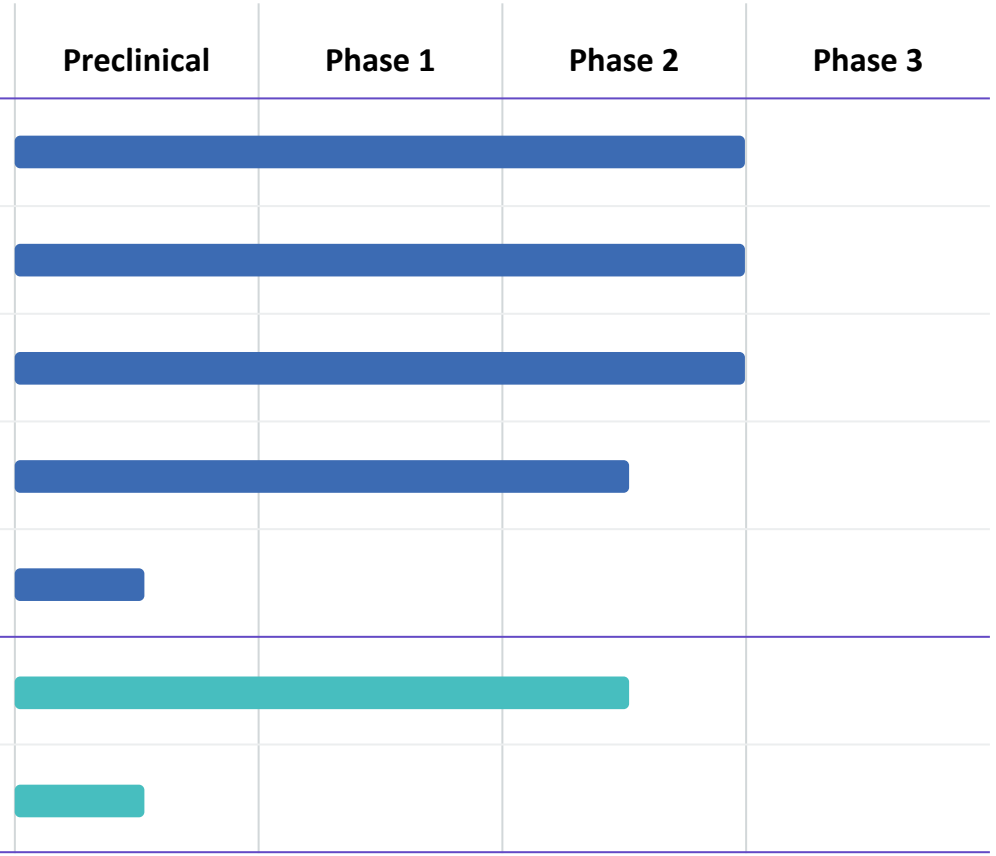
Direct generation of new data internally **to maximize pipeline and partnership value-drivers**

- **Licensing**
- **Augment Recursion OS**

**Includes a single oncology indication from our Roche and Genentech collaboration.*

Our pipeline reflects the scale and breadth of our approach

	Program	Indication	Target	Patient Population
Rare & Other	**REC-994**	Cerebral Cavernous Malformation	Superoxide	~ 360K[1]
	REC-2282	Neurofibromatosis Type 2	HDAC	~ 33K[2]
	REC-4881	Familial Adenomatous Polyposis	MEK	~ 50K[3]
	REC-3964	*Clostridioides difficile* infection	TcdB	~730K
	Epsilon	Fibrotic Diseases	Undisclosed	~ 50K[4,5,6]
Oncology	**REC-4881**	AXIN1 or APC Mutant Cancers	MEK	~ 65K[7]
	RBM39	HR-Proficient Ovarian & Solid Tumors	RBM39	~ 200K[8]



More than a dozen discovery and research programs in oncology or with our partners – first program optioned by Roche-Genentech in GI-oncology

All populations defined above are US and EU5 incidence unless otherwise noted. EU5 is defined as France, Germany, Italy, Spain, and UK. (1) Prevalence for hereditary and sporadic symptomatic population. (2) Annual US and EU5 incidence for all *NF2*-driven meningiomas. (3) Prevalence for adult and pediatric population. (4) Our program has the potential to address several indications. (5) We have not finalized a target product profile for a specific indication. (6) Incidence for US only. (7) 2L drug-treatable population. (8) 2L drug-treatable population comprising ovarian, prostate, breast, and pancreatic cancers with no HRR mutations.

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 **accelerated path** to approval

- Precision Oncology
- Rare Disease

Partnerships

Partnership Strategy

Partner in **complex therapeutic areas** requiring large financial commitment or competitive arbitrage

Leverage partner knowledge and clinical development capabilities

- **Neuroscience***
- **Undruggable Oncology**
- **Other large, intractable areas of biology (e.g. CV/Met)**

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

**Includes a single oncology indication from our Roche and Genentech collaboration.*

Exciting scientific collaborations span biopharma, tech & data

Therapeutic discovery

Roche Genentech
A Member of the Roche Group

Announced Dec. 2021

Neuroscience and a single oncology indication

- **$150M upfront** and up to or exceeding **$500M in research milestones and data usage options**
- Up to or exceeding **$300M in possible milestones per program** for up to **40 programs**
- **First program already optioned**
- **Mid to high single-digit tiered royalties** on net sales

BAYER

Announced Sept. 2020

Significant Update Announced Nov. 2023

Undruggable oncology targets

- **$30M upfront** and **$50M equity investment**
- Increased per program milestones which may be **up to $1.5B for up to 7 oncology programs**
- **Mid single-digit royalties** on net sales
- **Recursion owns all algorithmic improvements**

Platform, Technology and Data

NVIDIA

Announced July 2023

Computation and ML/AI

- **$50M equity investment**
- Partnership on **advanced computation** (e.g., foundation model development)
- **Priority access** to compute hardware or **DGXCloud Resources**
- **Phenom-Beta, a foundation model for phenomics from Recursion, now available on NVIDIA's BioNeMo platform**

TEMPUS

Announced Nov. 2023

Real-world data access

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

Enamine

Announced Dec. 2023

Cheminformatics and chemical synthesis

- 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

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 **accelerated path** to approval

- Precision Oncology
- Rare Disease

Partnerships

Partnership Strategy

Partner in **complex therapeutic areas** requiring large financial commitment or competitive arbitrage

Leverage partner knowledge and clinical development capabilities

- Neuroscience*
- Undruggable Oncology
- Other large, intractable areas of biology (e.g. CV/Met)

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**

*Includes a single oncology indication from our Roche and Genentech collaboration.

Recursion

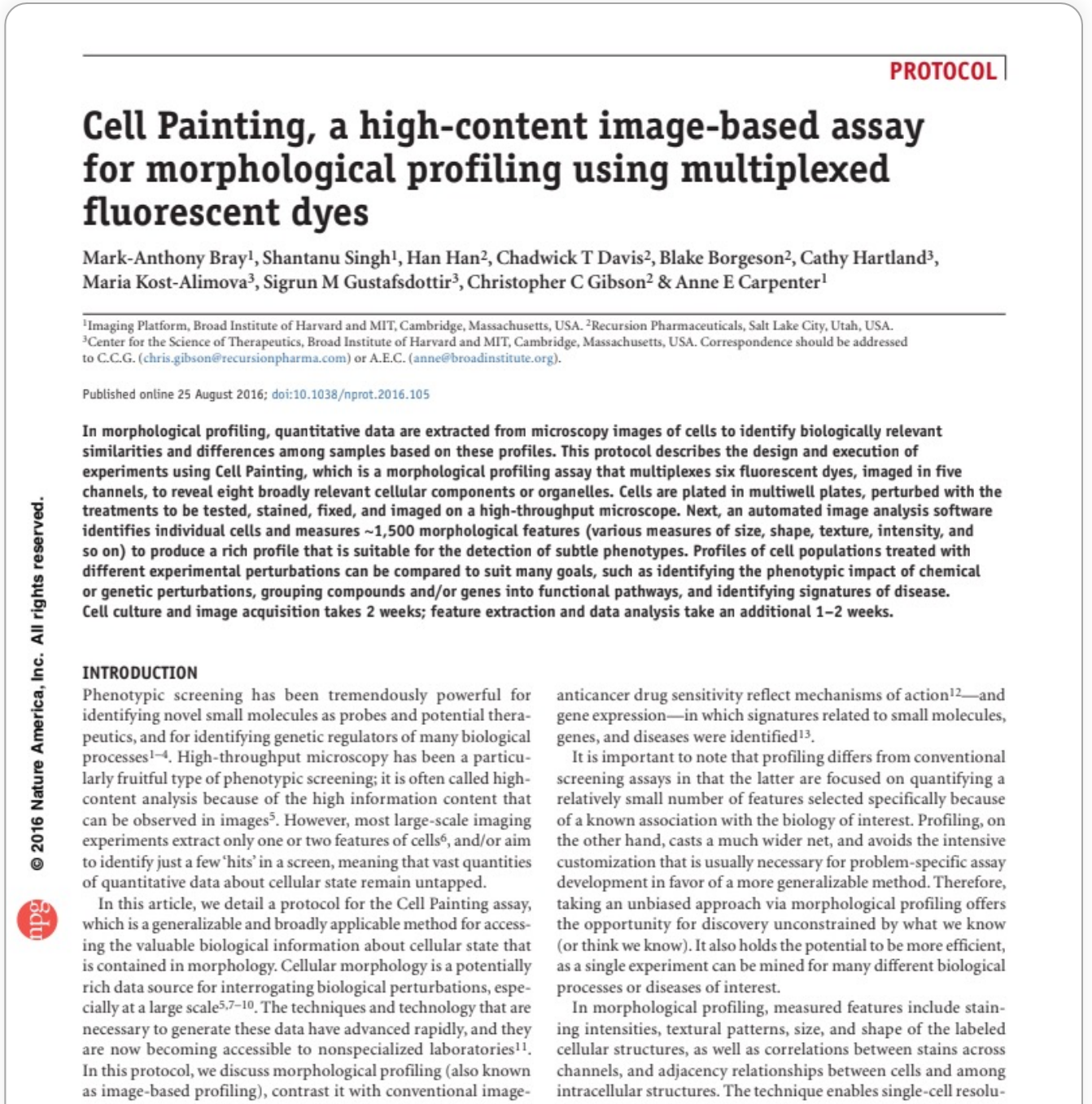
The Future of TechBio

TechBio Origins: Point Solutions

Most BioTech companies have built a point solution - they've developed a tool, process, model or analysis to accomplish an important step in drug discovery.

This is how we started too.

But discovering and developing medicines requires hundreds of steps…



PROTOCOL

Cell Painting, a high-content image-based assay for morphological profiling using multiplexed fluorescent dyes

Mark-Anthony Bray[1], Shantanu Singh[1], Han Han[2], Chadwick T Davis[2], Blake Borgeson[2], Cathy Hartland[3], Maria Kost-Alimova[3], Sigrun M Gustafsdottir[3], Christopher C Gibson[2] & Anne E Carpenter[1]

[1]Imaging Platform, Broad Institute of Harvard and MIT, Cambridge, Massachusetts, USA. [2]Recursion Pharmaceuticals, Salt Lake City, Utah, USA. [3]Center for the Science of Therapeutics, Broad Institute of Harvard and MIT, Cambridge, Massachusetts, USA. Correspondence should be addressed to C.C.G. (chris.gibson@recursionpharma.com) or A.E.C. (anne@broadinstitute.org).

Published online 25 August 2016; doi:10.1038/nprot.2016.105

In morphological profiling, quantitative data are extracted from microscopy images of cells to identify biologically relevant similarities and differences among samples based on these profiles. This protocol describes the design and execution of experiments using Cell Painting, which is a morphological profiling assay that multiplexes six fluorescent dyes, imaged in five channels, to reveal eight broadly relevant cellular components or organelles. Cells are plated in multiwell plates, perturbed with the treatments to be tested, stained, fixed, and imaged on a high-throughput microscope. Next, an automated image analysis software identifies individual cells and measures ~1,500 morphological features (various measures of size, shape, texture, intensity, and so on) to produce a rich profile that is suitable for the detection of subtle phenotypes. Profiles of cell populations treated with different experimental perturbations can be compared to suit many goals, such as identifying the phenotypic impact of chemical or genetic perturbations, grouping compounds and/or genes into functional pathways, and identifying signatures of disease. Cell culture and image acquisition takes 2 weeks; feature extraction and data analysis take an additional 1–2 weeks.

INTRODUCTION

Phenotypic screening has been tremendously powerful for identifying novel small molecules as probes and potential therapeutics, and for identifying genetic regulators of many biological processes[1–4]. High-throughput microscopy has been a particularly fruitful type of phenotypic screening; it is often called high-content analysis because of the high information content that can be observed in images[5]. However, most large-scale imaging experiments extract only one or two features of cells[6], and/or aim to identify just a few 'hits' in a screen, meaning that vast quantities of quantitative data about cellular state remain untapped.

In this article, we detail a protocol for the Cell Painting assay, which is a generalizable and broadly applicable method for accessing the valuable biological information about cellular state that is contained in morphology. Cellular morphology is a potentially rich data source for interrogating biological perturbations, especially at a large scale[5,7–10]. The techniques and technology that are necessary to generate these data have advanced rapidly, and they are now becoming accessible to nonspecialized laboratories[11]. In this protocol, we discuss morphological profiling (also known as image-based profiling), contrast it with conventional image-anticancer drug sensitivity reflect mechanisms of action[12]—and gene expression—in which signatures related to small molecules, genes, and diseases were identified[13].

It is important to note that profiling differs from conventional screening assays in that the latter are focused on quantifying a relatively small number of features selected specifically because of a known association with the biology of interest. Profiling, on the other hand, casts a much wider net, and avoids the intensive customization that is usually necessary for problem-specific assay development in favor of a more generalizable method. Therefore, taking an unbiased approach via morphological profiling offers the opportunity for discovery unconstrained by what we know (or think we know). It also holds the potential to be more efficient, as a single experiment can be mined for many different biological processes or diseases of interest.

In morphological profiling, measured features include staining intensities, textural patterns, size, and shape of the labeled cellular structures, as well as correlations between stains across channels, and adjacency relationships between cells and among intracellular structures. The technique enables single-cell resolu-

As these point solutions evolve they increase in complexity and scale

AUTOMATION

High-throughput screening

Our highly automated wet-labs systematically capture images of human cells in response to different perturbations

Up to
2.2M experiments
conducted every week

PROFILING SYSTEMS

Diverse biological and chemical inputs

We manipulate human cells with CRISPR/Cas9-mediated gene knockouts, compounds, and other reagents

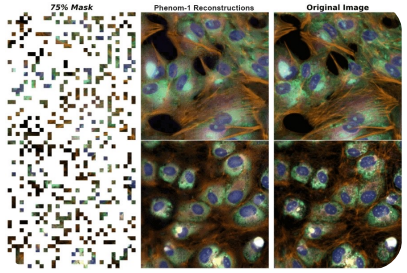
>50 human cell types
~2M physical compounds
Whole-genome CRISPR knockouts

Phenomics

FOUNDATION MODELS

Phenom-1

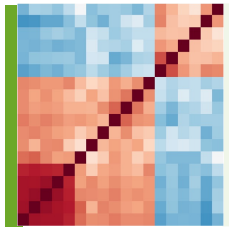
Groundbreaking models trained on >1 billion images and hundreds of millions of parameters learn to extract biologically meaningful signals from cell images



DIGITIZATION

Maps of Biology & Chemistry

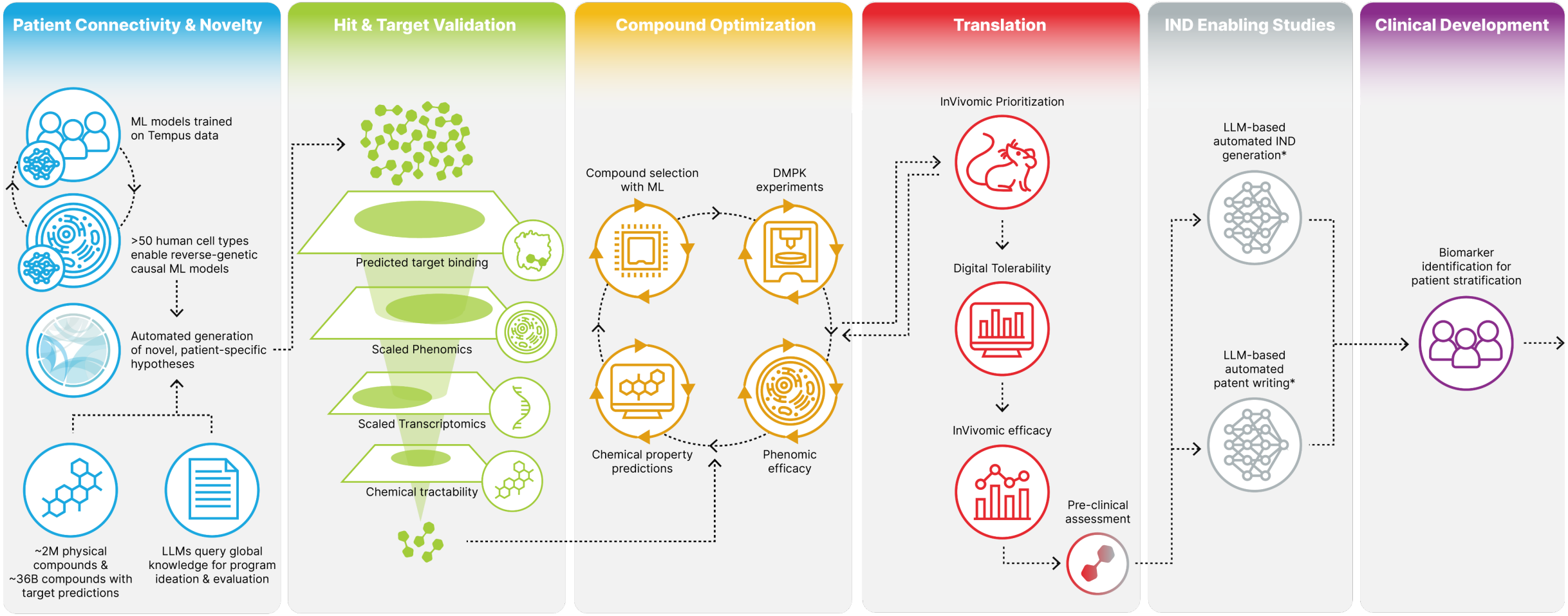
Models infer relationships between all possible combinations of genes and compounds, recapitulating known biology and revealing novel insights



>5 trillion relationships
across multiple biological and chemical contexts

Recursion

To truly industrialize drug discovery, point solutions must be integrated as modules across many diverse steps



*Currently being explored

Recursion

Each module is complex, and we continuously improve them

WET LAB

HT ADME Experiments

A highly automated DMPK module executes 3 critical assays across human and rat contexts.

ENRICH FOR QUALITY

Pre-synthesis Evaluation

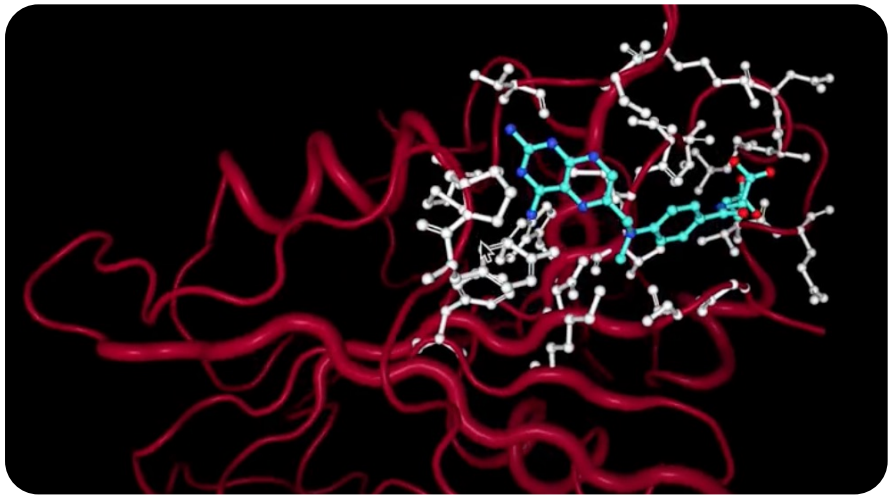
Prioritize compound synthesis for compounds predicted to have high likelihood of suitable pharmacokinetics

DMPK

LEARNING CYCLES

Predictive Models

Leverage Recursion's power for structure-based prediction of in vitro assays and in vivo compound profiles



ANIMAL PHARMACOKINETICS

In Vivo Validation

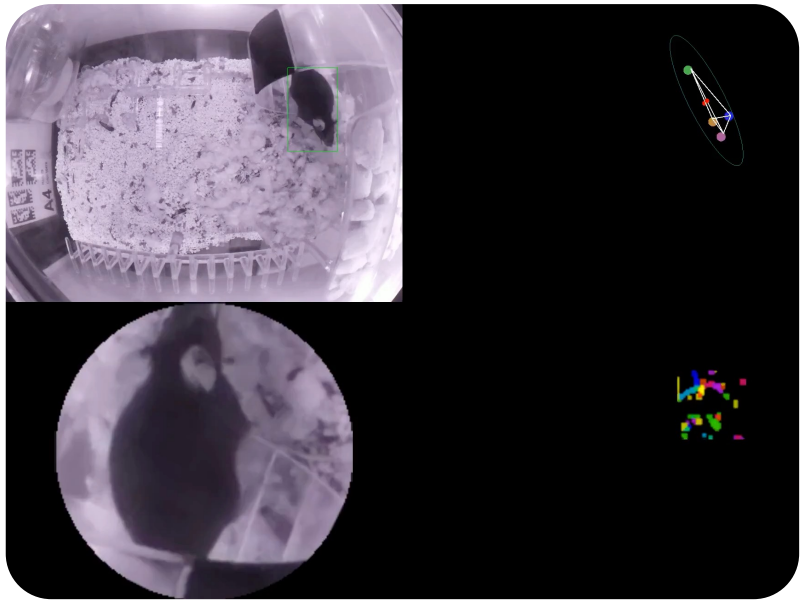
Establish in vitro-in vivo and in silico-in vivo correlations to minimize experimental toil.

Utilizing each module requires specialized teams and expertise

GOING DIGITAL

Industrialized program progression

Digitized data collection yields real-time, continuous, and non-invasive data recorded in the animal's home cage. Data generates high-dimensional assays, and ML connects studies for productivity. Overall, there is a drastic reduction in time, labor, and cost.



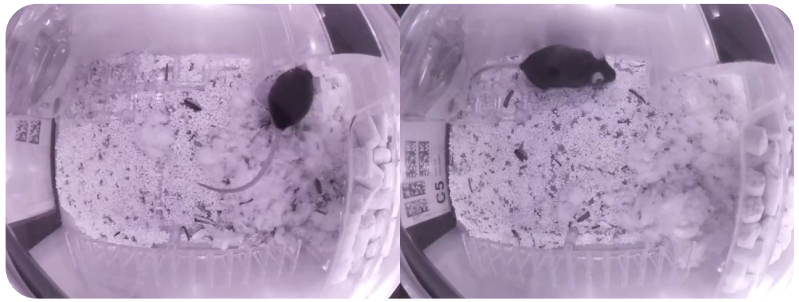
InVivomic prioritization

DETERMINING DOSAGE

Compound optimization

Rat and mouse studies with ML-based selection of optimal compound and dose from video.

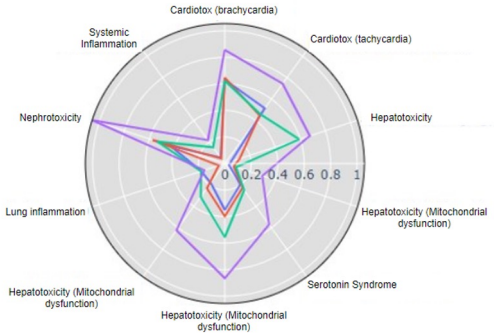
Not tolerated *Tolerated*



ML evaluation of mice against >10 liabilities.

SPEED & EFFICIENCY

Faster readouts for critical studies



Speed to insight, including tolerability liability

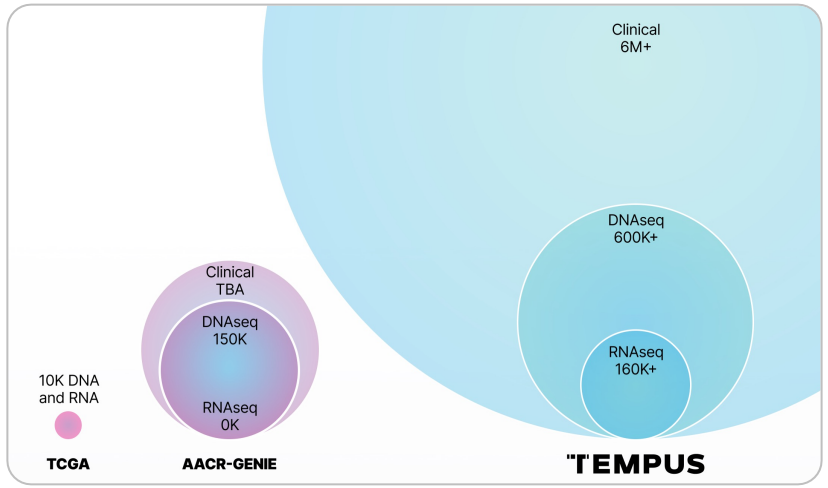
Recursion

We continuously add new modules to improve the Recursion OS

DATASET

>20 PB of real-world patient data

The Tempus partnership provides Recursion with preferential access to multi-modal data for >100,000 oncology patients totaling **over 20 PB**.



COMPUTE

BioHive-1 supercomputer

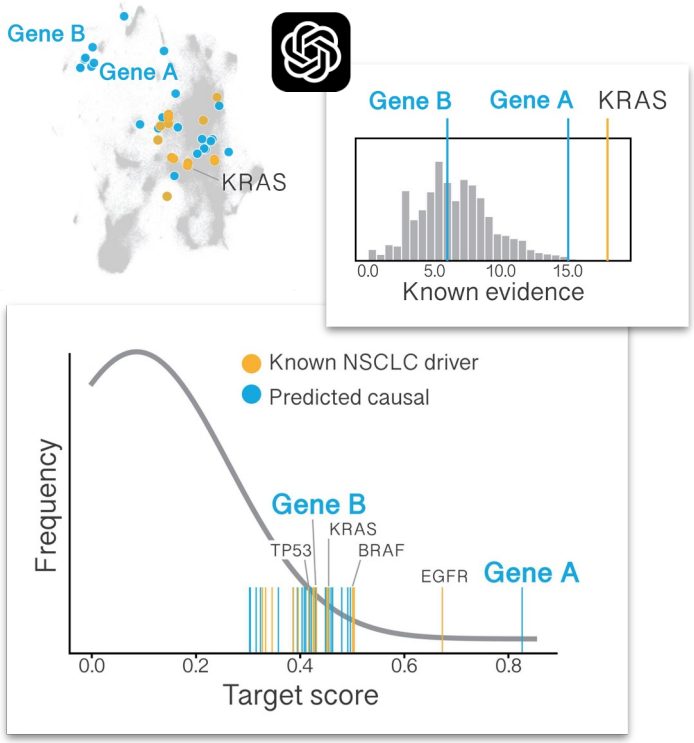
The Tempus data is computed and ML oncology models run on BioHive-1, Recursion's in-house supercomputer. BioHive-1 will be expanded into a **top 50 supercomputer** in 2024 in partnership with Nvidia.

ML trained on Tempus data

APPROACH

Combining forward & reverse genetics

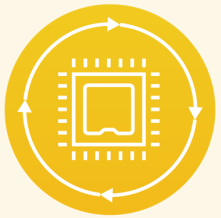
We are using Tempus' scaled multimodal real-world patient data to train AI models for target discovery, biomarker development, and patient selection.



