remix therapeutics

Modulating RNA Processing

TO TARGET THE UNDRUGGABLE

Investor Presentation

November 2026

NON-CONFIDENTIAL

NASDAQ: RMTX

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Remix Therapeutics: Targeting Disease Through RNA Modulation

Proprietary Drug Discovery Platform

- Founded by and led by an experienced team that has built and commercialized novel biotechnology companies
- Discovery platform designed to address RNA processing and disease drivers at their origin
- Diversified pipeline with potential across several oncology and CNS indications

Differentiated mRNA Degrader

- Lead program REM-422 is a selective, oral small molecule mRNA degrader
- Ongoing Phase 2 ARIA study in patients with recurrent, metastatic or unresectable Adenoid Cystic Carcinoma (ACC)
- Phase 1: 57% ORR at RP2D* with durable responses in MYB biomarker+ patients

*Datacut: 24Aug2026 (Phase 1 only)

Positioned to Deliver on Key Milestones

- Potentially registrational Phase 2 ongoing with ORR and DOR data for REM-422 expected in ~mid-2027
- Potential commercial readiness in 2028
- RP2D and initial data from AML/High-Risk MDS Phase 1 study
- Strong financial position providing runway into 2028 and through several clinical milestones

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REM-422: A De-Risked Oncology Asset at a Critical Inflection Point

- REM-422: The first drug candidate observed to inhibit MYB with meaningful clinical activity in ACC
  - **Adenoid Cystic Carcinoma (ACC):**
    - >1,500 US incidence and 13-15K US prevalence MYB driven cancer with no FDA approved treatments
    - REM-422 Phase 1: durable 57% ORR* in MYB biomarker+ patients, exceeding historic benchmarks[1,2,3]
    - FDA authorized Remix to proceed with an ongoing Phase 2 study with proposed key elements, including: recommended dose, biomarker positive population, and single arm study design for potential registration
    - Potentially registrational Phase 2 study in recurrent, metastatic or unresectable ACC ongoing, **full enrollment expected H2'26**
    - $600M+ US sales opportunity; Remix is preparing for **potential commercial readiness in 2028**
  - **r/r AML and high risk MDS (HR-MDS):**
    - ~25,000 treatable AML/HR-MDS patients in the US
    - REM-422 Ph1 dose escalation ongoing with multiple responses observed
- Late discovery stage MYC program demonstrating tumor regressions in vivo in MYC-dysregulated models
- Funded through ACC Phase 2 readout and potential NDA submission **as early as H2'27**

[1]Tchekmedyian et al., [2]J Clin Oncol. 2019, Locati et al., Cancer, 2020, [3] Laurie SA et al., Lancet Oncol 2011, *based on best overall response

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Experienced management team with deep domain expertise



PETE SMITH

Co-Founder, President & CEO

H3 Biomedicine, Takeda, Millennium



HEATHER WASSERMAN

Chief Business / Operating Officer

Eli Lilly, Human Genome Sciences



MYTHILI KONERU

Chief Medical Officer

Legend, Marker, Eli Lilly



DOM REYNOLDS

Chief Scientific Officer

H3 Biomedicine, Forma, Millennium



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Small molecule pipeline targeting high value oncology targets

Target/Compound	Mechanism	Indications	Pre-clinical	Phase I	Phase II	Phase III	Rights	Anticipated Milestone
MYB/REM-422	mRNA degrader	Adenoid Cystic Carcinoma					remix therapeutics	ORR / DoR Data Mid ’27
		AML/MDS					remix therapeutics	RP2D Data Mid '27
		Others (e.g. BRCA, lymphoma, CRC)					remix therapeutics	
Oncology program	mRNA degrader	MYC-dependent cancers (~25% of all cancers)					remix therapeutics	



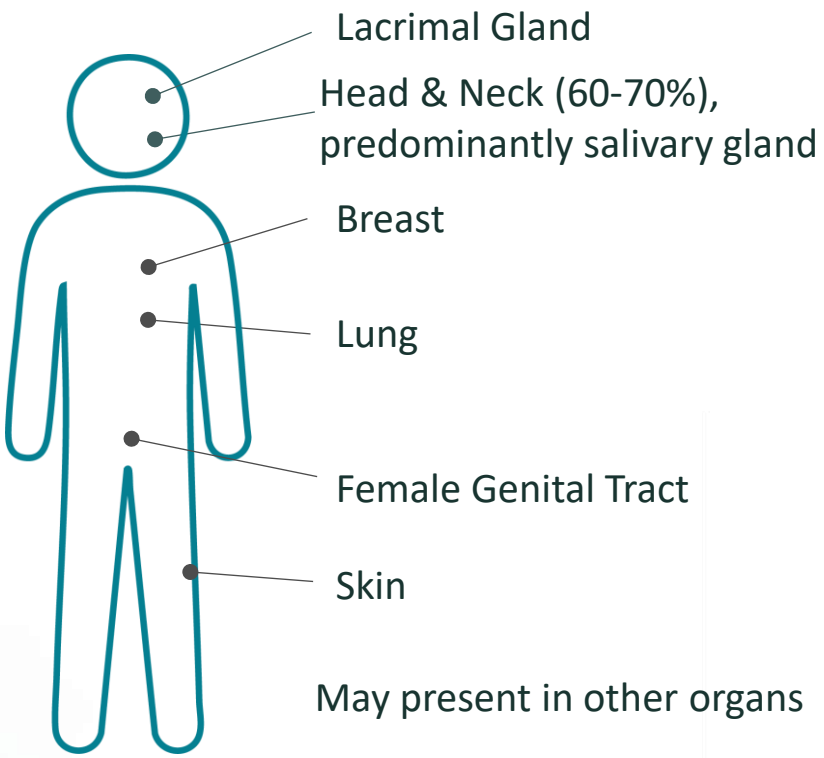
Roche

- Multi-target discovery collaboration across multiple therapeutic areas
- $30M upfront payment and $20M in milestone payments received
- Eligible for >$1B preclinical, clinical, commercial and sales milestone payments and tiered royalties

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Adenoid Cystic Carcinoma has a high unmet medical need

ACC Sites by Organ[5]



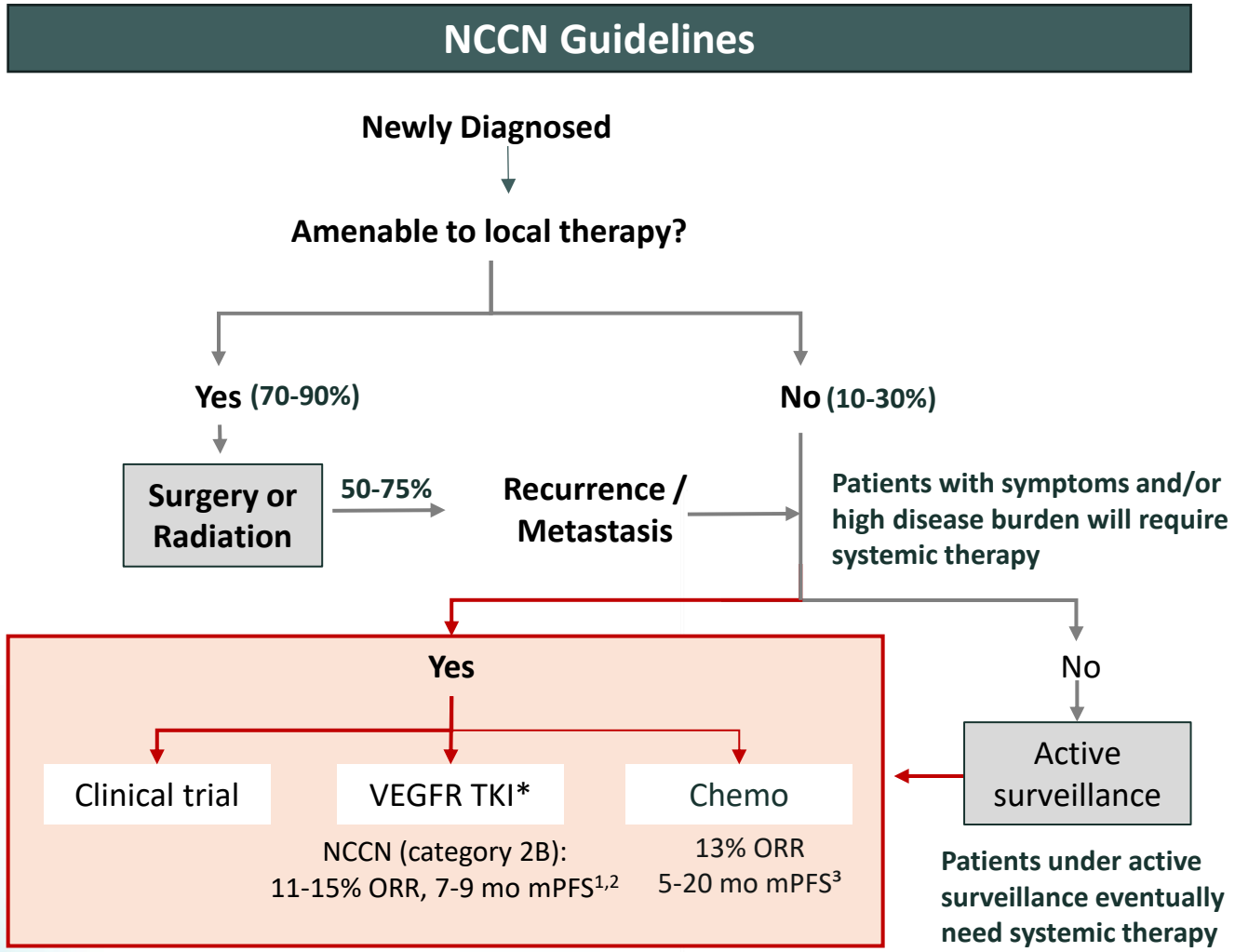
- Malignant epithelial tumor arising predominantly **in salivary glands** and other glandular tissues; high rates of perineural invasion and **relentless growth/metastasis**
- **1,500+ new patients**[1] per year in the US; prevalence is ~13K – 15K[2]
- MYB a key driver of disease in all subtypes, with **60-65% of ACC population MYB biomarker positive**[3]
- High unmet need, **no FDA-approved treatments**

Critical need for a precision therapy that targets the molecular driver of ACC

[1] Boyle et al., *J Clin Oncol.* 2020; Wang et al., *Cancer Epidemiol*. 2026; Epiphany Partners Inc., *EpiOncology Custom Analysis*. 2026, [2] Remix Market Research, [3] Remix Data on File, [4] Ferraroto et al., 2021, Remix data on file, [5] Li et al., *Cancer.* 2012;118(16):3945-3953

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Current SoC: Up to 75% of ACC patients will require systemic therapy, but only 11-15% ORR with available therapy



High Unmet Need for Targeted Treatment

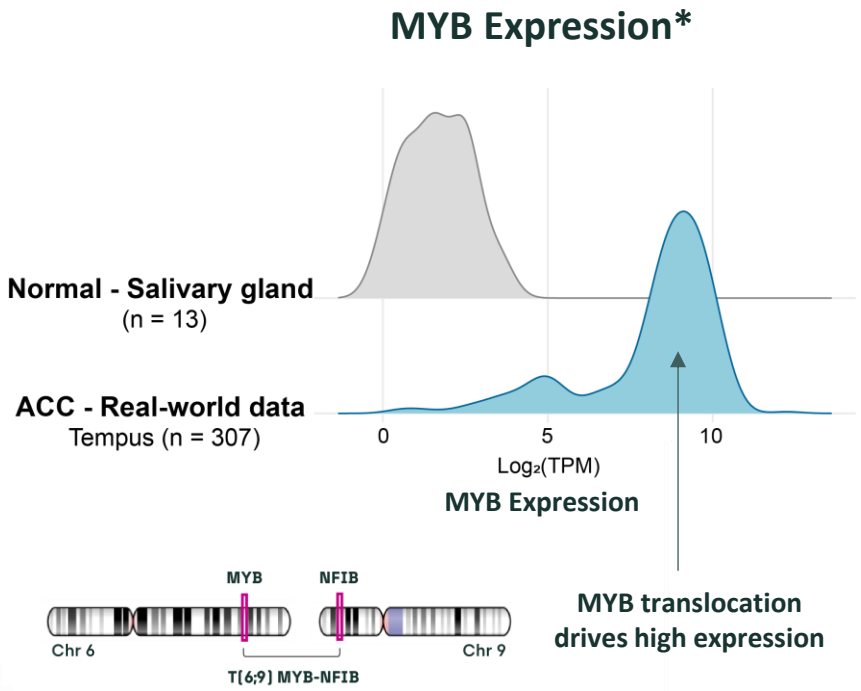
- Surgery standard of care
- Disease will recur in ~50-75%[4] of patients
- Nearly all patients with recurrence will require systemic treatment
- TKIs and chemo offer low response and tolerability challenges

