cullinan® THERAPEUTICS

CLN-049 Update at ASH

December 8, 2025

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Agenda

Introduction & Pipeline Strategy
Nadim Ahmed

CLN-049 and Results from ASH 2025
Jeff Jones

Risk-adapted Therapy in AML
David Sallman, MD

Strategic Perspective & Next Steps
Nadim Ahmed

Q&A

Presenters

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Chief Executive Officer

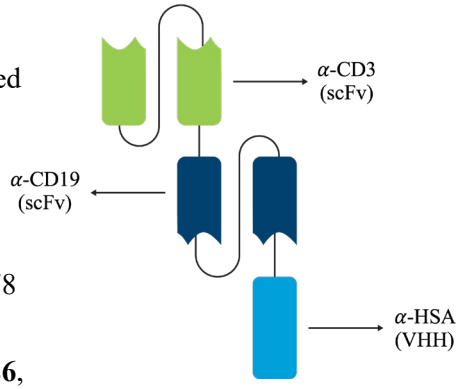
Jeff Jones, MD, MBA
Chief Medical Officer

David Sallman, MD
Associate Member, Myeloid Section Head, Moffitt Cancer Center & Research Institute

Advancing T cell engagers for high-impact, validated targets across immunology and oncology

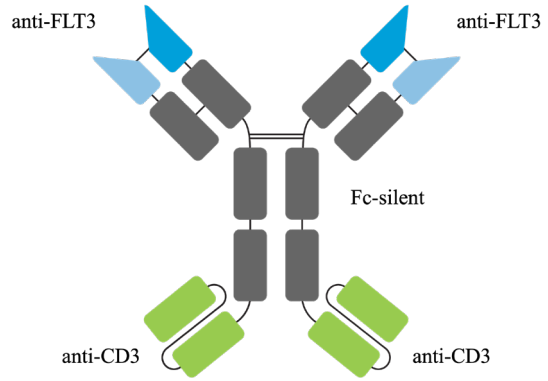
CLN-978 in autoimmune diseases:
CD19xCD3 bispecific T cell engager

- **Potential best-in-class** CD19xCD3 T cell engager in autoimmune diseases
- Prior observations from Phase 1 B-NHL study showed rapid, deep, and sustained B cell depletion and anti-tumor efficacy
- Off the shelf, potential disease modifying treatment across autoimmune diseases
- Experienced immunology team dedicated to CLN-978
- Phase 1 studies ongoing in SLE, RA and Sjögren's disease, with **initial data in SLE and RA in H1 2026**, with a focus on safety and B cell depletion



CLN-049 in AML and MDS:
FLT3xCD3 bispecific T cell engager

- **Potential first-in-class** T cell engager in AML, where FLT3 is a well-validated therapeutic target
- U.S. FDA **Fast Track designation** in R/R AML
- Internal deep expertise in hematology
- Phase 1 study ongoing in patients with relapsed/refractory AML or MDS
- Promising clinical activity observed, including multiple complete responses, (oral presentation at **ASH 2025)**



