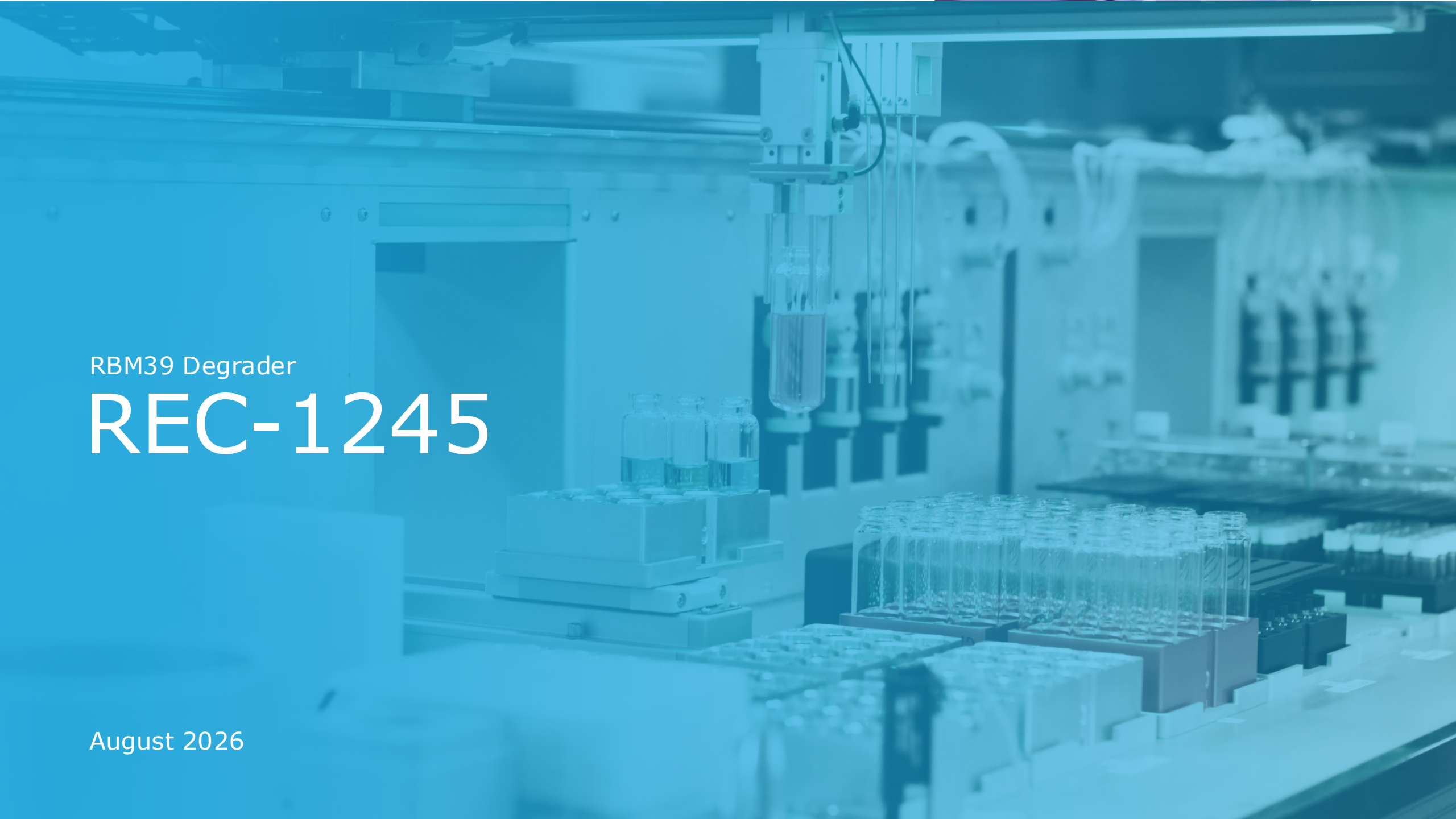


RBM39 Degrader

REC-1245

August 2026

RBM39: A broad opportunity in hard-to-treat cancers

Genomically unstable tumors are hard-to-drug, but depend on stress pathways to survive

- Genomic instability promotes tumor growth through multiple cellular pathways
- Often driven by transcription factors (e.g., MYCN) that lack a binding site for conventional inhibitors

*To survive, these **tumors become dependent on stress-response pathways**, a shared vulnerability across diverse cancers*