Recursion

The result is a palette of ever-evolving sophisticated modules


Phenomics


Compound selection with ML


Patient-specific hypotheses


Phenom-1


Chemical tractability


InVivomic prioritization


LLMs & literature search


InVivomic efficacy


Chemical property predictions


Predicted target binding


Tempus data ML models


Phenomic efficacy


Patient stratification


Transcriptomics


MatchMaker


Digital tolerability


DMPK


Compound ordering

We use different modules for different tasks: Find NCE for known target

Phenomics

Compound selection with ML

Patient-specific hypotheses

Phenom-1

Chemical tractability

InVivomic prioritization

LLMs & literature search

InVivomic efficacy

Chemical property predictions

Predicted target binding

Tempus data ML models

Phenomic efficacy

Patient stratification

Transcriptomics

MatchMaker

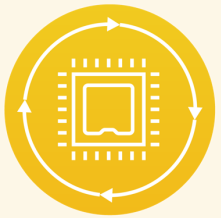
Digital tolerability

DMPK

Compound ordering

We use different modules for different tasks: Find novel target & drug it

Phenomics



Compound selection with ML

Patient-specific hypotheses

Phenom-1

Chemical tractability

InVivomic prioritization

LLMs & literature search

InVivomic efficacy

Chemical property predictions

Predicted target binding

Tempus data ML models

Phenomic efficacy

Patient stratification

Transcriptomics

MatchMaker

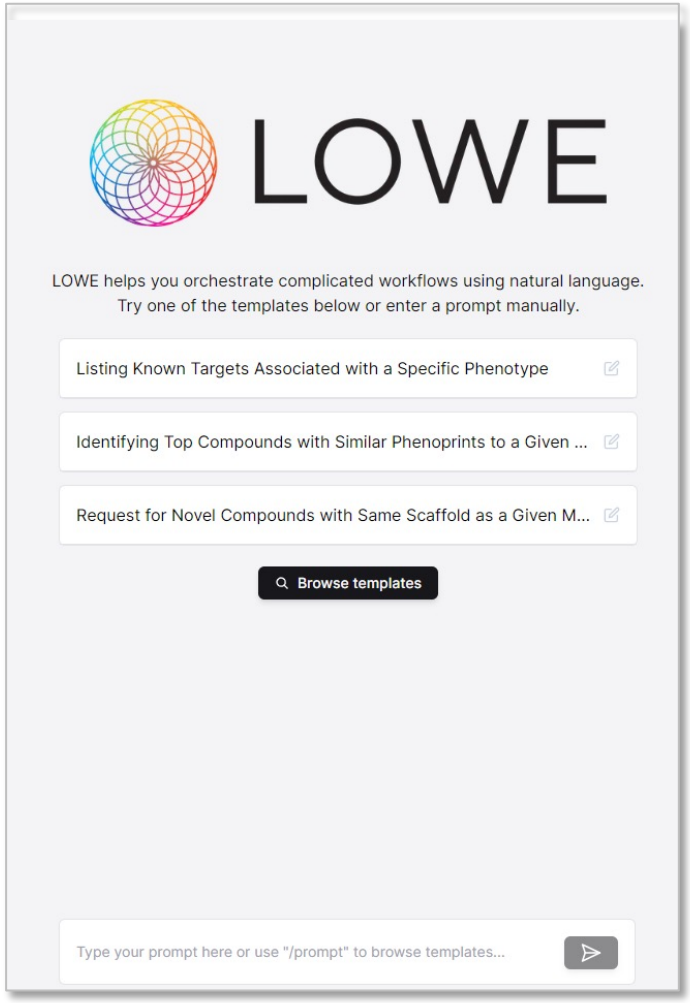
Digital tolerability

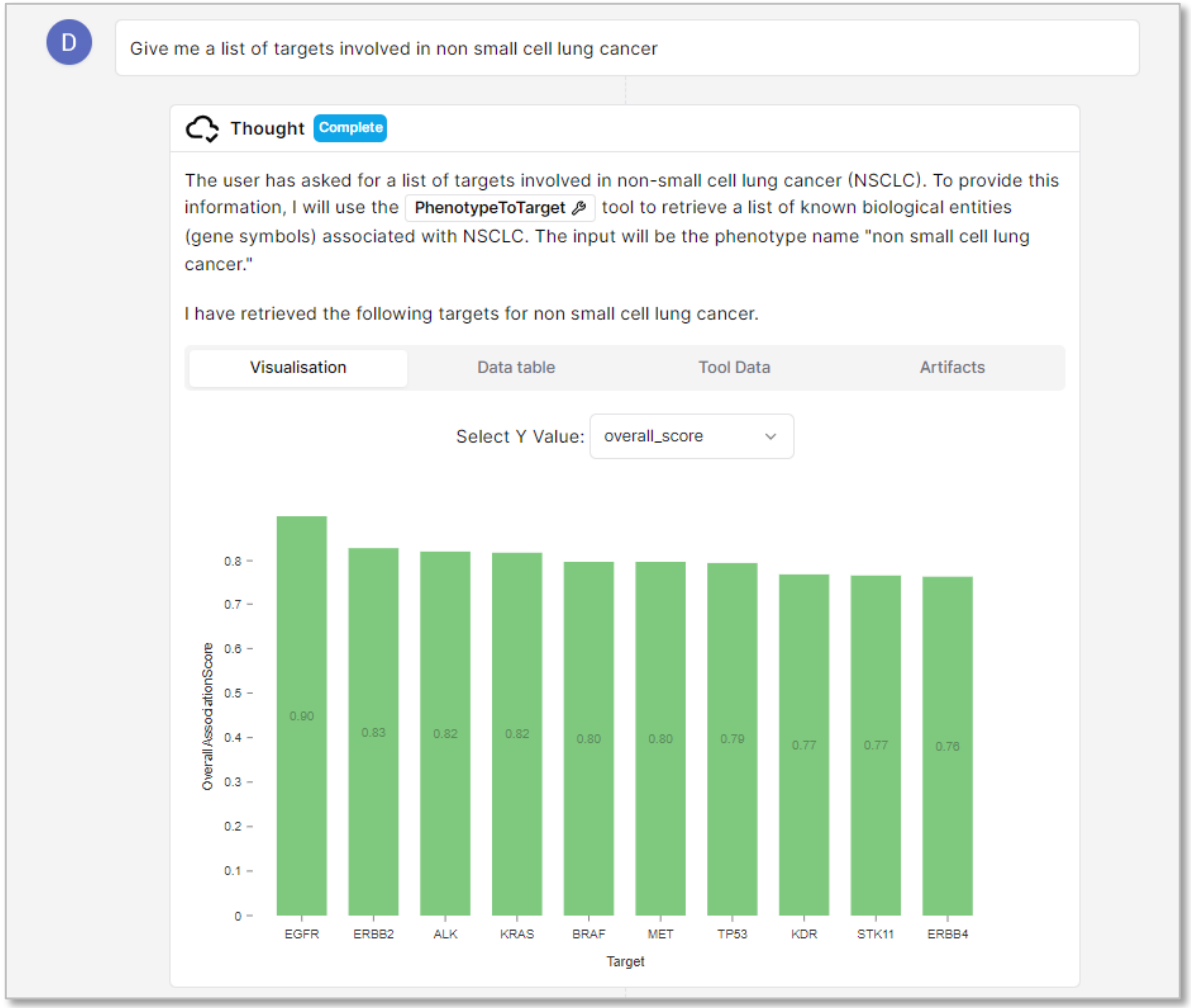
DMPK

Compound ordering

LOWE

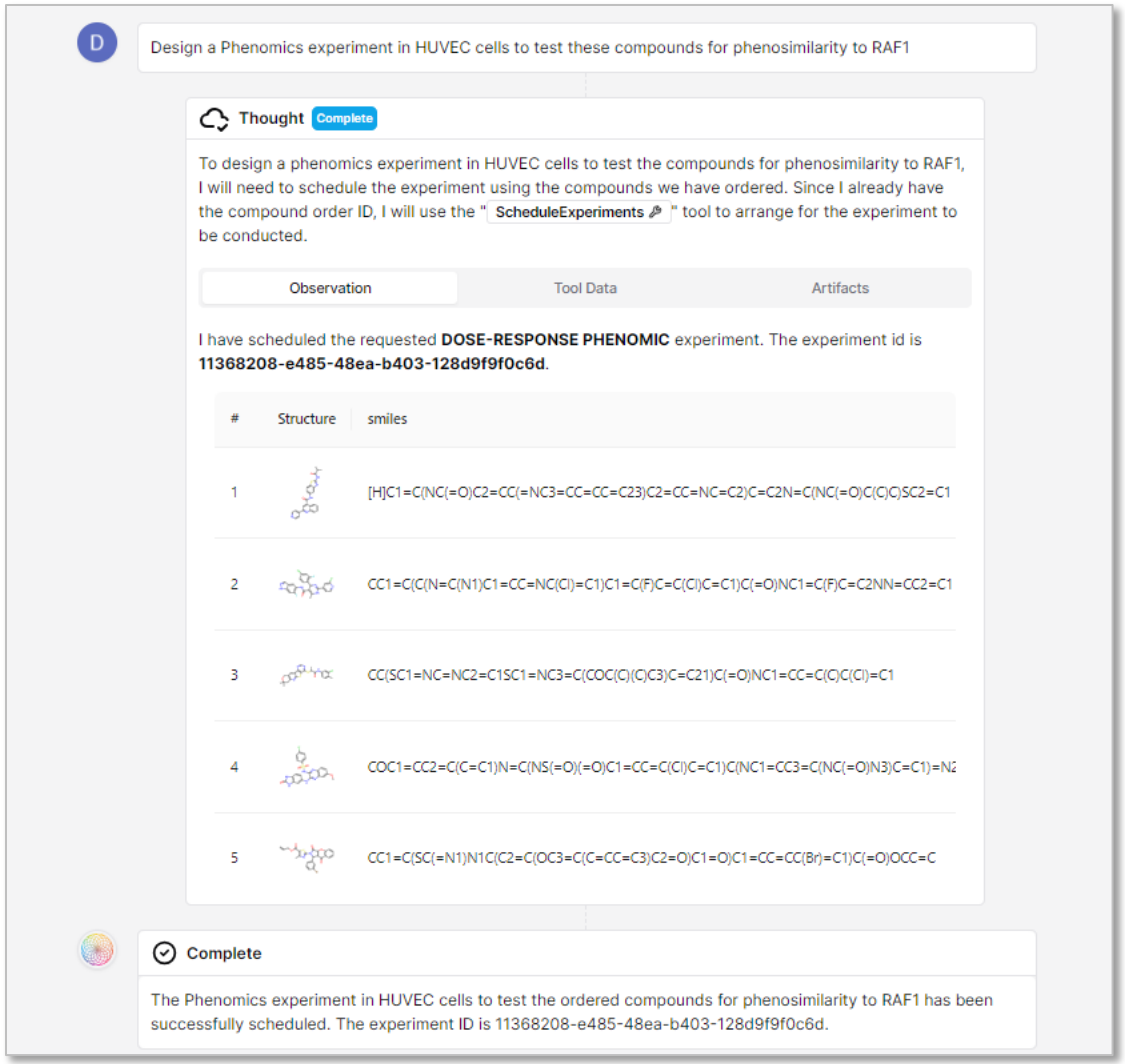
LOWE puts the power of the Recursion OS at your fingertips via natural language without any coding expertise required

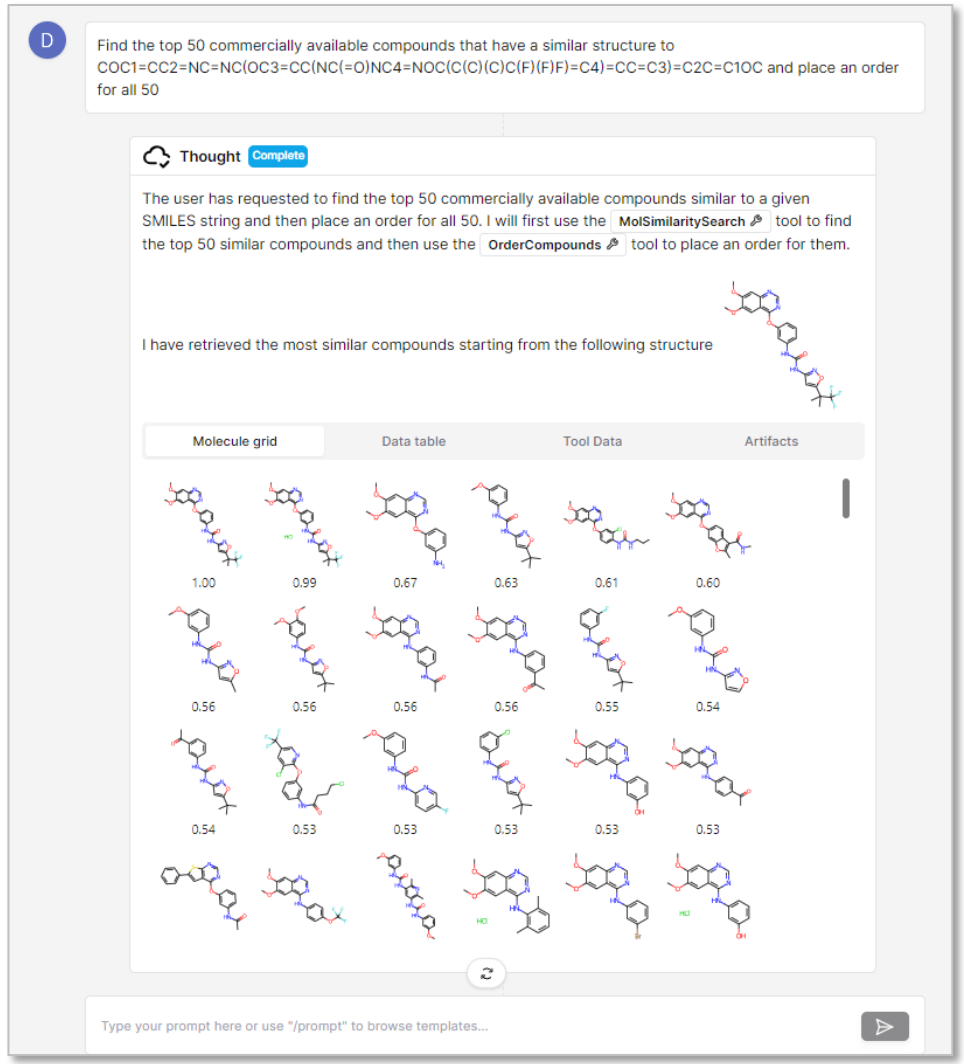




Recursion

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

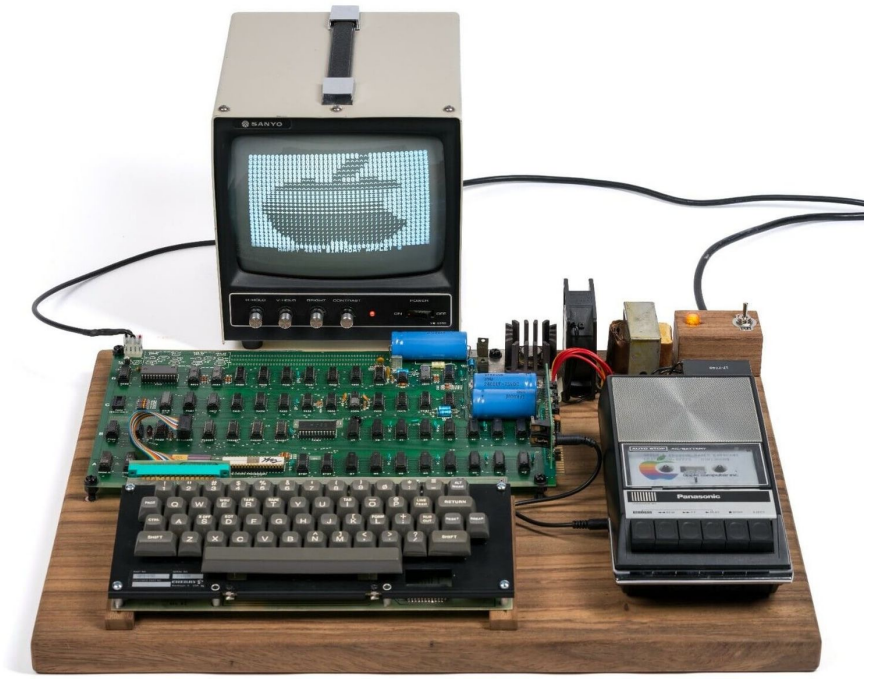


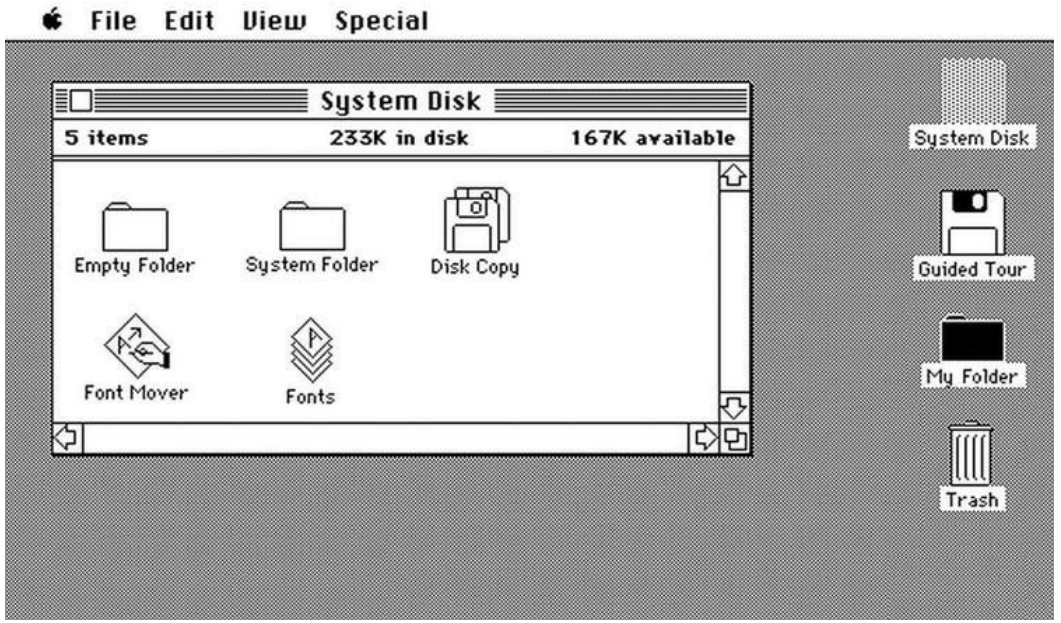


Recursion

The Recursion OS is now more than a collection of point solutions accessible to expert users

…it is increasingly integrated and accessible via a **Discovery User Interface** that can be used by any of our scientists from the comfort of their laptop…





Recursion

First-in-Disease Opportunities and Beyond

Clinical: CCM

SYCAMORE Clinical Trial : REC-994 for CCM Phase 2 Fully Enrolled

PREVALENCE & STANDARD OF CARE

~360,000

Symptomatic US + EU5, >1 million patients worldwide live with these lesions today

>5x larger US patient population than other rare diseases like Cystic Fibrosis (>31k patients)

No approved therapy

- Most patients receive no treatment or only symptomatic therapy
- Surgical resection or stereotactic radiosurgery not always feasible because of location and is not curative

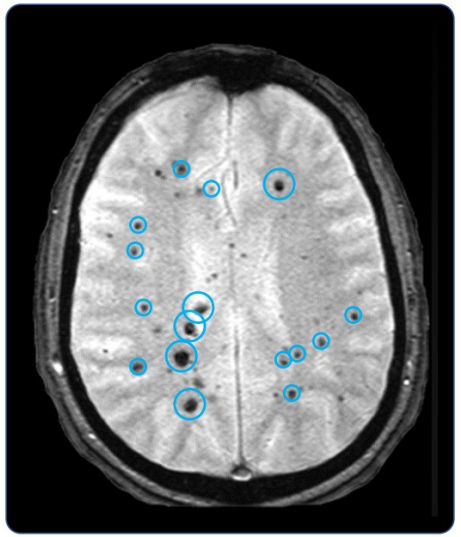
CAUSE

LOF mutations in genes *CCM1*, *CCM2* & *CCM3*, key for maintaining the structural integrity of the vasculature due to unknown mechanisms

PATHOPHYSIOLOGY & REASON TO BELIEVE

Vascular malformations of the CNS leading to focal neurological deficits, hemorrhage and other symptoms

Efficacy in Recursion OS as well as functional validation via scavenging of massive superoxide accumulation in cellular models; reduction in lesion number with chronic administration in mice



Vascular malformations (cavernomas)

Julia – living with CCM

KEY ELEMENTS

- Targeting **sporadic and familial symptomatic CCM** patients with *CCM1*, *CCM2*, and *CCM3* mutations
- Superoxide scavenger, small molecule
- Phase 2 readout **expected Q3 2024**
- US & EU **Orphan Drug Designation**
- Oral dosing

Clinical: CCM

SYCAMORE Clinical Trial : REC-994 for CCM Phase 2 Fully Enrolled

Phase 2 trial initiated in Q1 2022

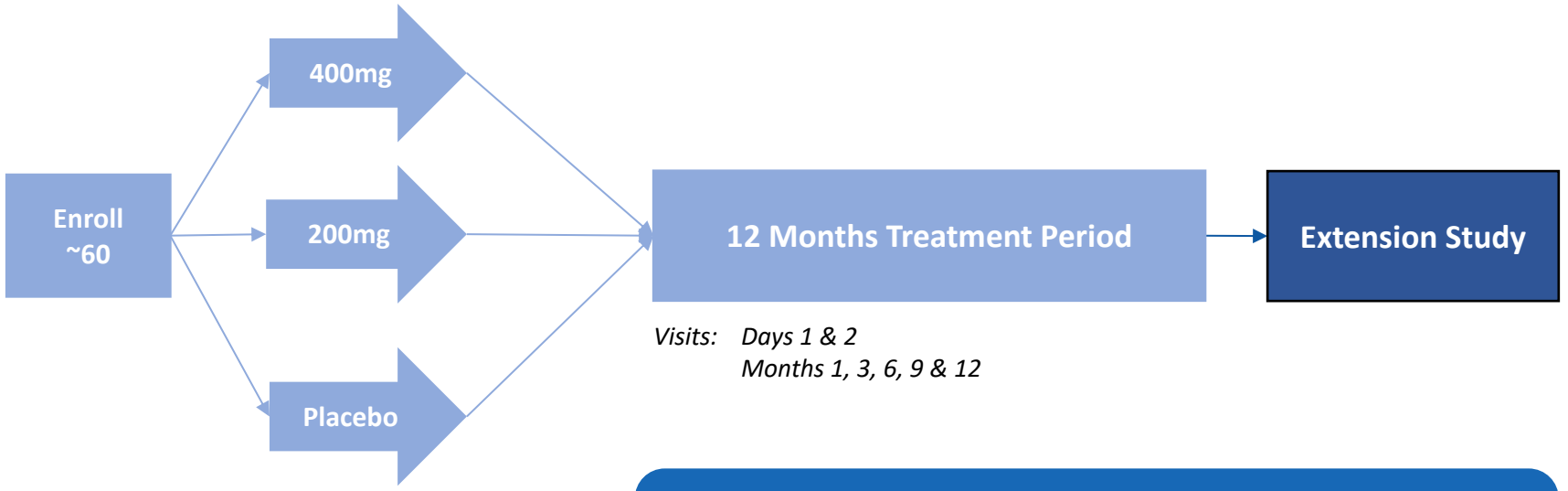
Enrollment Criteria

- MRI-confirmed CCM lesion(s)
- Familial or sporadic
- Symptoms directly related to CCM

Outcome Measures

- Primary: Safety and tolerability
  - Adverse events & symptoms
- Secondary: Efficacy
  - Clinician-measured outcomes (CGI and PGI)
  - Imaging of CCM lesions – number, size & rate of change
  - Impact of acute stroke (mRS, NIHSS)
  - Patient reported outcomes (SMSS, PROMIS-29, CCM-HI, symptom questionnaires)
- Exploratory: Biomarkers

Screening & Randomization 1:1:1	Treatment	Follow-up



Trial Update

- Enrollment is complete
- Vast majority of participants who completed 12 months of treatment continue to enter long-term extension study
- **Phase 2 readout expected Q3 2024**

Source: https://www.clinicaltrials.gov/ct2/show/NCT05130866?term=recursion&draw=2&rank=3; https://www.SycamoreCCM.com/

Clinical: NF2

POPLAR Clinical Trial : REC-2282 for NF2 Phase 2 Underway

PREVALENCE & STANDARD OF CARE

~33,000 **Treatable US + EU**

No approved therapy

- There are no approved drugs for NF2
- Surgery is standard of care (when feasible)
- Location may make complete resection untenable, leading to hearing loss, facial paralysis, poor balance and visual difficulty

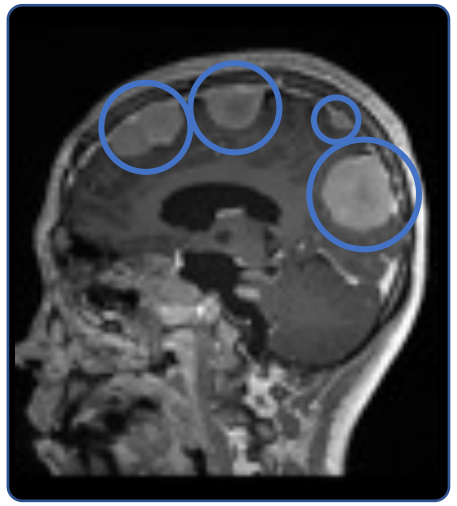
CAUSE

LOF mutations in NF2 tumor suppressor gene, leading to deficiencies in the tumor suppressor protein merlin

PATHOPHYSIOLOGY & REASON TO BELIEVE

Inherited rare **CNS tumor syndrome** leading to loss of hearing and mobility, other focal neurologic deficits

Efficacy in Recursion OS, cellular, and animal models; suppression of aberrant ERK, AKT, and S6 pathway activation in a Phase 1 PD Study in NF2 patient tumors



Intracranial meningiomas

Ricki – living with NF2

KEY ELEMENTS

- Targeting **familial and sporadic NF2 meningioma** patients
- HDAC inhibitor, small molecule
- Oral dosing
- Phase 2 readout **expected Q4 2024**
- **Fast-Track** and US & EU **Orphan Drug Designation**

Clinical: NF2

POPLAR Clinical Trial : REC-2282 for NF2 Phase 2 Underway

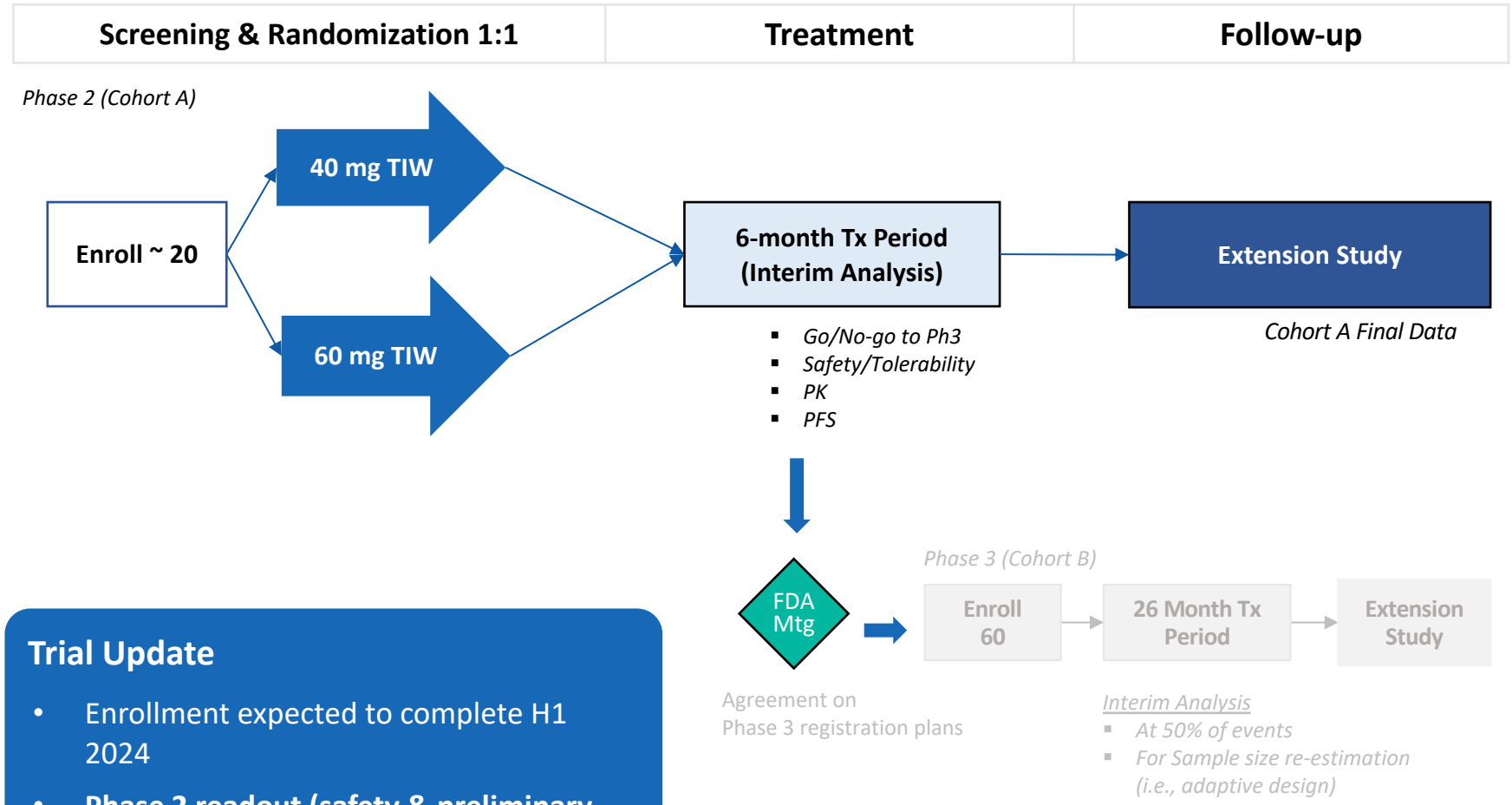
Phase 2/3 trial initiated in Q2 2022

Enrollment Criteria

- MRI-confirmed progressive meningioma
- Either of the below
  - Sporadic meningioma with confirmed NF2 mutation
  - Confirmed diagnosis of NF2 disease

