Recursion

Earnings 2Q26

AUGUST 5, 2026

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Other important factors and information are contained in Recursion's most recent Annual Report on Form 10-K, Quarterly Report on Form 10-Q, and the Company's other filings with the U.S. Securities and Exchange Commission (the "SEC"), which can be accessed at https://ir.recursion.com, or www.sec.gov. All forward-looking statements are qualified by these cautionary statements and apply only as of the date they are made. Recursion does not undertake any obligation to update any forward-looking statement, whether as a result of new information, future events or otherwise.

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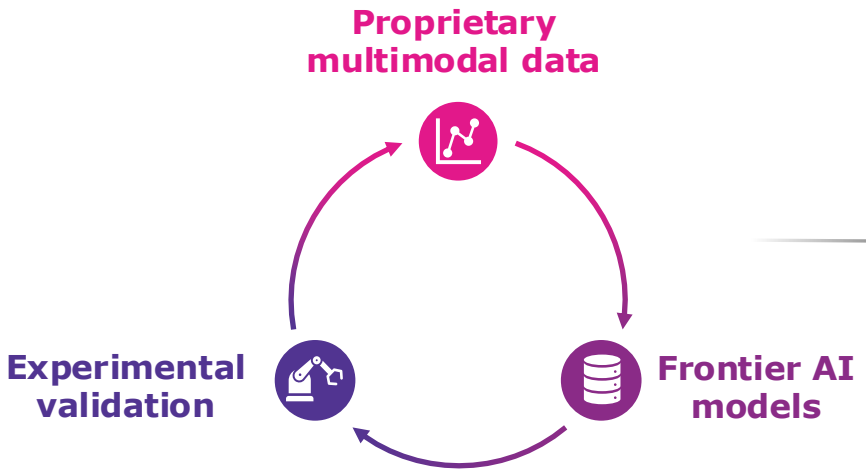
Cross-trial or cross-candidate information about other investigational assets and drug candidates are not based on head-to-head studies and are presented for informational purposes; data presented are based on publicly available information for other clinical trials and other drug candidates.

Recursion

Our AI-enabled product engine is delivering differentiated programs

Product engine

End-to-end AI-native product engine for drug discovery & development



Proprietary multimodal data from our own wet labs feeds an AI-native loop that improves every cycle, compounding into more, and more valuable output as AI advances

1 Pipeline

An advancing internal pipeline with multiple clinical value inflection points

REC-4881 in FAP — promising Phase 2 data, >$10B TAM[1]

4 additional clinical-stage assets in Phase 1/2 development

2 Partnerships

An external portfolio differentiated by our engine and validated by leading biopharma partners

>$500M of ***realized*** partner inflows to date

Sanofi — oral small molecules against challenging I&I and Oncology targets

Roche and Genentech — translating AI-driven insights into novel biology and potential first-in-class programs

1. Modeled total addressable market (not realized or forecasted sales): diagnosed prevalence (~50k) x rare-disease oral net price (benchmark GOMEKLI ~$22–30k/mo = ~$260–360k/yr; ATTR/PAH orals); sensitive to price & treated fraction

Recursion

Building differentiated medicines with independent long-term value

Proprietary Data

Our AI learns from data no one else has generated

- Proprietary multimodal data at industrial scale
- Data designed for AI, not just experiments
- Durable asset that compounds over time

Recursive Lab-in-the-Loop

Every prediction is experimentally challenged and improves the next cycle

- Biology ⇆ Design ⇆ Clinic
- Closed learning loop
- Faster iteration and de-risking

Differentiated Products

The engine repeatedly creates independent assets

- Internal clinical pipeline
- Partnered programs
- Medicines with standalone value

Beyond platform-only players, we **convert our program engine** into **differentiated products,** each having **independent long-term value**

We have delivered on our milestones this year, with strong momentum and catalysts ahead

Delivered year to date

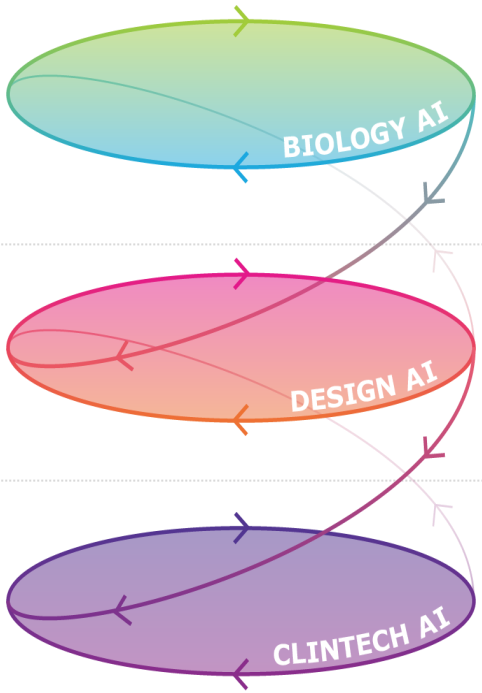
- ✓ **REC-4881 (MEK1/2)**
  Initial engagement with FDA
- ✓ **REC-1245 (RBM39 degrader)**
  Mono – early safety and PK
- ✓ **Sanofi: Oral FIC oncology program milestone**
  Oncology lead series ($4m)
- ✓ **NEW: Genentech options FIRST neuroscience target into early discovery program**
  Advancing a previously unexplored neuroscience target
- ✓ **NEW: REC-7735 (PI3Kα H1047R)**
  IND clearance

Key potential near-term milestones

- ☐ **REC-4881 (MEK1/2)**
  Additional Ph 2 clinical data at CGA-IGC
  Update on FDA engagement
- ☐ **REC-1245 (RBM39 degrader)**
  Additional Ph 1 dose escalation data
- ☐ **Sanofi: Potential Development Candidate**
  AI-driven drug design for challenging targets
- ☐ **REC-7735 (PI3Kα H1047R)**
  Phase 1/2 study initiation

Our AI-native product engine compounds advantage across every stage of R&D

	Industry baseline	Recursion's AI product engine	Recursion's advantage
Biology	• **Majority of biology still unknown** • Discovery limited by incomplete understanding of human biology		• **Largest proprietary dataset** (>50PB)[1] • Multiple **novel programs**
Design	• **~2,500 compounds** per program • **~4 years** to candidate[2]		• **~330 compounds** per program **(~90% fewer)** • **~1.5 years** to candidate
Clinical	• **Slow enrollment**, ops complexity • Low patient matching		• **30–60%** faster enrollment; • **10–40%** more eligible patients



Result: Faster, more capital-efficient discovery that's already produced 5 clinical-stage programs and >$500M in validated partnerships

1. Generated and aggregated >50 petabytes of high-quality, multimodal data
2. McGinnity et al, Drug Discovery Today, 2025

Recursion

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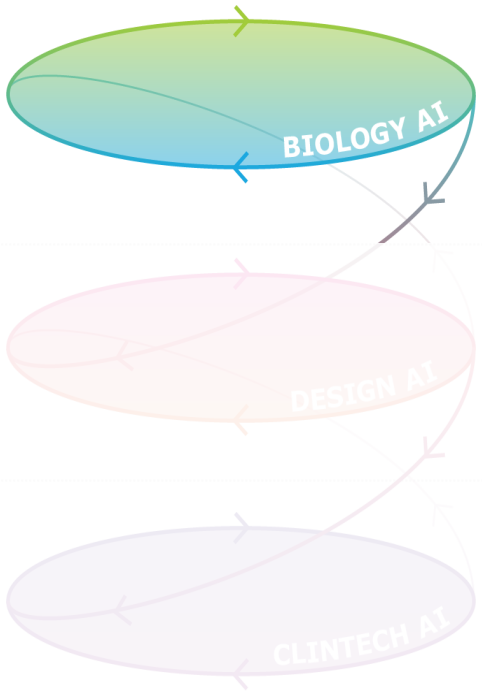
Biology	• **Majority of biology still unknown** • Discovery limited by incomplete understanding of human biology		**Genentech exercised FIRST unexplored Neuroscience target**
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Unexplored neuroscience target optioned by Genentech from the Recursion, Roche and Genentech partnership