*Lenvatinib, [1]Tchekmedyian et al., [2]J Clin Oncol. 2019, Locati et al., Cancer, 2020, [3] Laurie SA et al., Lancet Oncol 2011 [4]Putnam Associates, Qualitative Research (Q1 2021) & KOL discussions, SoC – Standard of Care

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REM-422 the first MYB inhibitor in clinical development

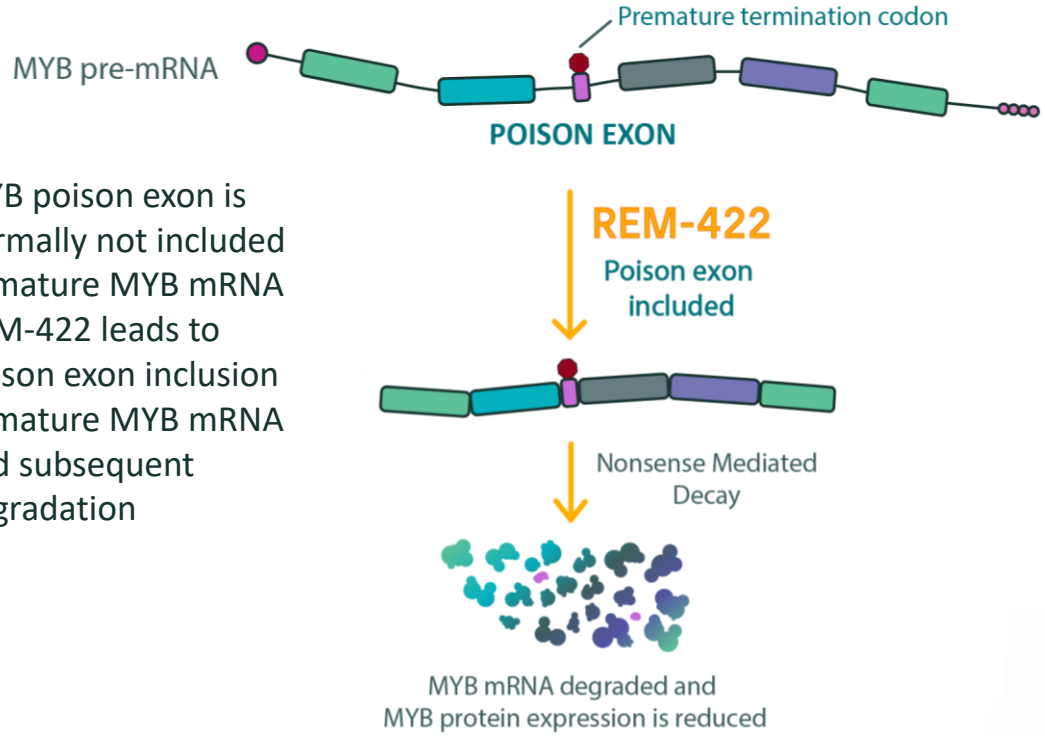
Majority of ACC patients express oncogenic MYB



*Source: Tempus Lens Real-world database

REM-422 designed to induce degradation of MYB mRNA

- MYB poison exon is normally not included in mature MYB mRNA
- REM-422 leads to poison exon inclusion in mature MYB mRNA and subsequent degradation

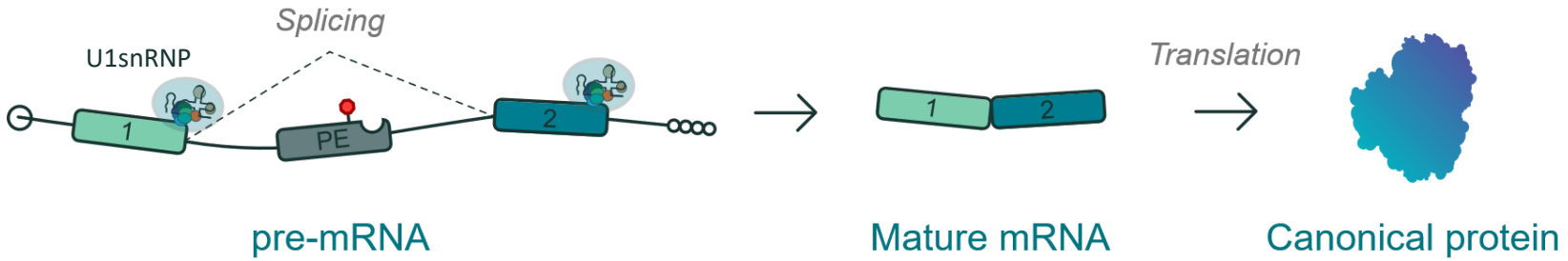


The MYB poison exon (PE) is detectable using an IUO assay in clinical trials

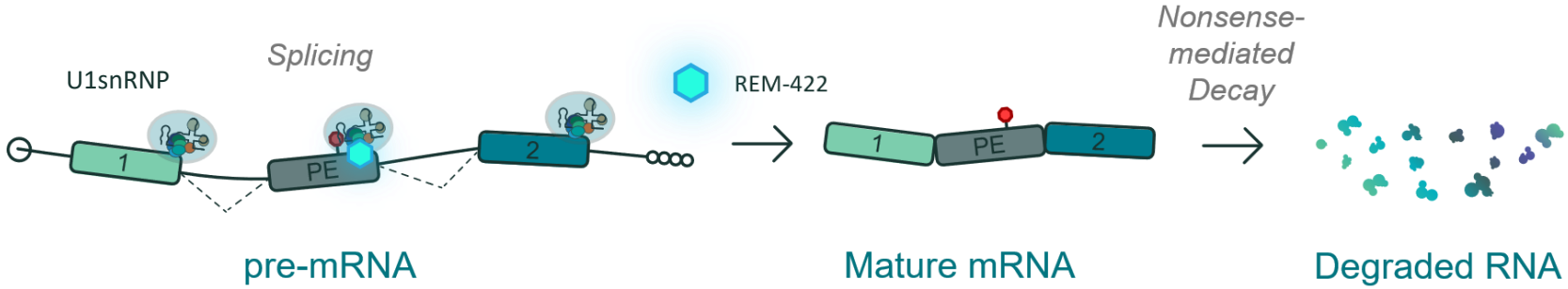
IUO = Investigational Use Only

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REM-422 Mechanism: mRNA degradation through Poison Exon inclusion



Poison exon (PE) excluded from mature mRNA due to unfavorable interaction between the U1snRNP splicing complex and the weak 5’ splice site of the PE



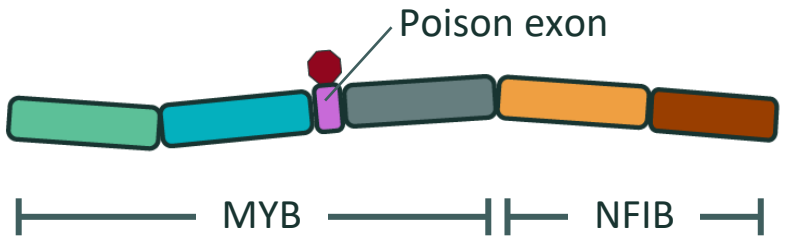
REM-422 acts as a specific small molecule molecular glue that enhances the interaction between the U1snRNP complex and the weak 5’ splice site leading to PE inclusion and subsequent degradation via the NMD pathway

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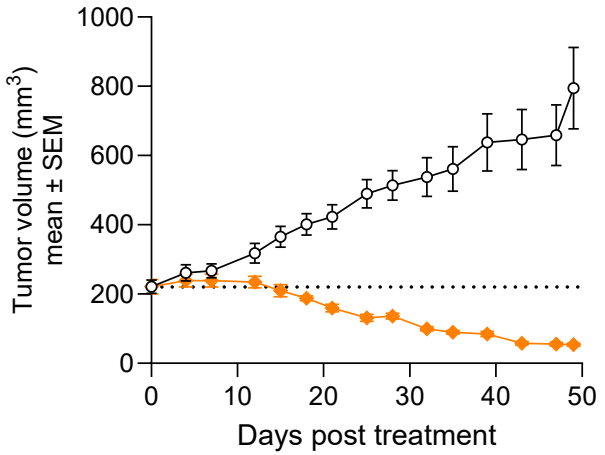
REM-422 demonstrated antitumor activity in biomarker positive ACC PDX models

ACC PDX model

Biomarker/Poison Exon positive

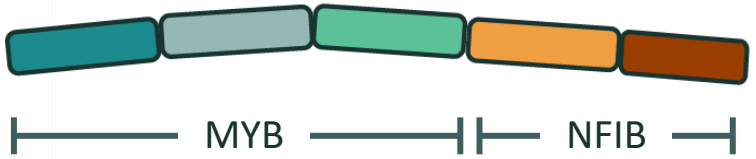


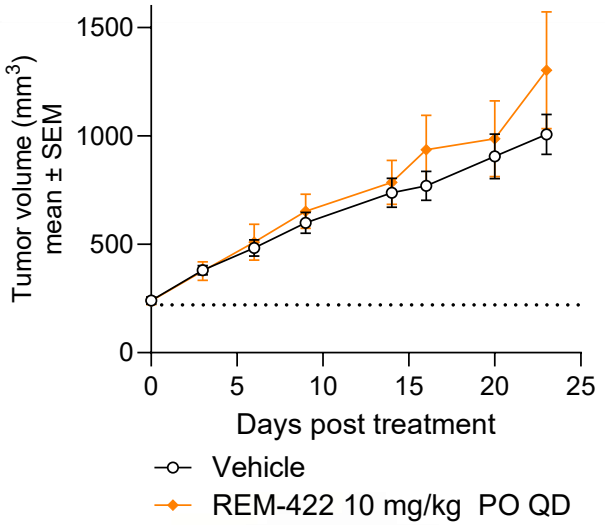
Tumor regressions observed in additional 2 ACC PDX models
Biomarker positive treated with REM-422



ACC PDX model

Biomarker/Poison Exon negative





REM-422 showed selective activity in Biomarker (poison exon) **positive** preclinical PDX models

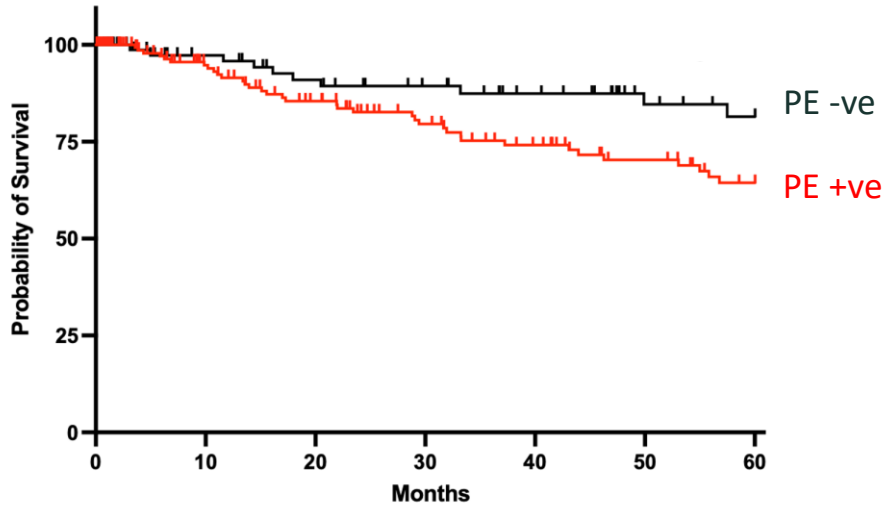
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A Poison Exon in MYB defines the majority of High-Risk ACC Patients

