



Recursion.

(L)earnings 2Q25

August 2025

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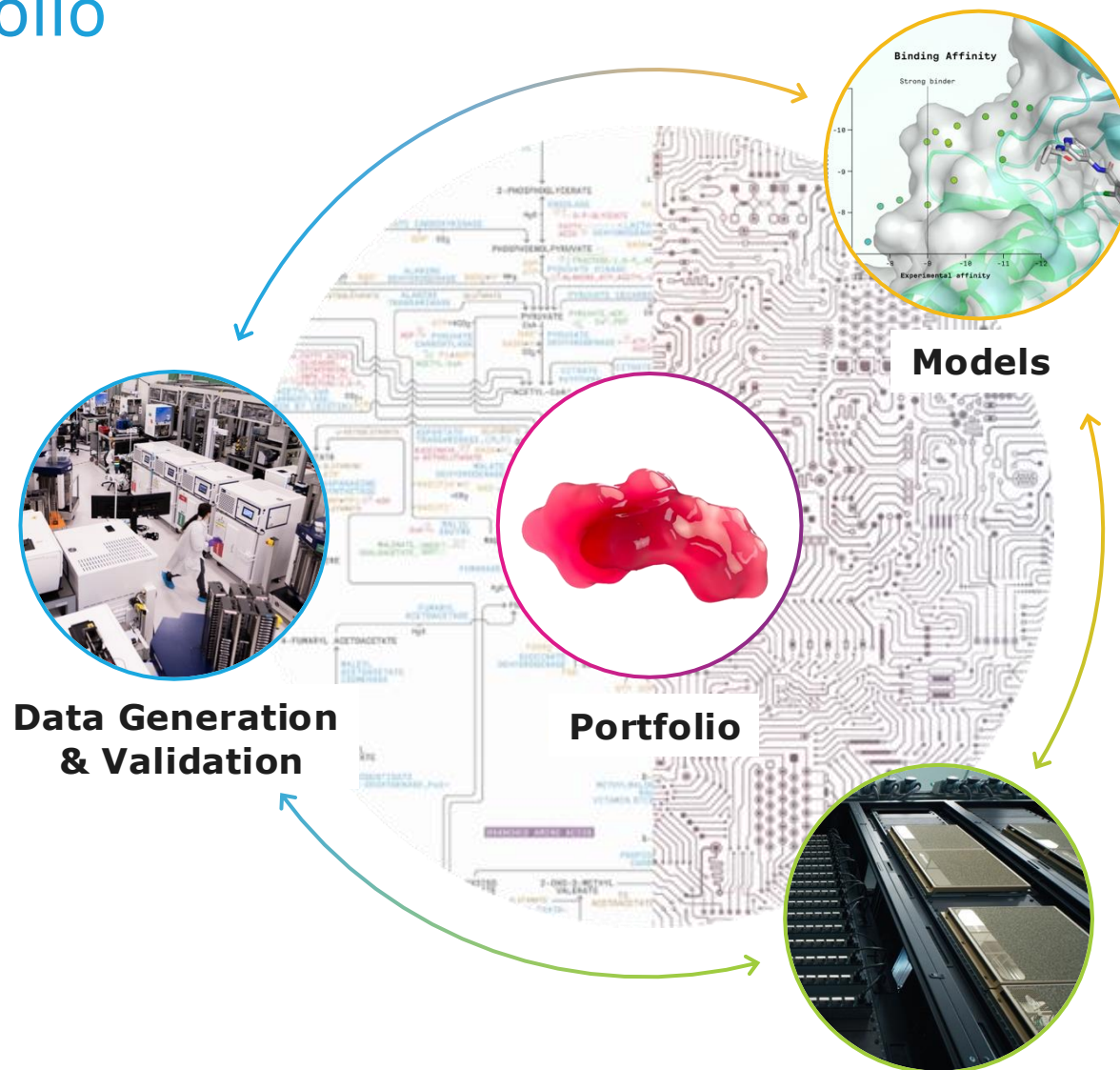
Cross-trial or cross-candidate comparisons against other clinical trials and other drug candidates are not based on head-to-head studies and are presented for informational purposes; comparisons are based on publicly available information for other clinical trials and other drug candidates.

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Recursion OS 2.0

Recursion's unbiased platform approach delivers a strong internal and partnered portfolio

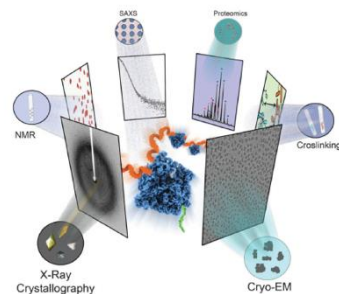
- Powered by **proprietary** and **fit-for-purpose** scaled data
- **End-to-end capabilities** spanning novel target discovery, precision chemistry and optimized clinical trials
- Built upon iterative cycles of **dry-lab predictions** and **wet-lab validation** to accelerate learning



Boltz-2: Open-source model with MIT commoditizing binding affinity prediction approaching FEP accuracy at 1000x speed

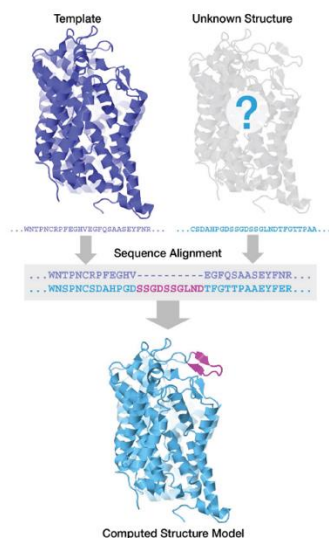
Designed for drug discovery and virtual screening

Method conditioning



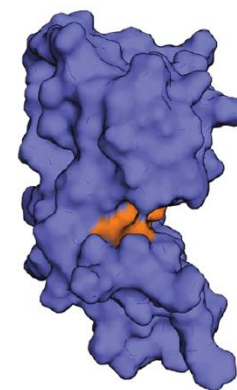
Allows users to specify an experimental modality to emulate

Templates



Template steering — allows users to input reference templates that embed prior knowledge

Contacts or pocket



Contact/pocket constraints — ensure output follows respects given conditions

Over **171K** downloads and **41.5K** unique users in less than two months

ClinTech: Industrialize clinical development by building an end-to-end platform to increase probability of success



Causal AI applied to human genomics

- **Patient-platform** connectivity
- Enables **target validation** and **patient stratification**
- Supports expansion into **new indications**



Intelligent clinical trial design

- **More robust trial planning** via clinical trial simulations
- **Potential for up to 30% more patients** receive optimal dose



AI-powered recruitment & execution

- **Potential for 50% faster enrollment projections** through high quality sites
- **2+ months faster** trial activation