$216 million in upfront and milestone payments achieved to date

2024

ACCEPTED

Roche and Genentech
Neuron AI biological map
*3Q24 - **$30M***

2025

ACCEPTED

Roche and Genentech
Microglia AI biological map
*4Q25 - **$30M***

2026

OPTIONED

Roche and Genentech
1st unexplored NS target

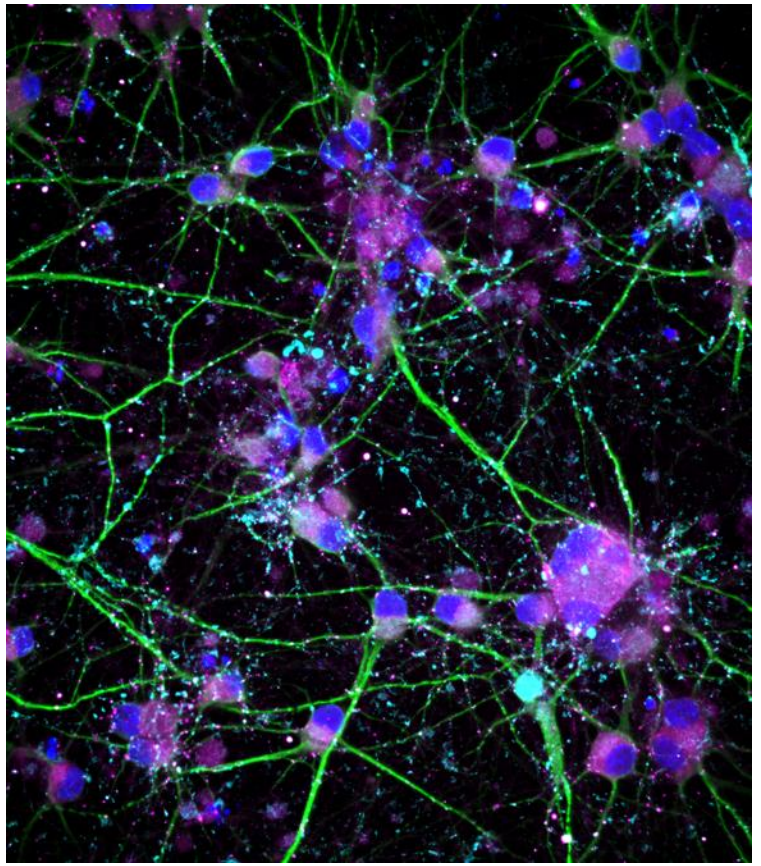
What's next:
Advancing into early discovery — applying Recursions AI-native chemistry engine to design a molecule against the validated target

KEY MILESTONE

1st

unexplored target, validated and optioned in Neuroscience

More than $300M
in development, commercialization, and net sales milestones for Recursion per small molecule program[1]



1. + tiered royalties up to high single digits

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Neuroscience: A growing disease burden, constrained by a limited target landscape

Vast unmet need persists as the industry repeatedly targets the same biology and limited success.

3B+ people affected

Neurological conditions are the world's leading cause of illness and disability.

Less than half the approval rate

CNS drugs face among medicine's highest attrition rates—before and during clinical trials—and are approved at less than half the rate of drugs in other therapeutic areas.

The brain is difficult to model

Human brain cells are complex, fragile and challenging to generate consistently at scale.

What does it take to build confidence and deliver a target?

QUESTION

Are you working in a disease relevant cellular context?

HOW RECURSION & ROCHE AND GENENTECH PARTNERED TOGETHER

Scaled, high-dimensional data set with the potential to contain multiple novel targets

1T+/~12 brains
hiPSC-derived neurons produced for the Neuromap

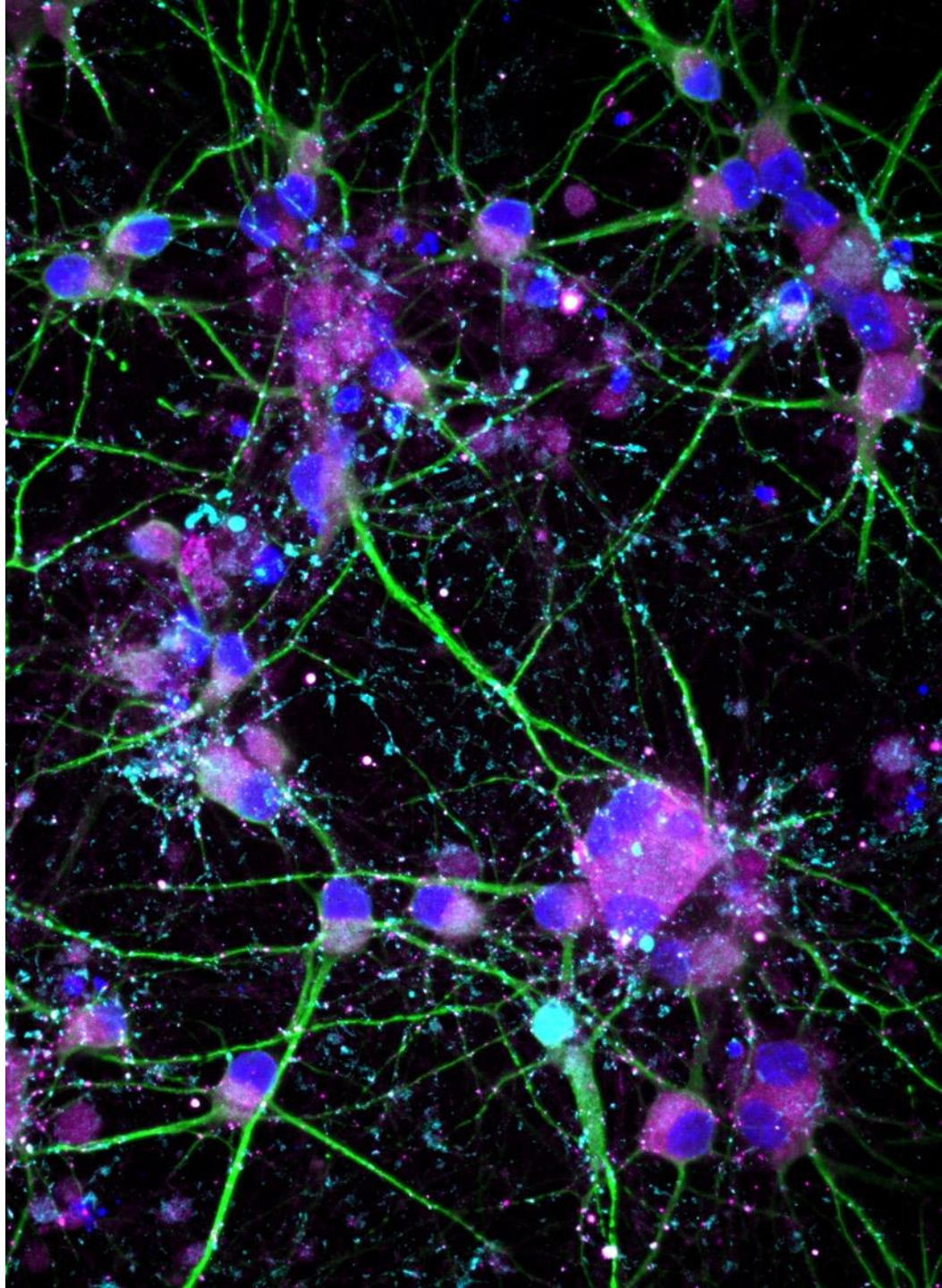
100B+
microglia produced for the Microglia Map

Disease-like perturbations
e.g. knockout, overexpression

46M
images across the genome — 2 first-of-its-kind whole-genome maps

WHY IT MATTERS

In biology, context matters. Recursion's moat is an atlas of disease-relevant datasets



What does it take to build confidence and deliver a target?