Molecular profiling data from 307 ACC patients



Poor prognosis regardless of ACC subtype



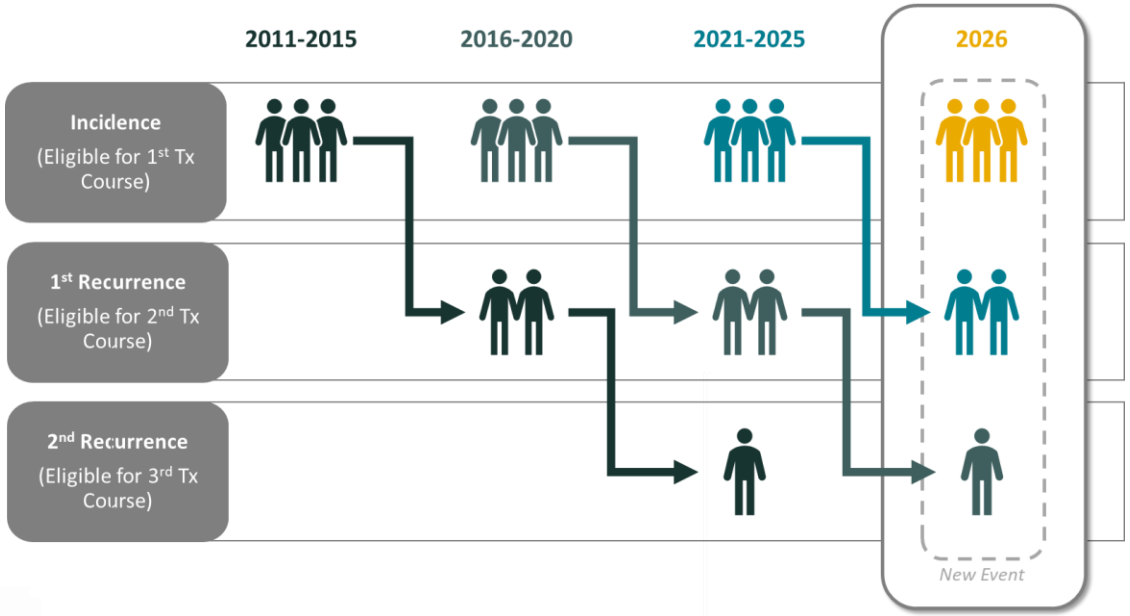
- MYB biomarker (Poison Exon) present in ~60-65% of ACC patients[1]
  - MYB biomarker present in Type I and Type II ACC[1]
- Associated with significantly worse OS[1]
  - Potentially predicts REM-422 sensitivity

[1]Source: Tempus AI and Remix Data on File

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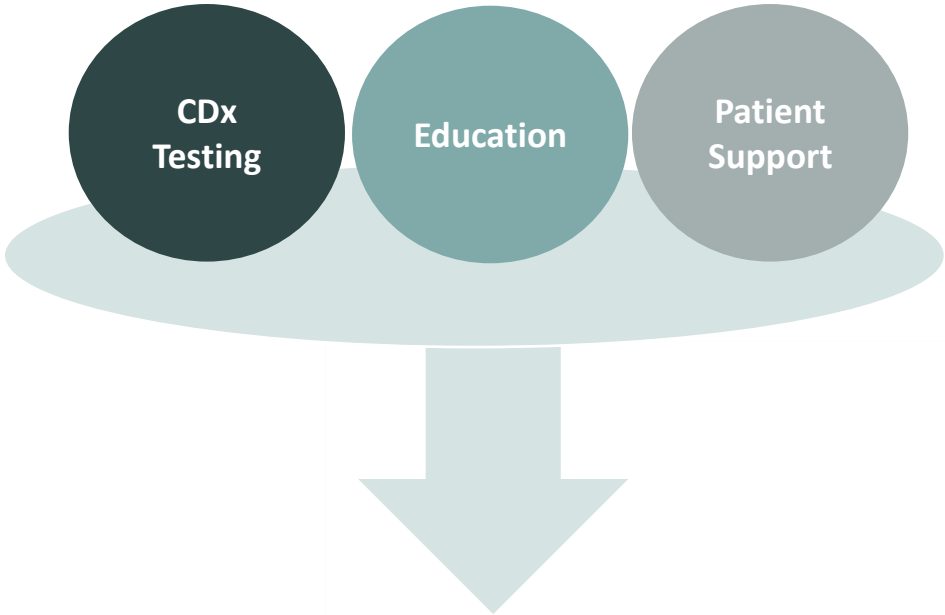
REM-422 offers potential to become the new standard of care for ACC

ACC Patient Flow[1]



- **Over the course of treatment journey, patients may have multiple recurrences**
- **Most patients who recur will eventually require systemic treatment**

Scaling Potential U.S. Commercial Capabilities for REM-422



REM-422 U.S. Sales Estimate: $600M+
Remix is preparing for potential commercial readiness in 2028

[1] Illustrative patient flow model

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REM-422 Phase 1 studies:

- Adenoid Cystic Carcinoma
- AML and High-Risk MDS

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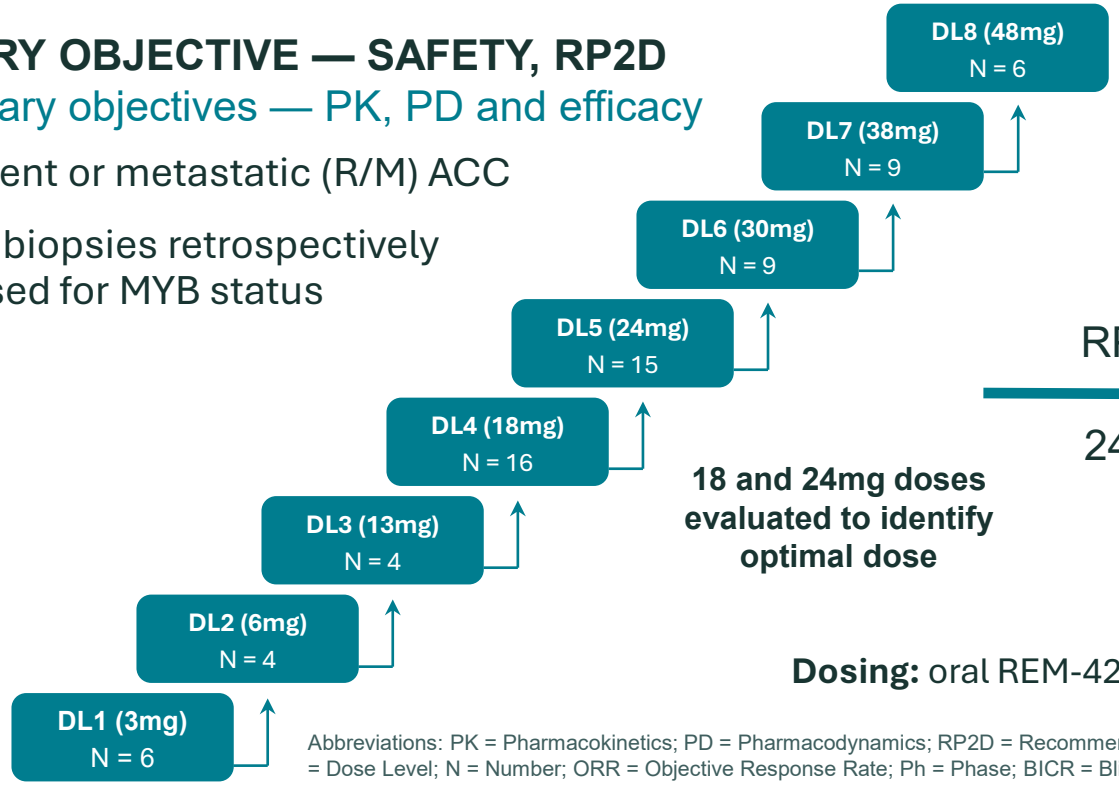
Ph1/2 ARIA (A study of REM-422 In Adenoid Cystic Carcinoma)

Ph1 DOSE ESCALATION (all-comers): N = 69

PRIMARY OBJECTIVE — SAFETY, RP2D

Secondary objectives — PK, PD and efficacy

- Recurrent or metastatic (R/M) ACC
- Tumor biopsies retrospectively assessed for MYB status



18 and 24mg doses evaluated to identify optimal dose

RP2D

24mg

Dosing: oral REM-422 once daily

Ph2 CONFIRMATORY COHORT (PE+): N ~ 50

PRIMARY OBJECTIVE — ORR (BICR)

Key Eligibility:

- R/M, locally advanced unresectable ACC
- RECIST disease progression within 12m
- Biomarker (PE) positive tumor

Abbreviations: PK = Pharmacokinetics; PD = Pharmacodynamics; RP2D = Recommended Phase 2 Dose; R/M = Relapsed/Refractory; ACC = Adenoid Cystic Carcinoma; DL = Dose Level; N = Number; ORR = Objective Response Rate; Ph = Phase; BICR = Blinded Independent Central Review.
Patients that started at dose levels of 30 mg and above were dose reduced to 24 mg

NCT #: NCT06118086

Ho A., et al. 2026

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Demographics and disease characteristics

	N = 69
AGE, MEDIAN (RANGE)	57 (20-82)
SEX	
Female	42 (61%)
Male	27 (39%)
RACE	
White	59 (85%)
Asian	6 (9%)
Not reported	4 (6%)
ECOG*	
0	43 (62%)
1	26 (38%)
PRIOR SYSTEMIC RX	
0	17 (25%)
1	16 (23%)
2+	36 (52%)

	N = 69
PRIMARY SITE	
Salivary	
• Major	40 (58%)
• Minor	14 (20%)
Non-Salivary	
• Trachea/Bronchial/Lung	8 (12%)
• Lacrimal	3 (4%)
• Breast	3 (4%)
• Esophageal	1 (1%)
HISTOLOGY	
Solid/high-grade transformation	11 (16%)
Non-solid	35 (51%)
Unknown	23 (33%)
ACC SUBTYPES&	
I	18 (26%)
II	34 (49%)
Unknown	17 (25%)
BIOMARKER*	
Positive	35 (51%)
Negative	30 (43%)
Unknown	4 (6%)

Datacut: 24Aug2026 (Phase 1 only)

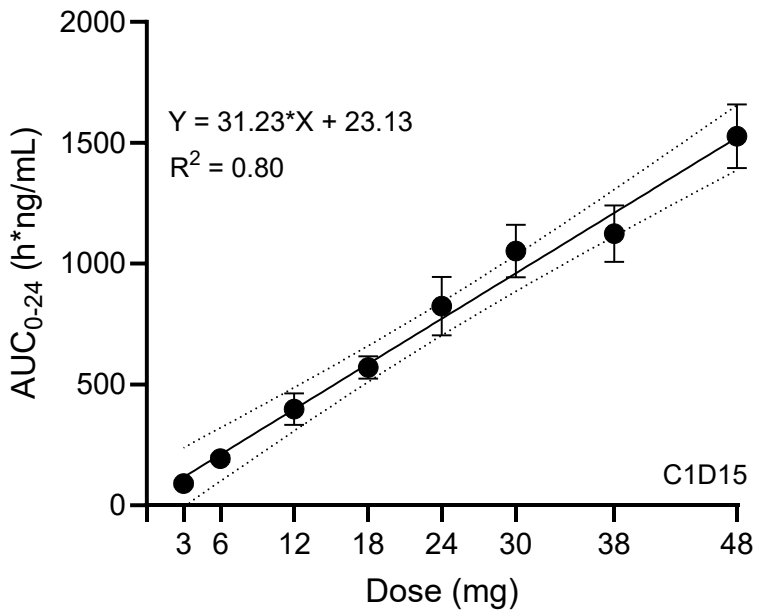
*MYB PE positive + MYB IHC high/PE unknown
&ACC Subtype as defined by Ferrarotto et al., 2021

Abbreviations: ECOG = Eastern Cooperative Oncology Group (Performance Status)

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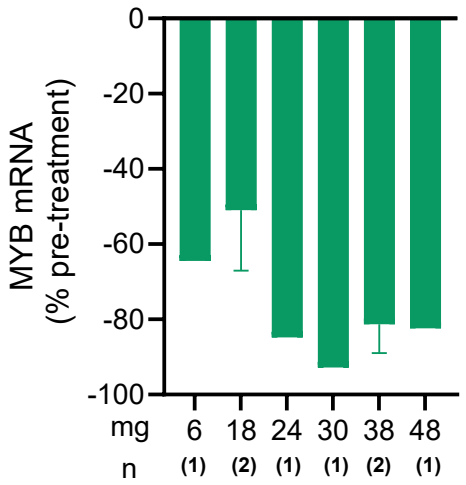
REM-422 PK/PD