Powered by *integrated tech stack, RWE, and agentic solutions*



Strategic Partnerships

TEMPUS



Delivering Pipeline Advancements and Partnership Value

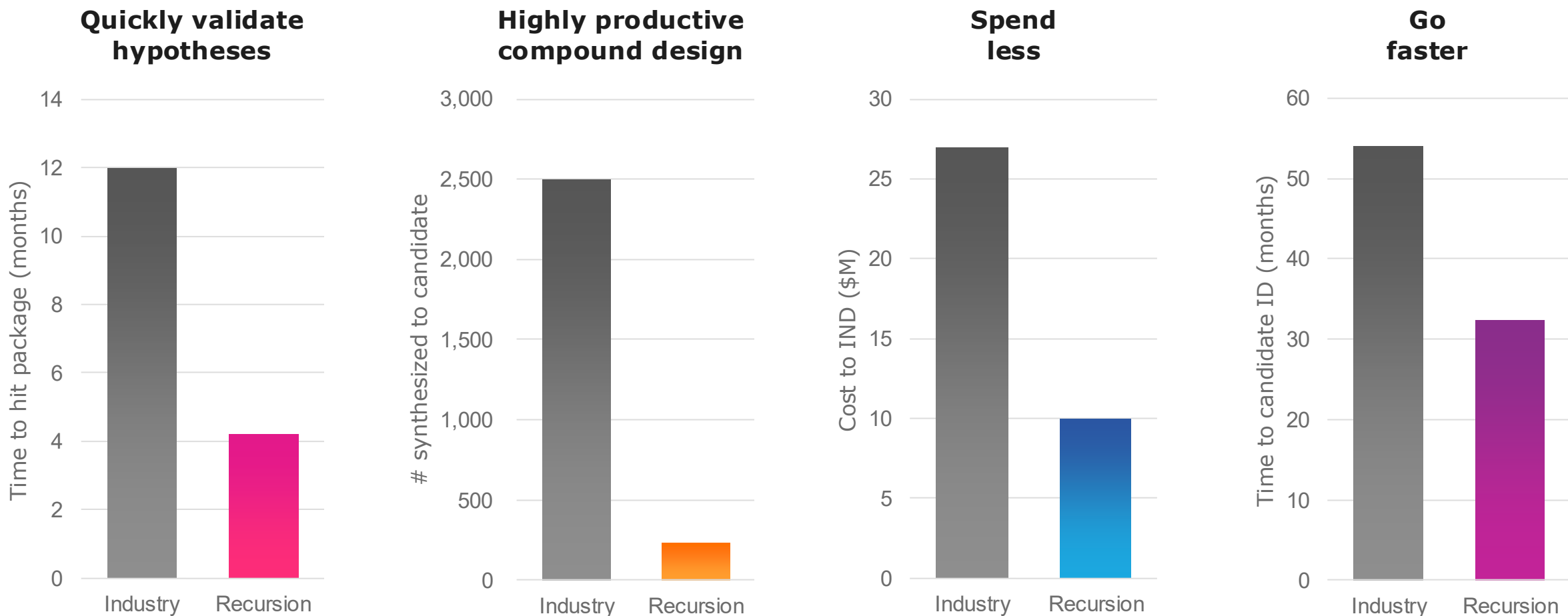
	Target	Disease Indication	Late Discovery	Preclinical	Phase 1/2	Pivotal/Phase 3
Oncology						
REC-617	CDK7	Advanced Solid Tumors	[Progress bar]			
REC-1245	RBM39	Biomarker-Enriched Solid Tumors & Lymphoma	[Progress bar]			
REC-3565	MALT1	B-Cell Malignancies	[Progress bar]			
REC-7735	PI3Kα H1047R	Breast Cancer	[Progress bar]			
Rare Disease						
REC-4881	MEK1/2	Familial Adenomatous Polyposis	[Progress bar]			
REC-102	ENPP1	Hypophosphatasia	[Progress bar]			

REC-4539 for Small-Cell Lung Cancer (target: LSD1) is on strategic pause.

Partnerships

	Therapeutic Area	Highlights
Roche and Genentech	Neuroscience & Oncology	First neuroscience phenomap in human cell context. One optioned Gi oncology program continues to advance. Potential for up to 40 compound programs
Sanofi	Oncology & Immunology	Milestones achieved across four distinct BIC and FIC discovery programs in 18 months; potential for several program milestones upcoming
Bayer	Oncology	Advancing multiple programs with previously “undruggable” targets to lead series milestones
Merck KGaA, Darmstadt, Germany	Oncology & Immunology	Multi-year collaboration to identify and advance first-in-class and best-in-class programs

Recursion brings medicines to clinic faster and at lower cost



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

Building Momentum: Portfolio Advancement Fueled by Platform Insights

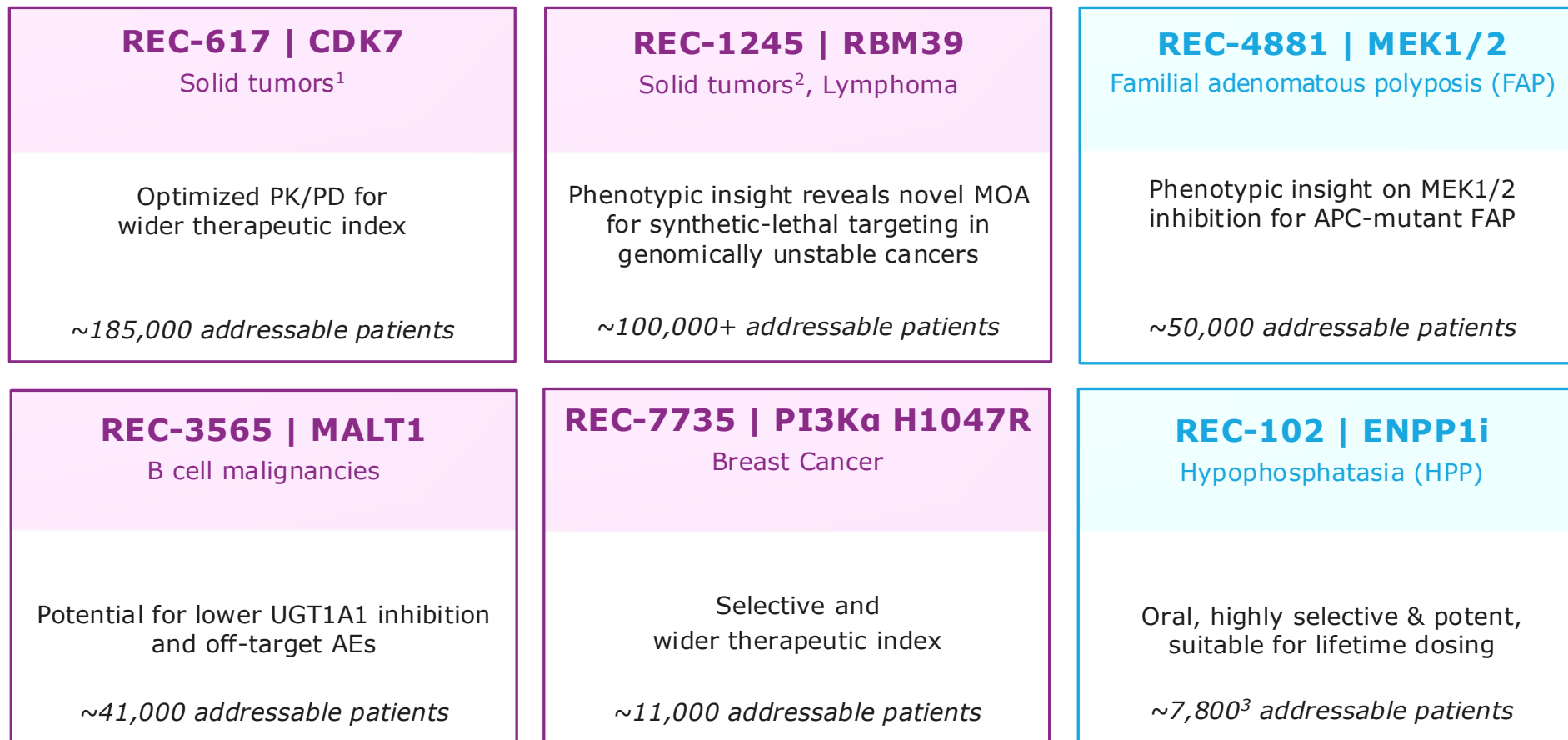
Delivering Pipeline Advancements and Partnership Value

	Target	Disease Indication	Late Discovery	Preclinical	Phase 1/2	Pivotal/Phase 3
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Advancing programs with strong therapeutic rationale, powered by Recursion OS