Product engine identified RBM39 as a target to exploit this vulnerability

- Anchored target ID on CDK12, given its role in transcription-associated DNA damage response
- Phenomics platform identified RBM39 degradation as phenocopying CDK12 loss
- RBM39 overexpression associated with worse survival in multiple solid tumors[1]

*RBM39 degradation induces splicing stress, **lethal in tumors already under high replication and transcriptional stress***

>100k

Addressable patients (US + EU5)

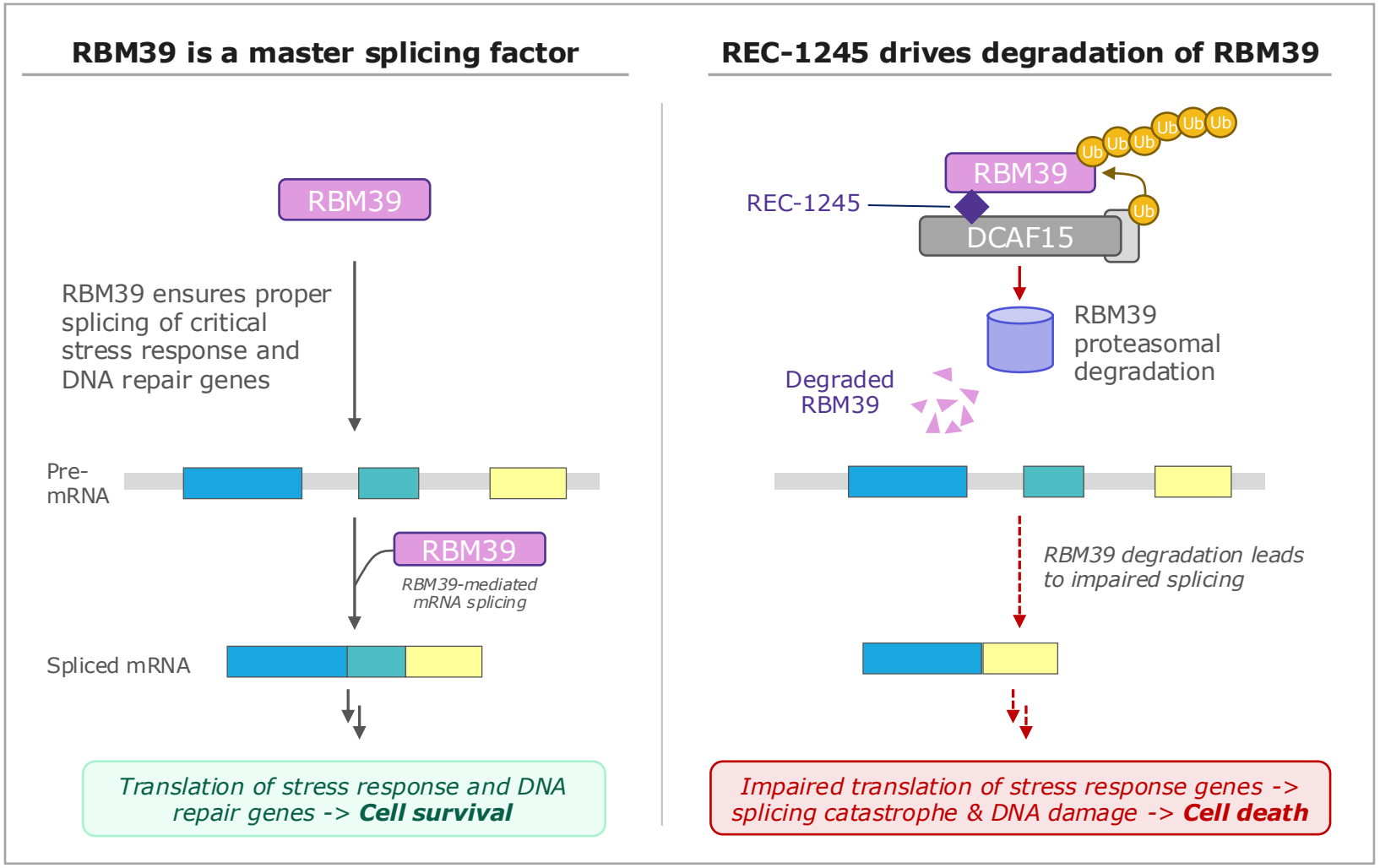
RBM39 inhibition relevant across a broad, defined patient population

- MSI-H/dMMR/TMB-H tumors *(post-checkpoint)*
- Genomically unstable solid tumors *(ovarian, endometrial, breast, pancreatic, colorectal)*
- Transcription factor–driven pediatric cancers *(Ewing sarcoma, neuroblastoma)*
- Advanced liver cancer *(HCC)*