REM-422 plasma exposure by dose level

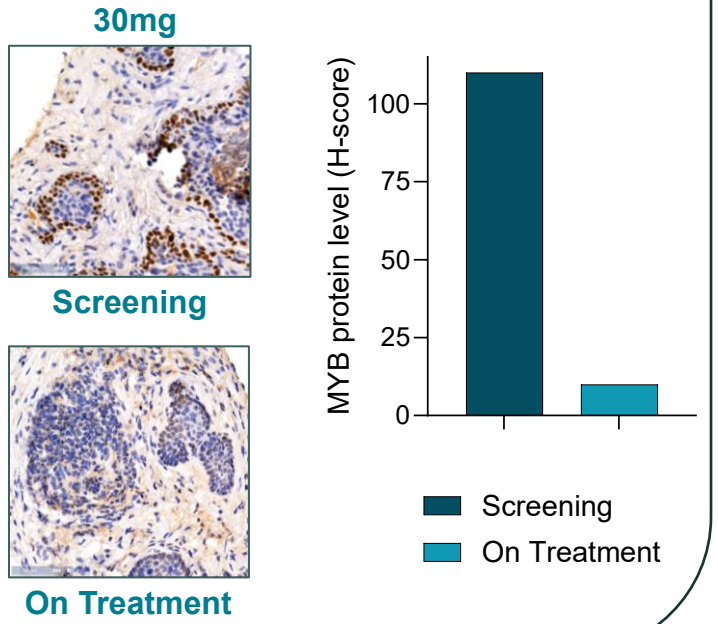


MYB levels in tumor

mRNA



Protein (IHC)



- Observed dose proportional increase in exposures
- Robust target engagement observed in tumor biopsies
- 24mg (RP2D) selected based on PK/PD, efficacy and safety results

Abbreviations: PK = Pharmacokinetics; PD = Pharmacodynamics; RP2D = Recommended Phase 2 Dose

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Overall summary of AEs by starting dose level

	3mg QD N = 6	6mg QD N = 4	12mg QD N = 4	18mg QD N = 16	24mg QD N = 15	30mg QD N = 9	38mg QD N = 9	48mg QD N = 6
TRAE	4 (67)	3 (75)	4 (100)	15 (94)	15 (100)	7 (78)	9 (100)	6 (100)
TRAEs ≥ Grade 3	1 (17)	0	1 (25)	3 (19)	1 (7)	2 (22)	6 (67)	4 (67)
REM-422 related SAEs	0	0	1 (25)	2 (13)	2 (13)	2 (22)	1 (11)	2 (33)
Interruptions due to TRAEs	0	1 (25)	1 (25)	9 (56)	10 (67)	2 (22)	7 (78)	5 (83)
Dose reduction due to TRAEs	0	0	1 (25)	3 (19)	1 (7)	1 (11)	4 (44)	2 (33)
Discontinuations due to TRAEs	0	0	0	1 (6)	0	2 (22)	2 (22)	0

- No DLTs observed at any dose level
- Tolerable Safety Profile particularly at 24mg (RP2D) with long-term dosing for 2+ years and ongoing

Datacut: 24Aug2026 (Phase 1 only)

Notes: TEAEs were reported using Medical Dictionary for Regulatory Activities, version 28.0; Percentages rounded to nearest whole number; # of subjects (%) reported in table
Abbreviations: TEAE = Treatment-Emergent Adverse Event; TRAE = Treatment-Related Adverse Event; SAE = Serious Adverse Event; DLT = Dose-Limiting Toxicity; N = number; RP2D = Recommended Phase 2 Dose

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TRAEs Gr3-4 by preferred term

	3mg QD N = 6	6mg QD N = 4	12mg QD N = 4	18mg QD N = 16	24mg QD N = 15	30mg QD N = 9	38mg QD N = 9	48mg QD N = 6	TOTAL N = 69
Lymphocyte count decreased	1 (17)	0	0	1 (6)	0	1 (11)	2 (22)	2 (33)	7 (10)
Neutrophil count decreased	0	0	0	0	0	0	1 (11)	1 (17)	2 (3)
White blood cell count decreased	0	0	0	0	0	0	0	1 (17)	1 (1)
Aspartate aminotransferase increased	0	0	0	0	0	0	0	1 (17)	1 (1)
Blood alkaline phosphatase increased	0	0	0	0	0	0	0	1 (17)	1 (1)
Anaemia	0	0	0	1 (6)	0	2 (22)	1 (11)	2 (33)	6 (9)
Fatigue	0	0	0	0	0	1 (11)	1 (11)	1 (17)	3 (4)
Face oedema	0	0	0	0	0	0	1 (11)	0	1 (1)
Malaise	0	0	0	0	0	0	1 (11)	0	1 (1)
Peripheral motor neuropathy	0	0	0	1 (6)	1 (7)	1 (11)	0	0	3 (4)
Peripheral sensory neuropathy	0	0	0	0	0	1 (11)	1 (11)	0	2 (3)
Muscular weakness	0	0	0	1 (6)	0	1 (11)	0	0	2 (3)
Acute kidney injury	0	0	0	0	0	0	1 (11)	0	1 (1)
Proteinuria	0	0	0	0	0	0	1 (11)	0	1 (1)
Epistaxis	0	0	1 (25)	0	0	0	0	1 (17)	2 (3)
Decreased appetite	0	0	0	0	0	0	1 (11)	0	1 (1)

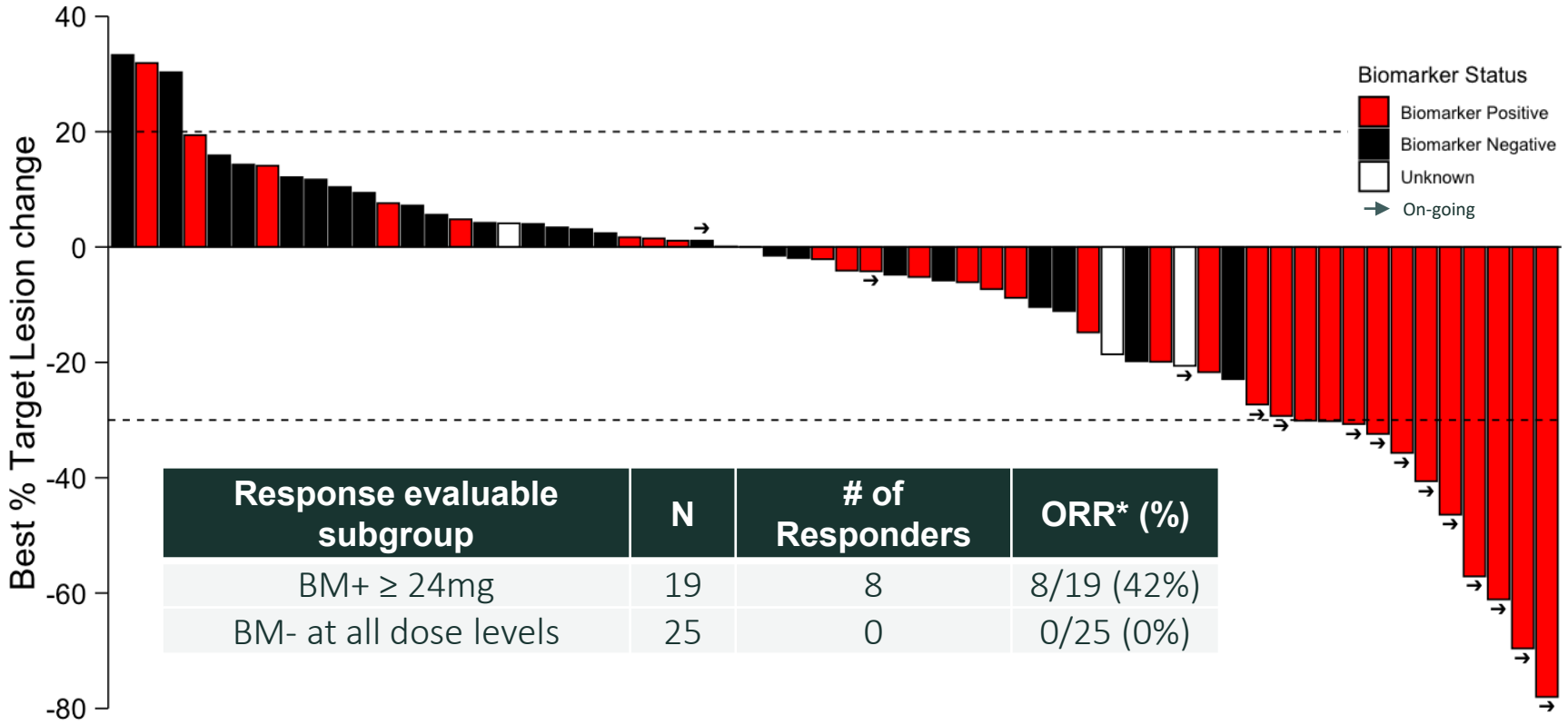
Minimal Gr3-4 TRAEs at 24mg (RP2D) underscores long-term safety and tolerability

Only 2 patients had Gr4 TRAEs: 1) 38mg: neutrophil and lymphocyte counts decreased 2) 48mg: lymphocyte count decreased

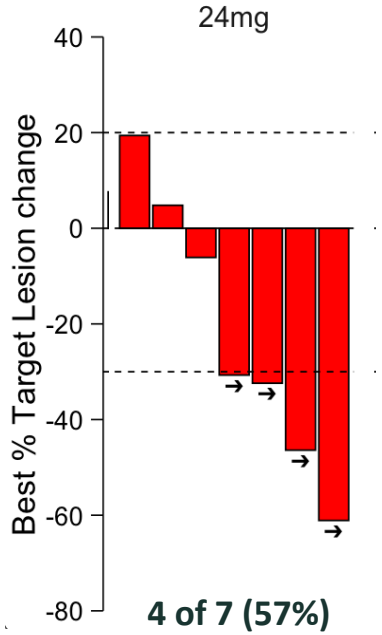
Datacut: 24Aug2026 (Phase 1 only)

Notes: TRAEs were reported using Medical Dictionary for Regulatory Activities, version 28.0; Percentages rounded to nearest number; # of subjects (%) reported in table
Abbreviations: TRAE = Treatment-Related Adverse Event, SOC = Systems Organ Class, QD = once a day; RP2D = Recommended Phase 2 Dose.