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

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

2. Biomarker-enriched

3. Diagnosed patients

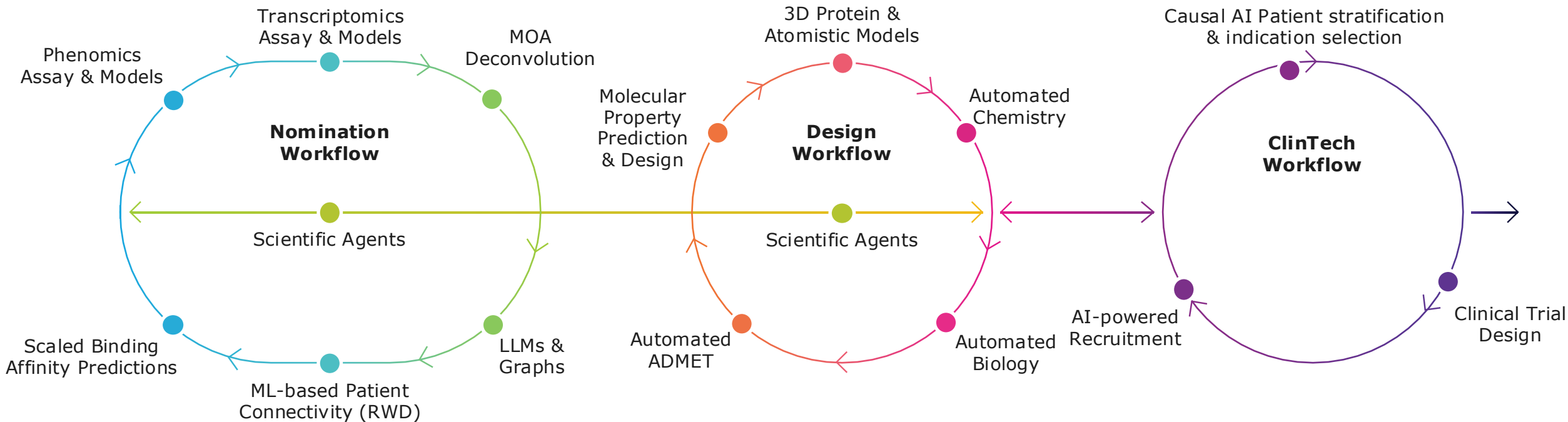
Note: REC-4539 | LSD1: Precision designed for reversibility and CNS penetration. *Strategic pause to ensure a competitive Target Product Profile*

Recursion OS 2.0: Efficiently delivering novel insights, precision design, and optimized clinical trials

AI-powered Biological Insights

AI-enabled Precision Design

AI-informed Clinical Development

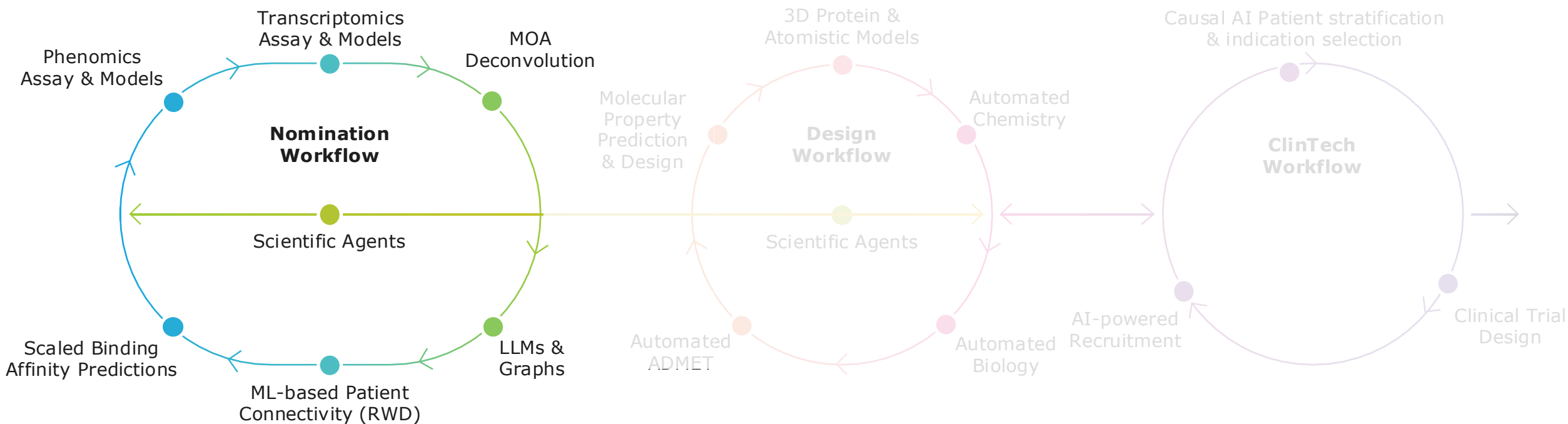


REC-1245 (RBM39 degrader): Novel biological insight drives clinical program

AI-powered Biological Insights

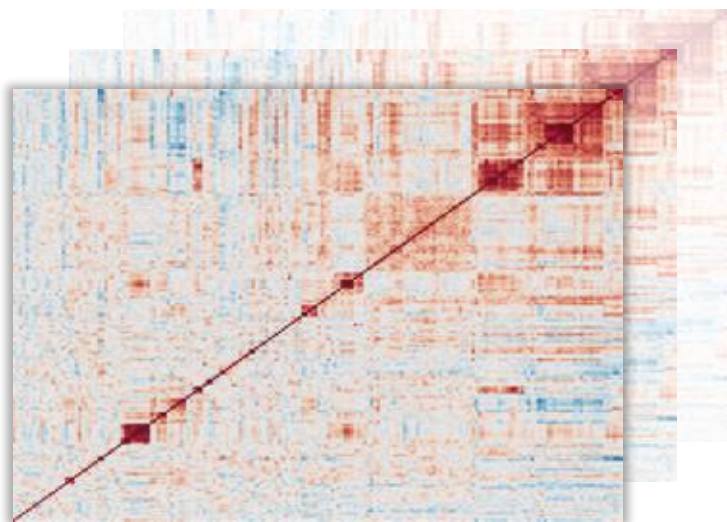
AI-enabled Precision Design

AI-informed Clinical Development



Platform links RBM39 biology to CDK12, a key regulator of DNA damage response genes (DDR) and replication stress (RS)

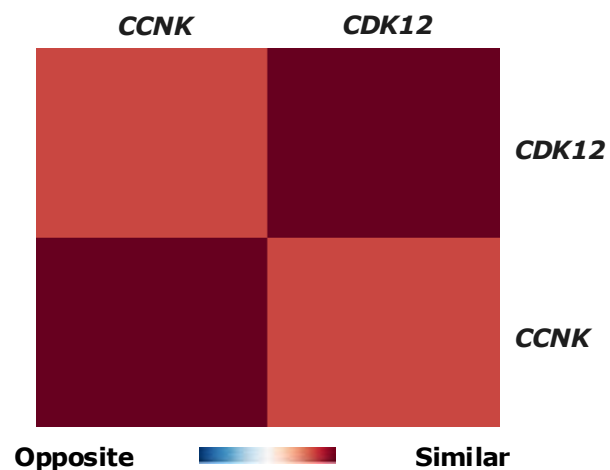
AI-guided Multimodal Mapping



Deep genome coverage across major DDR pathways in HUVEC PhenoMap¹

- **Whole Genome CRISPR** knockouts; close to **1 million** compounds profiled
- **Vast** DDR network resolution **unattainable** with single-readout screens