Outcome Measures

- Primary: Safety and tolerability
  - Progression-free survival
  - Time to progression
  - Duration of response
  - Overall response rate



Trial Update

- Enrollment expected to complete H1 2024
- **Phase 2 readout (safety & preliminary efficacy) expected in Q4 2024**

https://clinicaltrials.gov/ct2/show/NCT05130866

Clinical: FAP

TUPELO Clinical Trial : REC-4881 for FAP Phase 1b/2 Underway

PREVALENCE & STANDARD OF CARE

~50,000 **Diagnosed US + EU5**

No approved therapy

- Colectomy during adolescence (with or without removal of rectum) is standard of care
- Post-colectomy, patients still at significant risk of polyps progressing to GI cancer
- Significant decrease in quality-of-life post-colectomy (continued endoscopies, surgical intervention)

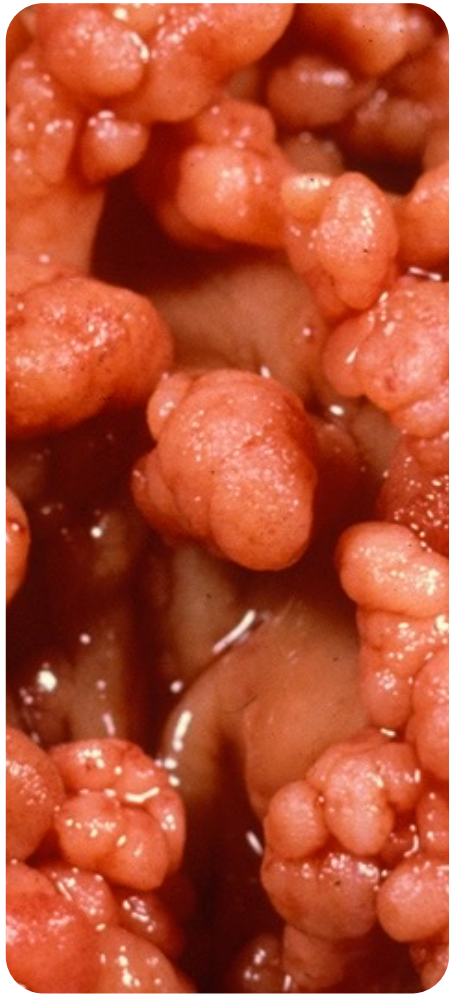
CAUSE

Inactivating mutations in the tumor suppressor gene *APC*

PATHOPHYSIOLOGY & REASON TO BELIEVE

Polyps throughout the GI tract with extremely high risk of malignant transformation

Efficacy in the Recursion OS showed specific MEK 1/2 inhibitors had an effect in context of *APC* LOF. Subsequent APC^{min} mouse model showed potent reduction in polyps and dysplastic adenomas



Polyps Found in Colon and Upper GI Tract

KEY ELEMENTS

- Targeting **classical FAP patients (with *APC* mutation)**
- MEK inhibitor, small molecule
- Oral dosing
- FPI for Part 2 **expected H1 2024**
- **Fast-Track** and US & EU **Orphan Drug Designation**

Clinical: FAP

TUPELO Clinical Trial : REC-4881 for FAP Phase 1b/2 Underway

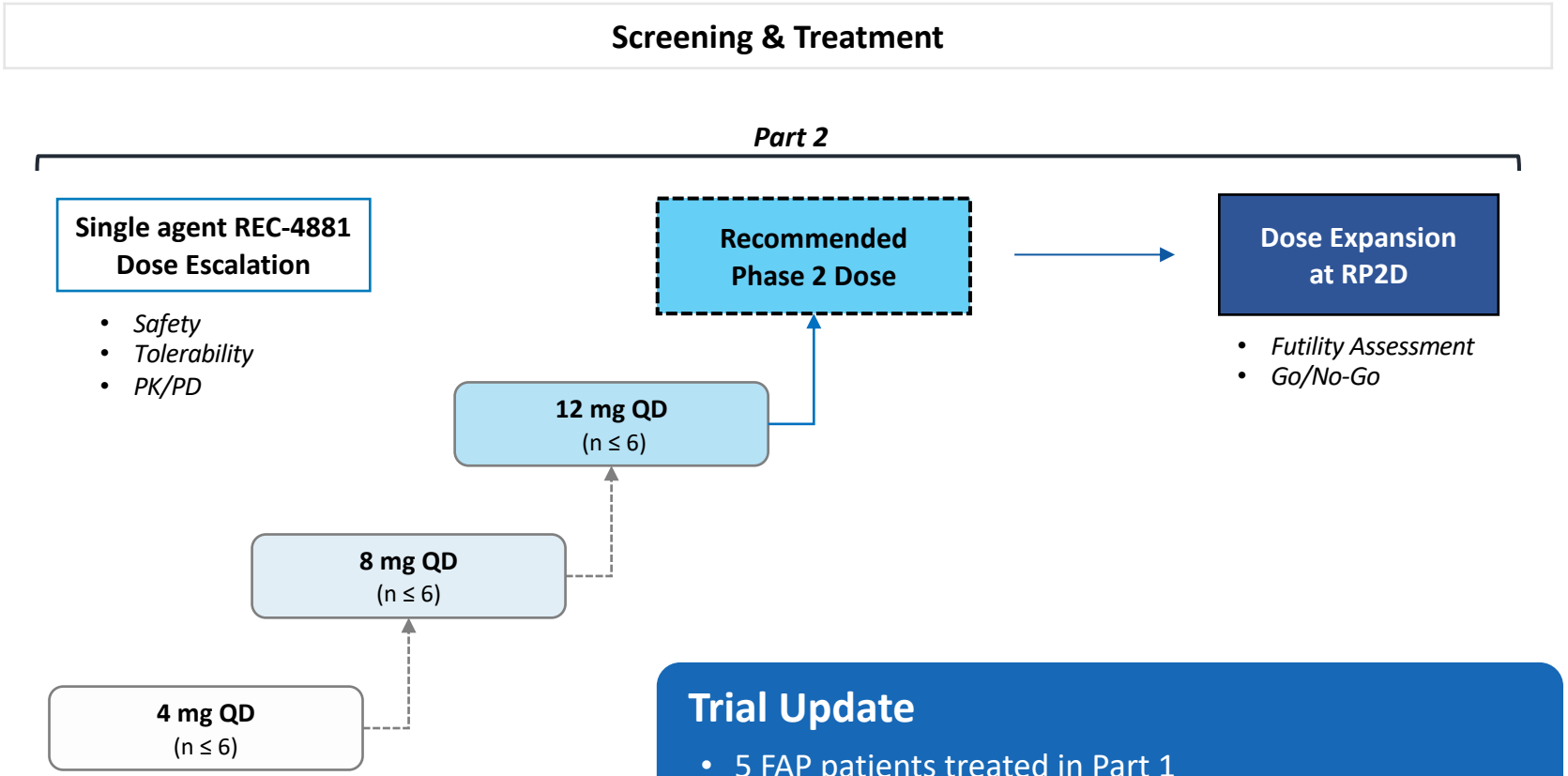
Part 1 Complete, Part 2 FPI Expected H1 2024

Enrollment Criteria

- Confirmed *APC* mutation
- ≥ 55 years old
- Post-colectomy/proctocolectomy
- No cancer present
- Polyps in either duodenum (including ampulla of vater) or rectum/pouch

Outcome Measures

- Primary:
  - Safety & Tolerability
  - Change from baseline in polyp burden at 12 weeks
  - RP2D
- Secondary:
  - PK/PD



Trial Update

- 5 FAP patients treated in Part 1
- 4mg dose appears pharmacologically active
- **Phase 2 initial readout expected H1 2025**

https://clinicaltrials.gov/ct2/show/NCT05552755

Clinical: AXIN1 or APC

LILAC Clinical Trial : REC-4881 for *AXIN1* or *APC* mutant cancers Phase 2

PREVALENCE & STANDARD OF CARE

~65,000 **Treatable US + EU5**

Substantial need for developing therapeutics for patients harboring mutations in *AXIN1* or *APC*, as **these mutations are considered undruggable**

To our knowledge, REC-4881 is the **only industry sponsored small molecule therapeutic** designed to enroll solid tumor patients harboring mutations in *AXIN1* or *APC*

CAUSE

LOF mutations in *AXIN1* or *APC* tumor suppressor genes

PATHOPHYSIOLOGY & REASON TO BELIEVE

Alterations in the WNT pathway are found in a **wide variety of tumors** and confer poor prognosis and resistance to standard of care

Efficacy in the Recursion OS and favorable results in PDX models harboring *AXIN1* or *APC* mutations vs wild-type leading to a significant PFS benefit in HCC and Ovarian tumors

KEY ELEMENTS

- Targeting ***AXIN1* or *APC* mutant cancers**
- MEK inhibitor, small molecule
- Oral dosing
- Phase 2 initiated late 2023
- **FPI expected Q1 2024**
- Initial **readout expected H1 2025**

Gross morphology of HCC

Clinical: AXIN1 or APC

LILAC Clinical Trial : REC-4881 for *AXIN1* or *APC* mutant cancers Phase 2

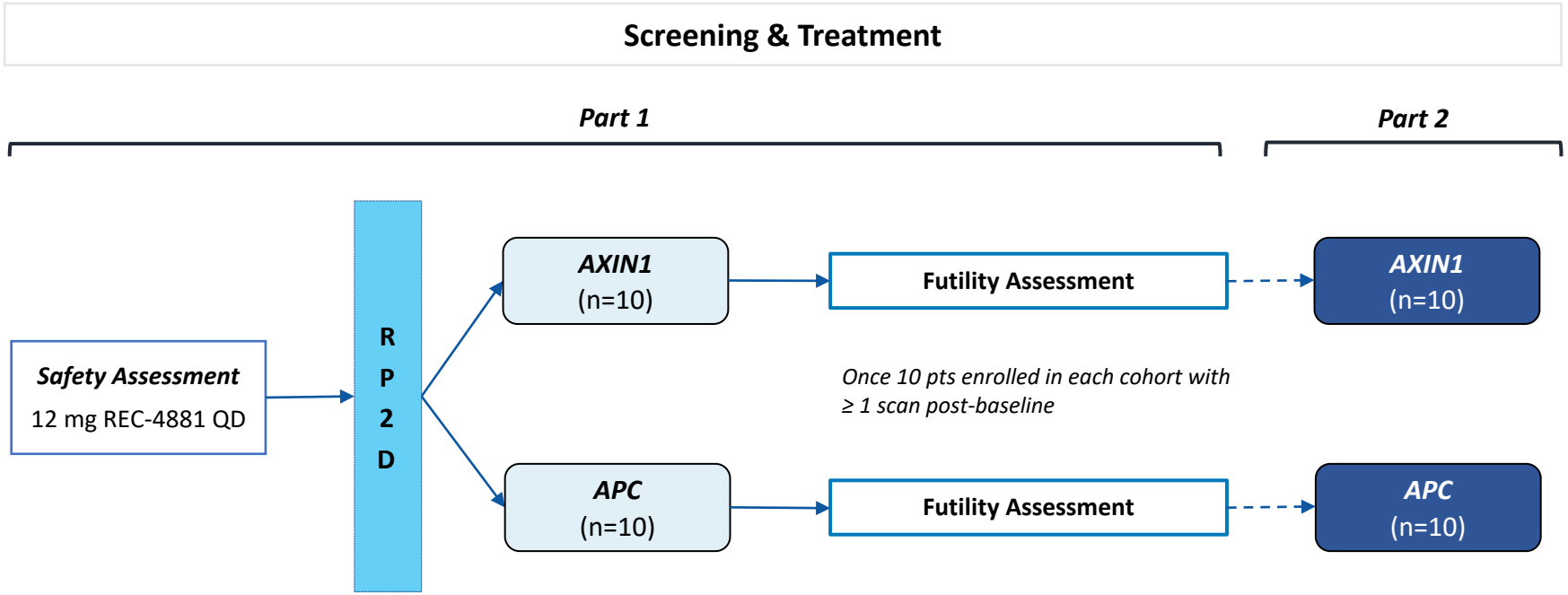
Expect FPI in Q1 2024

Enrollment Criteria

- Unresectable, locally advanced, or metastatic cancers
- ≥ 55 years old
- *AXIN1* or *APC* mutation confirmed by NGS (tissue or blood)
- CRC patients must be *RAS / RAF* wildtype
- No MEK inhibitor treatment within 2 months of initial dose
- ≥ 1 prior line of therapy
- ECOG PS 0-1

Outcome Measures

- Primary
  - Safety/tolerability
  - ORR (RECIST 1.1)
- Secondary
  - PK
  - Additional efficacy parameters



Trial Update

- Utilizing Tempus and FMI solutions for patient identification
- **Phase 2 initial readout expected H1 2025**

https://clinicaltrials.gov/ct2/show/NCT06005974

Clinical: *C. difficile*

Clinical Trial : REC-3964 for *C. difficile* Phase 1 Study Complete

PREVALENCE & STANDARD OF CARE

~730,000 **Diagnosed US + EU5**

Standard of care includes antibiotic therapies which can further impair gut flora, and lead to relapse

CAUSE

***C. difficile* toxins from colonizing bacterium** causes degradation of colon cell junction, toxin transit to bloodstream, and morbidity to host

KEY ELEMENTS

- Selective *C. difficile* toxin inhibitor, small molecule
- **Non-antibiotic approach** with potential for combination with SOC and other therapies
- Designed for **selective antitoxin pharmacology** to target infection
- Phase 1 HV study complete

PATHOPHYSIOLOGY & REASON TO BELIEVE

Highly recurrent infectious disease with severe diarrhea, colitis, and risk of toxic megacolon, sepsis, and death

Recursion OS identified a new chemical entity for recurrent *C. difficile* infection and potentially prophylaxis via glycosyl transferase inhibition with potential to be orally active

TRIAL UPDATE

- **Phase 1 PK study complete**
- REC-3964 was **well tolerated** and all AEs were Grade 1
- Expect to **initiate Phase 2 proof-of-concept study** in **2024**

Colleen – lived with rCDI

Clinical: *C. Difficile*

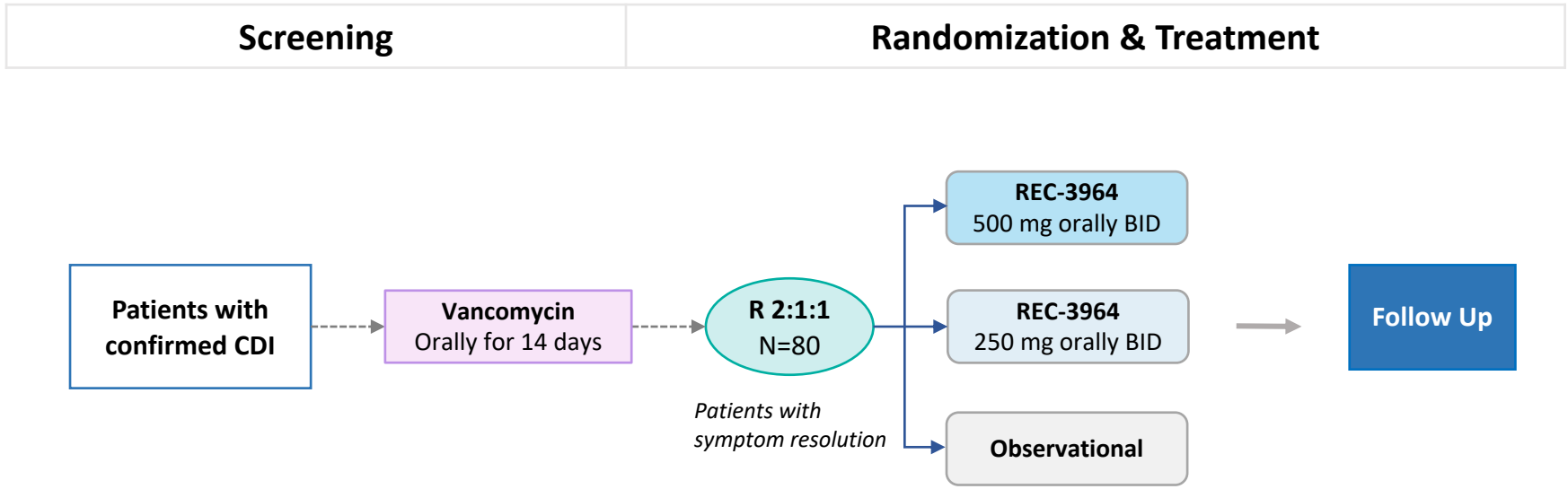
Planned Proof of Concept Phase 2 Design

Enrollment Criteria

- 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

Outcome Measures

- Primary
  - Rate of recurrence
- Secondary
  - Additional efficacy measures
  - Safety / tolerability
  - PK



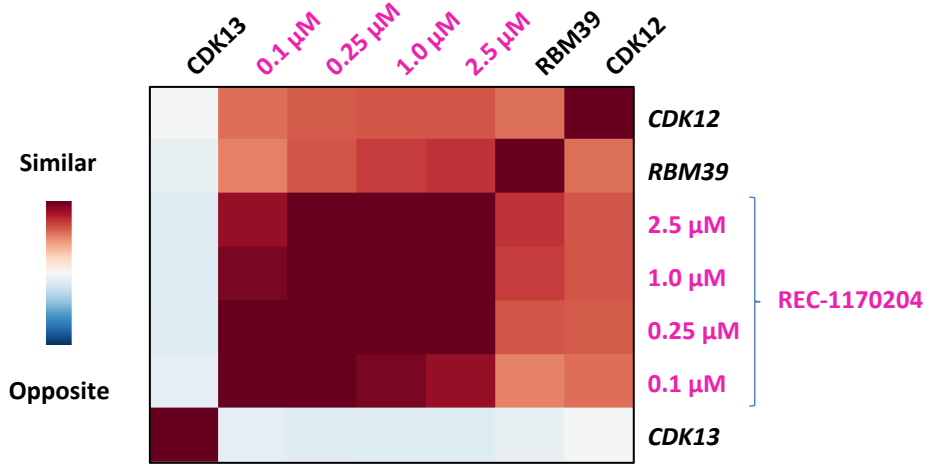
Trial Update

- NHV DDI study will precede initiation of Phase 2 POC
- Study designed to rapidly demonstrate proof of concept
- **Phase 2 initiation expected in 2024**

Recursion

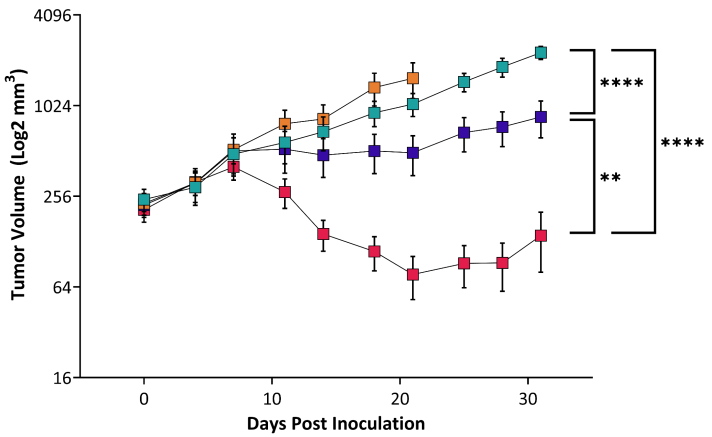
RBM39: HR-Proficient Ovarian Cancer & Other Solid Tumors

GOAL	Identify potential first-in-class tumor-targeted precision therapeutic NCE with novel MOA capable of potentially treating HR-proficient cancers
INSIGHT FROM OS	Inhibition of target RBM39 (previously referred to as Target γ) may mimic the inhibition of CDK12 while mitigating toxicity related to CDK13 inhibition
FURTHER CONFIDENCE	A Recursion-generated NCE showed single agent efficacy that is enhanced in combination with Niraparib in a BRCA-proficient PDX model
NEXT STEPS	IND submission expected in H2 2024

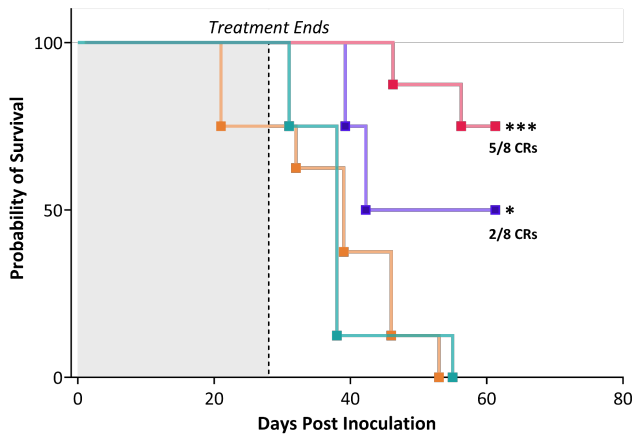


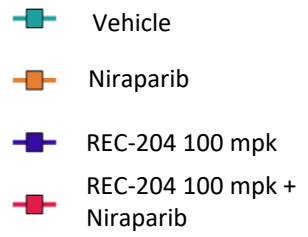
BRCA-proficient ovarian cancer PDX

OV0273 (PDX) in-vivo efficacy



Survival data





*Note: in the OV0273 PDX model, mice were treated with a representative lead molecule REC-1170204 (100 mg/kg, BID, PO) ± Niraparib (40 mg/kg, QD, PO) for 28 days. Single agent REC-1170204 or in combination with Niraparib resulted in a statistically significant response vs either Niraparib or vehicle arms. In addition, there was a statistically significant improvement in survival > 30 days post final dose. *p<0.05, ** p<0.01, **** p<0.0001*

Recursion

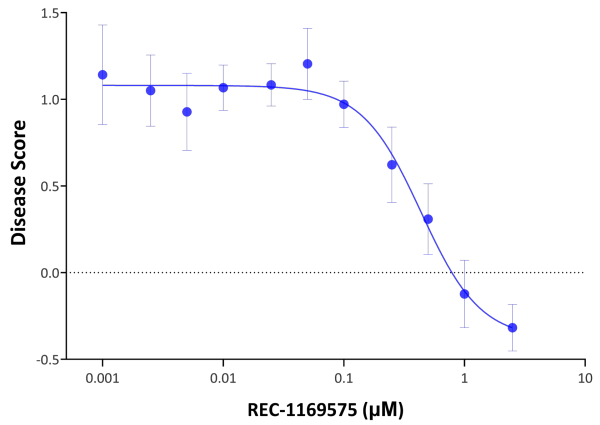
Target Epsilon: Novel Approach for Fibrotic Diseases

GOAL	Identify a potential first-in-class therapeutic NCE with a novel MOA capable of reversing disease-related fibrotic processes
INSIGHT FROM OS	Recursion-generated hits show concentration-dependent rescue in a disease relevant human PBMC assay and phenomimic genetic KO of *Target Epsilon*
FURTHER CONFIDENCE	Compelling efficacy demonstrated in a gold standard animal model of a fibrotic disease with significant unmet need
NEXT STEPS	Now entering IND-enabling studies

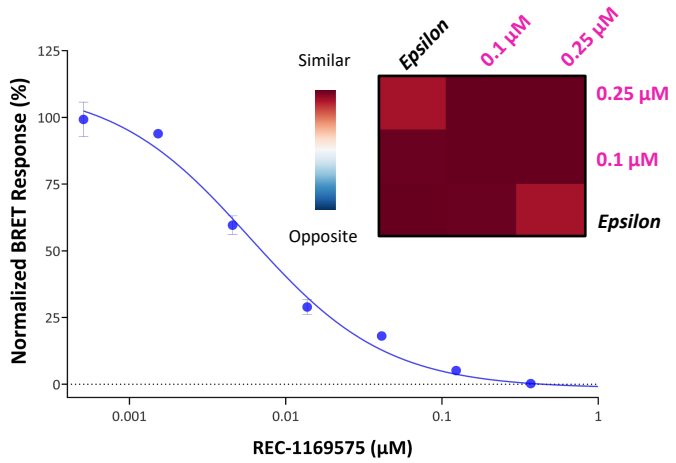
Reversal of Fibrocyte Differentiation Assay

- *Differentiation of human PBMCs into fibrocytes can be reversed by Pentraxin-2, a tissue repair protein, to mimic a healthy state*
- *Phenotypic features of healthy state can be replicated by small molecule rescue*

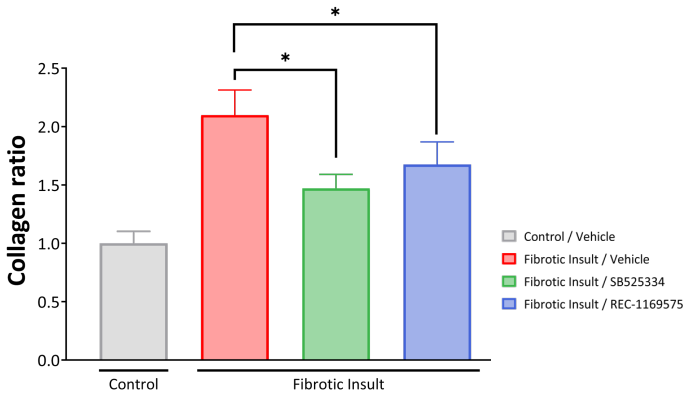
1 REC-1169575 demonstrated concentration dependent rescue in the human fibrocyte phenotypic assay [1]



2 REC-1169575 mimicked CRISPR-KO of *Epsilon* at low doses and validated in a target Epsilon engagement assay [2]



3 REC-1169575 significantly reduced collagen in a gold standard animal model of fibrotic disease [3]



*1. Disease Score of 1.0 reflects "disease state" while disease score of 0.0 reflects "healthy state." 2. Target Epsilon NanoBRET assay. 3. REC-1169575 administered 50 mg/kg BID PO. Differences between groups analyzed using Kruskal-Wallis test (*p< 0.05).*

Value Driven by Our Milestones and Team

2023 Successes

Pipeline

- Multiple Phase 2 trials began or continued enrolling patients
- Positive C Diff Phase 1 data
- Progress against multiple discovery and preclinical NCE programs moving towards the clinic

Platform

- LLMs deployed to automate significant portions of new program initiation
- Creation of Phenom-1, which we believe is the largest phenomics-based foundation model
- Predictions for ~36B ligand-protein interactions using MatchMaker
- Produced more than 1 trillion hiPSC-derived neuronal cells since 2022
- Scaled multi-timepoint phenomics and transcriptomics
- Already testing and improving causal models using patient-centric data from Tempus collaboration
- Creation of LOWE (LLM Orchestrated Workflow Engine)

Partnerships

- Roche-Genentech GI-oncology program option
- Bayer focus evolving to precision oncology
- In-licensed program from Bayer for novel target in fibrosis
- NVIDIA collaboration and investment
- Tempus collaboration signed
- Enamine collaboration signed

Business

- Cyclica and Valence acquisitions
- Expanded operations in SLC, Toronto & Montreal
- Announced expansion of Biohive capabilities (Top 50 supercomputer)
- Deliver with our team as One Recursion to continue as a leader of the TechBio industry

What to Watch for from Recursion: Potential Near-Term Milestones

- Potential for **additional INDs**
  - **HR-Proficient Cancers RBM39** in **H2 2024**
  - **In-licensed program from Bayer (Target Epsilon)** for a novel target in fibrotic diseases now entering **IND-enabling studies**
- Expected **Ph2 trial starts**
  - **Ph2 FPI** for **AXIN1 or APC mutant cancers** program expected in **Q1 2024**
  - **Ph2 initiation** for ***C. difficile*** **Infection** program in **2024**
- Expected **Ph2 readouts** for AI-discovered programs
  - **CCM** readout expected in **Q3 2024**
  - **NF2** safety & prelim efficacy expected **Q4 2024**
  - **FAP** safety & prelim efficacy expected **H1 2025**
  - **AXIN1 or APC mutant cancers** safety & prelim efficacy expected **H1 2025**
- Potential for **option exercises** for **map building** initiatives and **partnership programs**
- Potential for **additional partnership(s)** in large, intractable areas of biology (CV/Met)
- Potential to **make some data and tools** available to biopharma and commercial users
- Recursion OS moves towards **autonomous discovery**