QUESTION

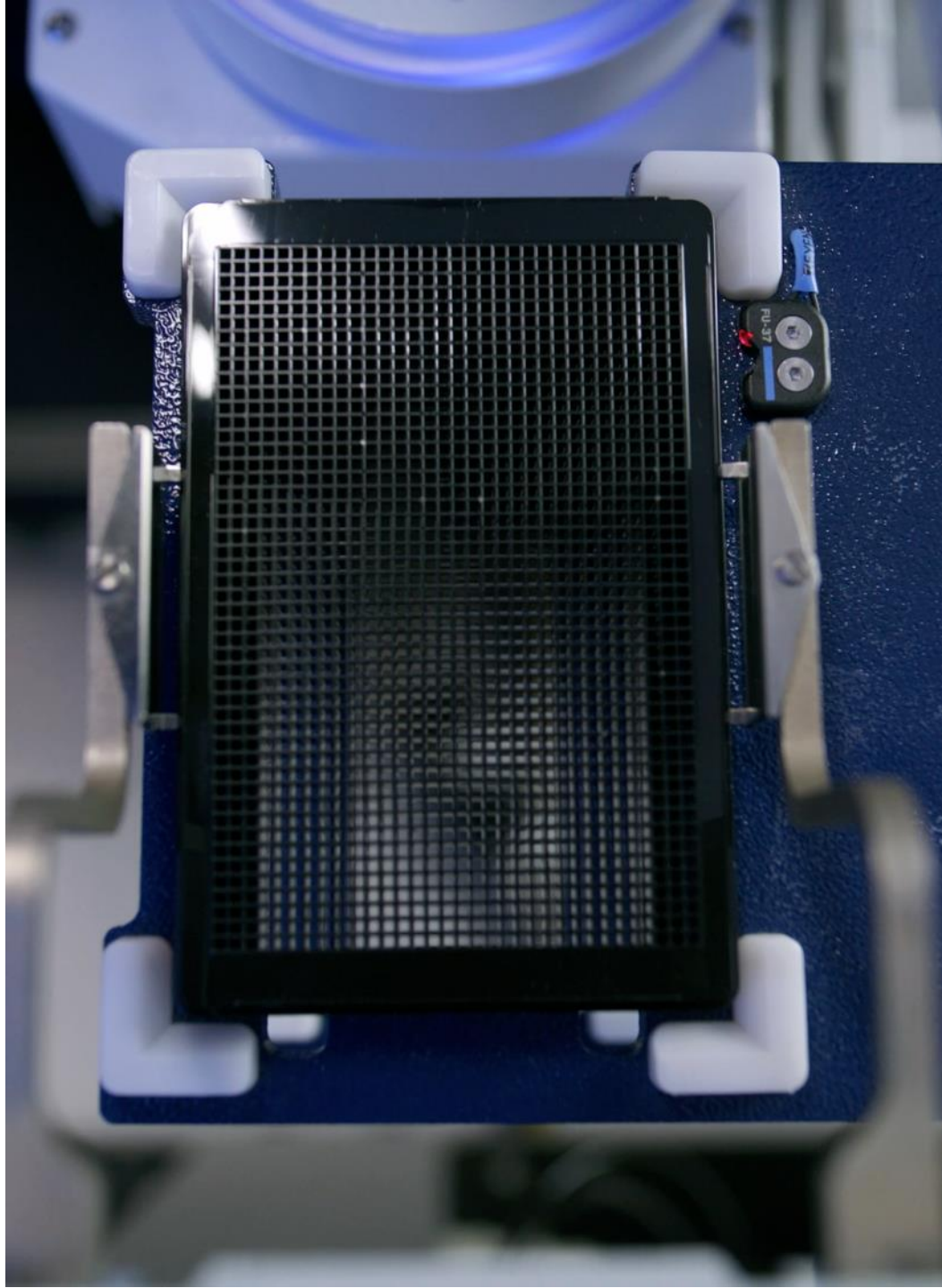
Are you grounding your research in known disease biology?

HOW RECURSION & ROCHE AND GENENTECH PARTNERED TOGETHER

- Hundreds of known-disease causing perturbations were added to experimental plates in the Map (e.g. disease-causing reagents and genetic mutations)
- All **queries are anchored around a known driver of neurological disease related pathology**

WHY IT MATTERS

Searches grounded with strong scientific evidence support clinical relevance at the outset



What does it take to build confidence and deliver a target?

QUESTION

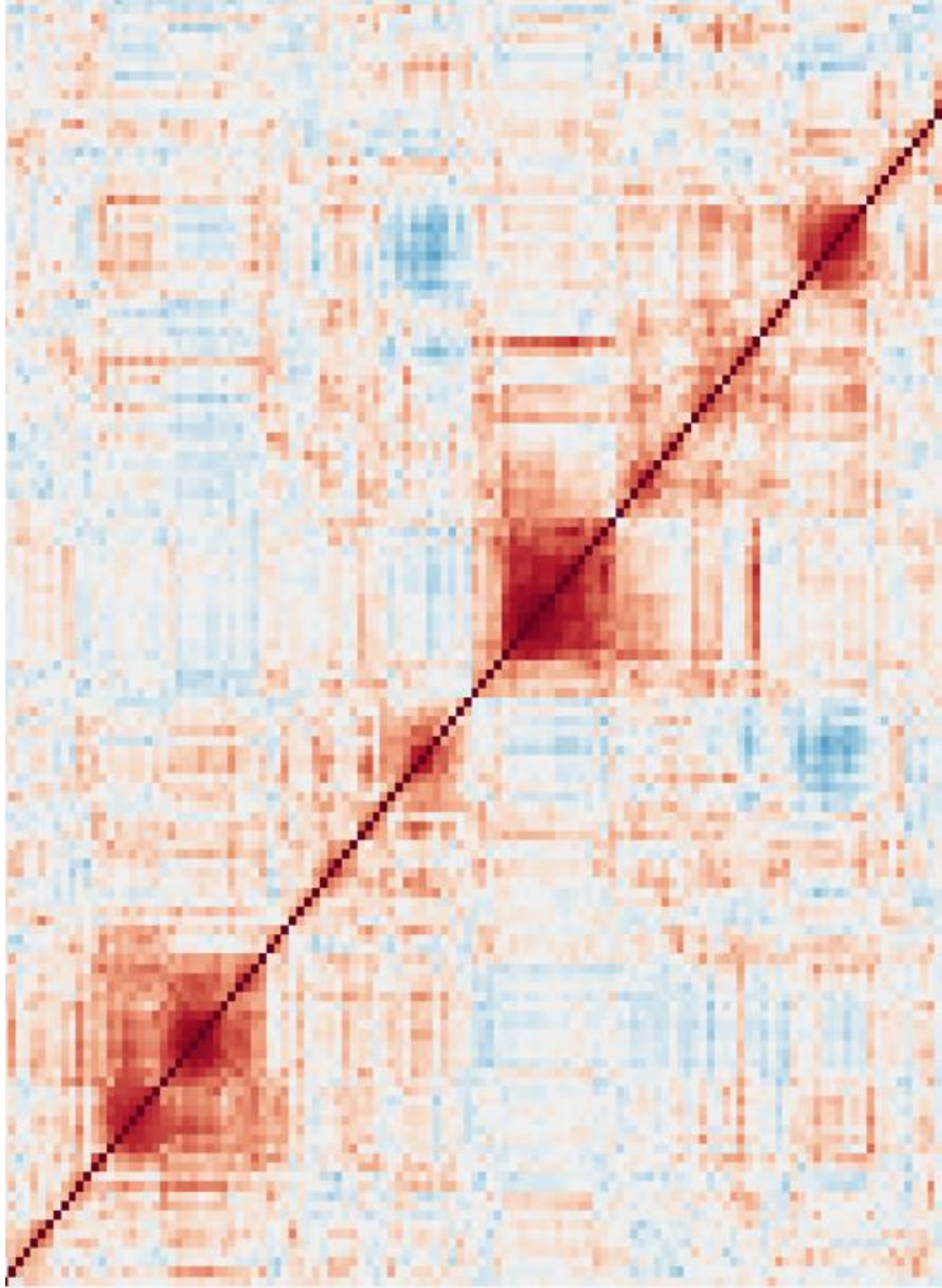
Can you find valuable insights others have missed?

HOW RECURSION & ROCHE AND GENENTECH PARTNERED TOGETHER

- Whole-genome screens allow us to apply biological expertise to interpret correlations across over 17,000+ genes for novel insights
  - 30 million high-dimensional cellular images to capture nuanced signatures for all perturbations in common format
  - Foundation models compare every perturbation within a common biological reference map to find genes associated with disease drivers of interest

WHY IT MATTERS

Every gene in the genome is ranked against disease-causing mutations of interest, allowing data —not individual hypotheses—to be the initial driver for which targets are most compelling.