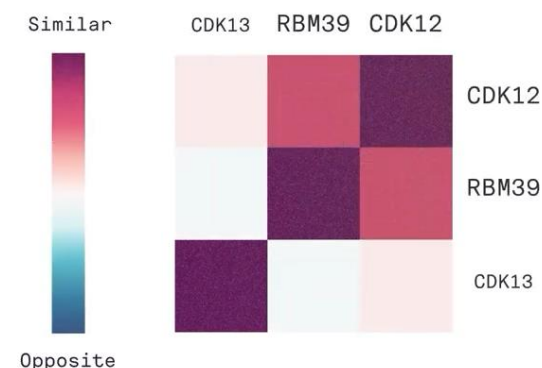
CDK12 Axis Recapitulated



CDK12 and Cyclin K (CCNK) share a similar signature in phenomic readout

- CDK12 kinase activity **requires** CCNK binding, a **well-established** dependency
- **Validates** approach and potential for novel **AI-driven mechanistic** and **patient** insights

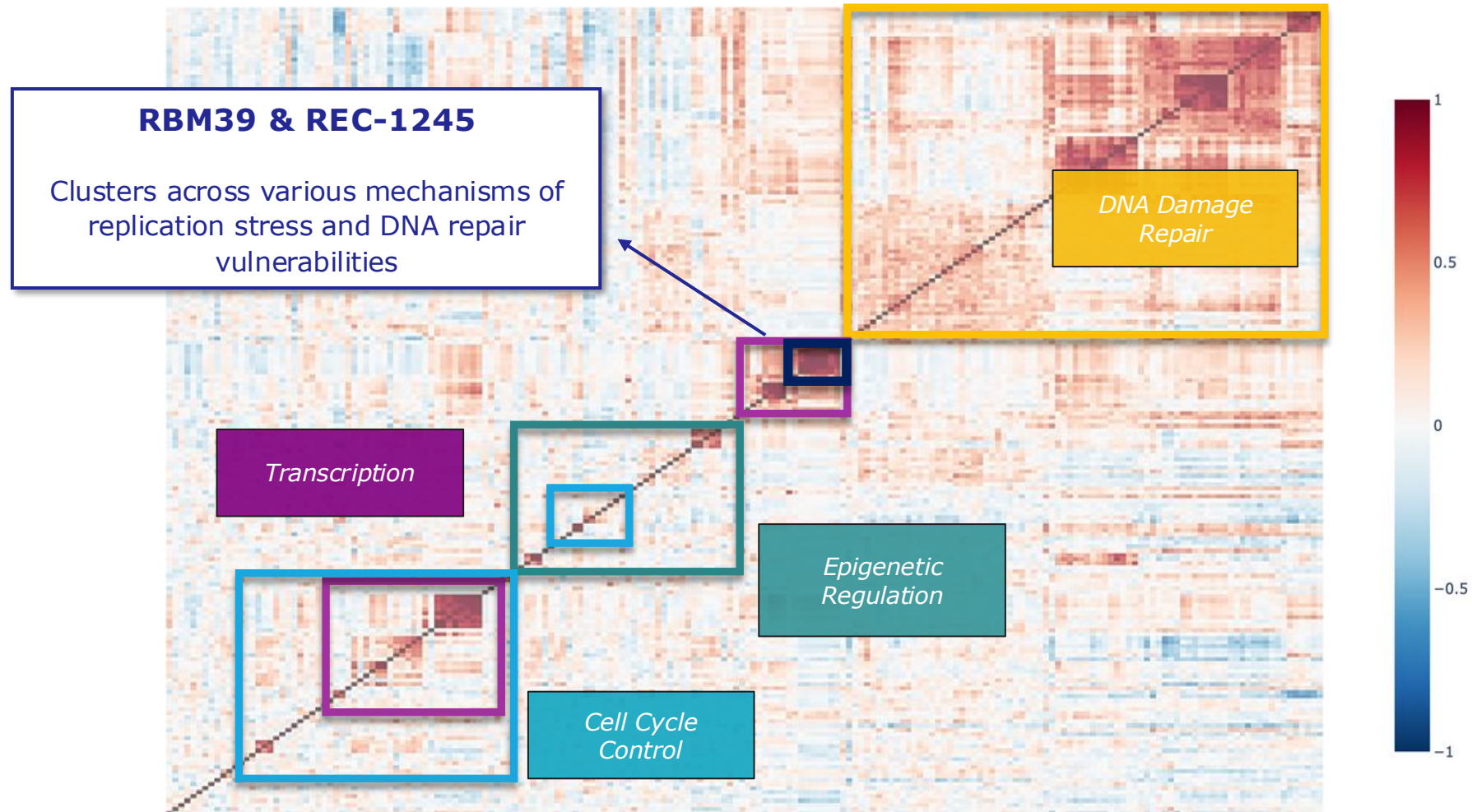
Genetic-Chemical Convergence



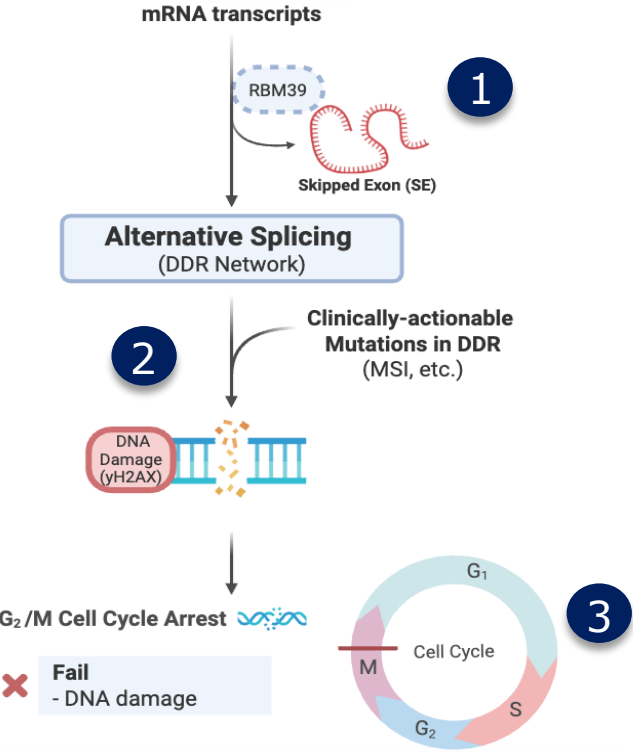
RBM39 loss mimics CDK12 inhibition, providing a druggable potential analog

- Suppresses DDR networks and cell-cycle pathways
- Potential for **tumor-selective** activity with a **wider** therapeutic index

Multi-Omics insights link RBM39 dependency to transcription-replication stress and DNA repair vulnerabilities



Selective degradation of RBM39 impairs splicing, compromising DDR pathways and transcriptional regulation



1. RBM39 Degradation

- **REC-1245** recruits **RBM39** & **DCAF15** for rapid proteasomal degradation
- Loss of this splice adaptor destabilizes the spliceosome

2. DDR Gene Silencing

- Long repair transcripts (*BRCA1/2*, *ATR*, etc.) retain introns and degrade
- Fork protection fails, marked by rising **γH2AX** and stalled replication

3. Mitotic Collapse

- Compromised **G2/M checkpoints** push cells into mitosis without repair
- Resulting chromosomal breakage drives **tumor cell death**

RBM39 degradation recapitulates **CDK12 loss**, potentially enabling a **synthetic-lethal opportunity** in genomically unstable cancers

Target tumors characterized by drivers of genomic instability arising from replication stress potentially linked to RBM39 dependency