Strong Financial Position

$392M in cash YE 2023

Cash refers to cash and cash equivalents at the end of Q4 2023

What it takes to make this happen – a new kind of team and culture

Team Members

>500 **Employees**

>50% **Advanced degrees**

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

~43% Female

~55% Male

~1% Non-Binary

Parity Pledge Signer
gender parity and people of color parity

Data shown reflective of Q4 2023, gender statistics include participating individuals

ESG Highlights

- ✓ ESG reporting on **Healthcare and Technology Metrics**
- ✓ **100% of electricity** powering our Biohive-1 supercomputer comes from renewable sources
- ✓ 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

Committed to ESG Excellence

Corporate ESG Performance
RATED BY ISS ESG
Prime

SUSTAINALYTICS
ESG 2024
INDUSTRY TOP RATED

MSCI ESG RATINGS
A
CCC | B | BB | BBB | A | AA | AAA

Our leadership team brings together experience & innovation to lead TechBio

Board of Directors

R Martin Chavez, PHD
Chairman of RXRX,
Board Member of Alphabet,
Vice-Chairman of 6th Street,
Former CFO/CIO of GS

Alphabet · 6 SIXTH STREET · Goldman Sachs

Chris Gibson, PHD
Co-Founder & CEO

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

Terry-Ann Burrell, MBA
CFO & Treasurer,
Beam Therapeutics

Beam THERAPEUTICS · J.P.Morgan

Rob Hershberg, MD PHD
Co-Founder/CEO/Chairman of HilleVax, Former EVP/CSO/CBO of Celgene

Celgene

Blake Borgeson, PHD
Co-Founder of RXRX

MIRI · BUILDASIGN.com

Zachary Bogue, JD
Co-Founder & Partner of Data Collective

DCVC

Executive Team

Chris Gibson, PHD
Co-Founder & CEO

Tina Marriott
President & COO

Roche · Genentech · ACHAOGEN

Michael Secora, PHD
Chief Financial Officer

LAURION Capital Management LP

David Mauro, MD PHD
Chief Medical Officer

CODIAK · CHECKMATE PHARMACEUTICALS

Ben Mabey
Chief Technology Officer

Laura Schaevitz, PHD
SVP & Head of Research

VIUM

Kristen Rushton, MBA
Chief Business Ops Officer

Myriad genetics

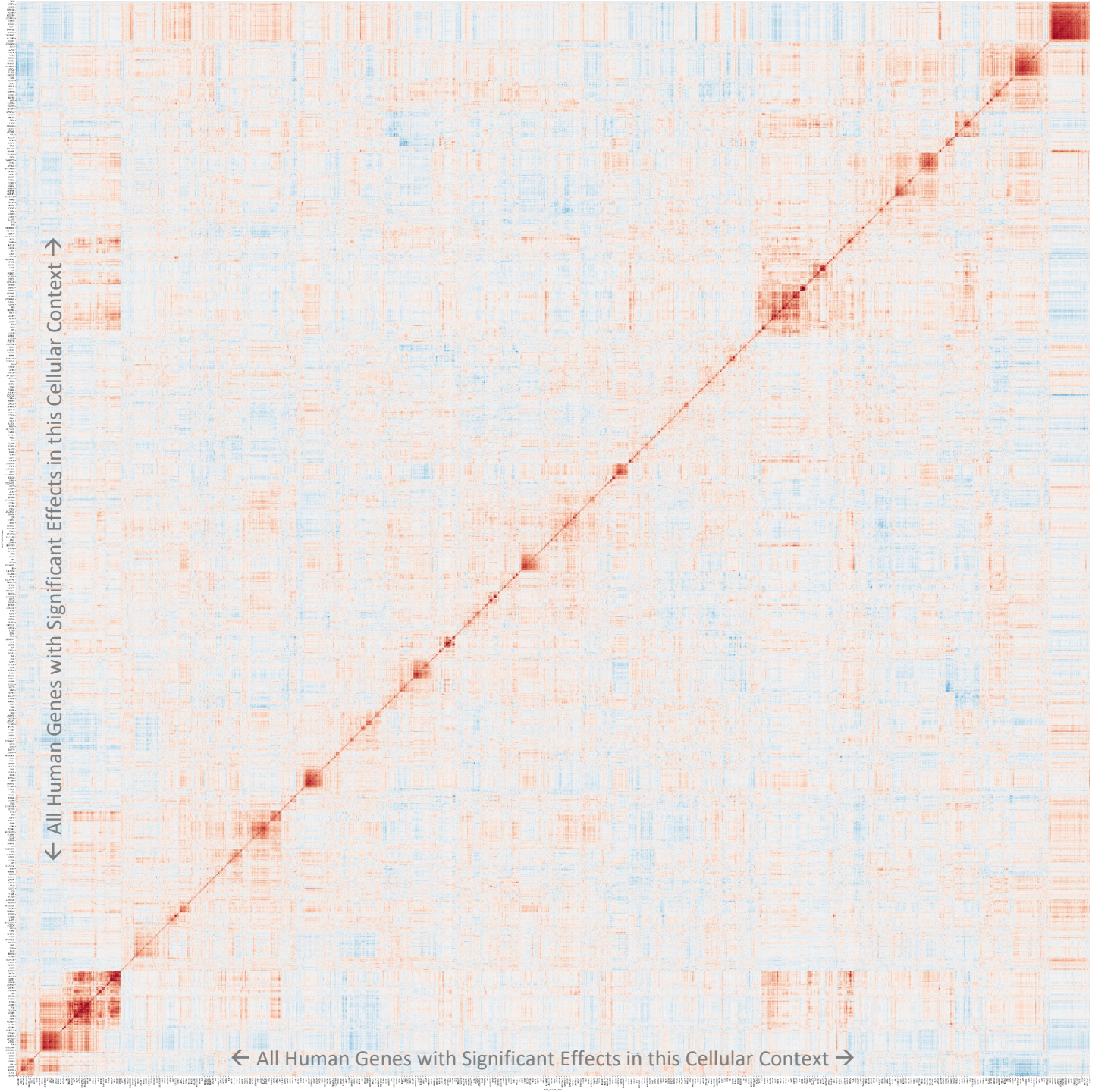
Nathan Hatfield, JD MBA
Chief Legal Officer

WILSON SONSINI

Matt Kinn, MBA
SVP Business Development

BCG · UBS

Appendix



Recursion

Genome-scale mapping

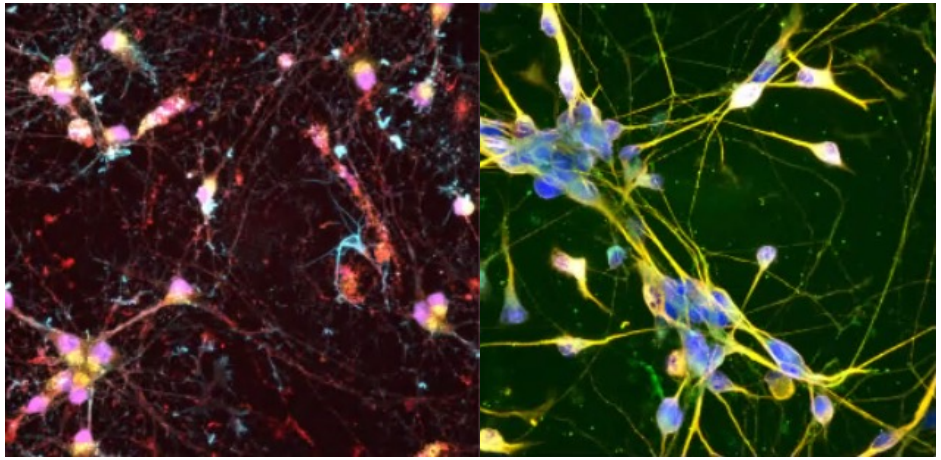
This is a **whole-genome arrayed CRISPR knock-out Map** generated in primary human endothelial cells

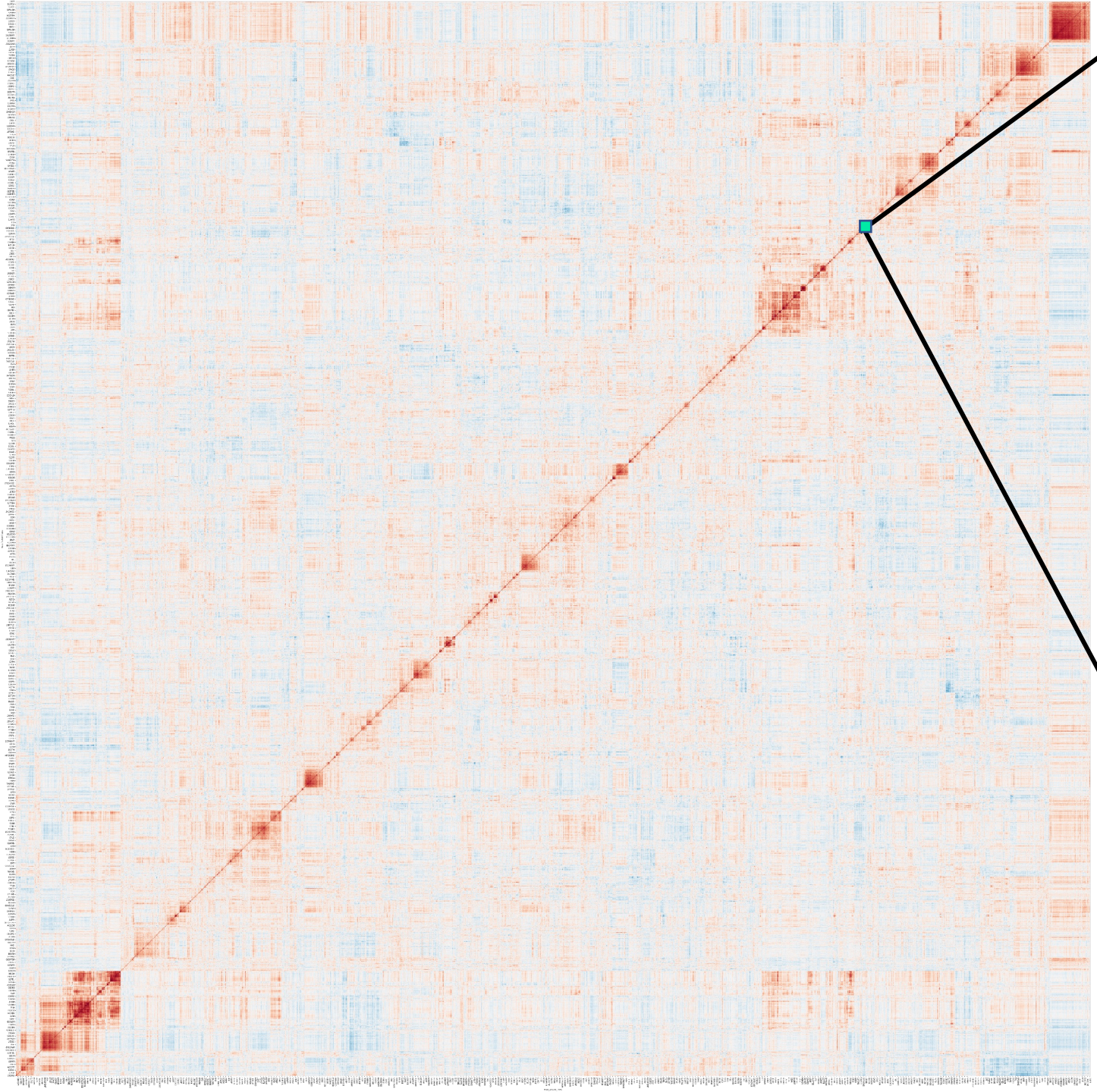
Every gene is represented in a pairwise way (each is present in columns and rows)

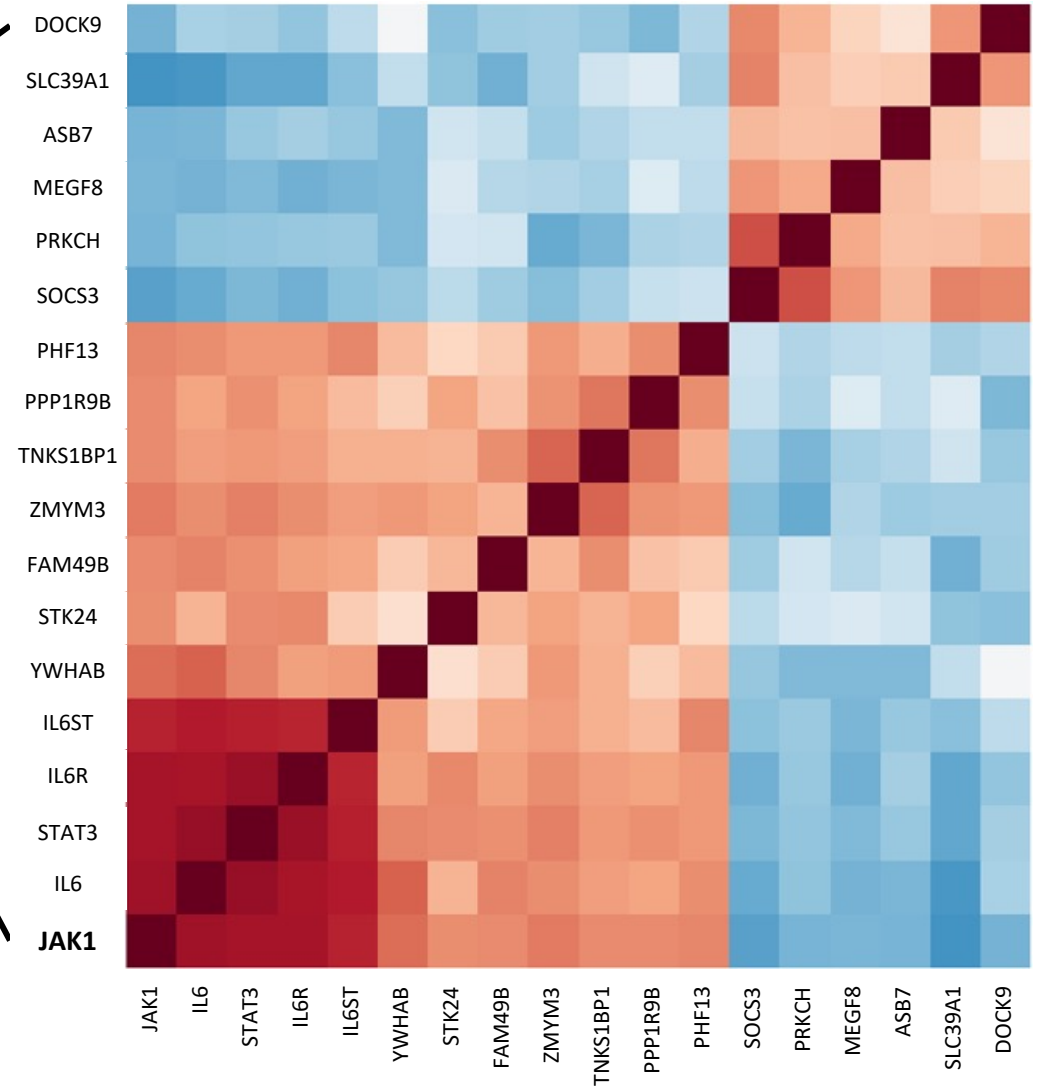
Dark Red indicates phenotypic similarity according to our neural networks while **Dark Blue** indicates phenotypic anti-similarity (which in our experience often suggests negative regulation)

We can add the phenotypes of hundreds of thousands of small molecules at multiple doses and query and interact with these maps using a web application

Thousands of examples of known biology and chemistry







One such example – the **JAK / STAT pathway** clustered by strength of interaction, including both **similar genes (red)** and **opposite genes (blue)**

Can wade into areas of **novel biology and chemistry…**

COVID-19 research: Recursion OS correctly predicted 9 of 10 clinical trials

Drug	Prediction	Correct?
Hydroxychloroquine	Negative	✓
Lopinavir	Negative	✓
Ritonavir	Negative	✓
Remdesivir	Positive	✓
Baricitinib	Positive	✓
Tofacitinib	Positive	✓
Fostamatinib	Positive	✓
Ivermectin*	Negative	✓
Fluvoxamine	Negative	✓
Dexamethasone	Negative	x

** Recursion did not screen ivermectin but did screen the related compounds selamectin and doramectin. Both of these tested negative; consequently, ivermectin was not expected to have efficacy. Fostamatinib recently read out positive Ph3 results in COVID but was discontinued in ACTIV-4.*

- Recursion conducted several AI-enabled experiments in **April 2020** to investigate therapeutic potential for COVID-19
- Included FDA-approved drugs, EMA-approved drugs, and compounds in late-stage clinical trials for the modulation of the effect of SARS-CoV-2 on human cells
- Experiments were compiled into the **RxRx19 dataset** (860+ GB of data) and **made publicly available** to accelerate the development of methods and pandemic treatments.

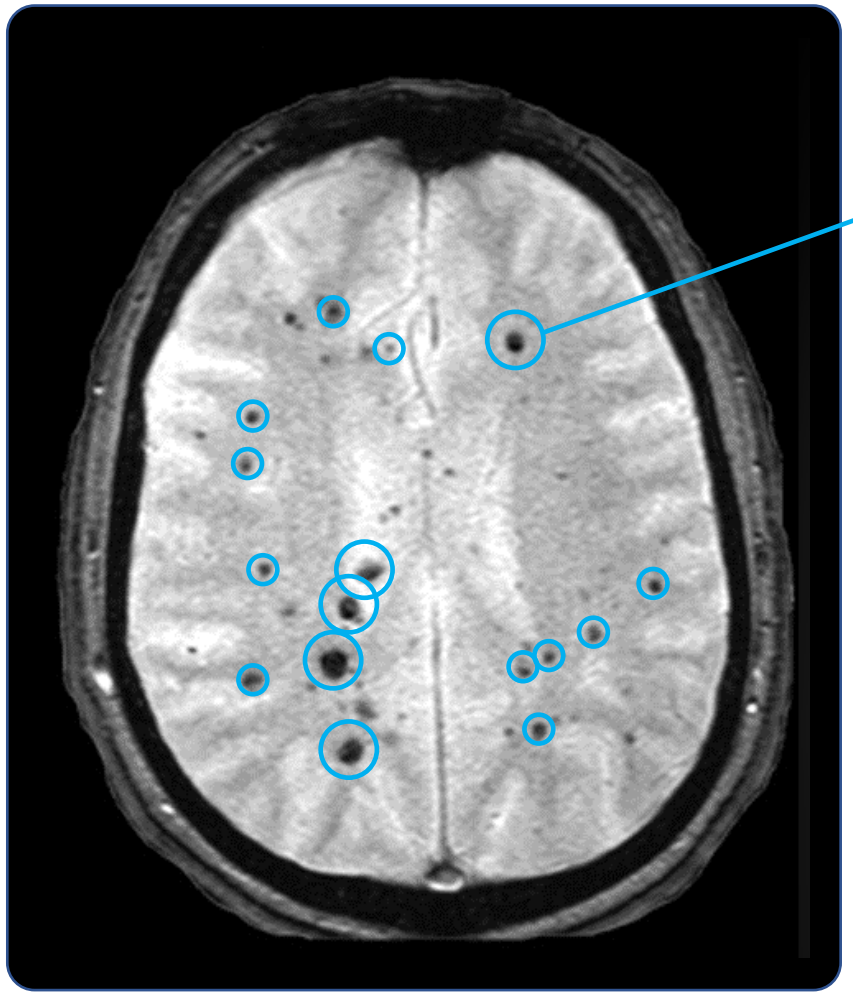
https://www.biorxiv.org/content/10.1101/2020.04.21.054387v1

REC-994 for the Treatment of Symptomatic Cerebral Cavernous Malformations (CCM)

Target / MOA	Superoxide Scavenger
Molecule Type	Small Molecule
Lead Indication(s)	Cerebral Cavernous Malformations
Status	Phase 2
Designation(s)	US & EU Orphan Drug
Source of Insight	Recursion OS

Clinical: CCM

Disease Overview : Cerebral Cavernous Malformations (CCM)



Description

- Large unmet need for a novel nonsurgical treatment
- Vascular malformations (cavernomas) in the brain and spinal cord
- High-risk for hemorrhage creates “ticking time bomb”
- Progressive increase in CCM size and number over time in those with familial disease
- Debilitating symptoms, including intractable seizure, intracerebral hemorrhage, focal neurological deficits

“Historically, cavernomas have been managed primarily with observation, surgical resection, and occasionally radiotherapy. However, for a number of reasons, many patients with cavernomas must endure a life with neurologic symptoms”

- **Ryan Kellogg**, MD, Investigator at the University of Virginia

Clinical: CCM

Disease Overview : Cerebral Cavernous Malformations (CCM)

Julia – living with CCM

Patient Population – Large and Diagnosable

- **>1 million patients** worldwide live with these lesions today
- Caused by loss of function mutation in one of three genes: *CCM1* (60%), *CCM2* (20%), and *CCM3* (20%)
- Inherited autosomal dominant mutation in 30-40%; or sporadic
- US symptomatic population is more than 5 times larger than other rare diseases like **Cystic Fibrosis** (>31k patients) and **Spinal Muscular Atrophy** (>33k patients)

No Approved Medical Therapy

- **No approved drugs** for CCM
- Most patients receive **no treatment** or only **symptomatic therapy**
- Surgical resection or stereotactic radiosurgery not always feasible because of location of lesion and is not curative

~360,000 Symptomatic US + EU5 patients

Sources: Angioma Alliance ; Flemming KD, et al . Population-Based Prevalence of Cerebral Cavernous Malformations in Older Adults: Mayo Clinic Study of Aging. JAMA Neurol. 2017 Jul 1;74(7):801-805. doi: 10.1001/jamaneurol.2017.0439. PMID: 28492932; PMCID: PMC5647645 ; Spiegler S, et al Cerebral Cavernous Malformations: An Update on Prevalence, Molecular Genetic Analyses, and Genetic Counselling. Mol Syndromol. 2018 Feb;9(2):60-69. doi: 10.1159/000486292. Epub 2018 Jan 25. PMID: 29593473; PMCID: PMC5836221.

Clinical: CCM

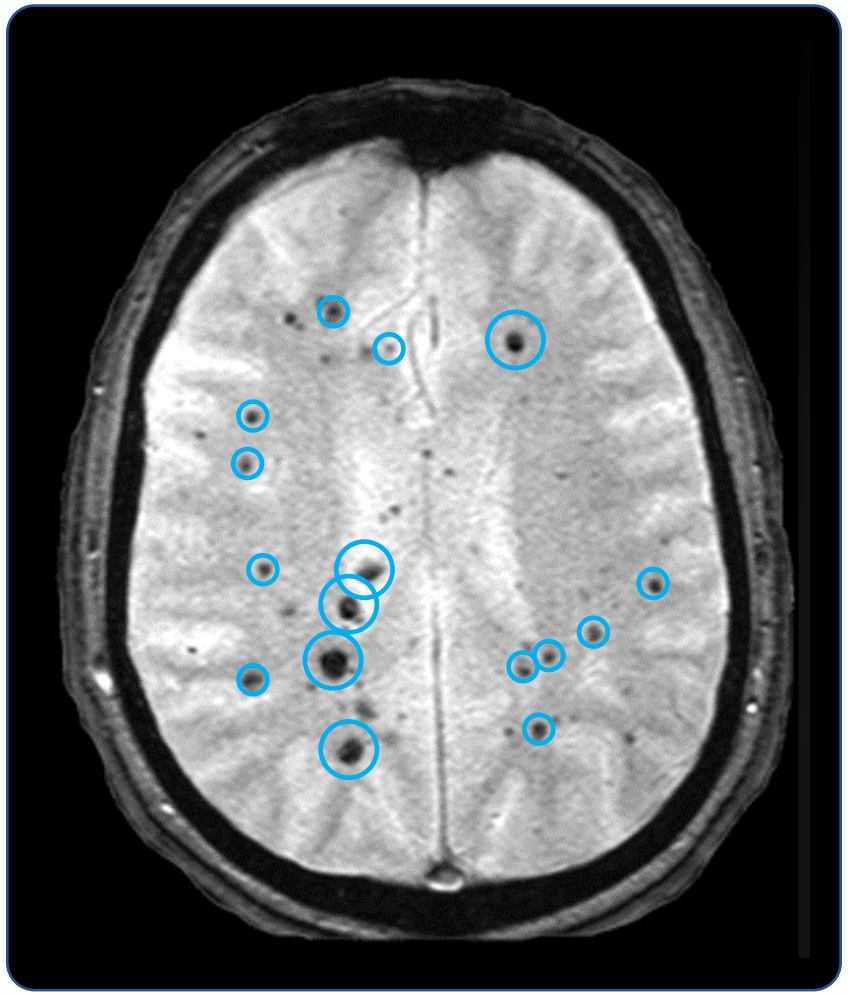
Disease Overview : CCM is an Under-Appreciated Orphan Disease

Non-oncology Orphan Indication	Product	U.S. + EU5 Prevalence
Cerebral cavernous malformation (CCM)	**REC-994 (Recursion)**	**>1,800,000 (Symptomatic: ~360,000)**
Idiopathic pulmonary fibrosis (IPF)	Esbriet (pirfenidone)	>160,000
Cystic fibrosis (CF)	VX-669/ VX-445 + Tezacaftor + Ivacaftor - Vertex	>55,000
Spinal muscular atrophy (SMA)	SPINRAZA (nusinersen)	>65,000

Sources: Angioma Alliance ; Flemming KD, et al . Population-Based Prevalence of Cerebral Cavernous Malformations in Older Adults: Mayo Clinic Study of Aging. JAMA Neurol. 2017 Jul 1;74(7):801-805. doi: 10.1001/jamaneurol.2017.0439. PMID: 28492932; PMCID: PMC5647645 ; Spiegler S, et al Cerebral Cavernous Malformations: An Update on Prevalence, Molecular Genetic Analyses, and Genetic Counselling. Mol Syndromol. 2018 Feb;9(2):60-69. doi: 10.1159/000486292. Epub 2018 Jan 25. PMID: 29593473; PMCID: PMC5836221; Maher T, et al Global incidence and prevalence of idiopathic pulmonary fibrosis. Respir Res. 2021 Jul 7;22(197). Doi: 10.1186/s12931-021-01791-z. PMID: 34233665. DRG 2022 Solutions, Report: Epidemiology, Cystic Fibrosis. CDC: SMA

Clinical: CCM

Therapeutic Approach to Cerebral Cavernous Malformations (CCM)



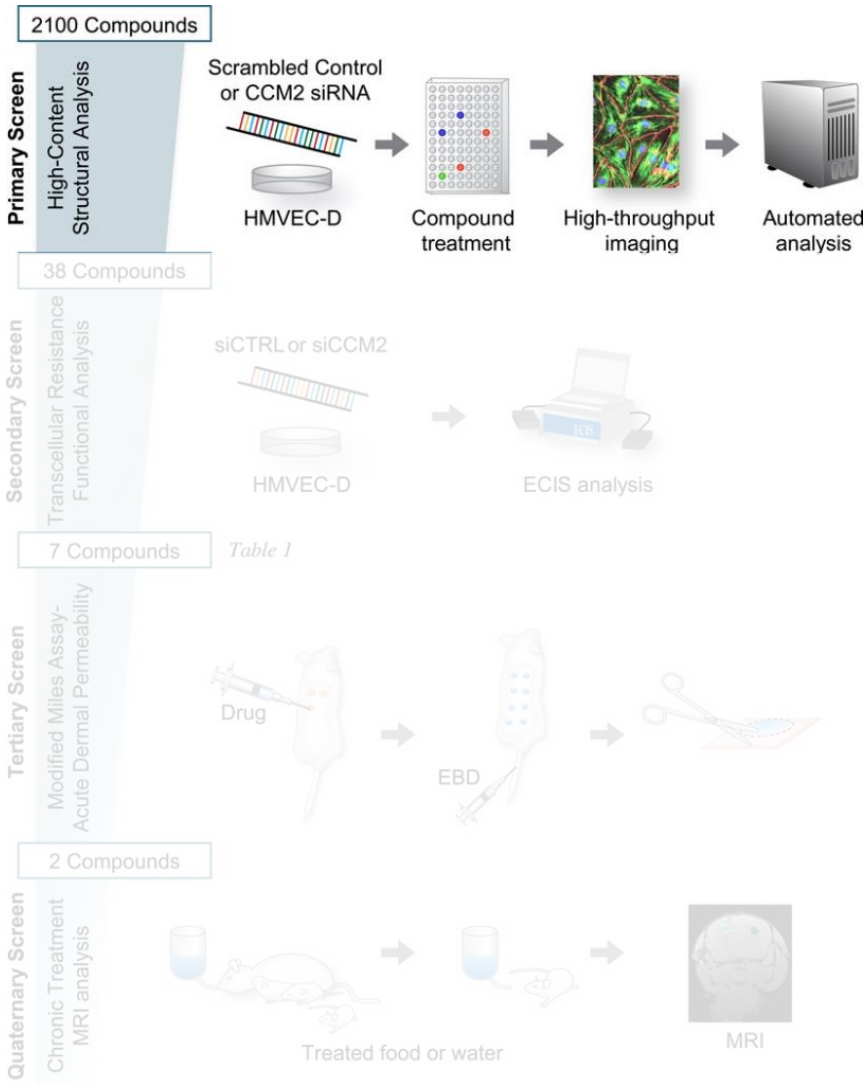
Novel therapeutic approach

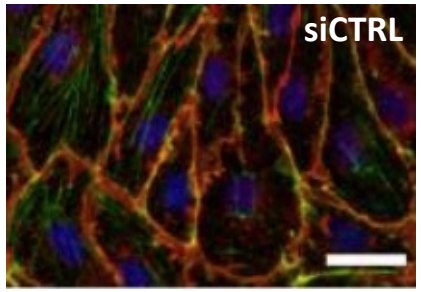
- Symptoms associated with both increased size of lesions, but also inflammation or activation of lesions within the immunoprivileged environment of the brain
- Lesions arise from the capillary bed and are not high-pressure (e.g., the lesion growth is unlikely to be primarily driven by the *law of Laplace*)

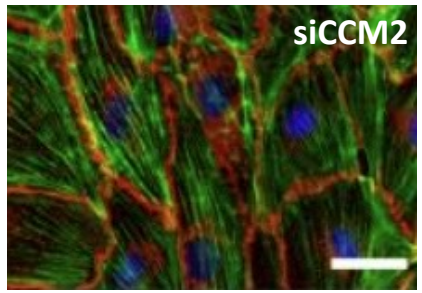
- *The Recursion Vascular Stability Hypothesis:*
  - Eliminating the lesions may <u>not</u> be required for significant patient benefit
  - Slowing or halting the growth of the lesions while mitigating lesion leakiness and endothelial cell activation to halt the feed-forward inflammatory reaction *may* mitigate some symptoms and be beneficial to patients

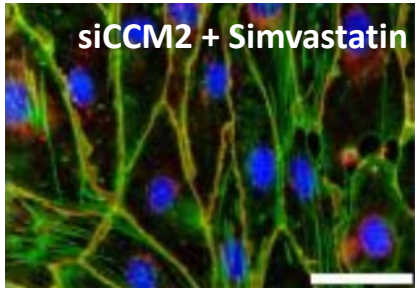
Clinical: CCM

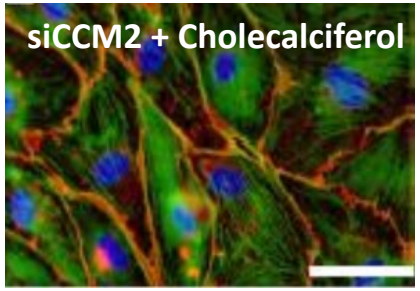
CCM – Applied prototyping of the Recursion OS

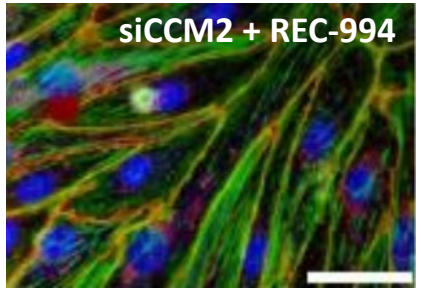












Using an early version of our Recursion OS in an academic setting, we identified about 39 molecules out of 2,100 screened that according to a machine learning classifier rescued a complex unbiased phenotype associated with CCM2 loss of function.