What does it take to build confidence and deliver a target?

QUESTION

How do you establish confidence that your target is disease-relevant?

HOW RECURSION & ROCHE AND GENENTECH PARTNERED TOGETHER

Validated with Genentech, and advancing into a co-developed discovery program

1

Pathway validation

Interrogate mechanism of action and true attractiveness of the target

2

Functional validation

Signs of a healthier cell: viability, metabolism, electrophysiological activity

3

Disease validation

The highest bar. Assessing if the target actually moves the disease phenotype

WHY IT MATTERS

No single experiment tells the whole story - need to create a body of evidence

Collaborative process to generate and validate neuroscience targets

1. Builds disease-relevant human models with high-dimensional data

Output: Disease-relevant biology at unprecedented scale

2. Explores the human genome at scale

Output: Unexplored biology emerges from maps, reviewed and prioritized by scientists

3. Navigates from known to unknown biology

Output: A ranked list of potentially novel, biologically connected targets.

4. Validation experiments with Genentech

Output: A target advanced into an early discovery program

Recursion

Today’s news

Internal Pipeline

Our platform-derived internal pipeline is differentiated and rich in near term catalysts

		Disease indication(s)	Addressable patients[1]	Late discovery / Preclinical / Phase 1/2 / Pivotal	Next catalyst	AI enabled differentiation: Biology / Design / Clinical
CLINICAL	**REC-4881** MEK1/2 (Oral)	Familial adenomatous polyposis (FAP)	**>50,000**		**2H26:** FDA update	*Novel disease-mechanism insight*
CLINICAL	**REC-1245** RBM39 (Oral)	Solid tumors & lymphoma[2]	**>100,000**		**2H26:** Additional Ph 1 dose escalation data	*Novel target + novel degrader*
CLINICAL	**REC-617** CDK7 (Oral)	Advanced solid tumors	**~150,000**		**1H27:** Combo – early safety and PK	*Hard to drug, enhanced therapeutic index*
CLINICAL	**REC-3565** MALT1 (Oral)	B-cell malignancies	**~41,000**		**1H27:** Mono – early safety and PK	*Hard to drug, enhanced therapeutic index*
CLINICAL	**REC-4539** LSD1 (Oral)	Solid tumors & hematology oncology	**~45,000**		**2H27:** Mono – early safety and PK	*Hard to drug, enhanced TI, CNS penetrant*
PRECLINICAL	**REC-7735** PI3Kα H1047R (Oral)	Solid tumors	**>80,000[3]**		**2H26:** IND cleared, FIH in 2H 2026	*Enhanced therapeutic index (Onc & non-Onc)*
PRECLINICAL	**REC-102** ENPP1 (Oral)	Hypophosphatasia (HPP)	**>7,800**		**2H26:** Completion of IND-enabling studies	*Novel target + novel molecule*

Platform capability utilized
Platform capability to be utilized further in dev't
Platform capability not utilized

Neither the safety nor efficacy of the investigational drugs described has been established
1. Addressable patient populations estimate based on annual US+EU5 and currently identified indication
2. Multiple biomarkers being explored
3. Includes non-metastatic PIK3CA-H1047Rm patients (US+EU5). Includes diabetic patients

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Translating proof to products

REC-4881

MEK1/2

REC-4881: Potential **first targeted medicine** for familial adenomatous polyposis (FAP)

SIGNIFICANT UNMET NEED

>50k

Diagnosed post-colectomy patients (US + EU5)[1]

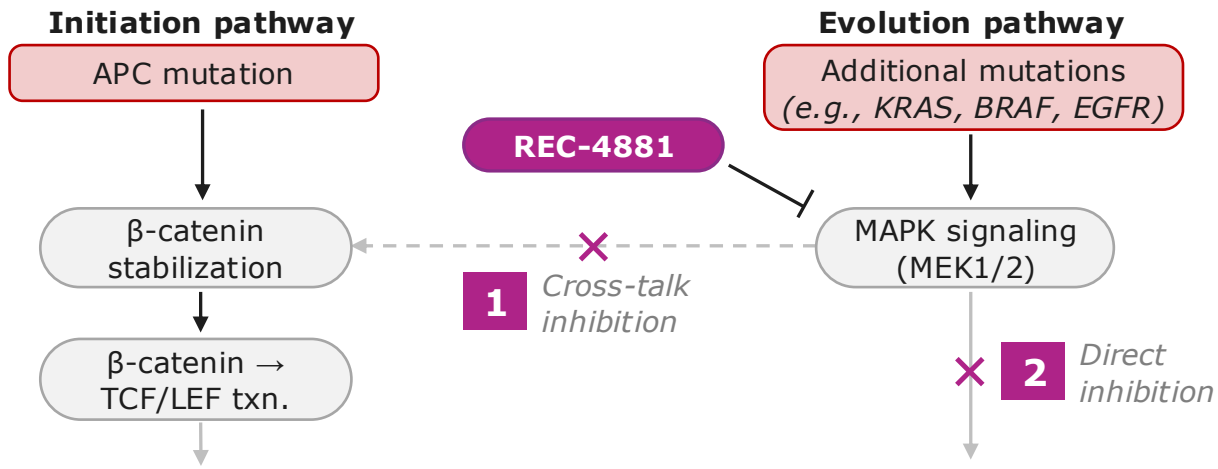
0

Approved therapies

>$10B

Total addressable market (US + EU5)[2]

REC-4881 MOA



REC-4881 designed to slow FAP progression through two mechanisms:

1 Modulates β-catenin transcriptional output by reducing MAPK–Wnt crosstalk

2 Blocks a common signaling pathway activated as adenomas acquire additional mutations (e.g., KRAS, BRAF)

1. EMA/NORD and Orphanet, adjusted for colectomy rate (Syngal S, et al. Am J Gastroenterol 2015;110(2):223–62. DOI 10.1038/ajg.2014.435). 20-30% of patients are diagnosed late or misdiagnosed 2. Modeled total addressable market (not realized or forecasted sales): diagnosed prevalence (~50k) x rare-disease oral net price (benchmark GOMEKLI ~$30k/mo for adults); sensitive to price and treated fraction
MoA Sources: Fujishita et al. Cancer Sci. 2015 Jun;106(6):692-699. doi: 10.1111/cas.12670; Blaj et al. Cancer Res. 2017 Apr 1;77(7):1763-1774. doi: 10.1158/00085472.CAN-16-2821.; Daniele Guardavaccaro and Clevers, Sci. Signal.5,pe15-pe15(2012).DOI:10.1126/scisignal.2002921; Cheng et al. Part Fibre Toxicol. 2026 Jan 16;23(1):4. doi: 10.1186/s12989-025-00656-3.Georgopoulos et al. J Cell Sci. 2014 Jul 1;127(Pt 13):2967-82. doi: 10.1242/jcs.150888

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Jenny's journey: A lifetime of surveillance and surgeries