Broad patient subsets with replication stress and DNA repair vulnerabilities¹

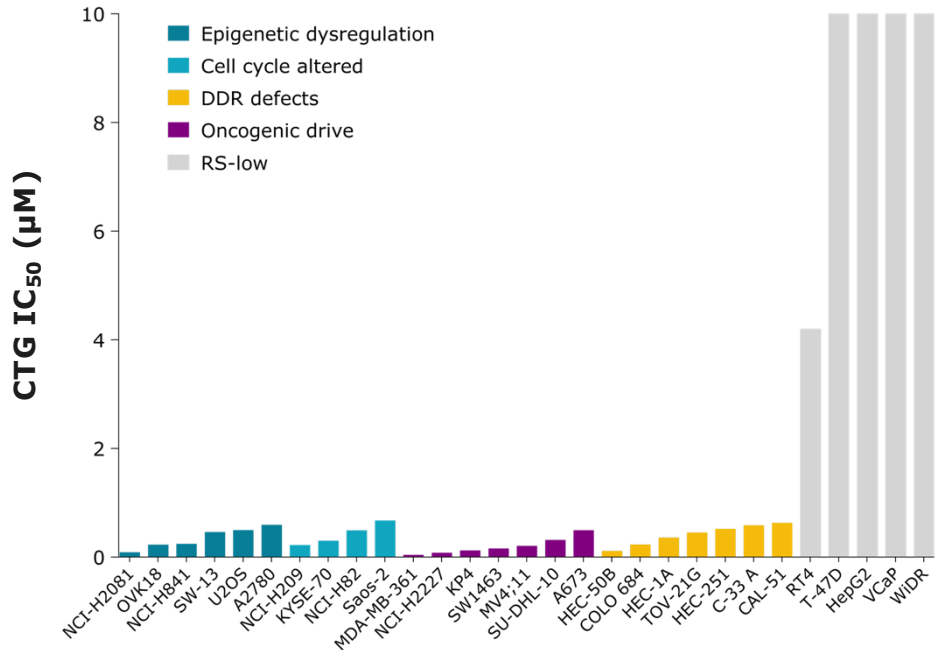


>100,000 patients in US & EU5 per year²

Replication stress and DNA repair vulnerabilities span several solid tumors and blood cancers

- Including colorectal, endometrial, lung, breast, sarcoma, bladder, ovarian, pancreatic, DLBCL and AML
- Correlates with clinically actionable alterations
 - E.g., MYC/MYCN amplification, SWI/SNF loss, MSI-H, HRR defects
- Potential to meaningfully expand beyond initial patient population

In vitro: cell viability with REC-1245³



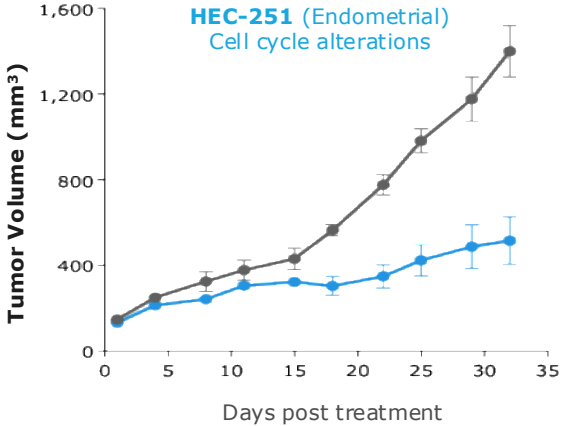
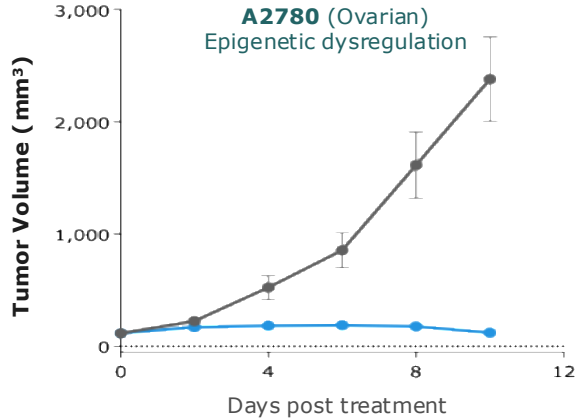
Emerging preclinical data uncover replication stress as a potential signature for REC-1245 sensitivity

- REC-1245 also demonstrates concentration-dependent increases in γH2AX in high replication stress driven human cancer cells *in vitro* (data not shown)

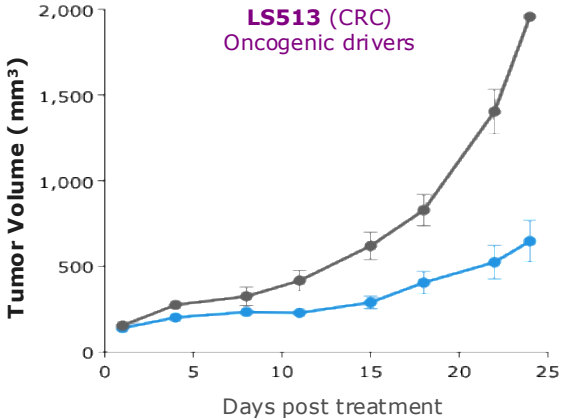
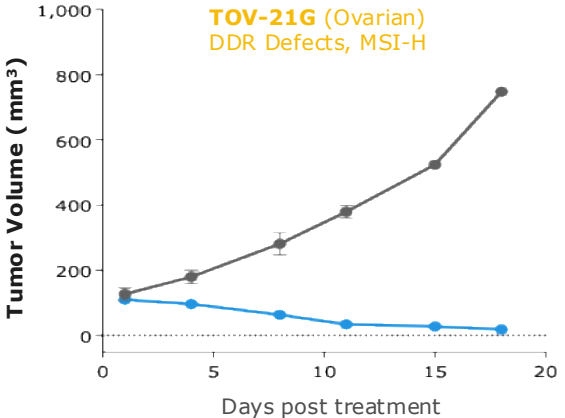
1. da Costa 2022, NRDD
 2. Internal company estimates. Assumes US+EU5 addressable incidence with biomarker-enriched solid tumors and other select histologies.
 3. Cell lines were assigned to broad pathway dysregulation contexts based on 1 or more documented alteration from CCLE/GSDC databases

Emerging preclinical data suggests greater sensitivity with REC-1245 in high replication stress and DDR vulnerable tumors

REC-1245 demonstrates reduction in tumor volume across different tumors with high-RS signatures and DNA repair vulnerability¹



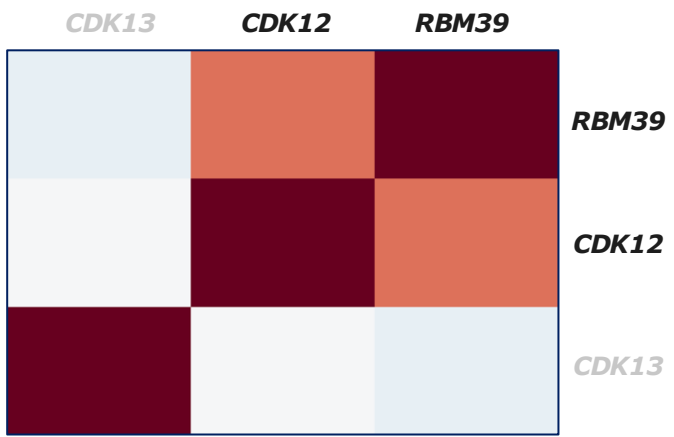
● Vehicle BID
● REC-1245 10 mg/kg BID



1. N=4 mice per group.

Platform derived insight to unlocking comprehensive genomic instability vulnerabilities