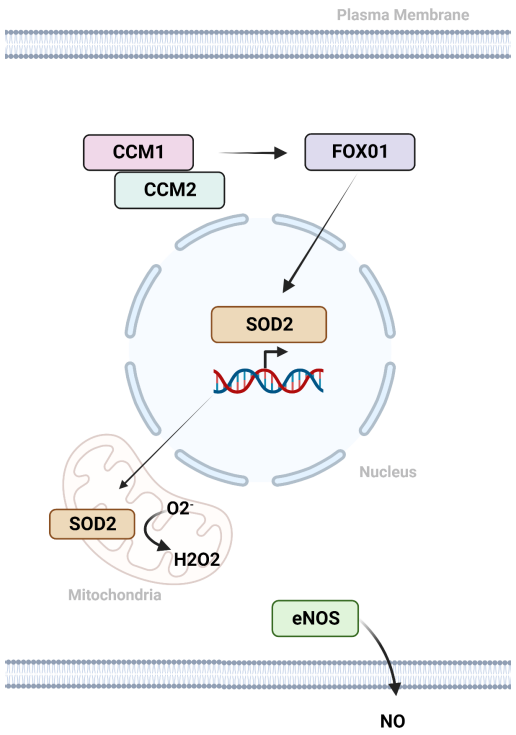
Through a set of follow-on confirmatory assays of increasing complexity, REC-994 stood out as one of two compounds we tested in a 5-month chronic CCM animal model where both compounds demonstrated significant benefit.

Gibson, et al. Strategy for identifying repurposed drugs for the treatment of cerebral cavernous malformation. Circulation, 2015

Clinical: CCM

REC-994 – Mechanism of Action

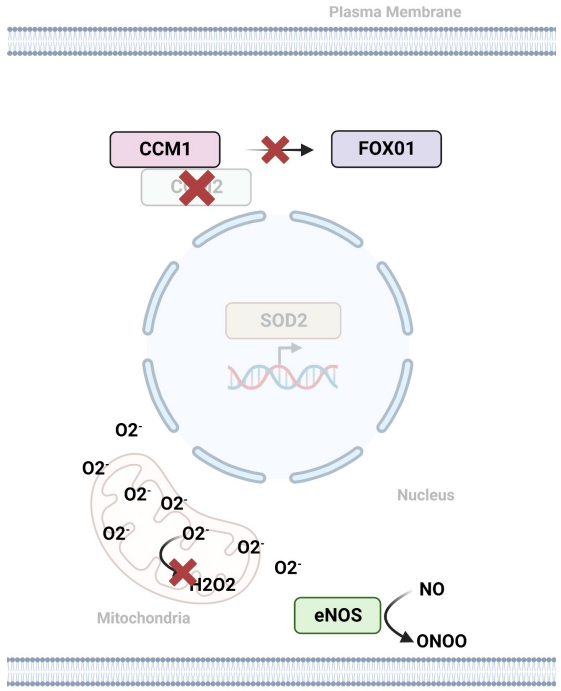
Healthy



By regulating SOD2, CCM1 (KRIT1) & CCM2 suppress:

- Endothelial cell activation
- Smooth muscle proliferation
- Leukocyte adhesion
- Platelet aggregation

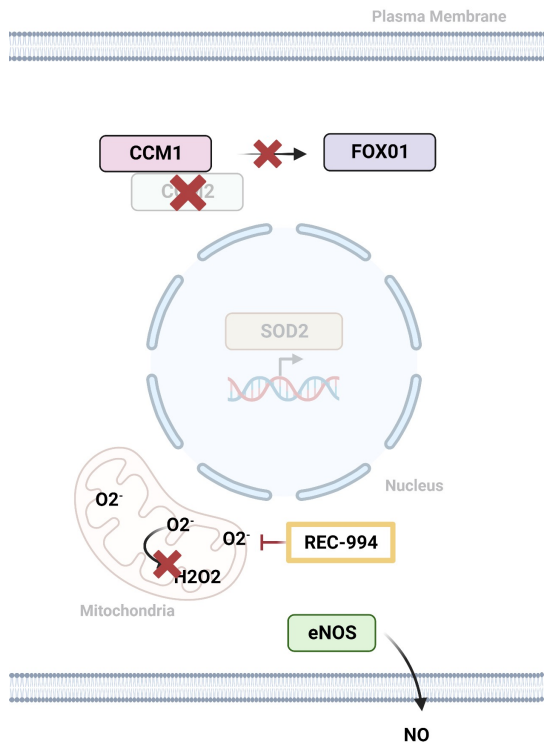
CCM



***CCM1* or *CCM2* loss of function leads to activated endothelium:**

- Decreased cell-cell junctional integrity and increased monolayer permeability
- Impaired vasodilation
- Cavernous angioma formation

REC-994 Impact



Dosing of REC-994 restores normal function:

- Normalized ROS balance
- Restores quiescent endothelial cell state
- Stabilizes endothelial barrier function

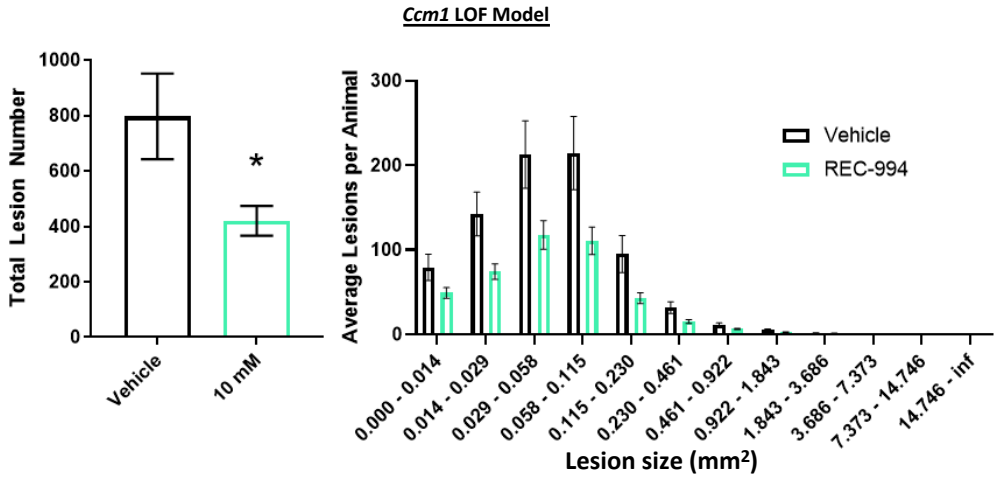
Adapted from REC-994 Investigator Brochure

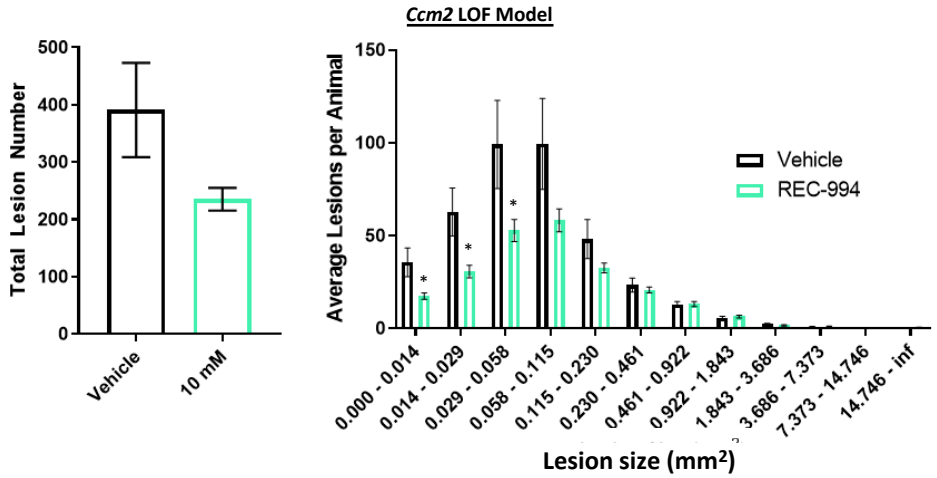
Clinical: CCM

Further Confidence : Preclinical Studies Confirm Insight

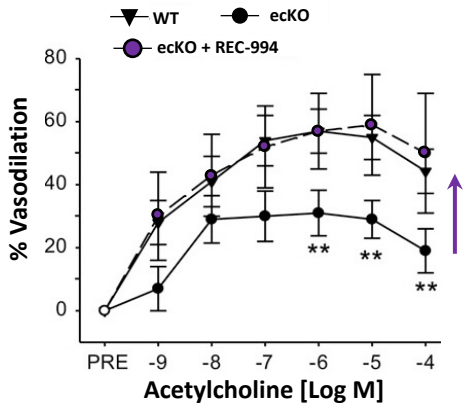
Preclinical Studies: REC-994 reduces lesion burden and ameliorates vascular defects in genetic mouse models of CCM

1 Reduces lesion number and size in *Ccm1* and *Ccm2* LOF mouse models



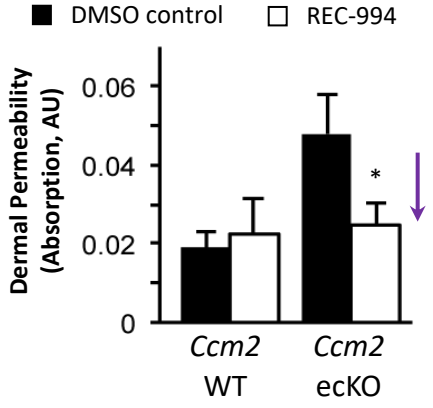


2 Completely rescues acetylcholine-induced vasodilation defect



REC-994 stabilizes the integrity of vasculature against challenges to permeability

3 Rescues dermal permeability defect in CCM2 mice



Vascular permeability is a clinically relevant feature of CCM lesions

Source: Data above from Gibson, et al. Strategy for identifying repurposed drugs for the treatment of cerebral cavernous malformation. Circulation, 2015 or Recursion internal data (Ccm1 mouse model)

Clinical: CCM

Further Confidence : Clinical Studies Confirming Safety

REC-994 Phase 1 Studies - well-tolerated with no dose-dependent adverse events in SAD and MAD

MAD Study	Placebo	50 mg	200 mg	400 mg	800 mg
Total Number of TEAEs	5	0	10	4	15
Total Subjects with ≥ one TEAE	4	0	3	3	4
Severity					
Mild	3	0	3	3	3
Moderate	1	0	0	0	1
Severe	0	0	0	0	0
Relationship to Study Drug					
None	3	0	0	2	1
Unlikely	1	0	1	1	2
Possibly	0	0	0	0	0
Likely	0	0	2	0	1
Definitely	0	0	0	0	0
Total Number of SAEs	0	0	0	0	0
Total Subject with ≥ one TEAE	0	0	0	0	0
Discontinued Study Drug Due to AE	0	0	0	0	0

Source: REC-994 for the Treatment of Symptomatic Cerebral Cavernous Malformation (CCM) Phase 1 SAD and MAD Study Results. Oral Presentation at Alliance to Cure Scientific Meeting. 2022 Nov 17

REC-994 *for* Symptomatic Cerebral Cavernous Malformations (CCM)

Target Product Profile:

Population not restrictive.
Targeting both sporadic and familial patients

Differentiated mechanism of action.
Decreases ROS and oxidative stress

Well-tolerated with no DLTs or SAEs in Phase 1.
No treatment-related discontinuations

Drug-like properties support oral dosing QD.
Therapeutic exposures achievable in humans

REC-994 for Cerebral Cavernous Malformations (CCM)

First-in-disease potential in CCM with a first-in-class orally bioavailable small molecule superoxide scavenger

Program Overview	• First therapeutic candidate advanced to an industry-sponsored Phase 2 trial (SYCAMORE) for CCM • Partnered with leading KOLs at University of Rochester to develop a CCM PRO instrument for clinical trials • Putative MOA decreases ROS and oxidative stress to rescue pathogenetic endothelial dysfunction
Clinical Updates	• Favorable safety and tolerability profile in Phase 1 dose-escalation with no DLTs and no SAEs • Phase 2 trial fully accrued ahead of schedule in June 2023, enrolling 62 symptomatic CCM patients • Majority of patients treated with REC-994 for ≥ 12 months have opted into LTE portion
Near-term Catalysts	• Phase 2 readout (safety, preliminary efficacy, pharmacokinetics) expected Q3 2024 • Results from Phase 2 expected to inform defined registration path with guidance from FDA
Commercial Opportunity	• ~360,000 symptomatic CCM patients living in US and EU5 with no pharmacological agents approved • Favorable competitive landscape with REC-994 2+ years ahead in development
IP & Exclusivity	• ODD in US and EU provides 7 and 10 years, respectively, of market exclusivity following approval • Method of use patents provide protection until 2035 (excluding extensions)

REC-2282 for the Treatment of Progressive Neurofibromatosis Type 2 (NF2) Mutated Meningiomas

Target / MOA	HDAC Inhibitor
Molecule Type	Small Molecule
Lead Indication(s)	NF2 Mutated Meningiomas
Status	Phase 2/3
Designation(s)	Fast Track; US and EU Orphan Drug
Source of Insight	Recursion OS

Clinical: NF2

Disease Overview : Neurofibromatosis Type 2 (NF2)

Ricki – living with NF2

Patient Population – Large and Diagnosable

- Rare autosomal dominant tumor syndrome resulting from biallelic inactivation of the *NF2* gene which leads to deficiencies in the tumor suppressor protein merlin
- **NF2 can be inherited or spontaneous** (>50% of patients represent new mutations); up to 1/3 are mosaic
- CNS manifestations: meningiomas and vestibular schwannomas; mean age at presentation: **~20 years**

No Approved Medical Therapy

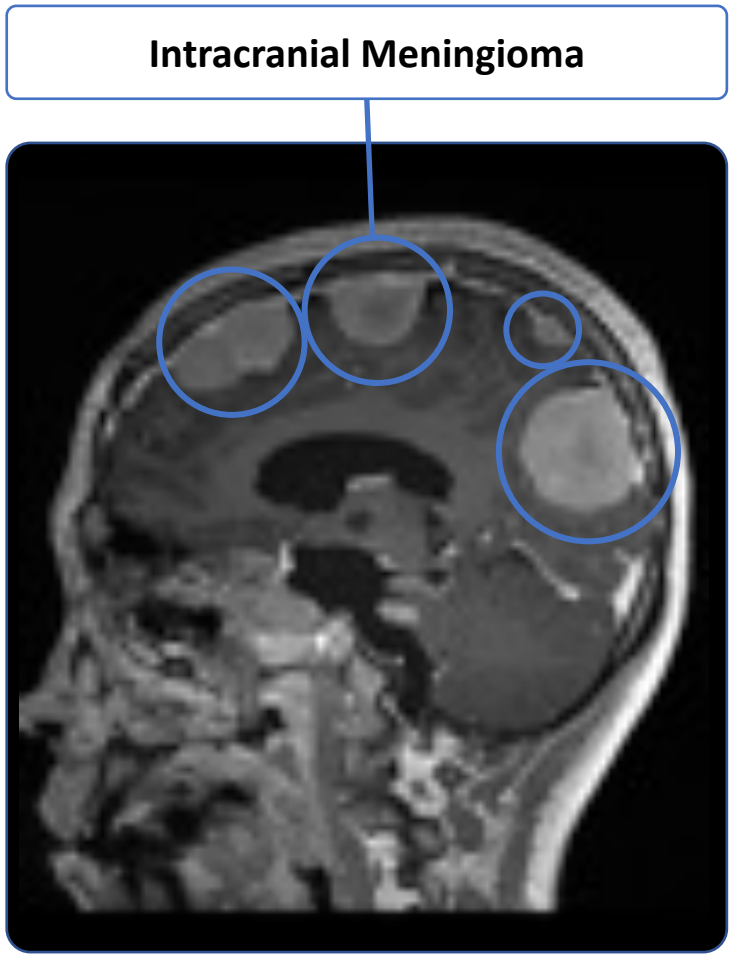
- **No approved drugs** for NF2
- **Surgery** is standard of care (when feasible)
- Location may make complete resection untenable, leading to hearing loss, facial paralysis, poor balance and visual difficulty

Source: https://rarediseases.org/rare-diseases/neurofibromatosis-2

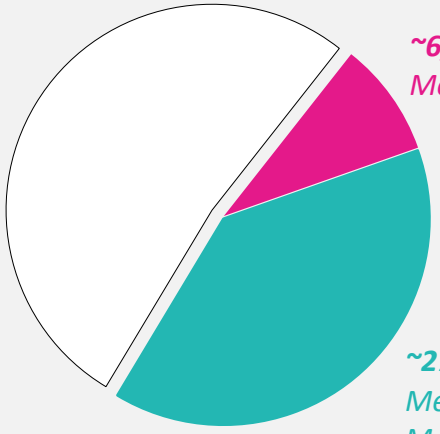
Clinical: NF2

Disease Overview : Neurofibromatosis Type 2 (NF2) Meningiomas

- Most tumors are benign and slow growing but location in CNS leads to serious morbidity or mortality
- Prognosis is adversely affected by early age at onset, a higher number of meningiomas and having a truncating mutation



>66,000 Patients have Meningiomas



~6,000 NF2 Patients have Meningiomas (Familial)

~27,000 Patients who have Meningiomas that Harbor NF2 Mutations (Sporadic)

~33,000

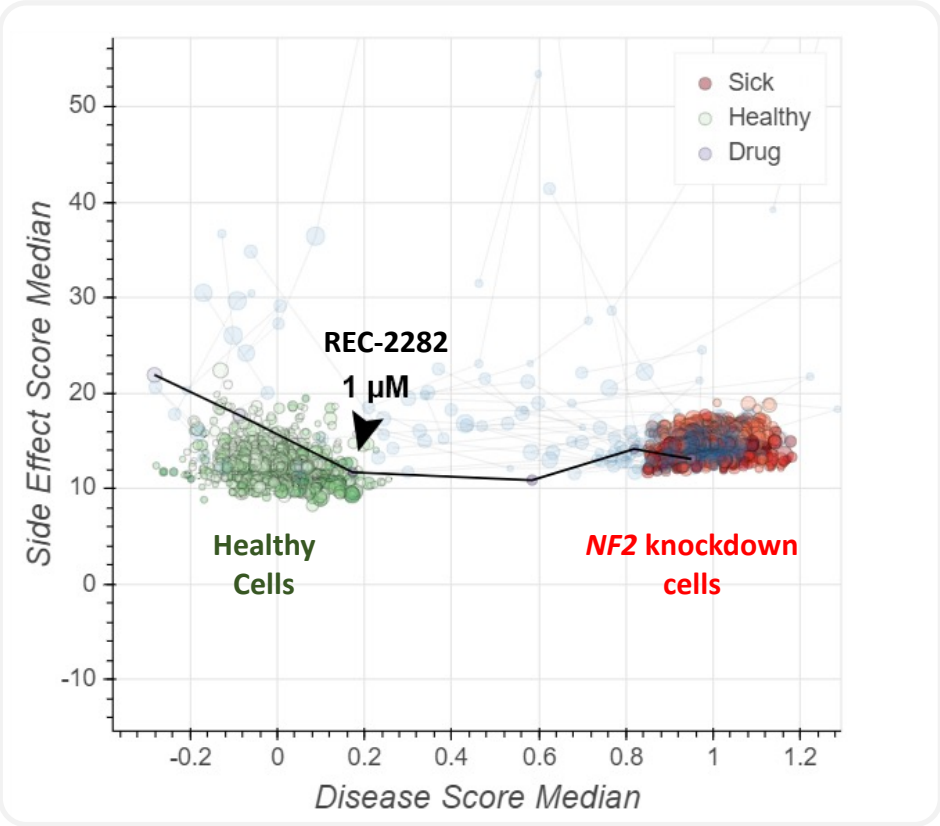
Treatable US + EU5 patients

- Threatens mortality; if amenable, surgical excision is primary intervention
- Many patients have multiple meningiomas that exhibit heterogenous behavior and asynchronous growth
- **Stasis or shrinkage of tumor could improve prognosis**

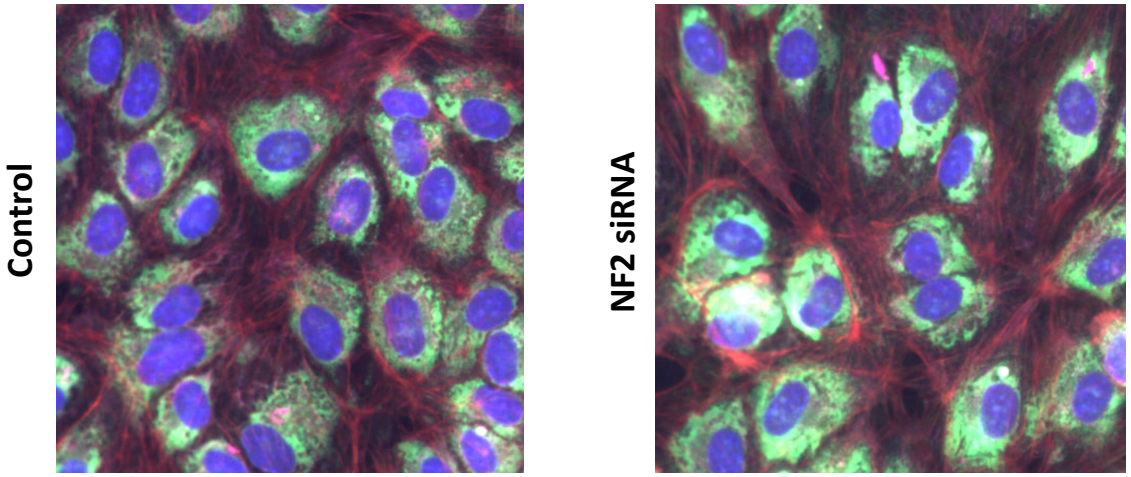
Source: Pemov, et al. Comparative clinical and genomic analysis of neurofibromatosis type 2-associated cranial and spinal meningiomas. Nature. 2020 Jul 28;10(12563). Doi: https://doi.org/10.1038/s41598-020-69074-z; NORD

Clinical: NF2

Insight from OS : REC-2282 Rescued Loss of *NF2*



REC-2282 identified as rescuing HUVEC cells treated with NF2

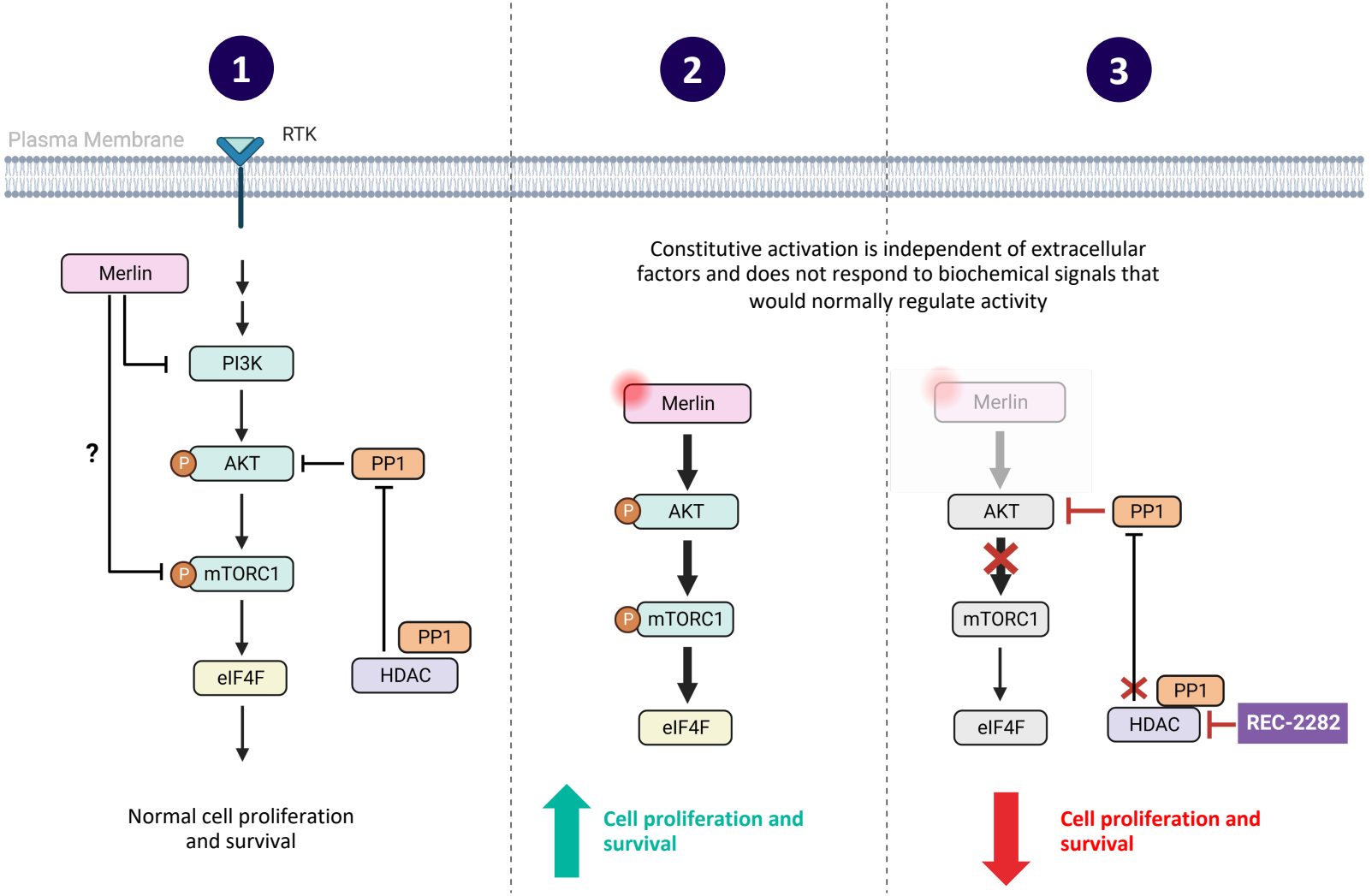


HUVEC, human umbilical vein endothelial cells; NF2, neurofibromatosis type 2; siRNA, small interfering RNA.