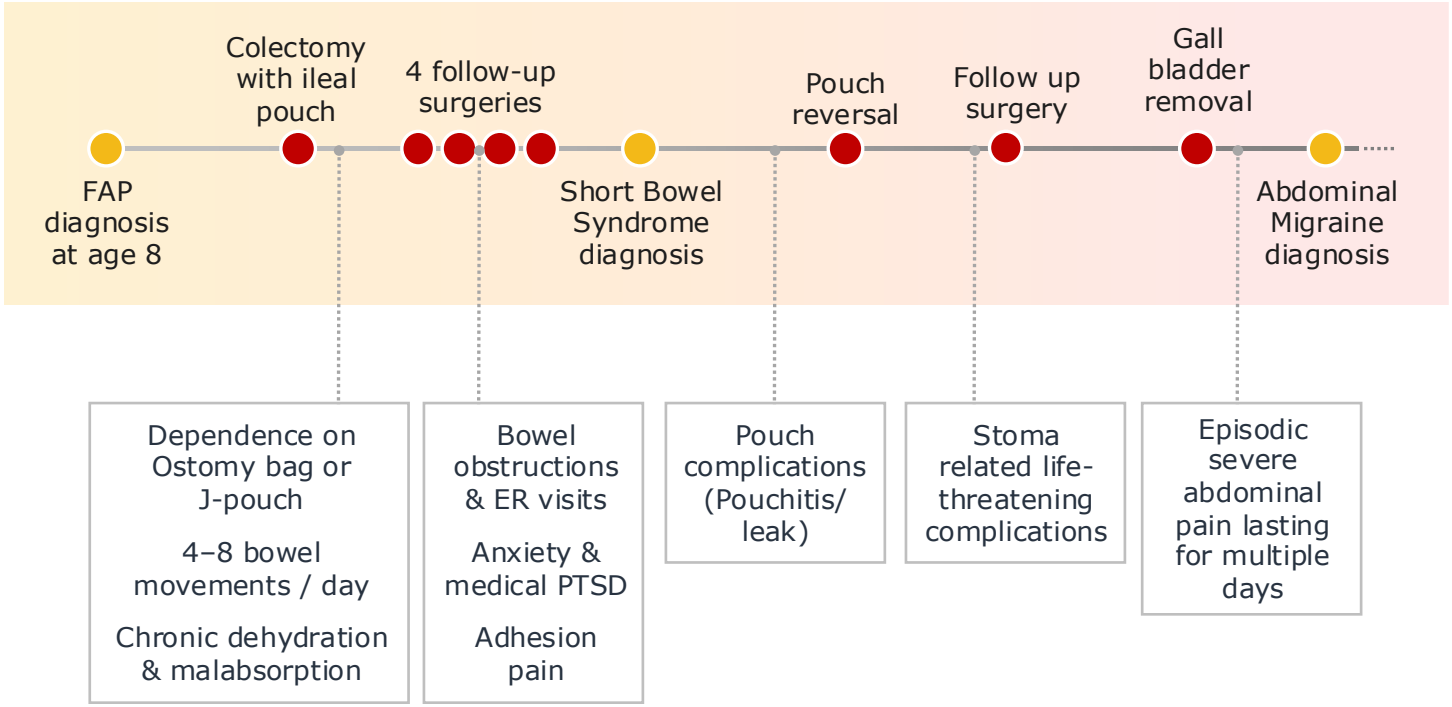
Jenny J.

Founder of Life's a Polyp, rare disease advocate, and patient living with FAP

"My biggest hope, for now and for the future, is just to make lives easier for FAP patients and families… ***to find new treatment modalities rather than just surgery****."*

"Adhesions cause pain from my surgeries. It causes obstructions. *I have to be careful with what I eat. When I do get an intestinal obstruction, I try to manage that at home. I [have] something always on the mind.* ***Mentally, it affects me every day because I have medical PTSD from my surgeries."***

8
Major Surgeries to Date



Note: Jenny's treatment journey may not be typical of all FAP patients

REC-4881: A potential paradigm shift for FAP — echoed by physicians treating these patients today

Rapid, durable efficacy

- **43%** median polyp burden reduction in 3 months
- **Reduction sustained off treatment** for 3 months

*"The **most compelling piece of data is the durability of REC-4881 from week 13 to 25**. The fact that the biological effect persists after the dosing ends."*
- Practicing Gastroenterologist, US academic medical center

Efficacy across the entire GI tract

- **Strong upper & lower GI efficacy**
  Upper GI is area of highest unmet need

*"A drug that impacts **both the retained rectum or the pouch, and the duodenum..** the combination of effectiveness **at both locations** is absolutely a critical observation"*
- Former Head of Colorectal Surgery, MSKCC

Tolerable safety, with oral, chronic dosing potential

- **1st-in-class potential**
  oral, chronically dosed therapy
- Safety consistent with MEK1/2 inhibition[1]: **No Grade ≥4 TRAEs**; Grade 3 in 15.8% of patients

*"This initial trial data could position [REC-4881] as something **that's taken daily, or perhaps we'll find it's effective in a cyclic fashion**, which might be a really **alluring option for patients** "*
- Practicing Gastroenterologist, US academic medical center

Dual**-mechanism MoA: **Targets 2 drivers of FAP
Wnt/APC initiation pathway and MEK1/2-driven evolution pathway

1. In approved oncology and non-oncology indications

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Near term value inflection across REC-4881: Regulatory update and key congress presentation

Key upcoming milestones

- **Ongoing**
  Enrollment of ≥18 cohort underway; dose optimization advancing
- **November 2026**
  Additional Phase 2 clinical data at CGA-IGC Annual Meeting
- **2H26**
  Update on FDA engagement to define potential registrational path
- **1H27**
  Additional Phase 2 clinical data

30th CGA-IGC Annual Meeting, Denver

- Leading annual meeting dedicated specifically to **hereditary GI cancer syndromes** including FAP
- Highly targeted audience: **Connects the entire care ecosystem** including gastroenterologists, surgeons, genetic counselors, and researchers

Session information

Presidential Plenary I
Mon. November 2, 2026 @ 13:30 - 15:00

Updated safety and efficacy data of REC-4881 monotherapy in familial adenomatous polyposis: Phase 1b/2 trial results contextualized with real-world registry data

Translating proof to products

REC-7735

PI3Kα

Recent pan-mutant PI3Kα inhibitors still have suboptimal therapeutic index

PI3Kα is a validated target but wildtype inhibition drives hyperglycemia and tolerability issues that limit efficacy

A validated oncology target[1]

PI3Kα-H1047R mutation is a validated driver across multiple cancers; H1047R mutations make up the largest, distinct subtype among PIK3CA-driven cancers

Wildtype inhibition drives toxicity[2]

Next-gen Pan-mutant inhibitors also block wildtype PI3Kα, which can drive tolerability issues and hyperglycemia (grade 1/2 hyperglycemia)

Hyperinsulinemia can reactivate PI3K in the tumor[3]

WT-driven hyperglycemia triggers compensatory hyperinsulinemia — which can reactivate PI3K signaling within the tumor, undercutting efficacy independent of dose or tolerability

Toxicity forces dose compromises[2,4]

Toxicity-driven dose interruptions and discontinuations, which can prevent patients achieving optimal pathway inhibition

WHAT WE'RE HEARING FROM KOLS

> ***"We have to move away from the idea that Grade 1 hyperglycemia is 'fine.'*** *Even at low levels, the accompanying hyperinsulinemia can reactivate the PI3K pathway within the tumor" – KOL*

> ***"The biggest unmet need is the diabetic patient****. We are still excluding the 20% of the population that arguably needs these drugs most because we haven't uncoupled the tumor's PI3K from the liver's glucose control" – KOL*

1. Madsen et al. Oncogenic *PIK3CA* promotes cellular stemness in an allele dose-dependent manner, *Proc. Natl. Acad. Sci. U.S.A.* 116 (17) 8380-8389, https://doi.org/10.1073/pnas.1821093116 (2019) | Conditional activation of Pik3ca(H1047R) in a knock-in mouse model promotes mammary tumorigenesis and emergence of mutations. Oncogene. 2013 Jan 17;32(3):318-26. Epub 2012 Feb 27. https://doi.org/10.1038/onc.2012.53 | Distinction: L. Zhao & P.K. Vogt, Helical domain and kinase domain mutations in p110α of phosphatidylinositol 3-kinase induce gain of function by different mechanisms, *Proc. Natl. Acad. Sci. U.S.A.* 105 (7) 2652-2657, https://doi.org/10.1073/pnas.0712169105 (2008).
2. Hanker et al. *Cancer Discov* (2019) 9 (4): 482–491. https://doi.org/10.1158/2159-8290.CD-18-1175
3. Hopkins, B.D., Pauli, C., Du, X. *et al.* Suppression of insulin feedback enhances the efficacy of PI3K inhibitors. *Nature* **560**, 499–503 (2018). https://doi.org/10.1038/s41586-018-0343-4
4. Shen S, Chen Y, Carpio A, Chang C, Iyengar NM. Incidence, risk factors, and management of alpelisib-associated hyperglycemia in metastatic breast cancer. *Cancer.* 2023; 129(24): 3854-3861. doi:10.1002/cncr.34928

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Our platform designed a mutant-selective PI3Kα H1047R inhibitor built to improve therapeutic index

REC-7735

>100x

selectivity for H1047R over wildtype

Precision for the mutation that matters most: H1047R is PI3Kα's single most frequent activating mutation