1. Colorectal cancer, Hepatocellular Carcinoma (HCC), and Sarcoma

Source: Zhang, Rui et al. "Systematic pan-cancer analysis identifies RBM39 as an immunological and prognostic biomarker." Journal of cellular and molecular medicine vol. 26,18 (2022): 4859-4871; Wang, Y., Yang, X., Yang, Z., Chen, Z., Jiang, H., Wang, Y., Shen, D., & Su, G. (2025). RBM39 Functions as a Potential Oncogene Through the NF-κB Signaling Pathway in Colorectal Cancer Cells. Journal of Cancer, 16(7), 2233–2249.

REC-1245: A first-in-class RBM39 molecular glue degrader



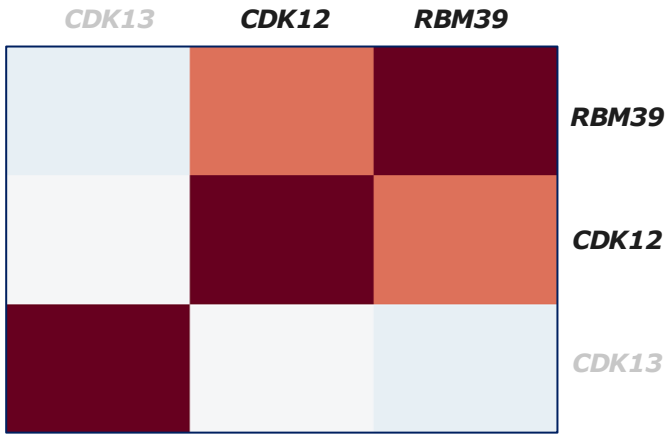
REC-1245 key attributes

- **First-in-class** molecular glue degrader
- Oral, **highly bioavailable**, and **highly selective**
- Hits **multiple oncogenic drivers** achieving synthetic lethal cancer cell killing
- ***Platform-derived biological insight and with candidate in 18 months after screening only 204 compounds***

Recursion

REC-1245: Novel platform derived insight to unlocking comprehensive genomic instability vulnerabilities

Recursion OS Insight

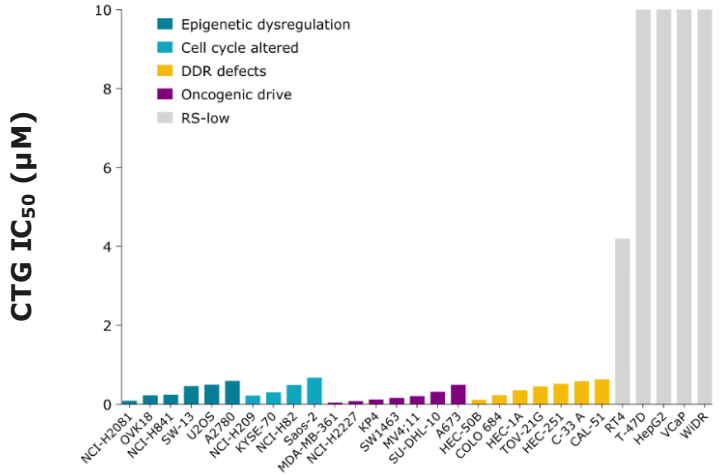


Platform-derived novel target and degrader

- RBM39 loss phenocopies CDK12 deficiency
- RBM39 degrader: advanced from **204 compounds to candidate in 18 months**
- Translates phenomic insight into mechanism, with **rapid and potent RBM39 degradation in human PBMCs within 24 hours**[1]

Preclinical Insight

In vitro: Cell viability with REC-1245[2]

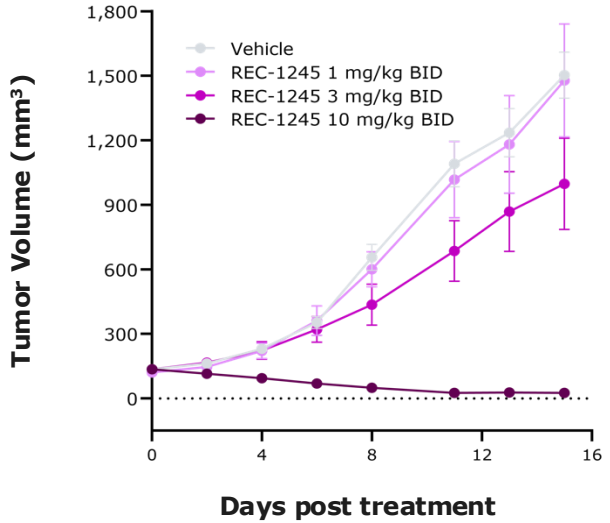


Potential REC-1245 sensitivity based on genomic instability

- Emerging preclinical data uncover **replication stress and DNA repair vulnerability** as potential signatures for REC-1245 sensitivity