Clinical: NF2

REC-2282 – Mechanism of Action

Orally Bioavailable, CNS-penetrating, Small Molecule HDAC Inhibitor



1. NF2 encodes for the protein Merlin and negatively regulates mTOR signaling

2. Loss of Merlin leads to increased signaling in the PI3K/AKT/mTOR pathway

3. Oncogenic mTOR signaling arrested with HDAC inhibitors

AKT, protein kinase B; eIF4F, eukaryotic initiation factor 4F; HDAC, histone deacetylase; mTor, mammalian target of rapamycin; mTORC1; mammalian target of rapamycin complex 1; NF2, neurofibromatosis type 2; PI3K, phosphoinositide 3-kinase; PP1, protein phosphate 1; Ras, reticular activating system.

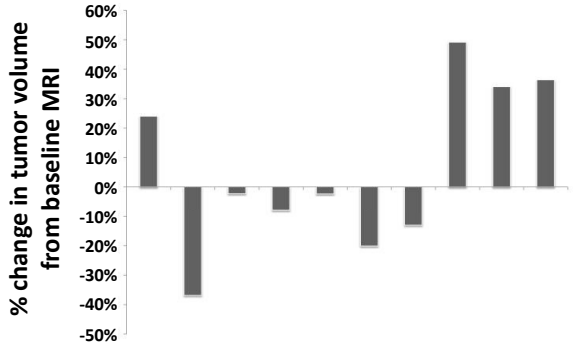
Clinical: NF2

Further Confidence : Preclinical Studies Confirming Insight

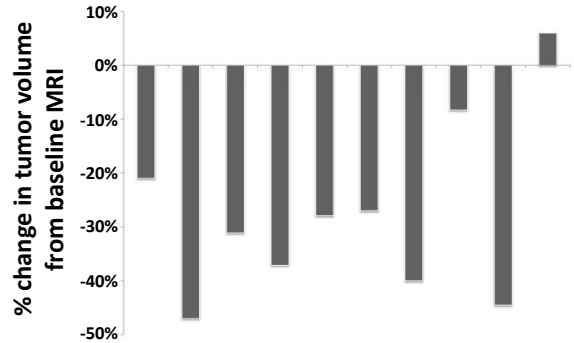
REC-2282 preclinical studies demonstrated clear in-vivo efficacy in multiple NF2 tumor types

1 **Shrinks vestibular schwannoma xenografts in nude mice**

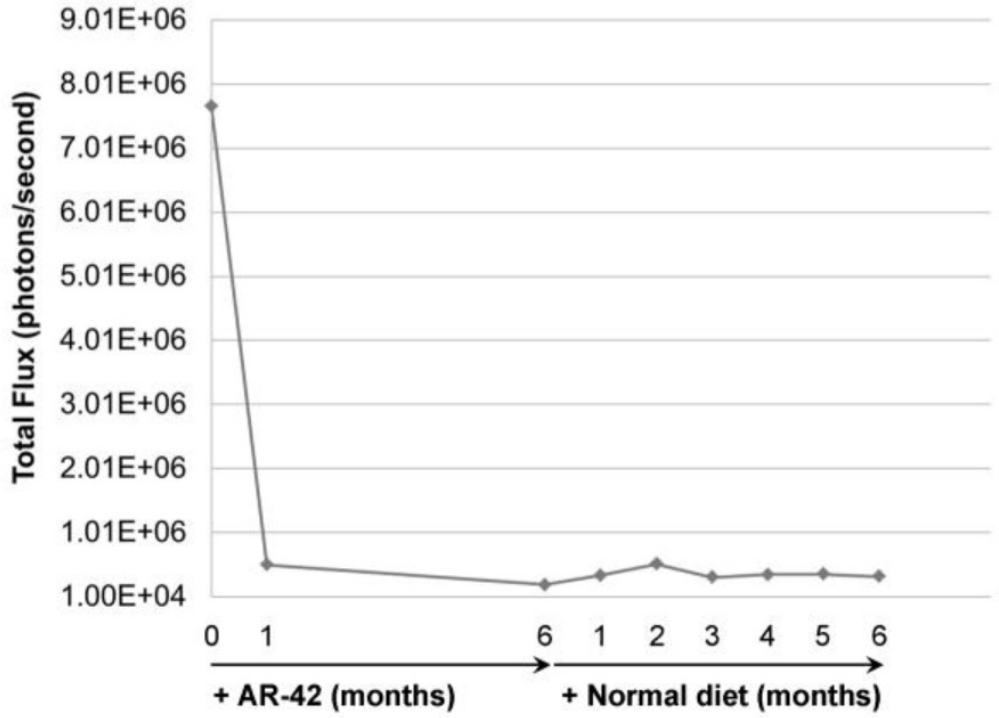
Vehicle
% Change tumor vol.



REC-2282
% Change tumor vol.



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

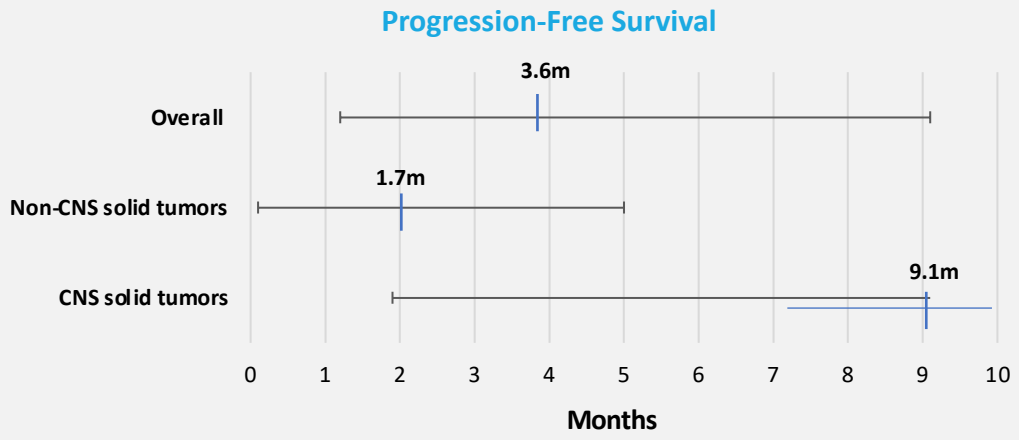


https://link.springer.com/article/10.1007/s00280-020-04229-3

Clinical: NF2

Further Confidence : Prior Studies Suggest Potential Therapeutic Benefit

- **Evaluable Patients: CNS Solid Tumors: NF2 N=5; Non-CNS Solid Tumors: N=10**
- PFS: CNS solid tumors = **9.1** months; Non-CNS solid tumors = **1.7** months
- Best overall response = **SD in 8/15 patients** (53%; 95% CI 26.6–78.7)
- Longest duration of follow-up without progression: > **27 months** (N=1)
- Most common AEs: **cytopenia, fatigue, nausea**



Well understood clinical safety ...

- Multiple investigator-initiated studies in oncology indications
- Lengthy human clinical exposure in NF2 – multiple patients on drug for several years
- Well-characterized side effect profile

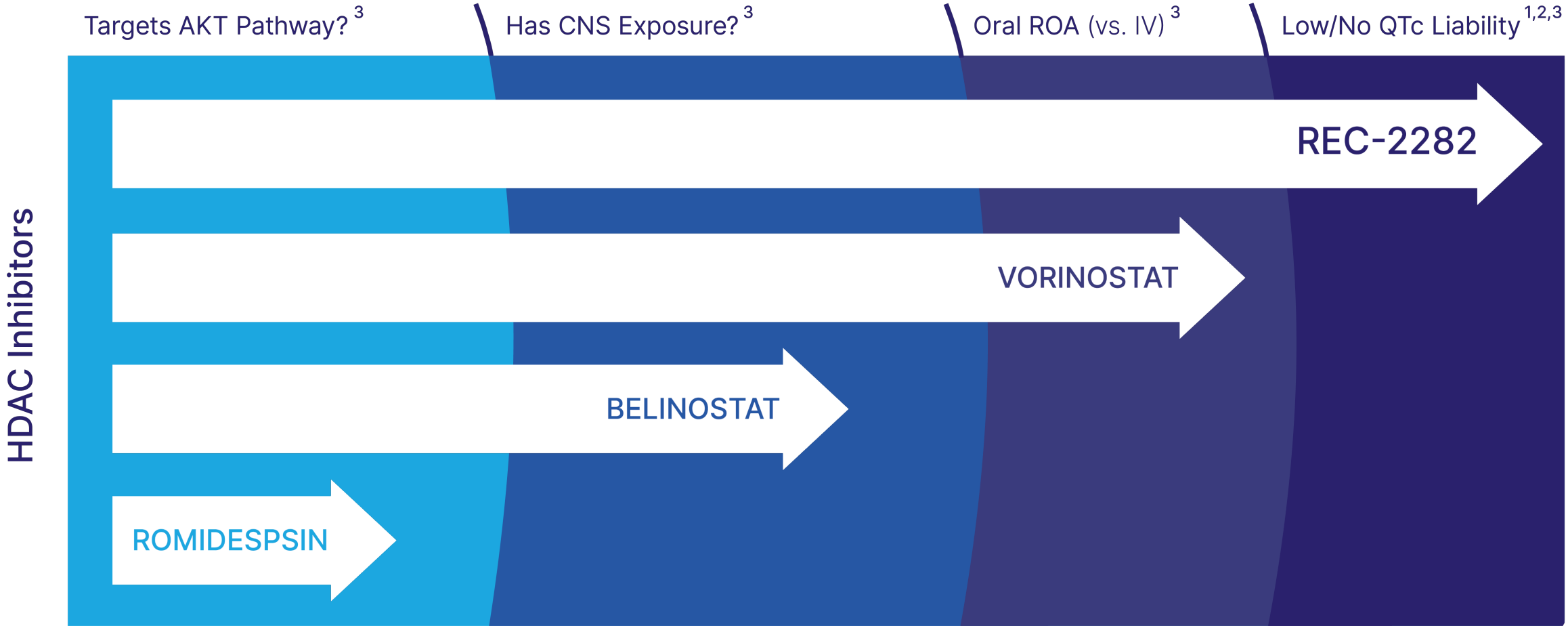
... with a drug-like profile

- Established and scalable API manufacturing process
- Multiple cGMP batches of 10mg and 50mg tablets have been manufactured
- Excellent long-term stability

Clinical: NF2

REC-2282 Appears Well Suited for NF2 vs Other HDAC Inhibitors

REC-2282 Would be First-In-Disease HDAC Inhibitor for Treatment of NF2 Meningiomas



[1] Sborov DW, et al. A phase 1 trial of the HDAC inhibitor AR-42 in patients with multiple myeloma and T- and B-cell lymphomas. Leuk Lymphoma. 2017 Oct;58(10):2310-2318.
[2] Collier KA, et al. A phase 1 trial of the histone deacetylase inhibitor AR-42 in patients with neurofibromatosis type 2-associated tumors and advanced solid malignancies. Cancer Chemother Pharmacol. 2021 May;87(5):599-611.
[3] Prescribing Information of Vorinostat/Belinostat/Romidepsin respectively

REC-2282 *for* Progressive Neurofibromatosis Type 2 (NF2) Associated Meningioma

Target Product Profile:

Brain-penetrant and orally bioavailable.
Preferential distribution into CNS tissues

Differentiated profile versus other HDACs.
Low/minimal QTc prolongation observed

Well-established safety profile.
50+ patients exposed across 4 ISTs

Preliminary evidence of PK/PD in Phase 1.
First-in-disease potential

REC-2282 for Neurofibromatosis Type 2 (NF2)

First-in-disease potential in NF2 with a best-in-class HDAC inhibitor

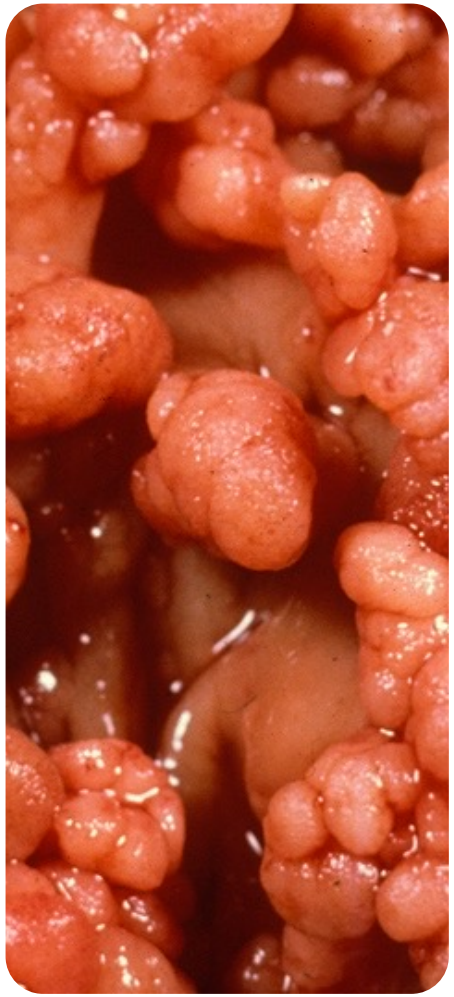
Program Overview	• Orally bioavailable small molecule inhibitor of class I and class IIB HDACs in Phase 2/3 (POPLAR) trial • Unique MOA that disrupts PP1-HDAC interface, attenuating pathophysiologic p-AKT without affecting total AKT • Fast Track Designation in *NF2* mutant meningioma granted by FDA in 2021
Clinical Updates	• Cohort A (Phase 2) enrollment ongoing targeting ~ 20 adults • Early Phase 1 study demonstrated mPFS of 9.1 months in patients with CNS tumors, including 5 NF2 patients • Therapeutic concentrations of REC-2282 achieved in plasma and CNS tumors in early Phase 1 studies
Near-term Catalysts	• Expected to complete Cohort A enrollment in adults by H1 2024 • Phase 2 readout (safety, preliminary efficacy, pharmacokinetics) expected Q4 2024
Commercial Opportunity	• ~ 33,000 NF2-associated meningioma patients in US and EU5 eligible for treatment with no approved therapies • Potential to expand into additional NF2 mutant populations including mesothelioma, MPNST and EHE
IP & Exclusivity	• ODD in US and EU provides 7 and 10 years, respectively, of market exclusivity following approval • Composition of matter patent provides protection until 2030 (excluding extensions)

REC-4881 for the Treatment of Familial Adenomatous Polyposis (FAP)

Target / MOA	MEK Inhibitor
Molecule Type	Small Molecule
Lead Indication(s)	Familial Adenomatous Polyposis
Status	Phase 1b/2
Designation(s)	Fast Track; US and EU Orphan Drug
Source of Insight	Recursion OS

Clinical: FAP

Disease Overview : Familial Adenomatous Polyposis



Polyps Found in Colon and Upper GI Tract

Patient Population – Easily Identifiable

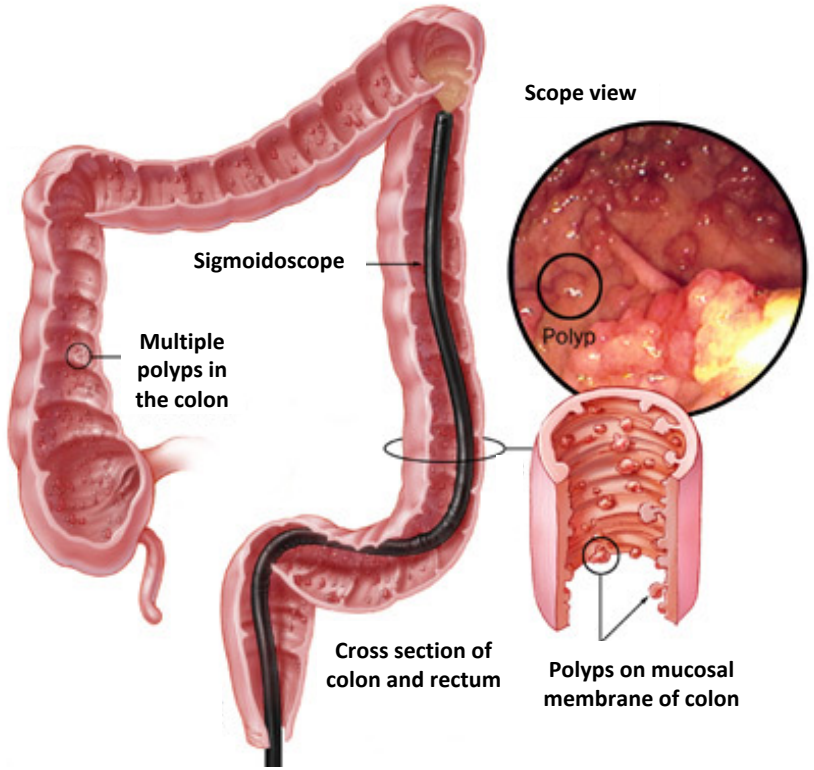
- Autosomal dominant tumor predisposition syndrome caused by a mutation in the APC gene
- Classic FAP (germline mutation) :
  - Hundreds to thousands of polyps in colon and upper GI tract
  - Extraintestinal manifestations (e.g., desmoid tumors)
  - 100% likelihood of developing colorectal cancer (CRC) before age 40, if untreated

~50,000 Diagnosed US + EU5 patients

https://www.hopkinsmedicine.org/health/conditions-and-diseases/familial-adenomatous-polyposis

Clinical: FAP

Disease Overview : Familial Adenomatous Polyposis – Standard of Care



No Approved Medical Therapy

- Standard of care: colectomy during adolescence (with or without removal of rectum)
- Post-colectomy, patients still at significant risk of polyps progressing to GI cancer
- Significant decrease in quality-of-life post-colectomy: continued endoscopies and surgical intervention

"Despite progress with surgical management, the need for effective therapies for FAP remains high due to continued risk of tumors post-surgery"

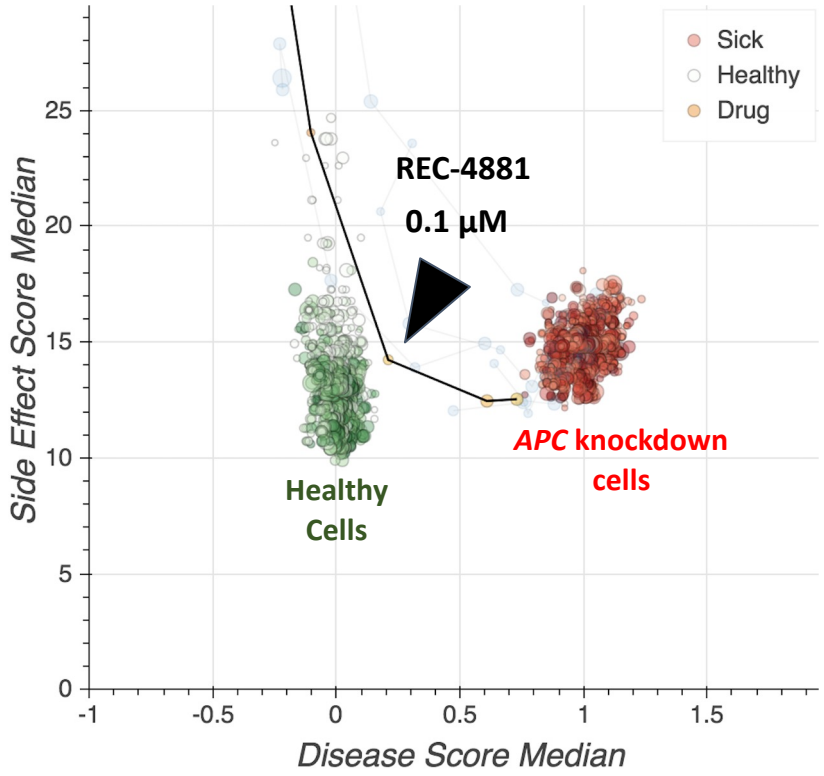
- **Niloy Jewel Samadder**, MD, Mayo Clinic

https://www.hopkinsmedicine.org/health/conditions-and-diseases/familial-adenomatous-polyposis

Clinical: FAP

Insight from OS : Rescued Loss of *APC*, Inhibited Tumor Growth

REC-4881 rescued phenotypic defects of cells with APC knockdown



- Compared to thousands of other molecules tested, REC-4881 rescued phenotypic defects substantially better (including better rescue than other MEK inhibitors) for APC specific knockdown

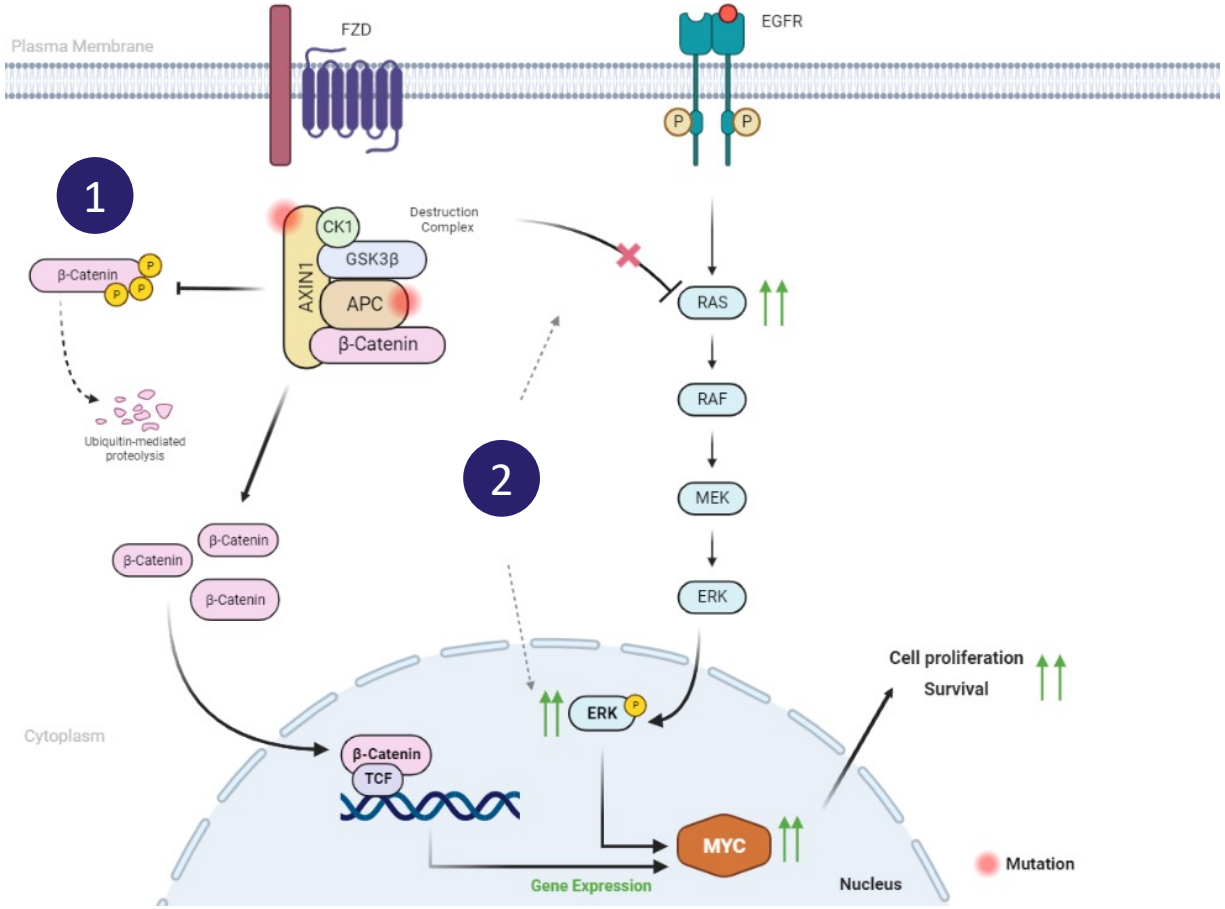
- Findings validated in tumor cell lines and spheroids grown from human epithelial tumor cells with *APC* mutation
  - 1,000x more selectivity in tumor cell lines with *APC* mutation
  - Inhibited growth and organization of spheroids

Clinical: FAP

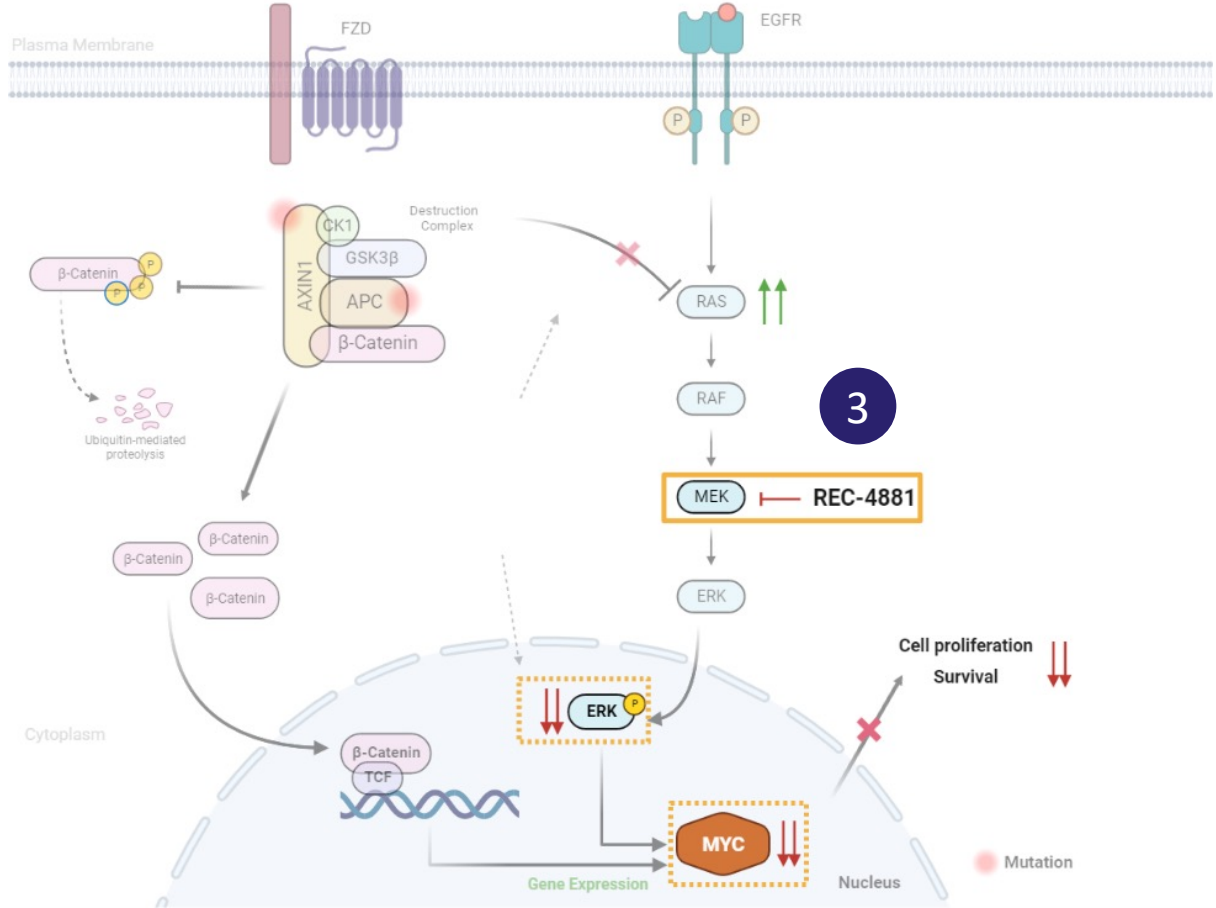
MoA : REC-4881 Blocks Wnt Mutation Induced MAPK Signaling

Orally Bioavailable, Small Molecule MEK Inhibitor

Disease State



REC-4881 Impact



3 REC-4881 inhibits MEK 1/2 and recovers the destabilization of RAS by the β-Catenin destruction complex, restoring the cell back to a Wnt-off like state

Jeon, WJ, et al. (2018). Interaction between Wnt/β-catenin and RAS-ERK pathways and an anti-cancer strategy via degradations of β-catenin and RAS by targeting the Wnt/β-catenin pathway. npj Precision Oncology, 2(5).