Platform identified a previously unpublished binding site and delivered a development candidate in only 10 months[1] with no identified off-target liabilities

Precision for H1047R turns >100x selectivity into a wider therapeutic index

A wider therapeutic index drives multiple benefits:

Deep target suppression	>100x selectivity drives full inhibition of H1047R at effective doses, potentially avoiding the toxicity ceiling that may require dose adjustments in pan-mutant inhibitors
Higher dose intensity & duration	Avoiding WT-driven toxicity lets patients stay on therapeutic doses longer, translating selectivity into deeper, more durable efficacy
Avoids hyperinsulinemia-driven reactivation	Limits the hyperinsulinemia that can reactivate PI3K signaling in the tumor and compromise efficacy, while easing treatment of prediabetic / diabetic patients

Preclinical proof: *dose-dependent tumor regression superior or comparable to other PI3Kα inhibitors with no hyperglycemia signal observed*[2]

1. ~10 months from first hit to development candidate, across only 242 compounds
2. Results generated in a breast cancer CDX model with an H1047A mutant PI3Kα and in naïve wildtype mice

Highly mutant-selective PI3K inhibitors with an improved TI have potential to benefit a greatly expanded patient population

Improved outcomes for indications with PI3Kα inhibitors

Ability to **treat additional indications** not reached by current therapies due to insufficient depth of inhibition, hyperglycemia, and tolerability

H1047R HR+ / HER2- breast cancer, across stages

- Pan-mutant PI3Kα inhibitors
- Tolerability concerns limit treatment duration, potentially reducing efficacy
- Historically, treatment has been mostly limited to non-diabetic and carefully selected, well-controlled diabetic patients due to hyperglycemia

~47,000 Patients[1] | **~16-21%** Diabetic[1]

H1047R metastatic solid tumors

- Including HER2+ BC, TNBC, colorectal, endometrial, ovarian, and head & neck cancers
- Hyperglycemia and tolerability concerns limit may limit treatment duration

~12,000 Patients | **~16-30%** Diabetic[2]

H1047R non-metastatic solid tumors

- Including HER2+ BC, TNBC, colorectal, endometrial, ovarian, and head & neck cancers
- Longer treatment duration in lower disease-burden patients raises tolerability requirements

~21,000 Patients | **~9-20%** Diabetic[2]

H1047R-driven non-onc. conditions

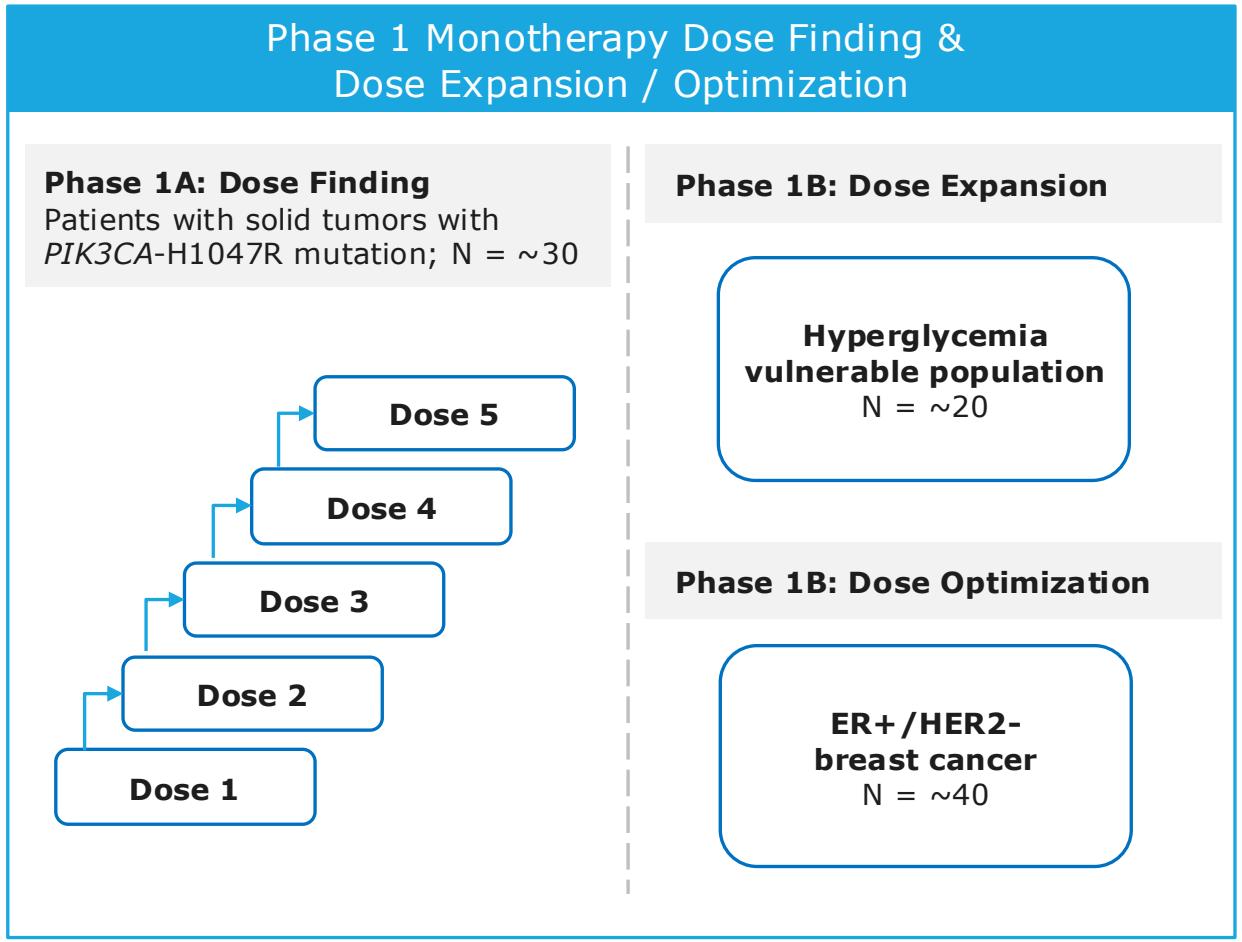
- Including PROS and PIK3CA-related vascular anomalies
- Current approved treatment can cause hyperglycemia, potentially limiting efficacy

~25,000 Patients

Note: TAM in US + EU5 for indications with H1047R mutations

1. Inclusive of early stage & metastatic cancer patients
2. % varies dependent on solid tumor indication

Phase 1 Dose Finding Clinical Trial: ZINNIA Phase 1/2 trial in patients with *PIK3CA* H1047R-mutant select solid tumors



- **Combination expansion** will be based on clinical safety profile and target engagement
- **Leveraging RWD** to optimize protocol and expand eligible patient population

Next steps:

- Initiate ZINNIA trial in 2H26
- Early safety and PK from monotherapy trial in 1H28