Recursion OS Insight

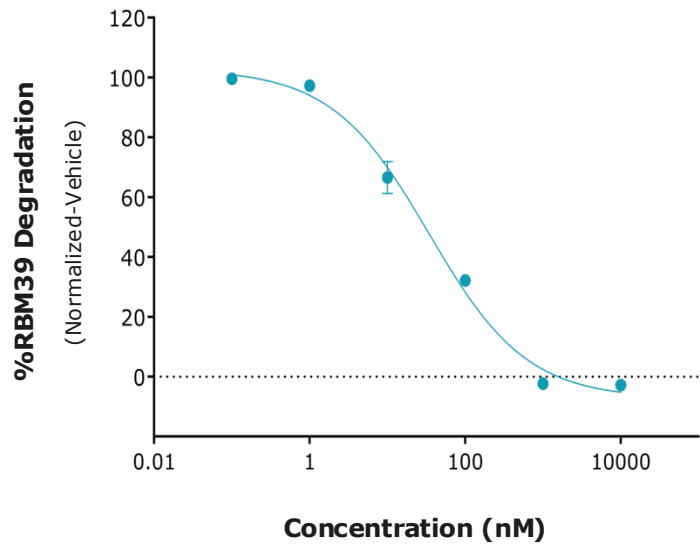


RBM39 loss mimics CDK12 deficiency

- 204 candidates synthesized to candidate ID (REC-1245)
- Advanced program from target ID to IND-enabling studies in 18 months

Mechanistic Validation

RBM39 Degradation

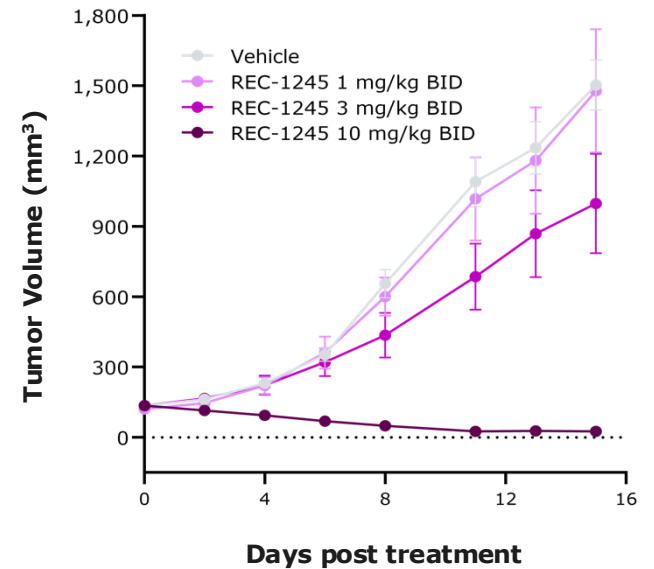


REC-1245 translates phenotypic insights

- Driving rapid and potent RBM39 degradation in human PBMCs within 24 hours

Preclinical Data

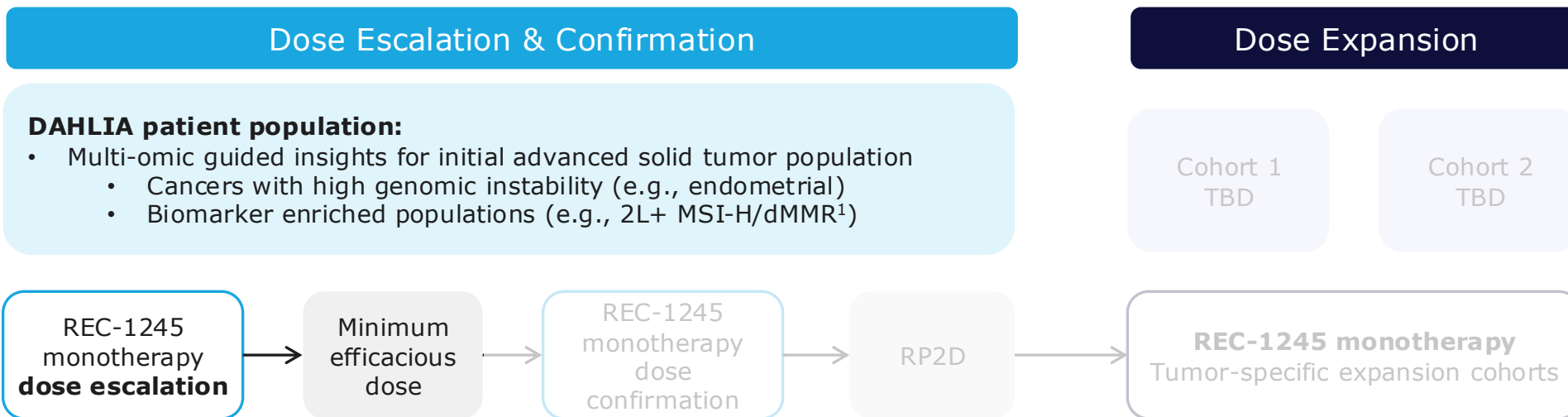
Ovarian CDX Model: OVK18 (MSI-H)



REC-1245 induces significant tumor regressions in an ovarian CDX

- Model driven by elevated replication stress

Phase 1/2 monotherapy dose escalation ongoing with early safety/PK data anticipated 1H26



Clinical Updates

Monotherapy data update expected in **1H26**

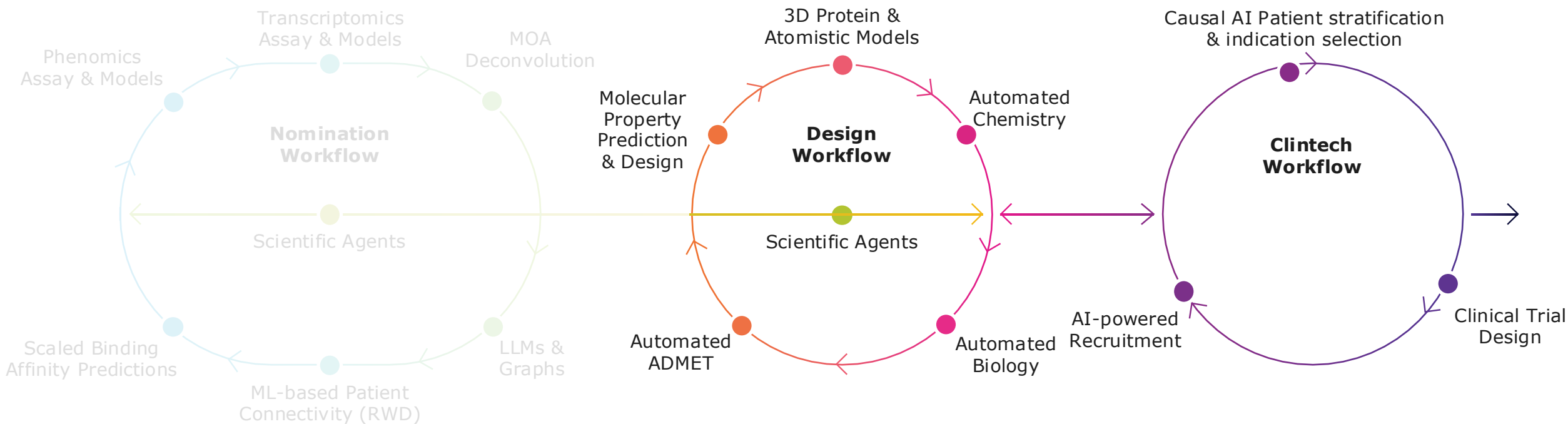
- First patient dosed in 4Q24
- Early safety and PK from monotherapy trial in 1H26

REC-617 (CDK7 inhibitor): Precision design for optimal therapeutic index and causal AI for indication validation