Change in tumor size from baseline by biomarker status



Response evaluable subgroup	N	# of Responders	ORR* (%)
BM+ ≥ 24mg	19	8	8/19 (42%)
BM- at all dose levels	25	0	0/25 (0%)



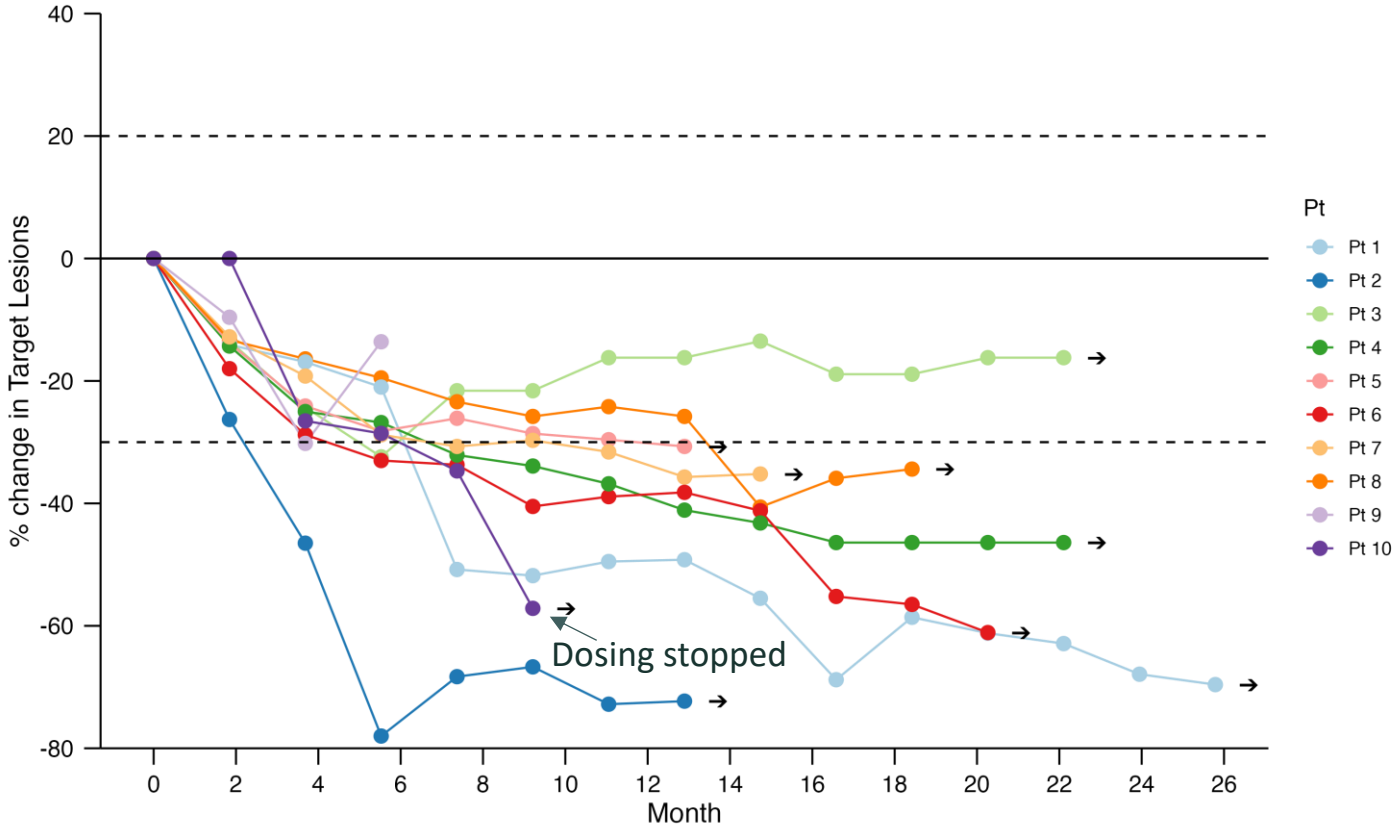
4 of 7 (57%)
BM+ patients at 24 mg
RP2D had response

- Biomarker positive patients: ORR = 57% at RP2D
- Biomarker negative patients: no objective responses across all dose levels

Datacut: 24Aug2026 (Phase 1 only)

Note: Efficacy dataset includes all patients with measurable target lesions at baseline and at least 1 post-treatment scan (N = 60). *ORR includes unconfirmed and confirmed responses

Sustained objective responses with treatment ongoing ~2+ years



Pt ID	Starting dose	Response	Time on Treatment (months)	Duration of Response (months)
1	12mg	cPR	**27+**	**19+**
2	18mg	cPR	**14+**	**9+**
3*	24mg	uPR	**24+**	**2**
4	24mg	cPR	**24+**	**15+**
5	24mg	cPR**	**14+**	**1+**
6	24mg	cPR	**23+**	**16+**
7	38mg	cPR	**16+**	**7+**
8	38mg	cPR	**20+**	**4+**
9	48mg	uPR	**5**	**2**
10***	48mg	cPR	**10+**	**4**

All patients with starting doses ≥30mg reduced to RP2D 24mg

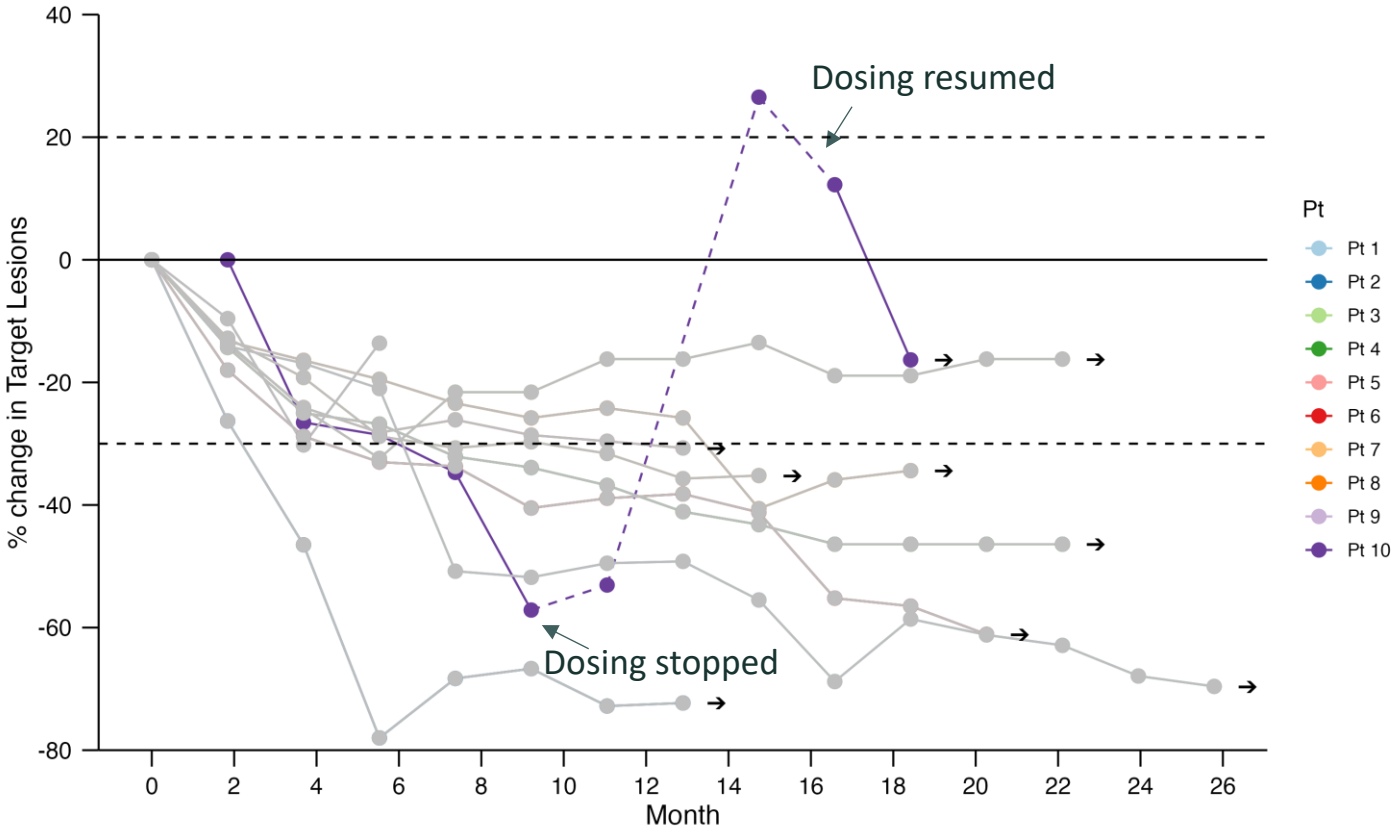
- **9/10 (90%)** sustained therapy for **10+ months**; **5/10 (50%)** for **20+ months**
- Median Duration of Response not reached
- Anti-tumor activity across ACC subtype, histologies, and after multiple lines of therapy (including ADCs)

Datacut: 24Aug2026 (Phase 1 only)

Notes:
*Treatment beyond progression; **confirmation of PR occurred after data cutoff date; ***updated data post-data cut; + = ongoing
Abbreviations: cPR= confirmed PR; uPR = unconfirmed PR

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Sustained objective responses with treatment ongoing ~2+ years



Pt ID	Starting dose	Response	Time on Treatment (months)	Duration of Response (months)
1	12mg	cPR	**27+**	**19+**
2	18mg	cPR	**14+**	**9+**
3*	24mg	uPR	**24+**	**2**
4	24mg	cPR	**24+**	**15+**
5	24mg	cPR**	**14+**	**1+**
6	24mg	cPR	**23+**	**16+**
7	38mg	cPR	**16+**	**7+**
8	38mg	cPR	**20+**	**4+**
9	48mg	uPR	**5**	**2**
10***	48mg	cPR	**10+**	**4**

All patients with starting doses ≥30mg reduced to RP2D 24mg

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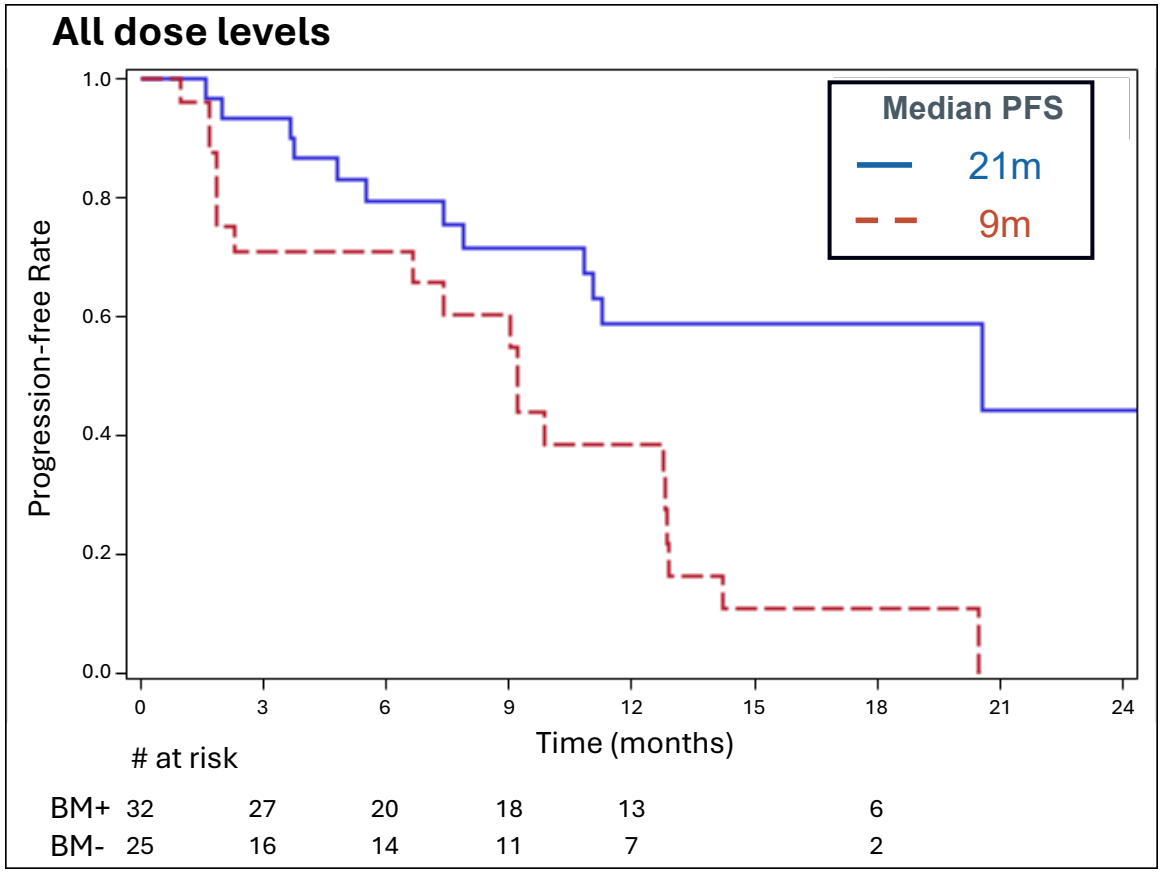
Datacut: 24Aug2026 (Phase 1 only)