Preclinical Data

Ovarian CDX Model: OVK18 (MSI-H)



REC-1245 induces significant tumor regressions in an ovarian CDX

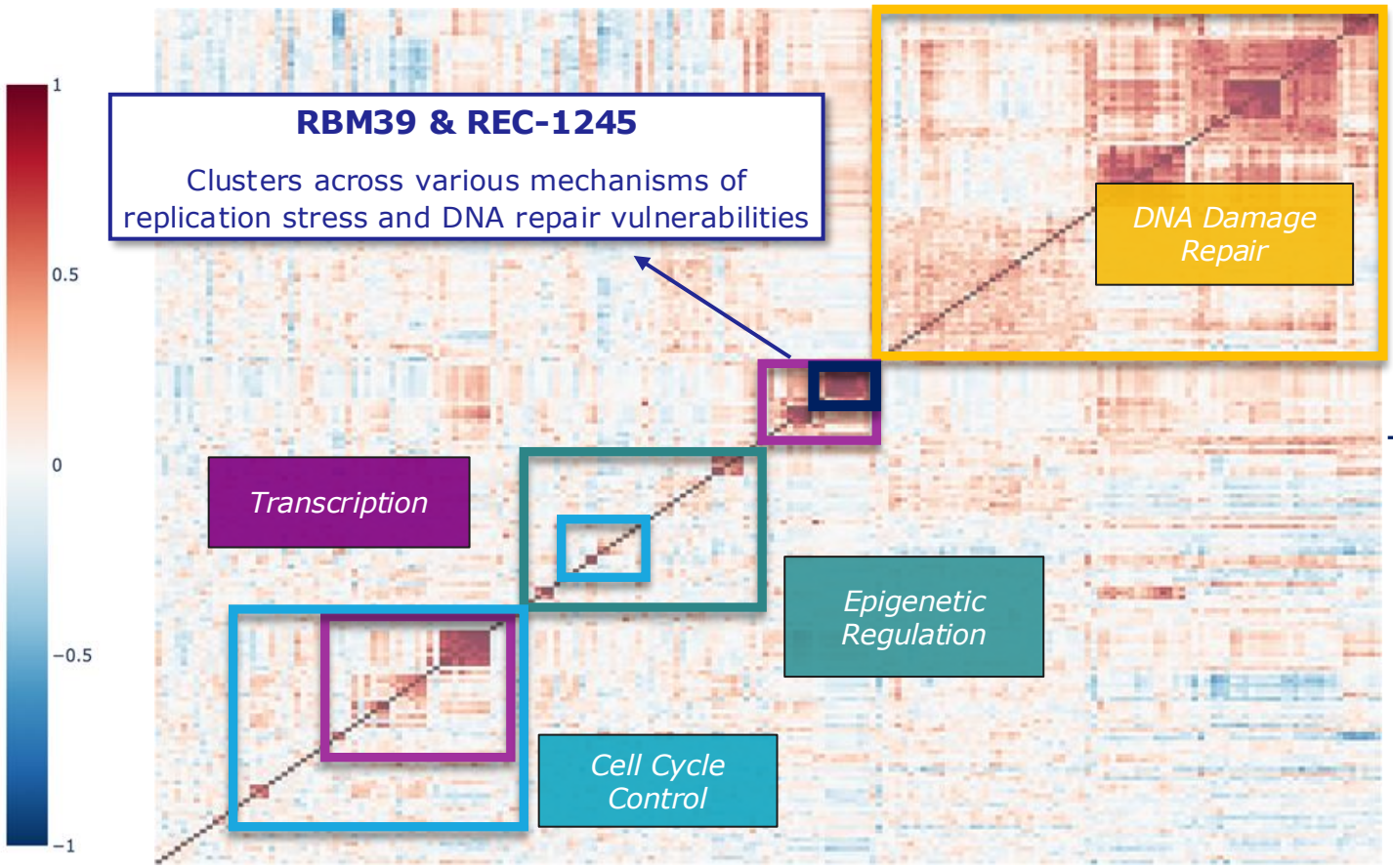
- Model driven by elevated replication stress