Clinical: FAP

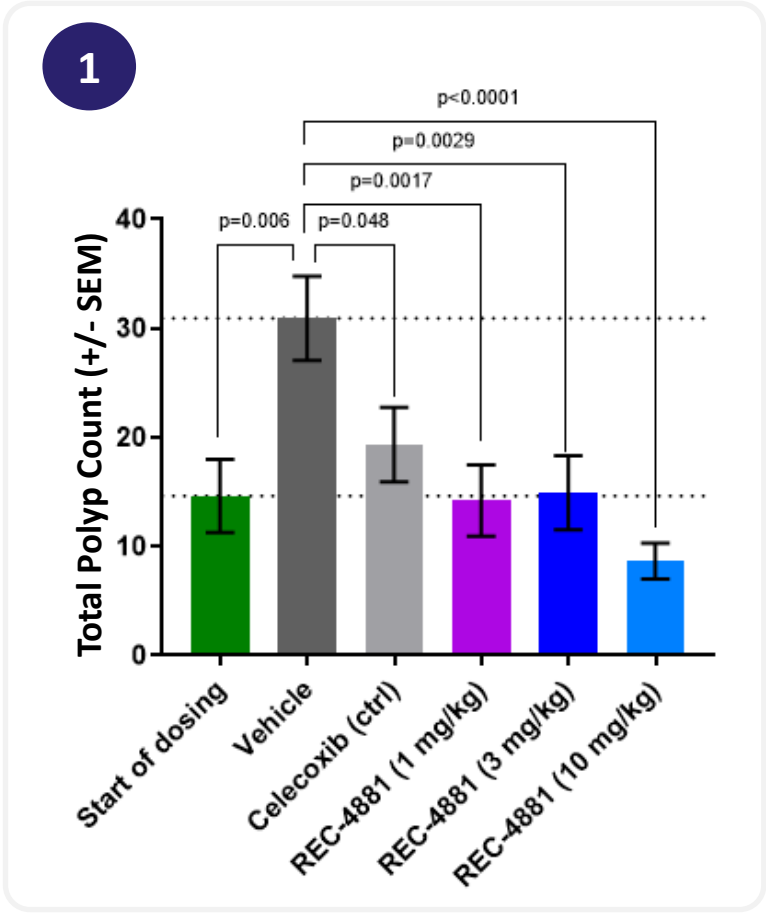
Further Confidence : Preclinical Studies Confirming Reduction in Polyp Count and High-Grade Dysplasia

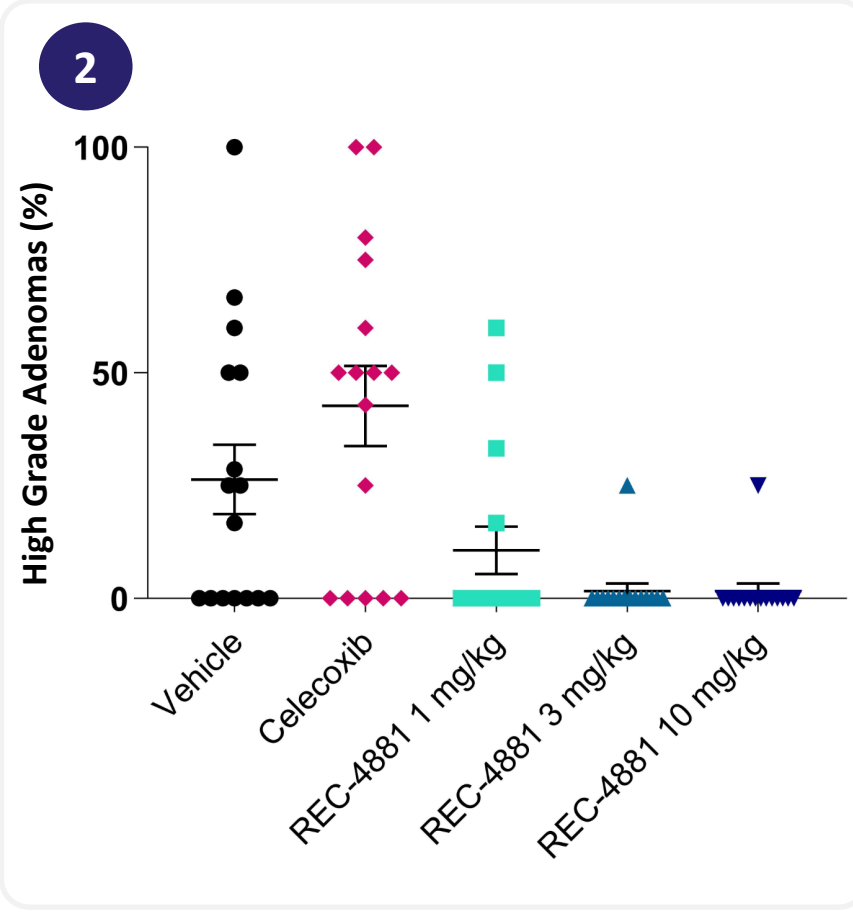
- In-vivo efficacy in APCmin mouse model
- Apc^{min} = FAP disease model
- Mice treated once daily for 8 weeks

After 8 weeks of treatment:

1 ↓ **Polyp Count**

2 ↓ **High-Grade Dysplasia**





APC, adenomatosis polyposis coli; ERK, extracellular signal-regulated kinase; FAP, familial adenomatous polyposis.

Clinical: FAP

Further Confidence : Clinical Data Generated by Recursion

REC-4881-101: Single-center, double-blind, placebo-controlled, dose-escalation study in healthy volunteers

- Group 1 (n=13): Food effect crossover (REC-4881 4 mg/PBO [fed/fasted]), followed by single dose REC-4881 8 mg/PBO [fed]
- Group 2 (n=12): Matched single ascending dose (REC-4881 4 mg/PBO; REC-4881 8 mg/PBO; REC-4881 12 mg/PBO)

Accomplished

Recursion formulation yields exposures comparable to Takeda's formulation (molecule in-licensed from Takeda)

No food effect

Dose proportional increases in exposure

Similar to C20001 study, observed pERK inhibition (i.e., target engagement) at 8 mg and 12 mg doses

Acceptable safety profile

Note: AE, adverse event; MEK, mitogen-activated protein kinase; NHV, normal healthy volunteer; pERK, phosphorylated extracellular signal-regulated kinase; SAE, serious adverse event.

REC-4881 *for* Familial Adenomatous Polyposis (FAP)

Target Product Profile:

Population specific for germline *APC* patients.
First precision targeted approach

Differentiated profile versus other MEKs.
Low clearance and minimal hepatic metabolism

Acceptable safety profile consistent vs other MEKs.
5 FAP patients treated in Part 1 of TUPELO

Drug-like properties support oral dosing QD.
Target engagement observed at 4mg

REC-4881 for Familial Adenomatous Polyposis (FAP)

First-in-disease opportunity in FAP with a potential best-in-class MEK 1/2 inhibitor

Program Overview	• Orally bioavailable, small molecule non-ATP competitive allosteric inhibitor of MEK 1/2 in Phase 1b/2 (TUPELO) • REC-4881 appears more active versus approved MEK inhibitors in disease relevant preclinical models • Fast Track Designation in FAP granted by FDA in 2022
Clinical Updates	• Part 1 completed with 4 mg QD generally well-tolerated and safety profile consistent with other MEK inhibitors • Early PD data indicates 4 mg is pharmacologically active – Part 2 protocol updated to dose escalation / expansion • Efficacy will evaluate change in polyp burden relative to baseline at 12 weeks
Near-term Catalysts	• FPI for Part 2 expected H1 2024 • Phase 2 initial readout (safety, preliminary efficacy, pharmacokinetics) anticipated H1 2025
Commercial Opportunity	• ~ 50,000 FAP patients in US and EU5 eligible for treatment with no approved therapies • Opportunity to treat moderate-to-severe population to potentially delay or prevent surgical intervention
IP & Exclusivity	• ODD in US and EU provides 7 and 10 years, respectively, of market exclusivity following approval • No known barriers to market access

REC-4881 for the Treatment of Solid Tumors with AXIN1 or APC Mutant Cancers

Target / MOA	MEK Inhibitor
Molecule Type	Small Molecule
Lead Indication(s)	Solid Tumors with AXIN1 or APC Mutant Cancers
Status	Phase 2
Source of Insight	Recursion OS

Clinical: AXIN1 or APC

Disease Overview : *AXIN1* or *APC* Mutant Cancers

Gross morphology of HCC tumor

- **Sustained Wnt signaling** is a frequent driver event found across a wide variety of solid tumors
- Dysregulation of β-catenin destruction complex due to inactivating mutations in *AXIN1* or *APC* **leads to sustained Wnt signaling promoting** cancer progression and survival[1]
- *AXIN1* or *APC* **mutant solid tumors** are considered clinically aggressive and resistant to standard treatments

"Nothing in HCC has immediate therapeutic relevance and the most common mutations are TERT, TP53, and Wnt (CTNNB1/AXIN1/APC) and combined these alterations define almost 80% of patients and are not targetable"

- KOL, Clinical Investigator, Texas

[1] *Bugter, J.M., et al. Nat Rev Cancer, 2021, 21, pp.5-21*

Clinical: AXIN1 or APC

Disease Overview : *AXIN1* or *APC* Mutant Cancers

Tumor Type	*AXIN1* Mutation Frequency[1]	*APC* Mutation Frequency[1]	Treatable Population[2] (US+EU5)
CRC	3%	70%	27,450
LUAD	4%	11%	14,000
Prostate	2%	11%	6,700
Bladder	3%	8%	5,100
HCC	12%	5%	3,100
Endometrial	8%	12%	2,600
Esophageal	2%	7%	2,600
PDAC	1%	2%	1,500
Ovarian	1%	3%	1,400
TNBC	1%	2%	300
			~65,000

Flexible Patient Selection Strategy and Study Design

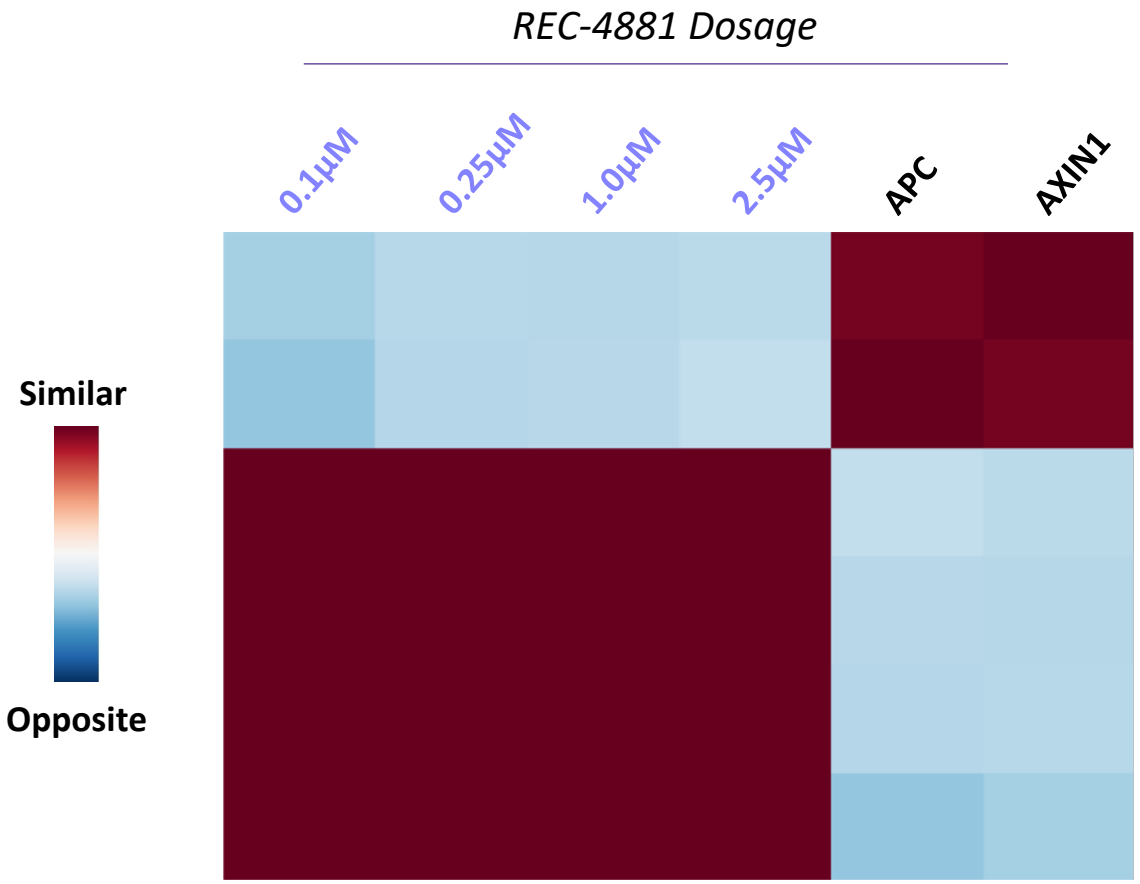
- *AXIN1* and *APC* genes covered by commercially available NGS panels and liquid biopsy detection assays
- FDA guidance supports utility of ctDNA as patient selection for the detection of alterations for eligibility criteria and as a stratification factor for trials enrolling marker-positive and marker-negative populations[3]
- Multiple tumor types will inform study design and patient selection

Preclinical data with REC-4881 at clinically relevant exposures in HCC and Ovarian PDX mouse models gives confidence to pursue other mutant cancer types

[1] Obtained from cbioportal.org. [2] Represents 2L treatable population estimates; obtained from DRG. [3] https://www.fda.gov/media/158072/download

Clinical: AXIN1 or APC

Insight from OS : Novel Insight around Established MoA



Heat map from Recursion OS

Hypothesis: Rescue of *AXIN1* may impact tumor progression and/or restore checkpoint sensitivity in cancers driven by *AXIN1* loss

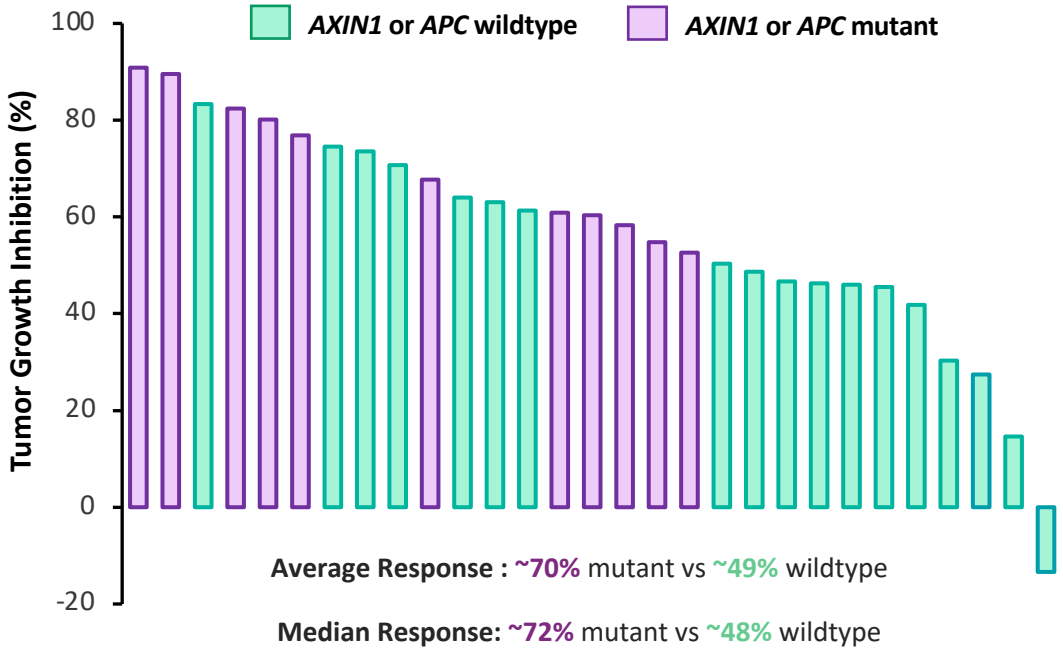
Recursion Differentiation: REC-4881 rescues tumor suppressor genes *APC* and *AXIN1*

- *APC* and *AXIN1* are negative regulators of Wnt signaling
- Both proteins form part of the B-catenin destruction complex. Strong clustering suggests map recapitulation of this biology

Clinical: AXIN1 or APC

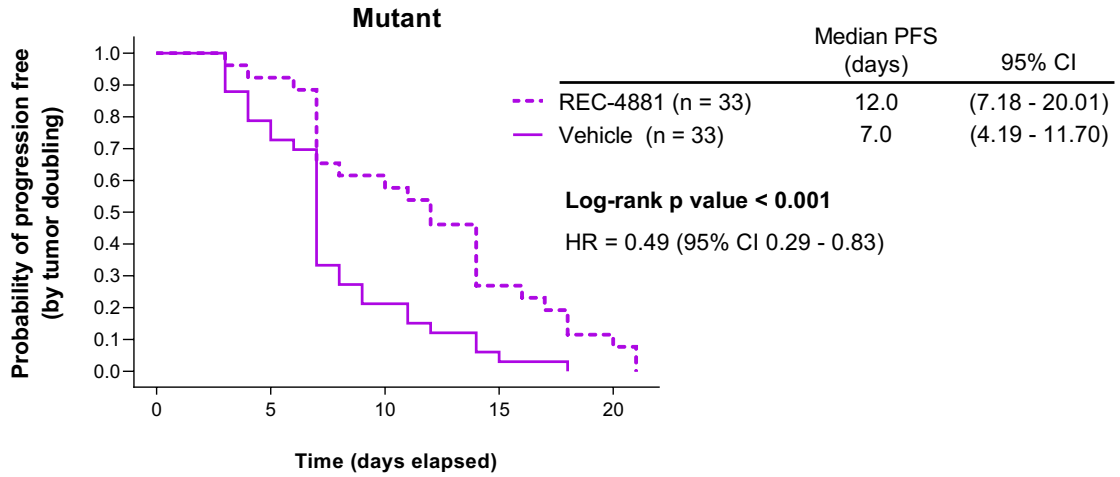
Further Confidence : Preclinical Studies Confirming Insight

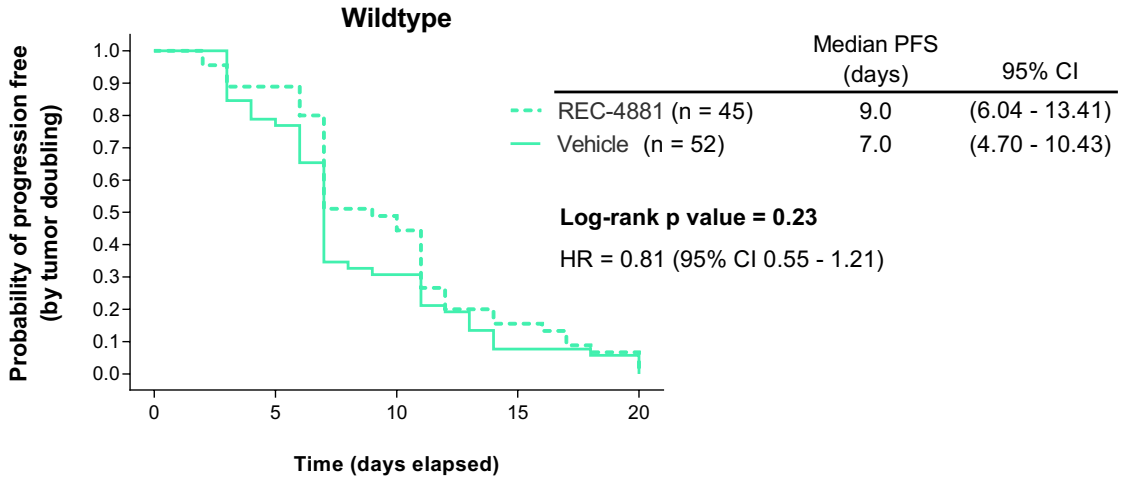
Efficacy found in PDX Models …



- Significantly greater antitumor activity observed with REC-4881 in mutant models versus wildtype
- Majority of mutant models ≥ 60% tumor growth inhibition, which is considered a benchmark for a response in the clinic[1]

… Led to Significant Progression Free Survival





Note: REC-4881 dosed at 3 mg/kg QD for up to 21 days. 3 mice per treatment per model (3 x 3 x 3) design. [1] *Wong, H., et al. Clin Cancer Res, 2012, 18:14, pp.3846-3855*

REC-4881 *for AXIN1 or APC* mutant cancers

Target Product Profile:

Potential to obtain tumor agnostic label.
First in disease opportunity

Differentiated versus other MEKs.
Low clearance and minimal hepatic metabolism

Acceptable safety profile versus other MEKs.
51 solid tumor patients treated in Phase 1

Drug-like properties support oral dosing QD.
Pharmacologically active at low doses

REC-4881 for AXIN1 or APC Mutant Cancers

First-in-disease opportunity in AXIN1 or APC mutant cancers with a potential best-in-class MEK 1/2 inhibitor

Program Overview

- Orally bioavailable, small molecule non-ATP competitive allosteric inhibitor of MEK 1/2 in Phase 2 (LILAC)
- First therapeutic candidate advanced to a Phase 2 signal finding study in AXIN1 or APC mutant cancers
- Recursion's first clinical trial in oncology and the first that used inferential search for hypothesis generation

Clinical Updates

- Safety run-in of REC-4881 to identify RP2D prior to allocation
- Protocol designed to assess activity in two independent cohorts of AXIN1 or APC mutant tumors
- Efficacy will evaluate ORR as measured by RECIST 1.1

Near-term Catalysts

- FPI expected in Q1 2024
- Phase 2 readout (safety, preliminary efficacy, and PK) anticipated H1 2025

Commercial Opportunity

- ~ 65,000 *AXIN1* or *APC* mutant patients in 2L in US and EU5 eligible for treatment with no approved therapies
- AXIN1 and APC genes covered by commercially available NGS panels and liquid biopsy detection assays

IP & Exclusivity

- Method of use patent pending with protection until 2043 (excluding extensions)
- No known barriers to market access

REC-3964 for the Prevention of Recurrent *C. difficile* Infection (rCDI)

Target / MOA	Selective *C. difficile* Toxin Inhibitor
Molecule Type	Small Molecule
Lead Indication(s)	Prevention of rCDI
Status	Phase 2
Source of Insight	Recursion OS

Clinical: *C. difficile*

Disease Overview : *C. Difficile* Infection (CDI)

Colleen – lived with rCDI

Patient Population – Large, Diagnosable and Easy to Identify

- Symptoms caused by clostridioides difficile tissue-damaging toxins released in the colon
- Patients who experience >3 unformed stools are diagnosed via NAAT* for toxin gene or positive stool test for toxins
- Patients who are at highest risk are those on antibiotics, and frequently visit hospitals or are living in a nursing home
- More than **80% of cases** occur among patients **age 65** or older

Large, Unmet Need with Significant Cost Burden

- RCDI** occurs in **20-30% of patients treated with standard of care**
  - 40% of those patients will continue to recur with 2+ episodes
- **>29,000 patients** die in the US each year from CDI
- Cost burden of up to **$4.8bn annually**

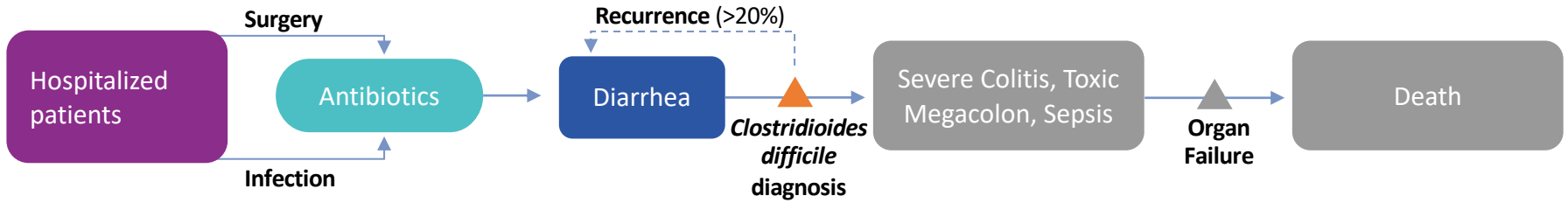
~730,000 **Diagnosed US + EU5 patients**

*Source, CDC *NAAT = Nucleic Acid Amplification Test; **rCDI = recurrent CDI*

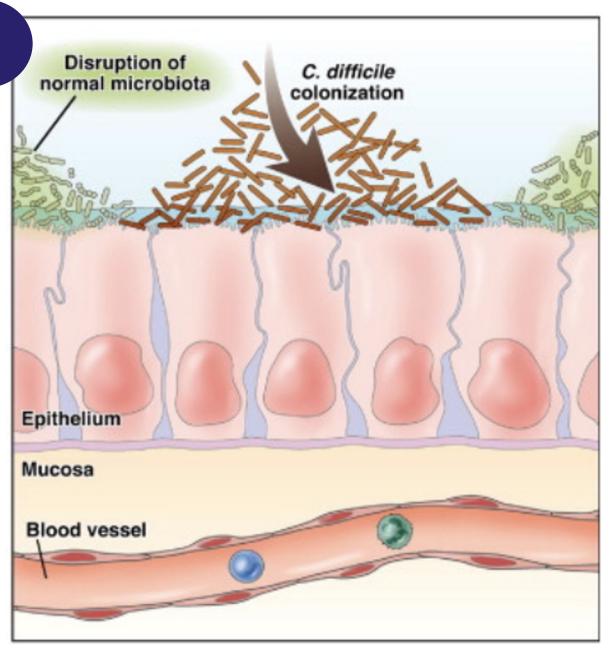
Clinical: *C. difficile*

Disease Overview : *C. Difficile* Infection (CDI)

C. Difficile **is the leading cause of antibiotic-associated diarrhea**

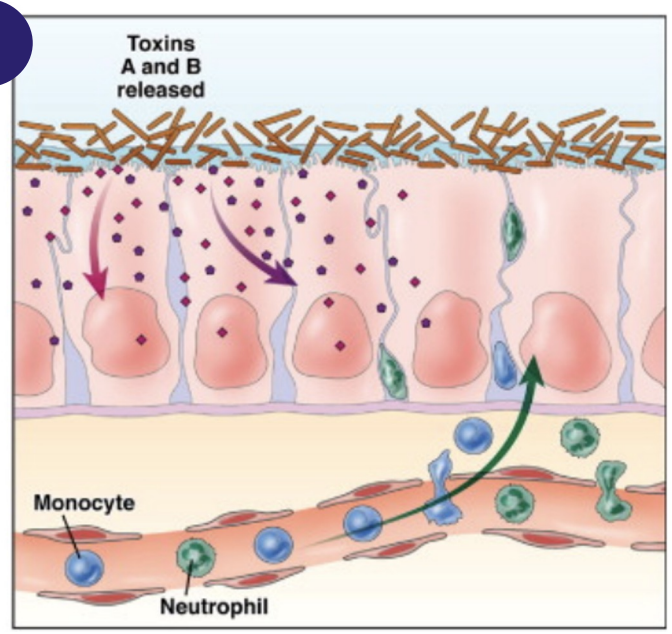


1



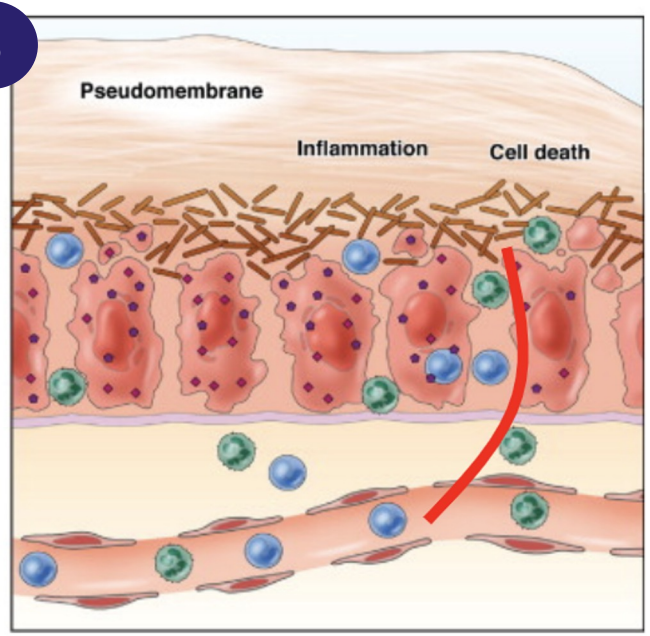
Disruption of microbiota and colonization of *C. diff*