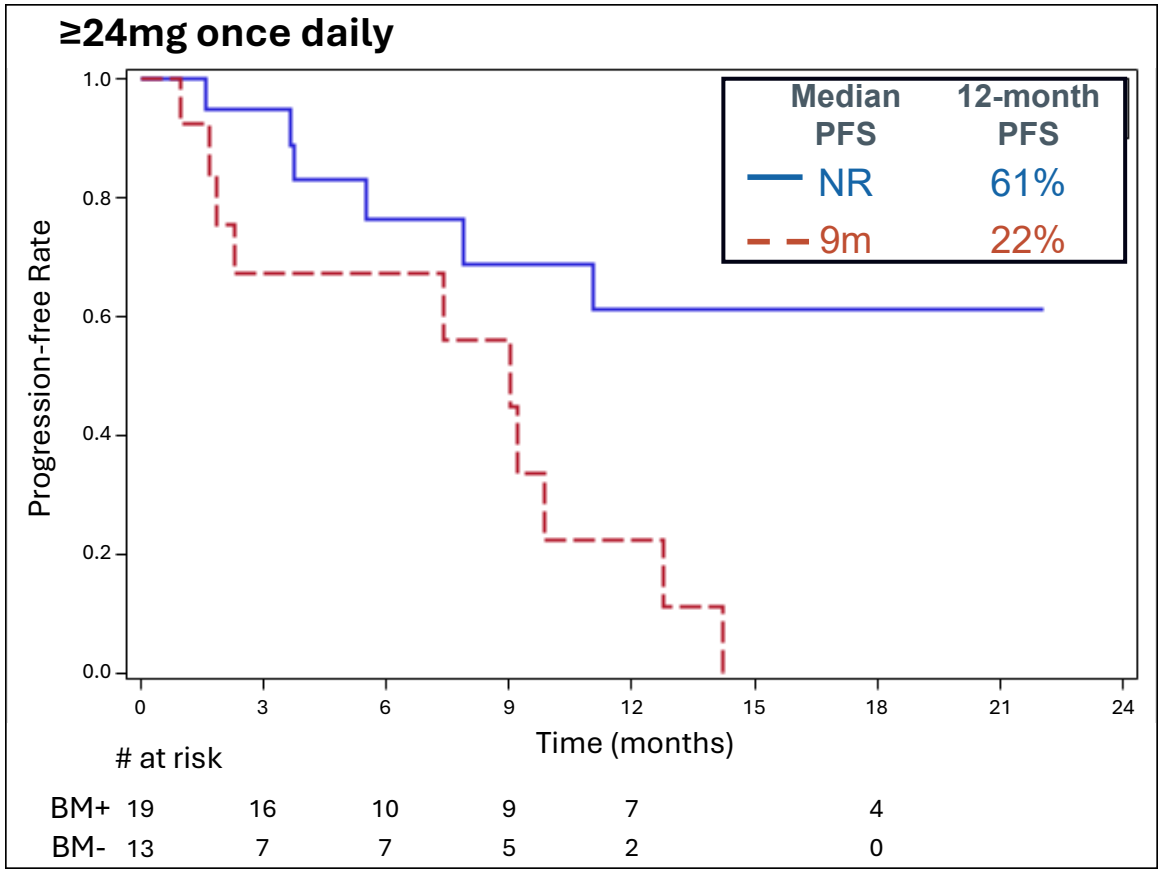
Notes:
*Treatment beyond progression; **confirmation of PR occurred after data cutoff date; ***updated data post-data cut; + = ongoing
Abbreviations: cPR= confirmed PR; uPR = unconfirmed PR

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Progression-free survival by biomarker status

BM positive
BM negative





- Other systemic agents: ORR 0–15% and median PFS ~6-9 months
- Prolonged PFS in BM+ compared to BM- ACC patients

Datacut: 24Aug2026 (Phase 1 only)

Ho A., et al. 2026

NR = not reached, m = months.

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REM-422 ACC Program Summary

- Adenoid Cystic Carcinoma is a high unmet medical need tumor with **no FDA approved therapies**
  - Prior therapies demonstrate an ORR range 0-15%, mPFS ~7 months highlighting need for more effective therapies

- **ORR of 57% at RP2D** with durable responses observed at last data cut-off, treatment ongoing in 90% responders, including patients at **~2+ years**

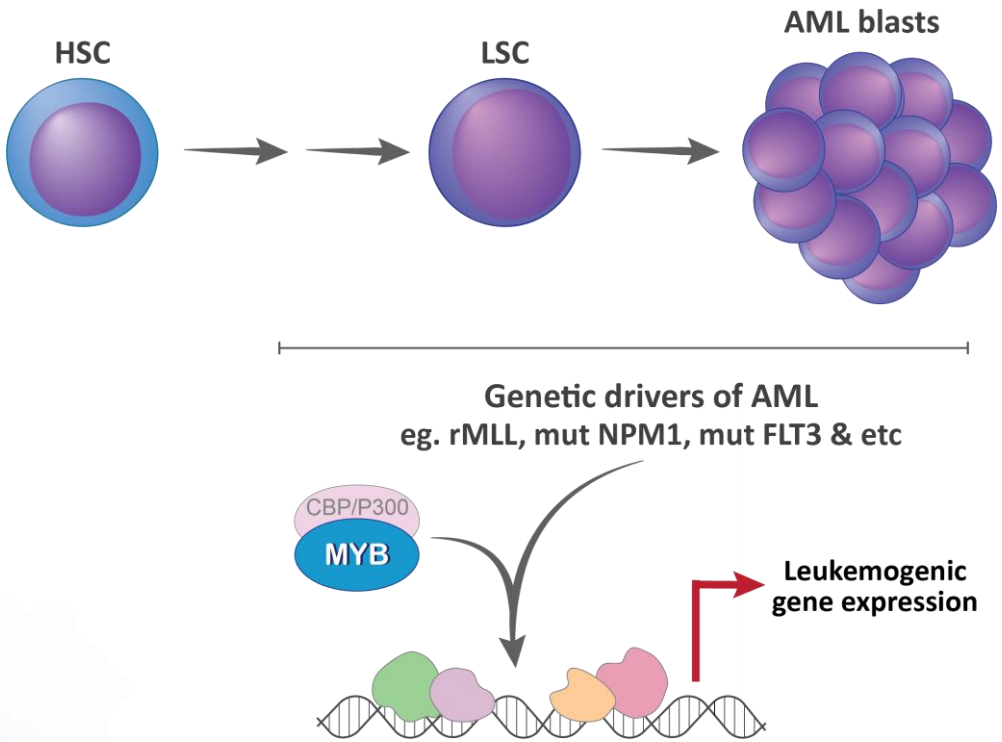
- FDA authorized Remix to proceed with an ongoing Phase 2 study with proposed key elements, including: **recommended dose, biomarker positive population, and single arm study design for potential registration**

- **Potentially registrational** Ph2 study ongoing with **full enrollment anticipated in H2'26**

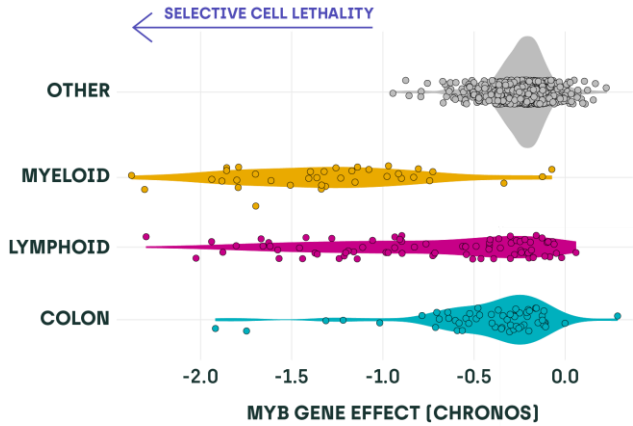
- As Phase 2 data matures, we expect to continue dialog with the FDA to discuss most efficient registrational path; ORR and DOR data expected in mid-2027

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AML and High-Risk MDS are MYB-driven malignancies



- Acute myelogenous leukemia (AML) & high-risk myelodysplastic syndrome (HR-MDS)
  - ~25,000 treatable AML/HR-MDS patients in the US
- MYB is a master transcriptional regulator of leukemogenesis



Cancer Dependency Map data

AML cell lines have a lineage-wide dependency on MYB

- REM-422 is active across multiple genetic subtypes in AML preclinical models (e.g. NPM1, FLT3, rMLL, IDH, p53, Ras etc)
  - No poison exon biomarker selection needed

remix therapeutics

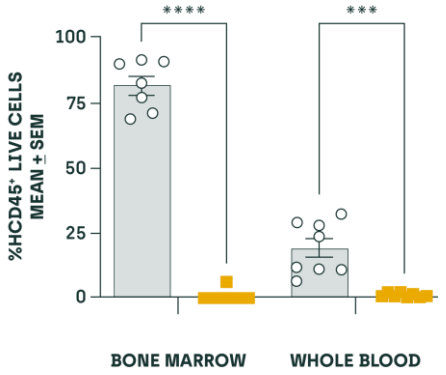
REM-422: Robust monotherapy/combination activity in AML models