Tumor regression replicated across multiple in vivo models with multiple tumor types, demonstrating breadth of response

1. Data not shown.
2. Cell lines were assigned to broad pathway dysregulation contexts based on 1 or more documented alteration from CCLE/GSDC databases

Recursion

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



Biological insight converted into a novel molecular glue degrader in 18 months

- RBM39 degrader: advanced from **204 compounds to candidate in 18 months**
- REC-1245 drives **rapid and potent RBM39 degradation in human PBMCs within 24 hours**[1]
- REC-1245 induces **significant tumor regressions in an ovarian CDX model** driven by elevated replication stress

Recursion

Phase 1 Dose Escalation Update: Early data from monotherapy dose escalation with REC-1245

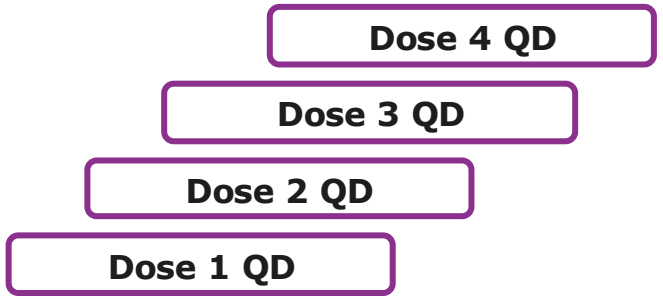
Key inclusion criteria

- **Unresectable, locally recurrent, or metastatic select solid tumors or select relapsed/refractory lymphoma**
- Progressed following, or intolerant to, available SoC treatments

ALL PATIENTS	N=16
Age (median)	**65**
Range	57-77
Advanced solid tumors	**16**
Tumor biomarker	
MSI-H and/or dMMR	7
MSS	9
Prior systemic therapy lines (median)	**4.5**

Ph 1A Monotherapy Dose-Escalation
Continuous once-daily dosing summary

Additional dose levels enrolling



Primary objective
- PK and safety

Secondary objective
- Anti-tumor activity

→ **ClinTech:** Ongoing RWE efficacy contextualization leveraging high-fidelity longitudinal EHR and claims data

Note: Data-cut off date 2026-03-31

Recursion

REC-1245 preliminary safety data: well-tolerated with no DLTs across all evaluated doses as of data cutoff

Treatment-Related Adverse Event (TRAE)	
	Patients (n=16)
Patients with **Any TRAE**	**10 (62.5%)**
Grade 1-2	9 (56.3%)
Grade 3	1 (6.2%)
Grade 4-5	0 (0.0%)

Preliminary safety and tolerability summary

- REC-1245 was **well-tolerated**
- **No DLTs reported** across evaluated doses to date
- **No serious TRAE** was reported
- **90%+ of TRAEs were Grade 1 or 2**
  - Most common GI-related: constipation (12.5%, n=2), nausea (12.5%, n=2), vomiting (12.5%, n=2)
  - Most common non-GI related: fatigue (18.8%, n=3)
- 6.2% (n=1) of patients experienced Grade 3 nausea and vomiting

Note: Data-cut off date 2026-03-31; All data summaries are reported for Safety Evaluable Population

REC-1245: Preliminary PK/PD summary

Early data suggests REC-1245 has **predictable, dose dependent exposure**

- **Predictable, dose-dependent exposure** across evaluated patients to date
- **PK is supportive of QD dosing** and exposures continue to increase with dose
- Expect to **achieve exposures consistent with tumor regression** in mice **within the next two dose levels**
- Pharmacodynamic assessments demonstrate **target engagement**

Note: Data-cut off date 2026-03-31; N=16

REC-1245: First-in-class RBM39 degrader for hard-to-treat, genomically unstable cancers

First-in-class mechanism of action

- First-in-class RBM39 degradation provides a **tractable route into hard-to-drug biology** by disrupting stress-response and DNA-repair splicing
- **Exploits synthetic-lethal vulnerability** in resistant, genomically unstable cancers, with **>100k addressable patients** across biomarker defined populations

Platform-derived insight and molecular design

- **Phenomics identified RBM39 degradation as phenocopying CDK12 loss,** providing a tractable route into hard-to-drug DDR biology
- Advanced from **204 screened compounds to candidate in 18 months**

Phase 1 study ongoing, additional data expected 2H `26

- **Encouraging PK / PD**, with predictable dose-dependent exposure, QD-supportive pharmacokinetics, and evidence of target engagement
- Additional dose levels enrolling, with **additional data expected in 2H26**