AI-powered Biological Insights

AI-enabled Precision Design

AI-informed Clinical Development



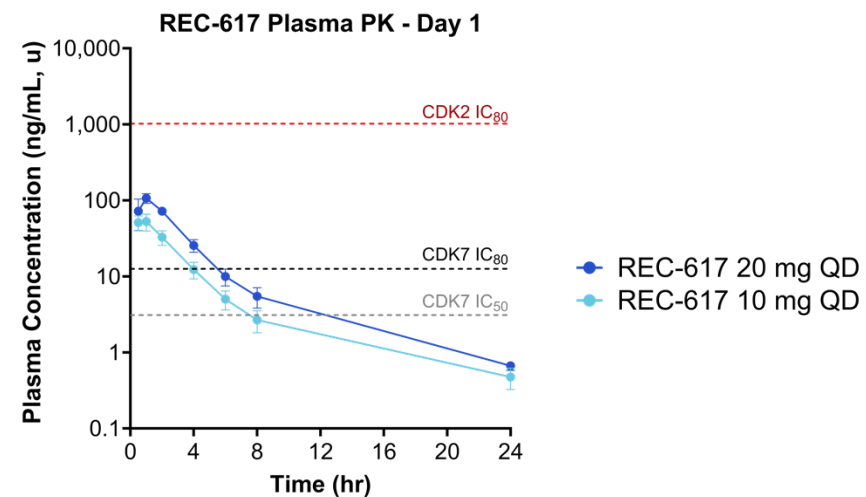
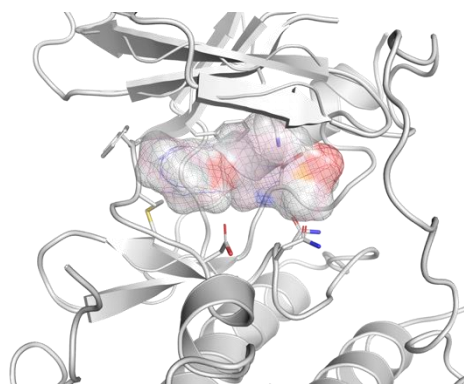
AI-guided precision design for optimized therapeutic index

High permeability, rapid absorption, low efflux

Designed to **optimize PK/PD** and **maximize potential therapeutic index**

REC-617 achieves peak **exposures** in the clinic **exceeding** CDK7 IC₈₀ and well **below** CDK2 IC₈₀

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



- Generative AI models used to design novel scaffolds
- Active learning and experimental data leveraged rapidly to optimize ADME
- **136 novel compounds** to Candidate ID in **<12 months**

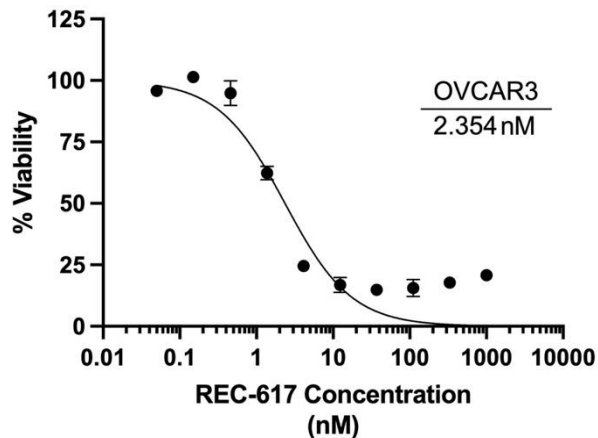
- AI-designed molecule achieves rapid, potent CDK7 inhibition

As of the November 2024 data cut, REC-617 showed one confirmed partial response and multiple cases of stable disease, with a favorable safety profile and no maximum tolerated dose reached

AI-enabled causal inference strengthens preclinical data for indication selection of ovarian cancer for ELUCIDATE

Cell Panels

Cell Line: OVCAR3

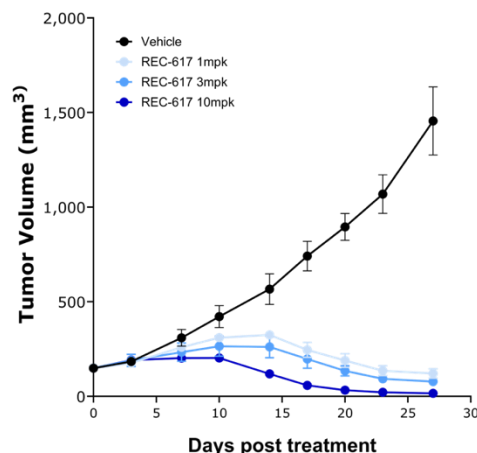


Ovarian cell line sensitive to CDK7 inhibition with REC-617

- Unbiased analysis of over 360 cell lines in glo titer assay

In vivo Models

CDX Model: OVCAR¹

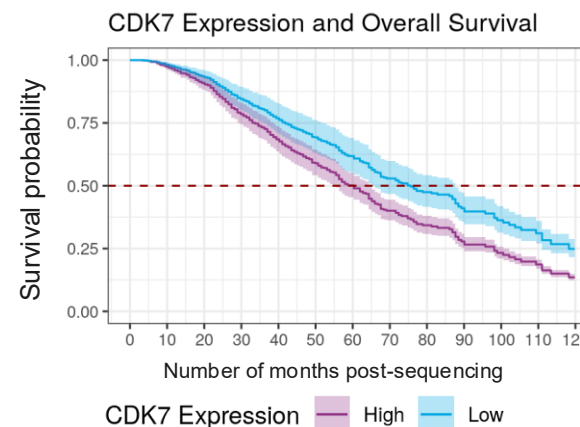


Potent tumor regression with REC-617 treatment

- <10 hours of exposure above IC80 to optimize benefit-risk

Causal Inference using Omics and Clinical data

Patient Data: Ovarian Cancer²



CDK7 emerges as a likely driver of poor survival in ovarian cancer based on a causal inference framework leveraging multi-omic and clinical data

- Over ~32K patient records using DNA, RNA and clinical outcomes

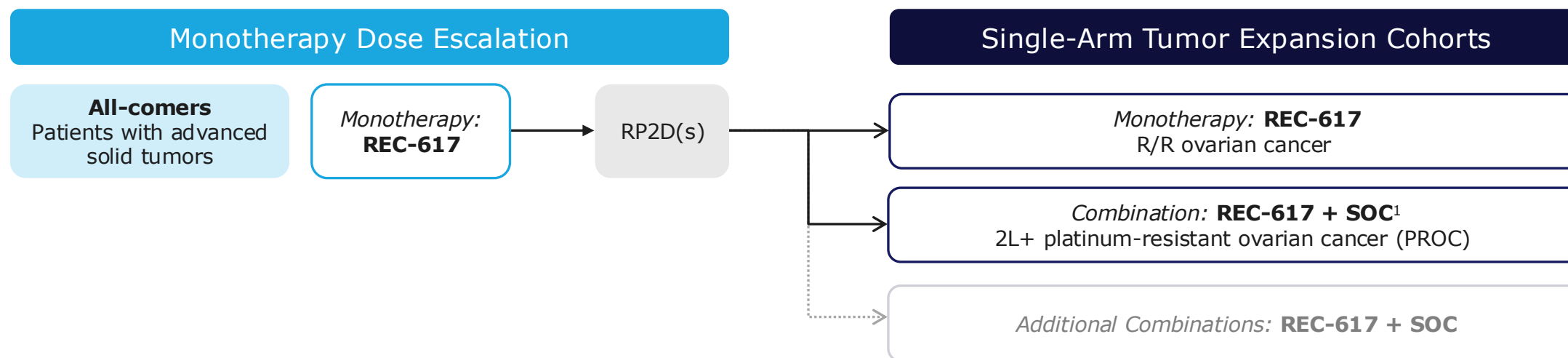
Impact

- Supports preclinical findings with **causal inference using omics and patient data**
- **1st indication:** 2L+ platinum-resistant ovarian cancer (PROC)

1. Besnard et al, AACR (2022)

2. Causal inference framework based on a network-informed directed acyclic graph (DAG) to assess CDK7's impact on clinical outcomes. Patients were indexed on their date of NGS sequencing and followed until death or censoring with 10 + years of patient follow available. The model adjusts for relevant clinical and genomic confounders, including BRCA status, treatment history, and tumor genomics.