Monotherapy Activity

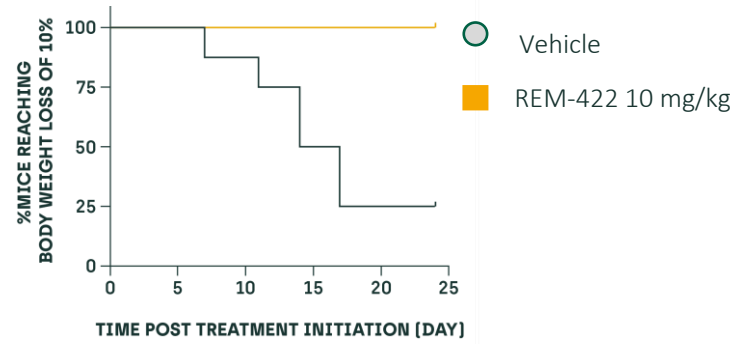
ERADICATED hCD45+ AML BLASTS



M4 (myelomonocytic) AML pt relapsed after chemotherapy with complex cytogenetics

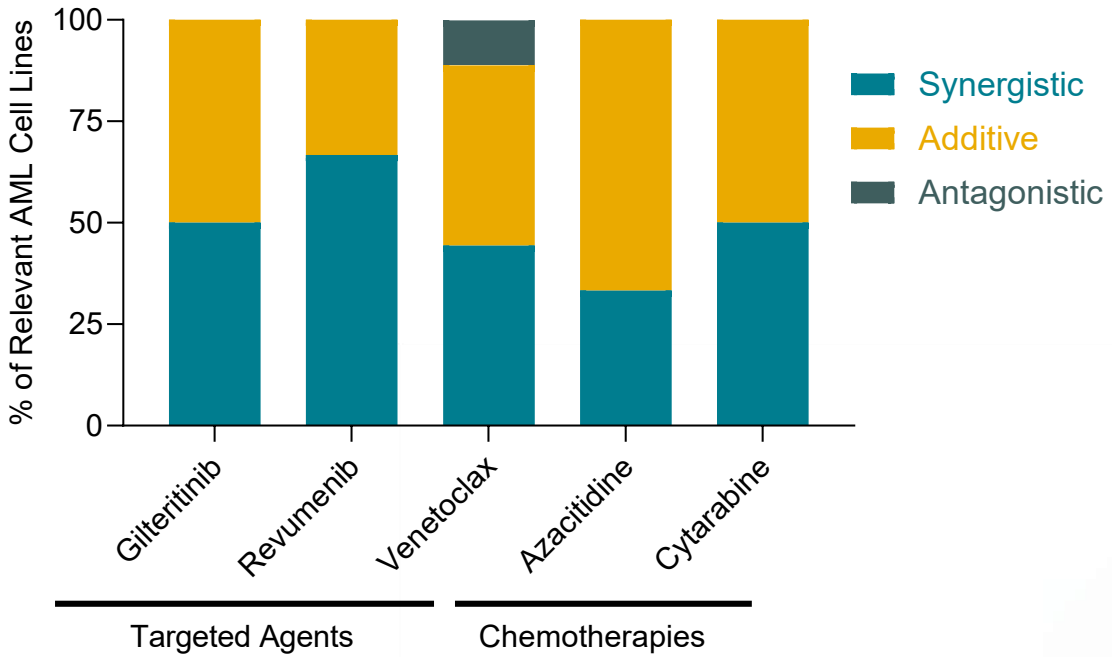


SURVIVAL BENEFIT IN PDX MODEL



Combination Activity

ADDITIVE/SYNERGISTIC ACTIVITY IN LEUKEMIA CELL PANEL

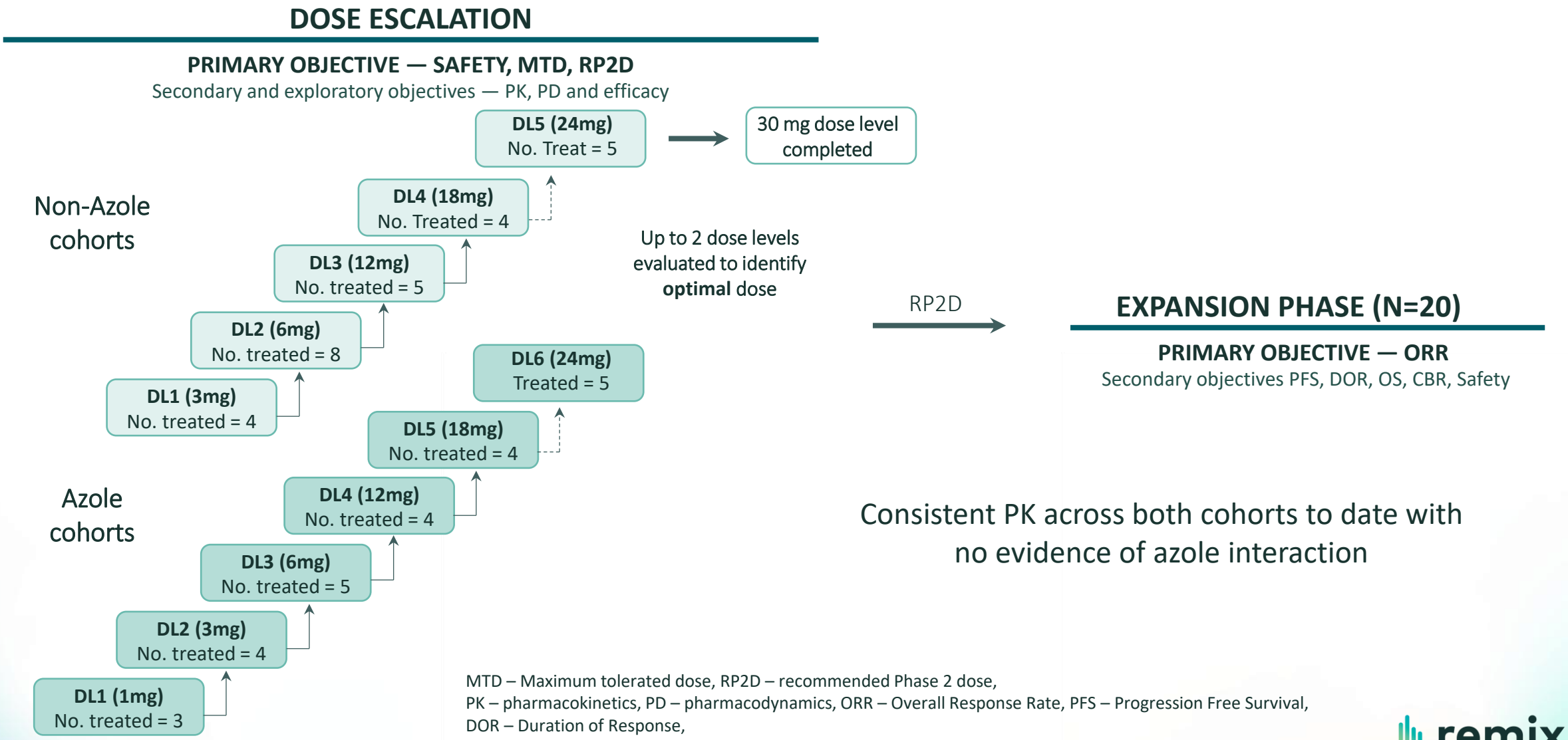


REM-422 has shown preclinical activity as a monotherapy and is additive/synergistic activity with multiple therapies

NOTE: Preclinical results may not be predictive of clinical outcomes

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Ph1 Study in Patients with R/R AML or HR-MDS



Consistent PK across both cohorts to date with no evidence of azole interaction

MTD – Maximum tolerated dose, RP2D – recommended Phase 2 dose,
PK – pharmacokinetics, PD – pharmacodynamics, ORR – Overall Response Rate, PFS – Progression Free Survival,
DOR – Duration of Response,
OS – Overall Survival, CBR – Clinical Benefit Rate

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Preliminary anti-tumor activity; RP2D not reached

Indication	Dose (mg)	Prior Therapies	Cytogenetics /Mutations	Response
HR-MDS	6	Decitabine/cedazuridine, fludarabine, MUD, decitabine/cedazuridine	P53, MSH3, complex karyotype, monosomy 17	CR (durable 15 months ongoing)
AML	12	Venetoclax/Azacitidine	STAG2, TET2, SRSF2, CUX1, MPL	CR_i (durable for 6 months)
AML	12	Multiple including HSCT, Ven, MTX	PTPN11, PHF6	MLFS
HR-MDS	30	CD70 Ab/Aza, MUD, decitabine/cedazuridine	P53, DNMT3A, MRE11	CR_{uni}
HR-MDS	30	Magrolimab/Aza, Aza, lenalidomide	P53, ASXL1	CR_L

Dose dependent reduction of MYB mRNA levels was observed in bone marrow biopsies
Additional patients with blast count reductions observed in ongoing dose escalation study

CR = Complete Remission, Cri Complete Remission with Incomplete Recovery, CRuni = Complete Remission Unilineage, CRL = Complete Remission with Limited Count Recovery, MLFS = Morphological Leukemia-Free State

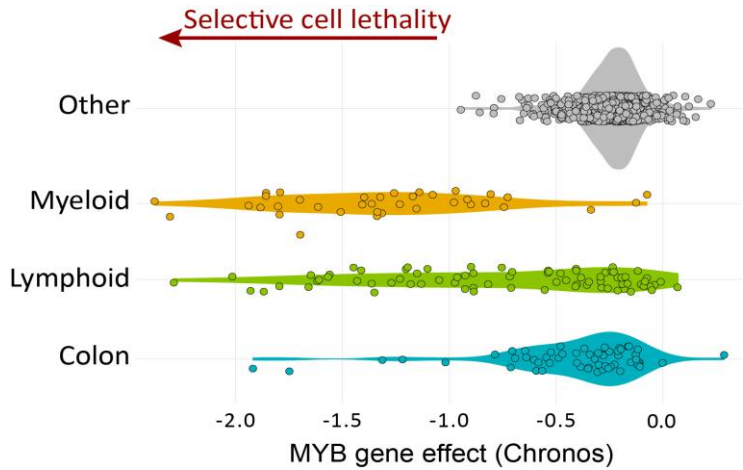
From live database last updated Mar 18, 2026

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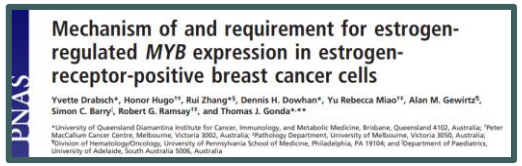
REM-422 indication expansion potential in heme and solid tumors

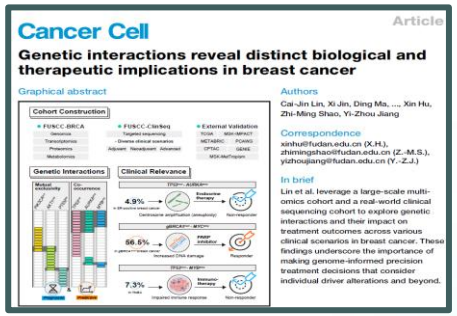
Disease	MYB dysregulation
Breast Cancer	MYB high expression
Lymphoma	Overexpression
Colon Cancer	Overexpression
Tumor agnostic	Fusion & amplification

MYB DEPENDENCY ACROSS SEVERAL LINEAGES (DepMap)



MYB DYSREGULATION IN BREAST CANCER





PMIDs: 17690249 and 38593782

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Pipeline programs

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DATA SCIENCE



+ Integrated database of >350k internal and external transcriptome datasets
+ Proprietary algorithms & AI/ML

BIOLOGY & BIOMOLECULAR SCIENCES



+ Functional & genetic validation of targets
+ Fit for purpose high-throughput multiplexed screens

MEDICINAL & COMPUTATIONAL CHEMISTRY



+ Proprietary small molecule library targeting RNA/protein complexes
+ RNA-protein complex structural biology

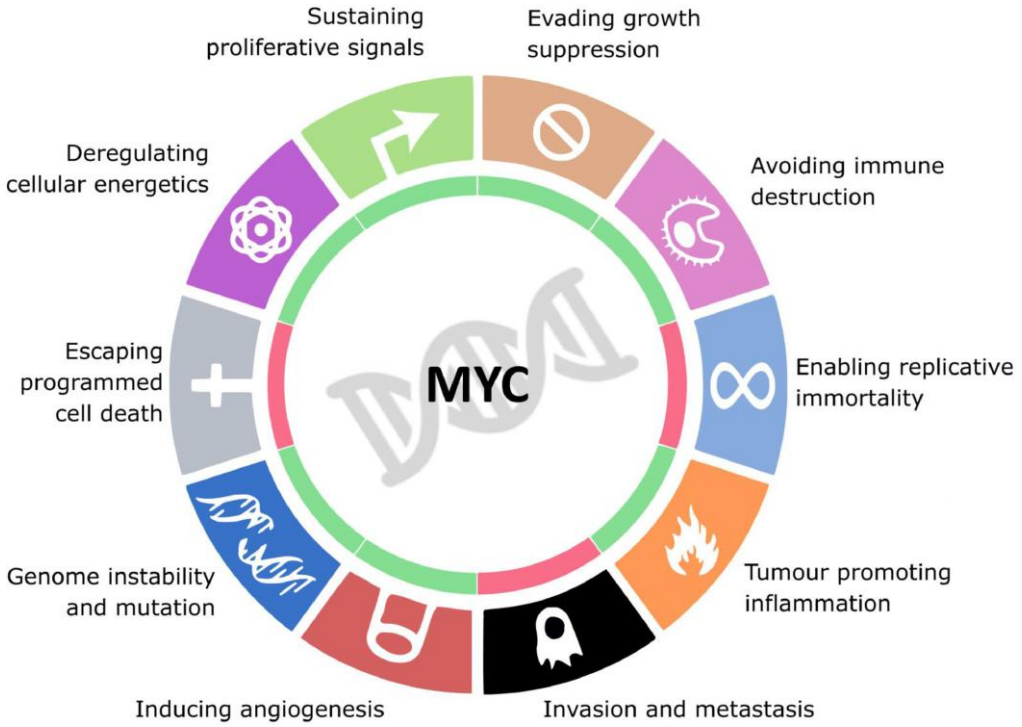
Genetically Defined Diseases

Patient Selection Criteria



REMIX TARGETS & DRUGS

Targeting MYC, an oncogenic transcription factor dysregulated across cancer



Indication	Total US New Cases (k/yr)	MYC dysregulation
Diffuse large B-cell lymphoma (DLBCL)	16-20	c-MYC translocation
Burkitt lymphoma (BL)	3	
Ovarian	21	MYC amplification frequencies >20% (c-MYC, N-MYC, or L-MYC)
Esophageal	22	
Gastric	30	
Head and neck	58	
Pancreatic	67	
Colorectal	154	>1/3 of patients have c-MYC overexpression
Prostate	313	

Llombart et al., eBioMedicine, 2021

- MYC amplification/activation occurs in ~28% of all tumors
- **Remix developing a novel mRNA degrader approach targeting a regulator of MYC to prevent signaling via all 3 MYC paralogs**

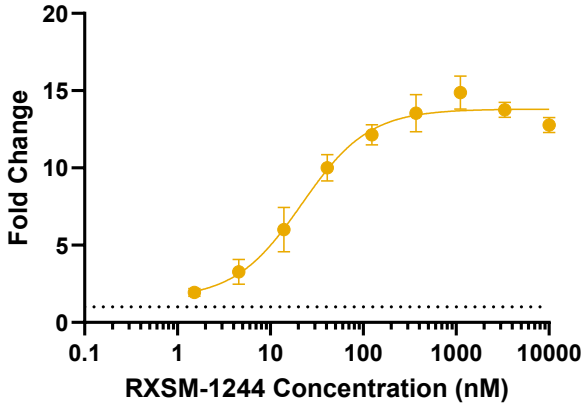
Wang, *et al.*, 2024 Blood Cancer J
Blum *et al.*, 2004 Blood
Clipson *et al.*, 2015 J Pathol Clin Res
Schaub *et al.*, 2018 Cell Systems
US case numbers per SEER