2



Release of C. diff toxins

3

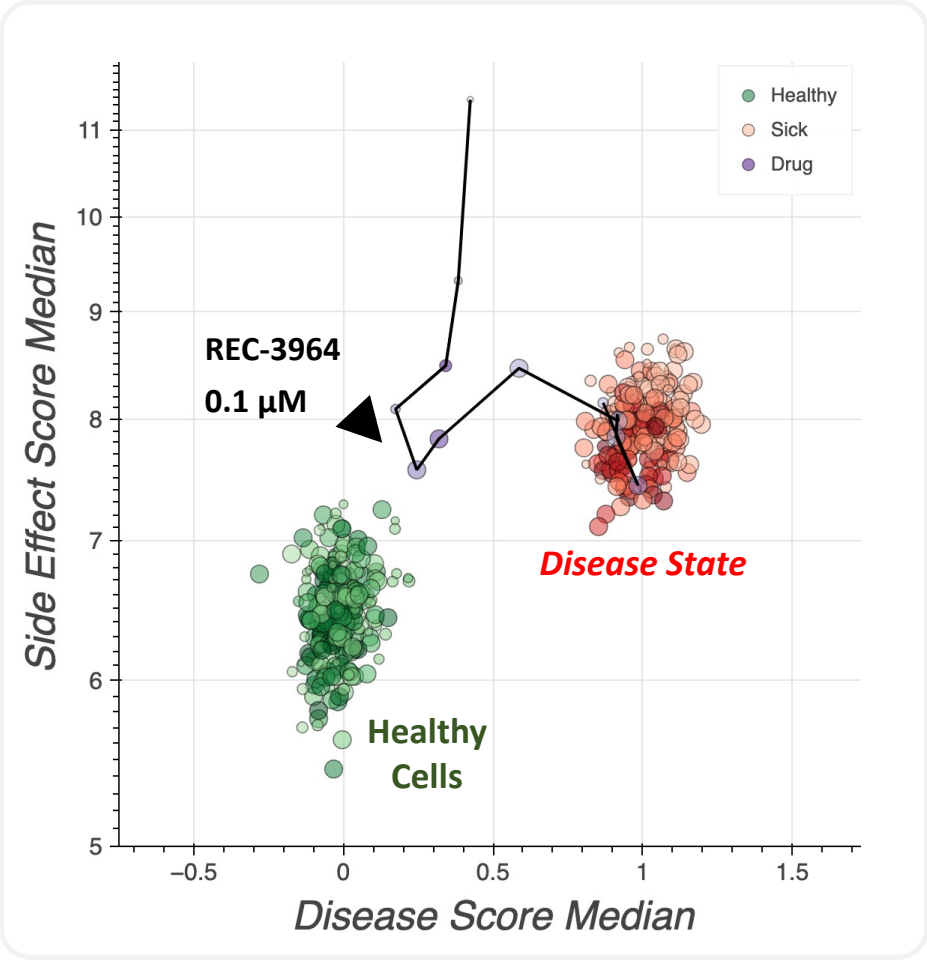


Degradation of colon cell junction & toxin transit to bloodstream

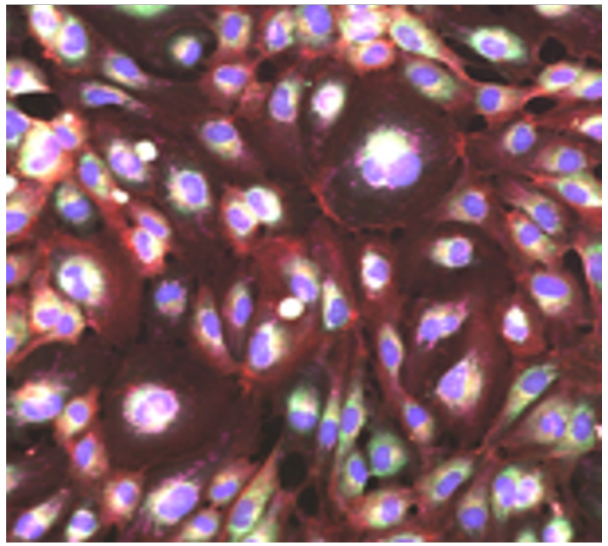
Source: McCollum, D,, Rodriguez, JM . Detection, Treatment, and Prevention of Clostridium difficile Infection. Clinical Gastroenterology and Hepatology 2012 Mar 19. https://doi.org/10.1016/j.cgh.2012.03.008

Clinical: *C. difficile*

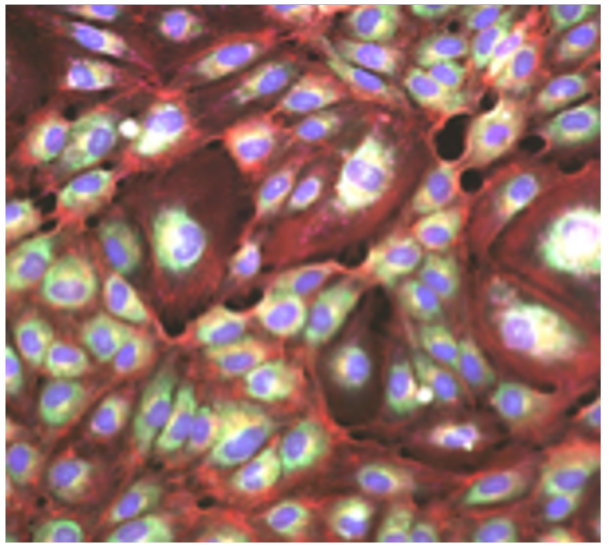
Insight from OS : REC-3964 Rescued Cells Treated with *C. difficile* Toxins



REC-3964 identified as a NCE that demonstrated strong rescue in HUVEC cells treated with *C. difficile* toxin



C. difficile toxin B phenotype



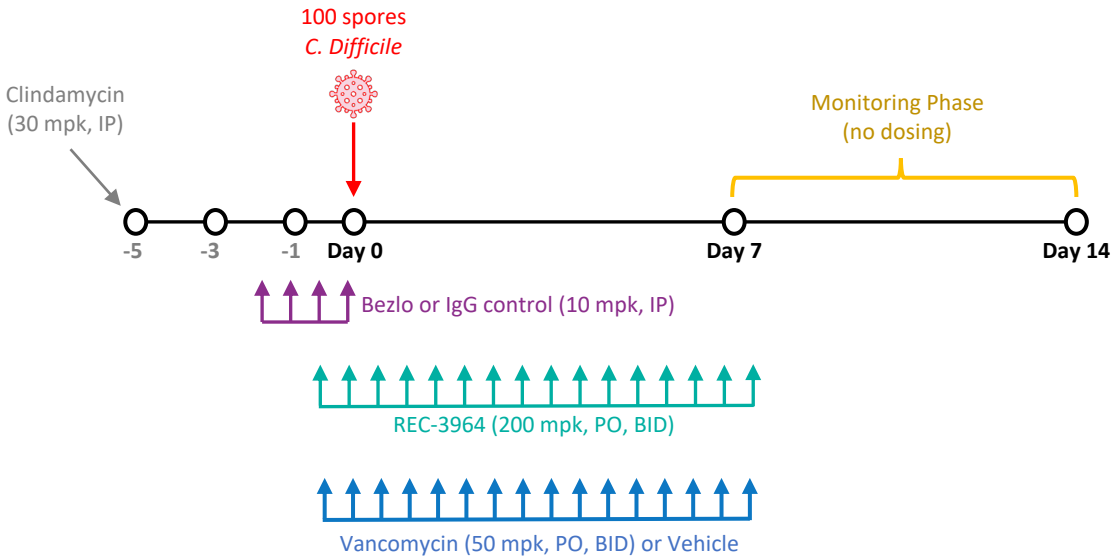
Healthy Control

Clinical: *C. difficile*

Further Confidence : Preclinical Studies Confirmed Recursion OS Insight

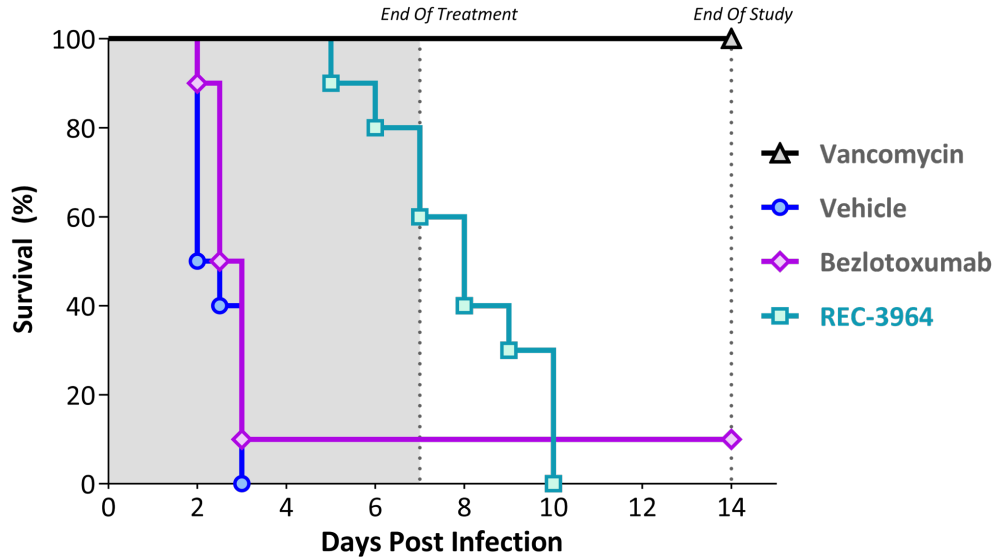
REC-3964 is Superior to Bezlotoxumab in a Human Disease Relevant CDI Hamster Model

Schematic representation of timelines for CDI model



- N = 10 hamsters per group
- *C. difficile* strain 630 was used as genetic experiments confirmed virulence via toxin B[1]
- Clinical observations were conducted from Day 0 to Day 7, with surviving animals monitored until Day 14

REC-3964 significantly extended survival over SOC



- REC-3964 potently inhibits toxin B with residual activity against toxin A, while bezlotoxumab is specific to toxin B.
- Significant difference in probability of survival vs bezlotoxumab alone at the end of treatment (**p<0.001**, log-rank test)

[1] *Lyras, D, et al. Nature, 2009, 458, pp.1176-1179.*

Clinical: *C. difficile*

Clinical Trial : REC-3964 for *C. difficile* Phase 1 Study Complete

Trial Design

- Randomized, Double-blind Trial

Population

- Healthy Participants
- SAD (n = 48)
  - 36 participants treated with REC-3964
  - 12 participants treated with placebo
- MAD (n = 42)
  - 34 participants treated with REC-3964
  - 8 participants treated with placebo

Primary Objectives

✓ Assess the safety & tolerability of SAD and MAD of REC-3964

✓ Evaluate the PK profile of REC-3964 after single and multiple doses

Phase 1 Topline

- REC-3964 oral administration was **well tolerated** by all subjects tested
  - ✓ **3%** (n=1) of participants in SAD with drug-related AEs
  - ✓ **12%** (n=4) of participants in MAD with drug-related AEs
  - ✓ All AEs were deemed **Grade 1**
  - ✓ **No SAEs** were observed
  - ✓ **No discontinuations** related to treatment
- REC-3964 exhibited a **favorable PK profile**
  - ✓ Exposures (AUC) **increased approximately dose-proportionally** across the dose ranges tested (50 mg – 1200 mg)
  - ✓ Half-life ranged from **~7-10 hours;** BID dosing expected to reach targeted trough concentrations

Clinical: *C. difficile*

Further Confidence : Clinical Studies Confirming Safety

REC-3964 was well-tolerated with no treatment-related SAEs

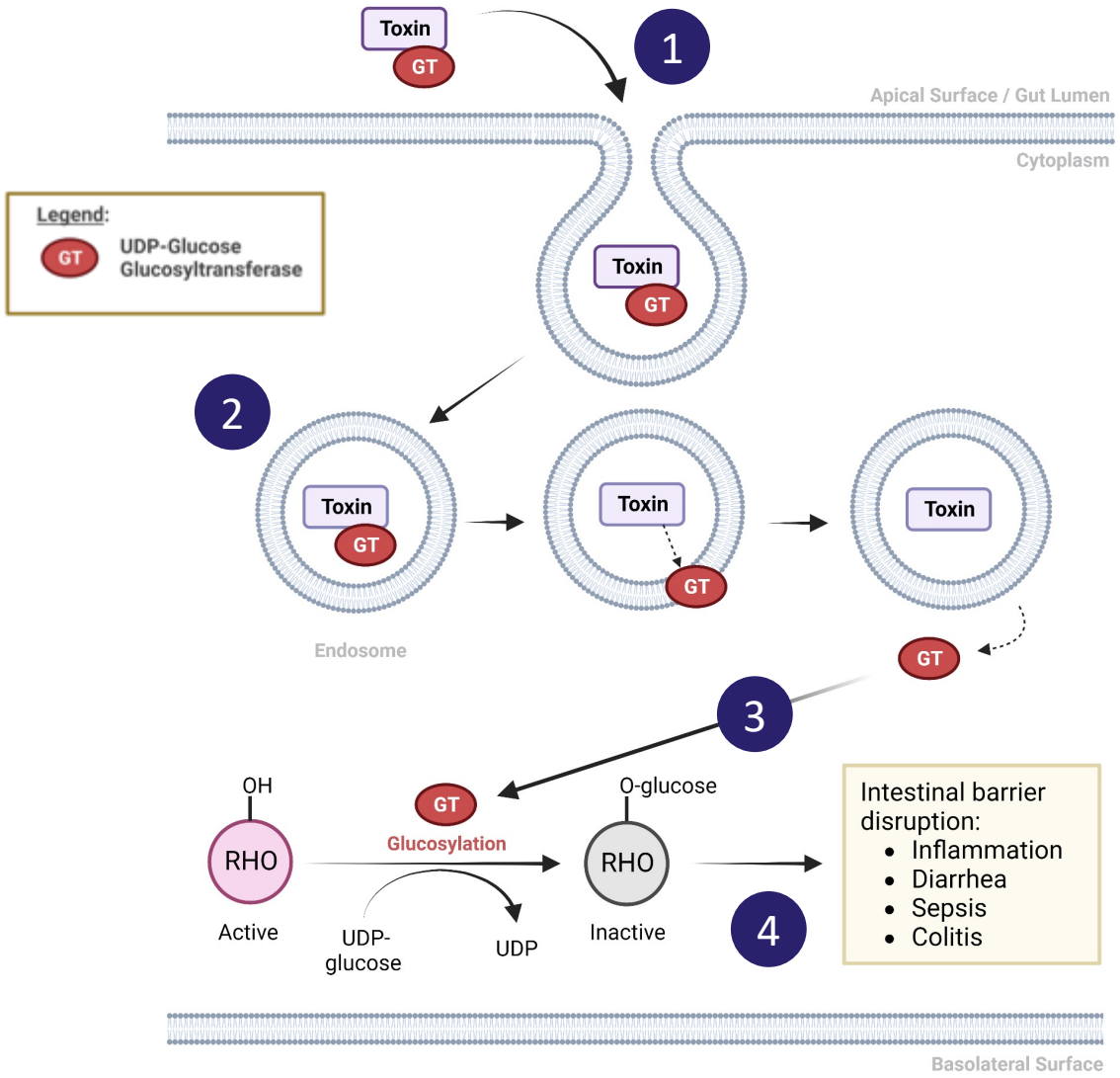
MAD Study	Placebo (N=8) n (%)	100 mg (N=10) n (%)	300 mg (N=8) n (%)	500 mg (N=8) n (%)	900 mg (N=8) n (%)	REC-3964 Overall (N=34) n (%)	MAD Overall (N=42) n (%)
Total Number of TEAEs	17	24	5	9	7	45	62
Total Participants with ≥ 1 TEAE	6 (75.0)	8 (80.0)	4 (50.0)	5 (62.5)	4 (50.0)	21 (61.8)	27 (64.3)
Relationship to Study Drug							
Not Related	4 (50.0)	6 (60.0)	3 (37.5)	4 (50.0)	4 (50.0)	17 (50.0)	21 (50.0)
Related	2 (25.0)	2 (20.0)	1 (12.5)	1 (12.5)	0	4 (11.8)	6 (14.3)
Abdominal Distension	2 (25.0)	1 (10.0)	1 (12.5)	1 (12.5)	0	3 (8.8)	5 (11.9)
Flatulence	0	1 (10.0)	0	0	0	1 (2.9)	1 (2.4)
Severity							
Grade 1	6 (75.0)	8 (80.0)	4 (50.0)	5 (62.5)	4 (50.0)	21 (61.8)	27 (64.3)
Grade 2	0	0	0	0	0	0	0
Grade ≥ 3	0	0	0	0	0	0	0
Total Number of SAEs	0	0	0	0	0	0	0
Discontinued Study Drug Due to AE	0	0	0	0	0	0	0

TEAEs = treatment emergent adverse events; Grade 1 = Mild, Grade 2 = Moderate, Grade 3 = Severe, Grade 4 = Life Threatening, Grade 5 = Fatal

Clinical: *C. difficile*

REC-3964 : Selective Inhibitor of *C. difficile* Toxins

REC-3964 is Recursion's 1st Small Molecule NCE to Reach the Clinic



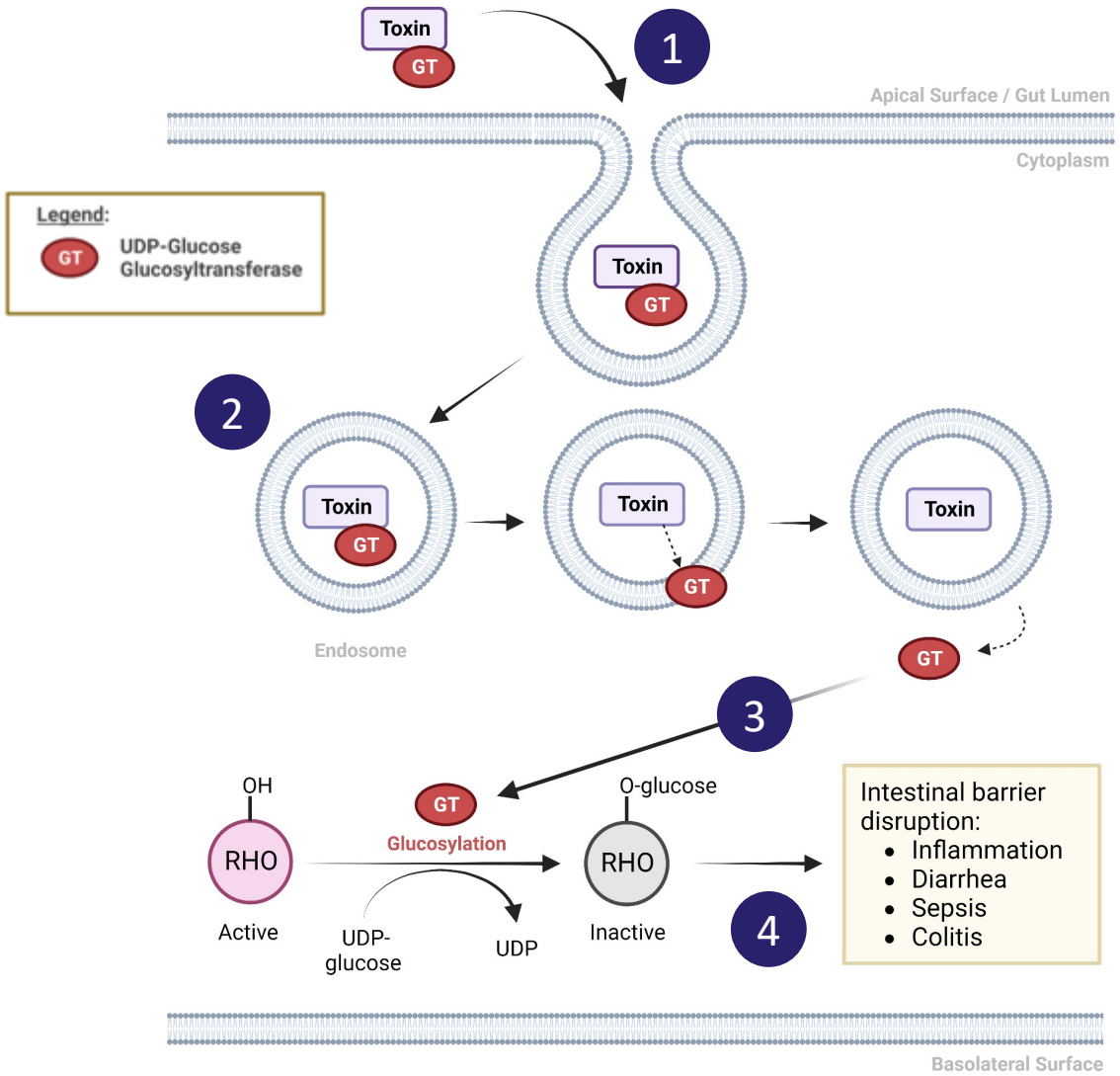
1. CDI toxins bind to cell surface receptors and trigger endocytic event
2. Autocatalytic cleavage event releases CDI toxin's glucoyltransferase enzymatic domain into the cytosol of the infected cell
3. The glucosyltransferase locks Rho family GTPases in the inactive state
4. Inactivation of Rho GTPases alters cytoskeletal dynamics, induces apoptosis, and impairs barrier function which drives the pathological effects of CDI

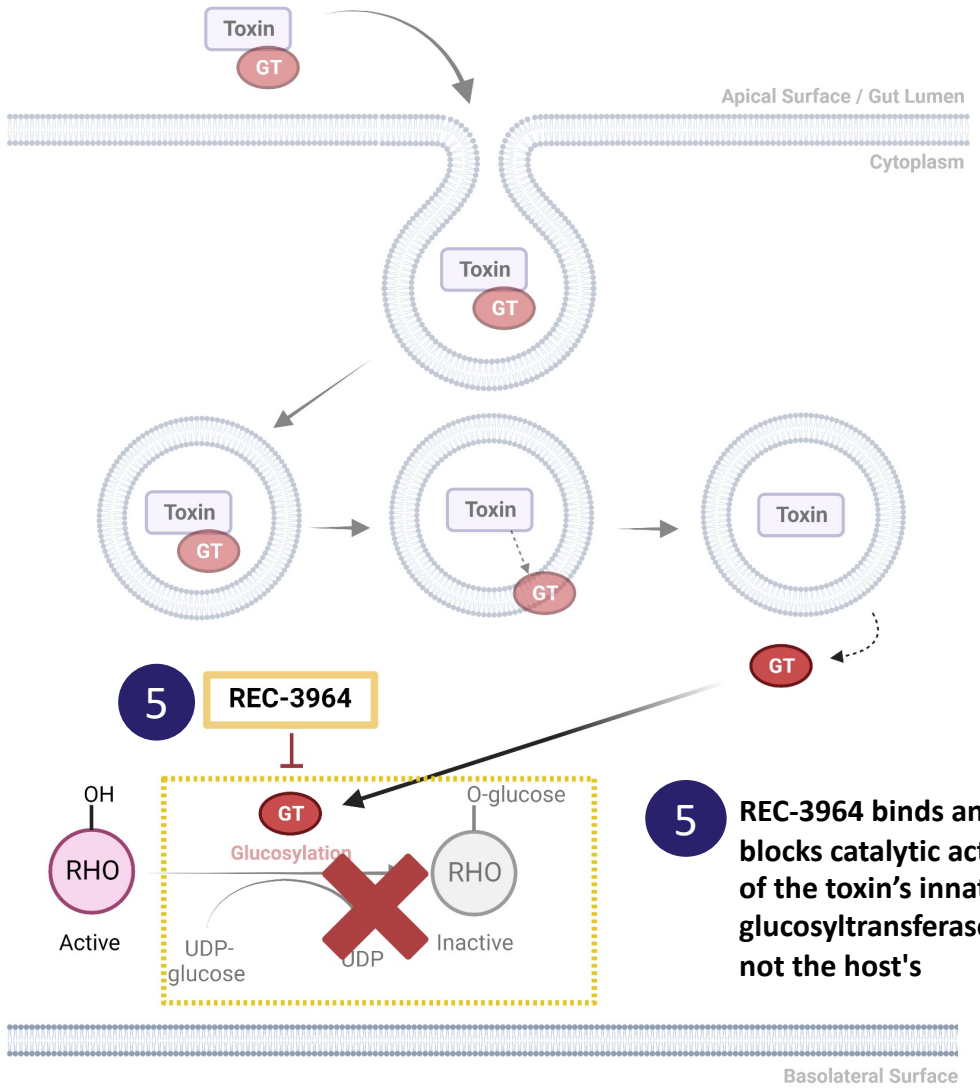
Adapted from Awad, MM. et al. (2014). Clostridium difficile virulence factors: Insights into an anaerobic spore-forming pathogen. Gut Microbes. 5(5), 579-593.

Clinical: *C. difficile*

REC-3964 : Selective Inhibitor of *C. difficile* Toxins

REC-3964 is Recursion's 1st Small Molecule NCE to Reach the Clinic





5 **REC-3964 binds and blocks catalytic activity of the toxin's innate glucosyltransferase, but not the host's**

Adapted from Awad, MM. et al. (2014). Clostridium difficile virulence factors: Insights into an anaerobic spore-forming pathogen. Gut Microbes. 5(5), 579-593.

REC-3964 *for*

Prevention of recurrent *C. difficile* infection (rCDI)

Target Product Profile:

Population addresses high unmet need.
Targeting patients with recurrent CDI

Differentiated mechanism of action.
Host independent and bacterial toxin selective

Well-tolerated with no DLTs or SAEs in Phase 1.
No treatment-related discontinuations

Drug-like properties support oral dosing BID.
Therapeutic exposures observed in humans

REC-3964 for Prevention of recurrent *C. difficile* infection (rCDI)

First-in-class potential for prevention of rCDI

Program Overview	• Orally bioavailable, small molecule *C. difficile* toxin inhibitor and the first NCE developed by Recursion • Differentiated MOA selectively targets bacterial toxin while sparing the host to minimize adverse events • Robust preclinical efficacy demonstrating superiority vs bezlotoxumab in the gold standard hamster model
Clinical Updates	• Favorable safety and tolerability profile in Phase 1 dose-escalation with no DLTs and no SAEs • Minimal adverse events seen in Phase 1, and all deemed Grade 1 • BID dosing provides therapeutic exposures expected to reach targeted trough concentrations
Near-term Catalysts	• Full Phase 1 data to be presented at a medical conference in H1 2024 • Phase 2 proof-of-concept study planned for initiation in 2024
Commercial Opportunity	• > 100,000 high-risk rCDI patients in US and EU5 with limited treatment options to prevent recurrent disease • Ability to address populations not eligible for FMT or microbiome-based therapies due to comorbidities
IP & Exclusivity	• Composition of matter patent allowed with protection until 2042 (excluding extensions) • No known barriers to market access

RBM39 Inhibition for the Treatment of HR-Proficient Ovarian Cancer and Other Solid Tumors

Target / MOA	RBM39 Molecular Glue Degrader
Molecule Type	Small Molecule
Lead Indication(s)	HR-Proficient Cancers
Status	Pre-IND
Source of Insight	Recursion OS

RBM39 Degradation

for HR-Proficient Ovarian Cancer & Other Solid Tumors

Target Product Profile:

Opportunity to address high unmet need.
PARP naïve and PARP resistant population

Monotherapy label with combination potential.
Acceptable TI in human cancer xenografts

Encouraging safety and tolerability profile.
Minimal off-target effects vs first-gen molecules

Robust RBM39 degradation correlated with benefit.
FIH studies enable rapid clinical path to POC

RBM39 Program for HR-Proficient Ovarian Cancer & Other Solid Tumors

Lead candidate is a potential first-in-class RBM39 degrader being developed for HR-proficient tumors

Program Overview	• Recursion OS identified RBM39 as a novel target capable of mimicking CDK12 biology independent of CDK13 • Lead molecule has demonstrated durable regressions across HRP and HRD cell line and patient derived xenografts • Program advanced from target identification to IND-enabling stages in under 18 months
Non- Clinical Updates	• No significant in vitro safety concerns with favorable tolerability in disease relevant animal models • Target engagement assays demonstrate strong correlation between RBM39 degradation and tumor reduction in vivo • Excellent physiochemical properties and reasonable human projected doses support cost-effective CMC campaign
Near-term Catalysts	• IND submission expected in H2 2024
Commercial Opportunity	• ~200,000 patients in US and EU5 harbor cancers that lack HRR mutations and have progressed on frontline therapies • First-in-class potential as a single agent or in combination with other agents (PARP, IO, chemo, etc.)
IP & Exclusivity	• Composition of matter patent pending with protection until 2043 (excluding extensions) • No known barriers to market access