Phase 1/2 single-arm combination in ovarian cancer initiated 1H25



Clinical Updates

Monotherapy data update expected in 2H25

- Early data indicates **favorable safety profile**
- Maximum Tolerated Dose (MTD) **not yet reached**

✓ Combination escalation initiated 1H25

- **2L PROC** selected as first cohort
- Site selection & activation in process

Partnered Discovery

Delivering on differentiated programs

Advancing partnership discovery by leveraging Recursion 2.0



4 Program milestone payments achieved in last 18 months



Several programs **advancing towards development candidate** over next 12-15 months



Recursion & Bayer have **nominated multiple early discovery precision oncology programs** against previously “undruggable” targets



Genentech
A Member of the Roche Group

5 Phenomaps in Neuroscience, GI Oncology

- 1 trillion iPSC-derived cells
- 100 billion GI Oncology relevant cells
- 5,000 transcriptomes



Continued **program advancement** in a GI oncology indication and multiple neuroscience **programs into target validation** advanced by leveraging the RecursionOS and Genentech's biology expertise

Merck KGaA
Darmstadt, Germany

Multiple-year collaboration to **identify first-in-class and best-in-class** targets

Potential for over **\$100 million** in partnership milestones by end of 2026

Financial Update

Well positioned to deliver on upcoming milestones

2Q25 Highlights

Cash, cash equivalents and restricted cash

- \$533.8 million in cash¹ as of June 30, 2025

Streamlining of operations and infrastructure

- Expected to reduce pro forma operating expenses by ~35% from 2024 to 2026²

Operational cash inflows

- \$7 million Sanofi milestone payment
- \$28.6 million R&D tax credit

2025 & 2026

Guidance

- Potential for over \$100 million in partnership inflows by end of 2026³
- Expected 2025 cash burn⁴ of <\$450 million
- Expected 2026 cash burn⁴ of <\$390 million

Expected cash runway into the fourth quarter of 2027

1. Cash, cash equivalents and restricted cash

2. YE2024 reported OpEx for Recursion and Exscientia combined, excluding non-cash GAAP items (e.g. share-based compensation). 2026 estimate of <\$390 million cash burn

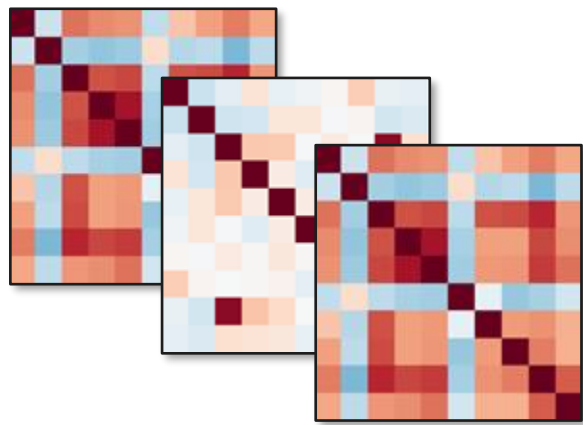
3. Risk-adjusted cash inflows from partnerships included in estimated cash runway

4. Cash burn, defined as operating cash flow less capital expenditures, excluding partnership and financing inflows, transaction expenses and severance

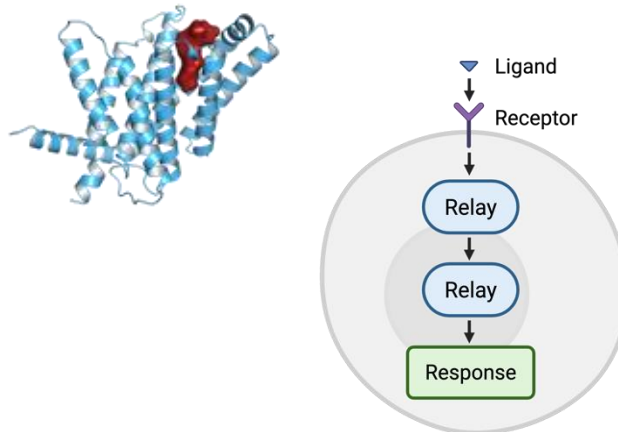
Looking ahead

Continuing our evolution to deliver differentiated, high-confidence programs

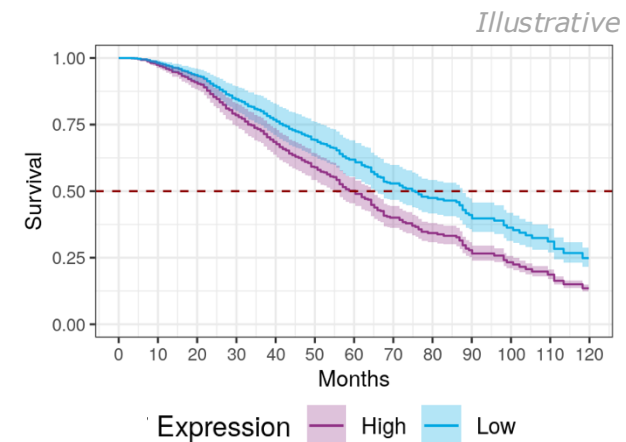
Unique biologic insights identified from **maps with different cell types**



MoA and Target deconvolution using Boltz-2, QM/MD, & CRISPR screens



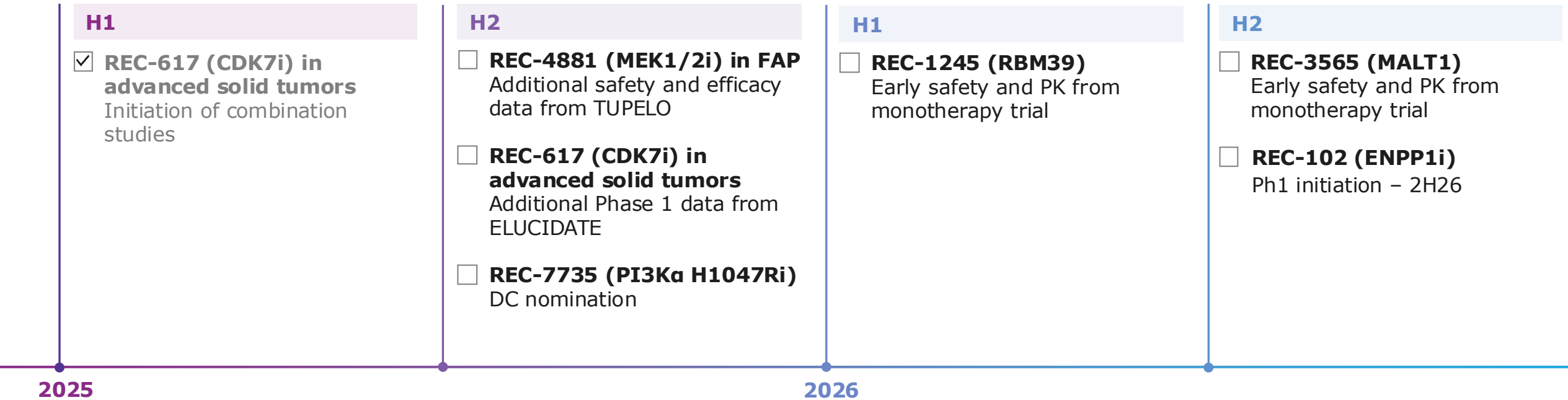
Patient strategy supported by **translational models & RWE**



Differentiated, high quality programs with novel pathway biology, chemistry design, and competitive positioning **emerging from evolving Recursion 2.0 platform**

Internal and external pipeline momentum

FY 2025 and 2026 upcoming pipeline catalysts



Partnership Catalysts

- Potential for **additional phenomap options**
- Potential for **multiple new project initiations**
- Potential for **multiple programs optioned** by partners





Recursion.

Q & A