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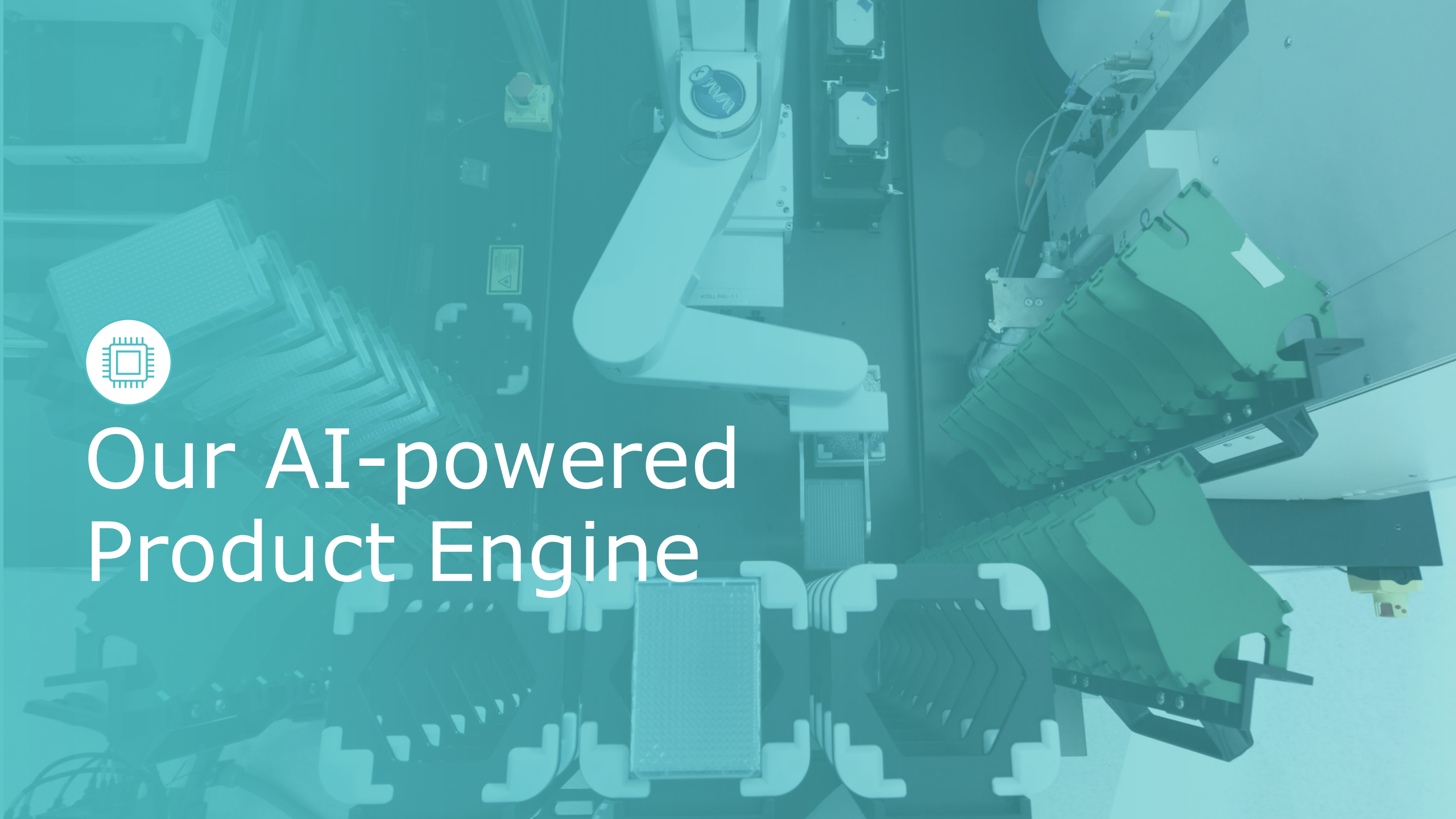
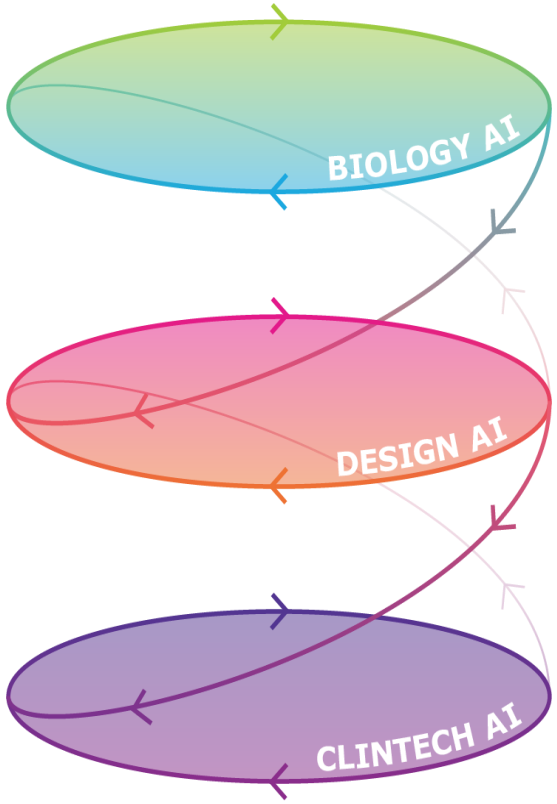


Our AI-powered Product Engine

Frontier AI and agents amplifies the value of Recursion's proprietary data and product engine



Select examples of early agent implementations

Target discovery agent

Pairs frontier reasoning with proprietary multimodal maps to surface novel targets

→ *Enabling scientists to mine and extract insights from proprietary maps in **hours rather than weeks***

Drug design agents

Design agent reasons over SAR & structural data to identify what to solve next and how

→ Reduce structural analysis **from 4h -> 30m**

*Agent-generated hypotheses now **driving design cycles in active programs***

Clinical strategy orchestration agent

Agentic decision layer coordinates patient, site, operational, CMC, and biometrics data

→ *Agent-supported enrollment driving a **1.3 to 1.6x increase in enrollment rates** vs. historical benchmarks*

Today's agents are an early foundation. As frontier AI advances, the value of Recursion's proprietary data and infrastructure compounds

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AGENTIC DESIGN

How we design our drugs matters as much as the drugs themselves.

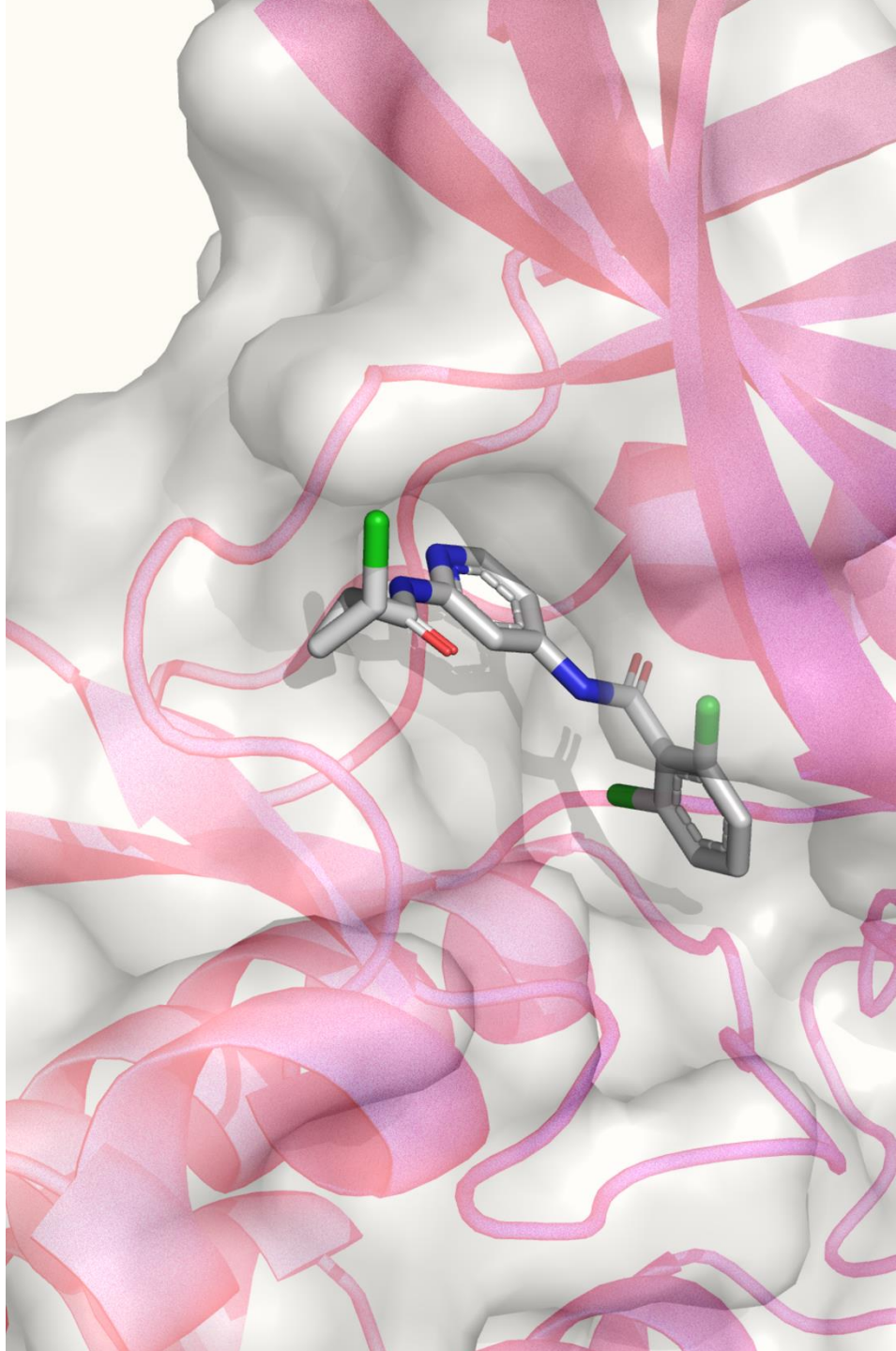
Link to Video

Nesso-1: Open source – Affinity prediction that keeps pace with rapid design cycles