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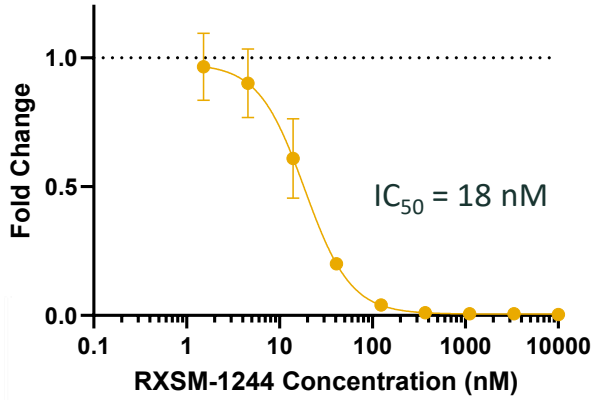
Discovery program targeting oncogenic high prevalence MYC signaling dysregulation across multiple solid and heme malignancies

Novel mRNA degraders potently reduce mRNA and protein levels through a poison exon inclusion mechanism

Poison Exon inclusion

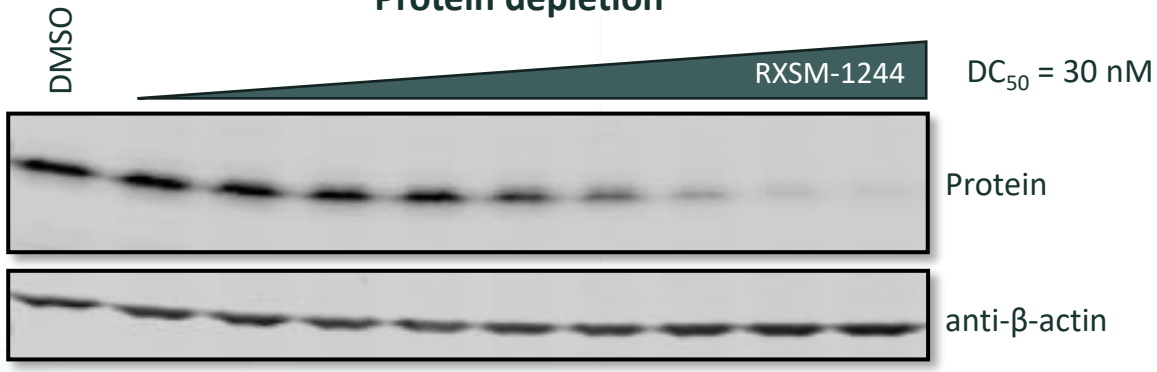


mRNA reduction



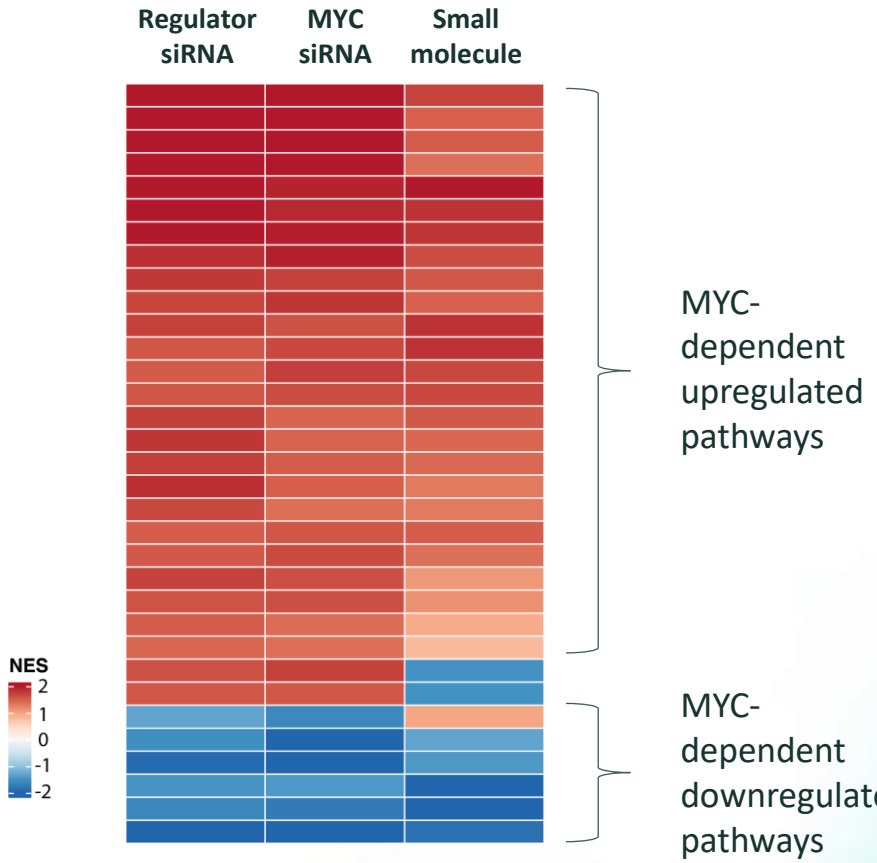
qPCR, 24h, n = 3

Protein depletion



Western Blot, 72h

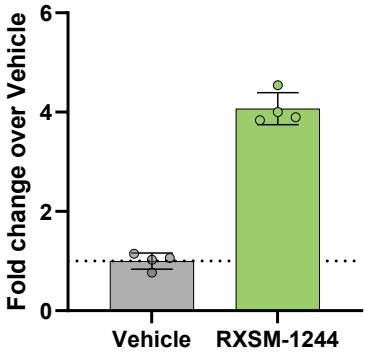
Small molecule mRNA degrader phenocopies genetic knockdown



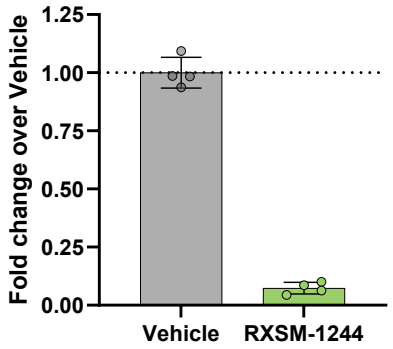
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RXSM-1244 demonstrated potent mRNA and protein target engagement and robust tumor growth inhibition in a mouse CDX model

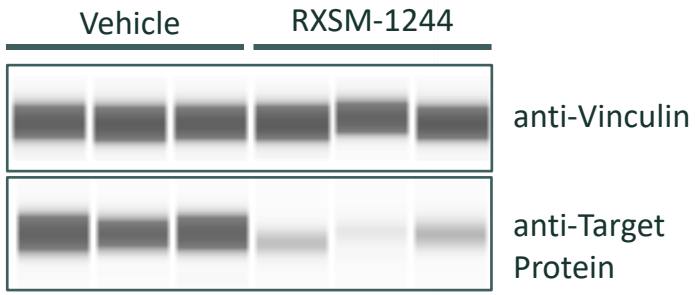
poison exon inclusion



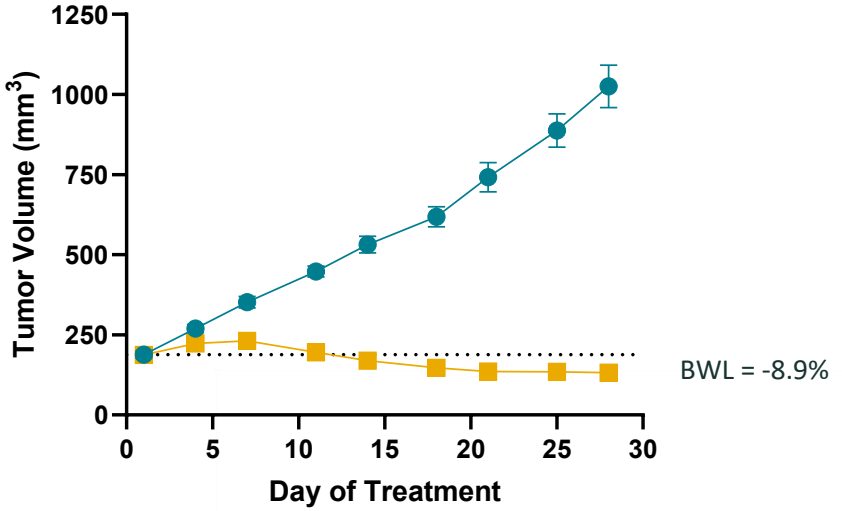
mRNA reduction



protein reduction



MYC-amplified CDX

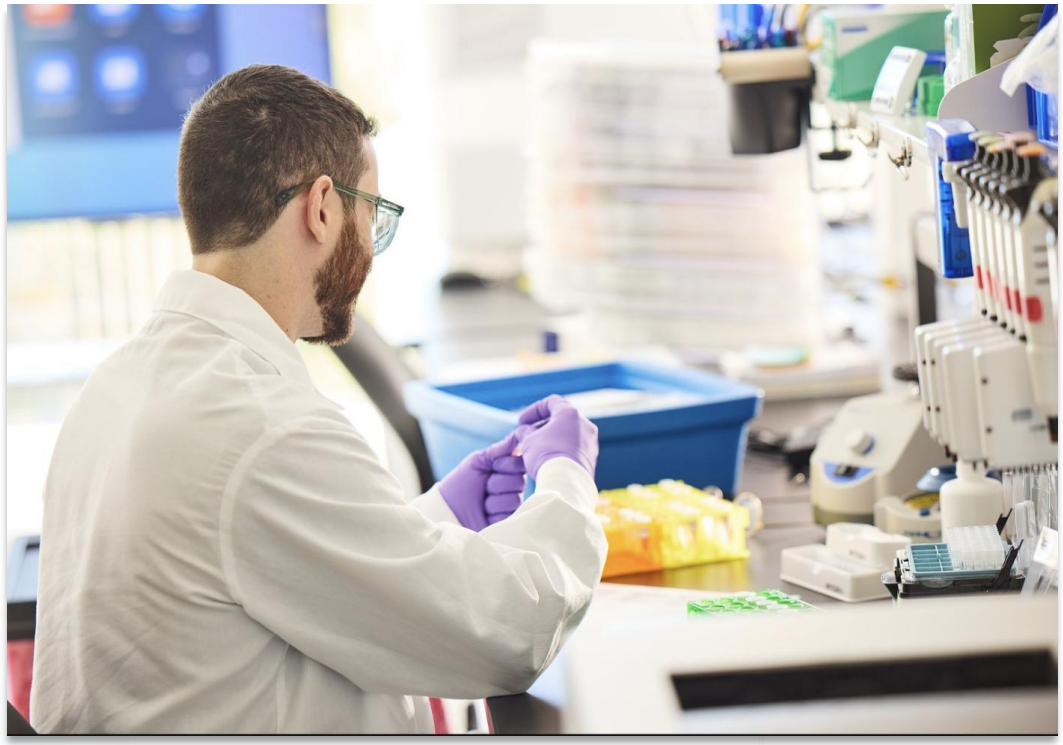


- RXSM-1244 showed well behaved rodent PK profile
- Observed *in vivo* poison exon inclusion, concomitant mRNA degradation and protein reductions
- Induced regressions in multiple MYC addicted cell-line and patient derived xenograft models

QDx3 study. mRNA and protein analysis from samples collected 6 hr post-last dose

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Remix is positioned to become a global leader in RNA-targeted medicines



Differentiated Platform
- Designed to address RNA processing and disease drivers at their origin

REM-422: MYB mRNA degrader
- Potent, selective oral small molecule MYB mRNA degrader currently in clinical studies for both ACC and AML or HR-MDS

Key upcoming value drivers
- Potentially registrational Phase 2 ongoing with ORR and DOR data for REM-422 expected in ~ mid-2027
- RP2D and initial data from Phase 1 AML/High Risk MDS study in ~mid-2027

Well-capitalized with a disciplined capital allocation approach
- Under current operating plan, expected cash runway into 2028

remix therapeutics

remix therapeutics

Investor Relations: Will.OConnor@precisionaq.com

Thank you.

NASDAQ: RMTX