***New*: Nesso-1 applied for live programs on platform supported by disease-relevant data**

- **Boltz-2 accuracy** on public benchmarks at **~10-20× faster** inference
- **>20x more compounds screened** for the same cost as current state-of-the-art approaches
- Proprietary data **lifts model prediction performance by ~20%**

Recursion Advantage:
Rapidly combining proprietary wet-lab data with new models and deploying them into real programs **shortens the design cycle** and tightens the **make–test–learn loop** — improving speed, cost, and probability of success



Nesso-1: Accelerating Open-Source Binding Affinity Predictions, Recursion, July 2026. https://www.valencelabs.com/wp-content/uploads/2026/07/nesso1.pdf)

Bolstering our team with key new hires

Hoifung Poon, Ph.D.
Chief AI Officer

- **Seasoned AI Research Executive:** 15+ years at Microsoft, most recently General Manager of Microsoft Research, leading biomedical AI research centered on the "virtual patient", a high-fidelity digital twin of the patient journey
- **Strategic AI Leadership:** Will lead Recursion's AI strategy end to end, unifying frontier research and applied AI into a single effort across drug discovery and clinical development
- **Proven Track Record:** Led breakthrough foundation models in digital pathology and spatial omics (GigaPath, GigaTIME; Nature and Cell); open-weight models with tens of millions of downloads, deployed in clinical use at major health systems

Donovan Chin, Ph.D.
Senior Vice President, Drug Design

- **Cross-Modality Drug Discovery Leader:** 20+ years spanning small molecules, RNA-targeted therapeutics, proximity approaches and novel peptide modalities, most recently leading the AI and physics-based computational drug discovery strategy at Parabilis and Arrakis, respectively
- **Proven Track Record:** At Parabilis, led the computational strategy behind Helicons, a novel class of constrained α-helical peptides; at Arrakis, pioneered computational approaches unlocking small-molecule engagement of previously inaccessible RNA structures
- **Deep Pharma Foundation:** Began his career at Novartis, building computational drug discovery capabilities across oncology, cardiovascular and metabolic disease, and infectious disease

Pairing bold ambition with disciplined execution

Financials

Building more value with less cost

Reducing 2026 cash operating expense[1] guidance to <$375m
(from <$390m)

An outcomes-based, fit-for-purpose organization:

- Prioritized capital towards operations with clear and measurable impact, focused on long-term pipeline and technology differentiation
- Leadership in AI and automation with differentiation from proprietary data and integrated modelling
- Rebuilt corporate systems to measure how every dollar invested leads to an outcome

$556.8 million in cash[2]
as of June 30, 2026

Revised guidance represents
~40% reduction in cash opex from pro forma 2024 to FY 2026

Expected **cash runway into early 2028** without additional financing

1. Cash operating expense—defined as net cash used in operating activities less partnership inflows and transaction costs—is a non-GAAP financial measure. See Appendix for reconciliation of non-GAAP financial measures.
2. Cash, cash equivalents and restricted cash

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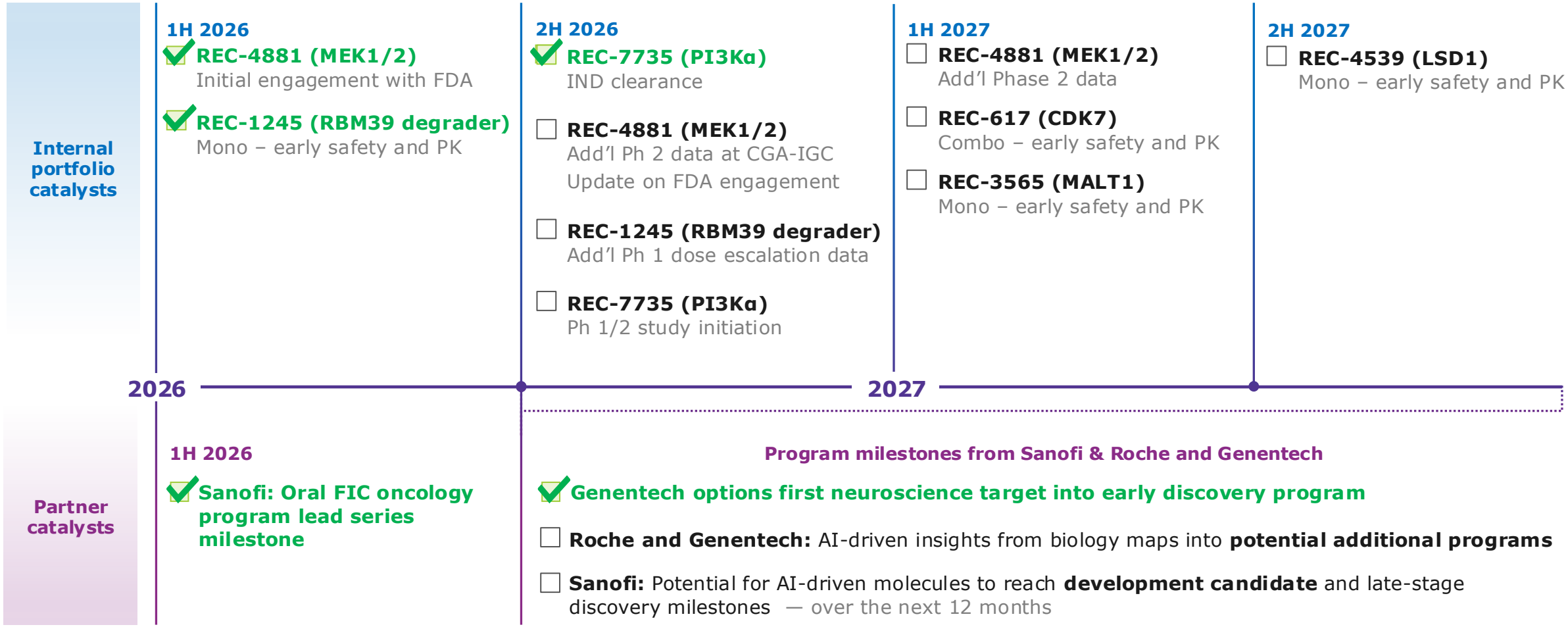
Looking ahead

Expected upcoming milestones

2026 and 2027 catalysts



Note: IND-enabling studies ongoing for REC-102 (ENPP1)

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THANK YOU

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Q & A

Appendix

Non-GAAP Financial Measures

To supplement our financial statements prepared in accordance with U.S. GAAP, we monitor and consider operating cash expense, which is a non-GAAP financial measure. We define operating cash expense as the net cash used in operating activities, excluding non-ordinary course transaction costs and partnership cash inflows. This non-GAAP financial measure is not based on any standardized methodology prescribed by U.S. GAAP and is not necessarily comparable to similarly-titled measures presented by other companies. We believe operating cash expense to be a liquidity measure that provides useful information to management and investors about the amount of cash consumed by the operations of the business. A limitation of using this non-U.S. GAAP measure is that operating cash expense does not represent the total change in cash and cash equivalents for the period because it excludes cash provided by or used for other investing and financing activities. We account for this limitation by providing information about our capital expenditures and other investing and financing activities in the statements of cash flows in our financial statements. Additionally, we reconciled operating cash expense to net cash used in operating activities, the most directly comparable U.S. GAAP financial measure. In addition, it is important to note that other companies, including companies in our industry, may not use operating cash expense, may calculate operating cash expense in a different manner than we do or may use other financial measures to evaluate their performance, all of which could reduce the usefulness of operating cash expense as a comparative measure. Because of these limitations, operating cash expense should not be considered in isolation from, or as a substitute for, financial information prepared in accordance with U.S. GAAP.