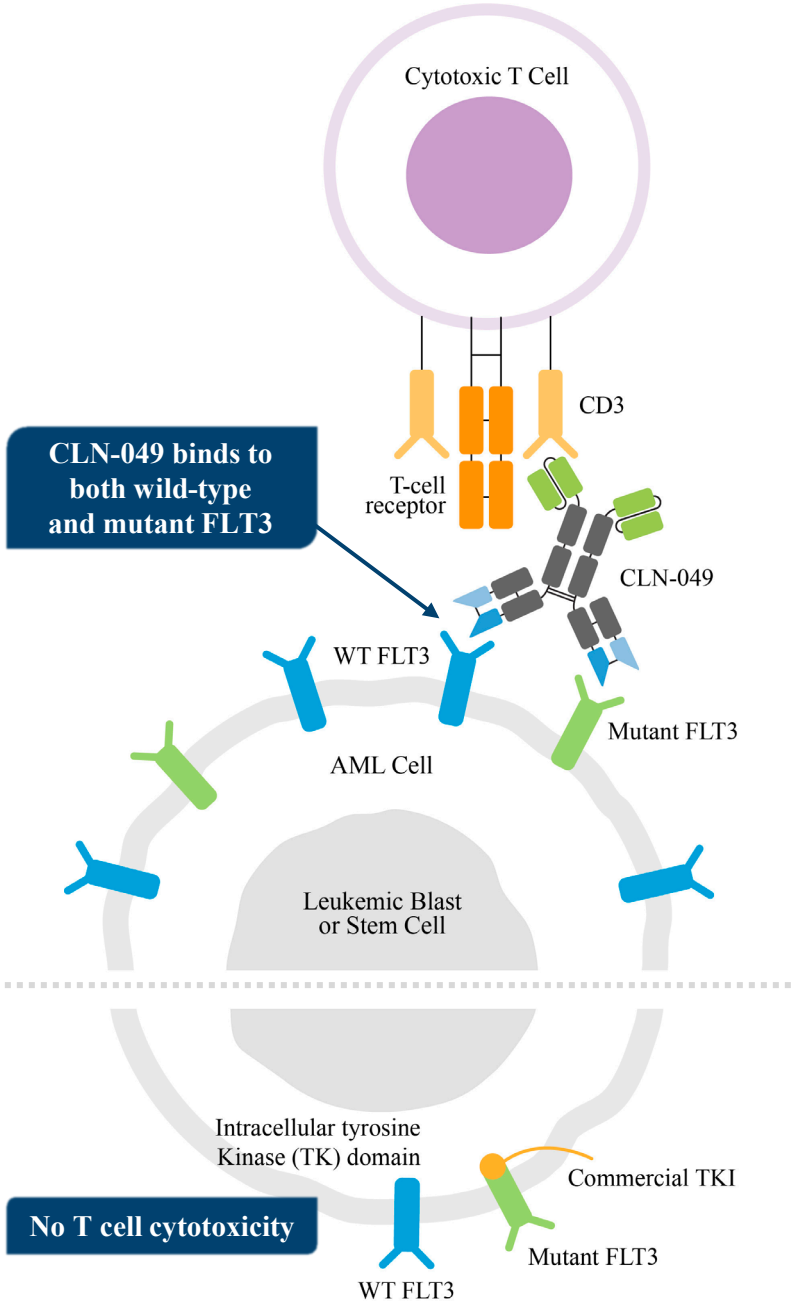
Cash and investments of $475 million on hand at September 30, 2025* to fund these priority programs, expected to fund operations into 2029

*Unaudited. Includes cash, cash equivalents, investments, and interest receivable.

CLN-049

FLT3xCD3 bispecific T cell engager

Jeff Jones, MD, MBA
Chief Medical Officer



CLN-049 is an optimal AML immunotherapy

1. **First-in-class opportunity:** CLN-049 binds to the extracellular domain of FLT3, both wildtype and mutated forms, redirecting a patient's own T cells to recognize and eliminate leukemic cells.

2. **Potential to treat a broad AML population:** FLT3 is expressed on more than 80%[1] of AML blasts and only a limited number of normal hematopoietic precursors and dendritic cells.

3. **Promising therapeutic potential:** FLT3 is expressed on leukemic stem cells as well as blast cells, which may increase response durability. Since FLT3 is an oncogenic driver, target loss is unlikely.

4. **Potential for reduced toxicity risk:** FLT3 expression is very low on most mature normal myeloid cells. FLT3 expression is also very low on normal pluripotent stem cells.

1. Gebru, M.T., Wang, HG. Therapeutic targeting of FLT3 and associated drug resistance in acute myeloid leukemia. J Hematol Oncol 13, 155 (2020). https://doi.org/10.1186/s13045-020-00992-1

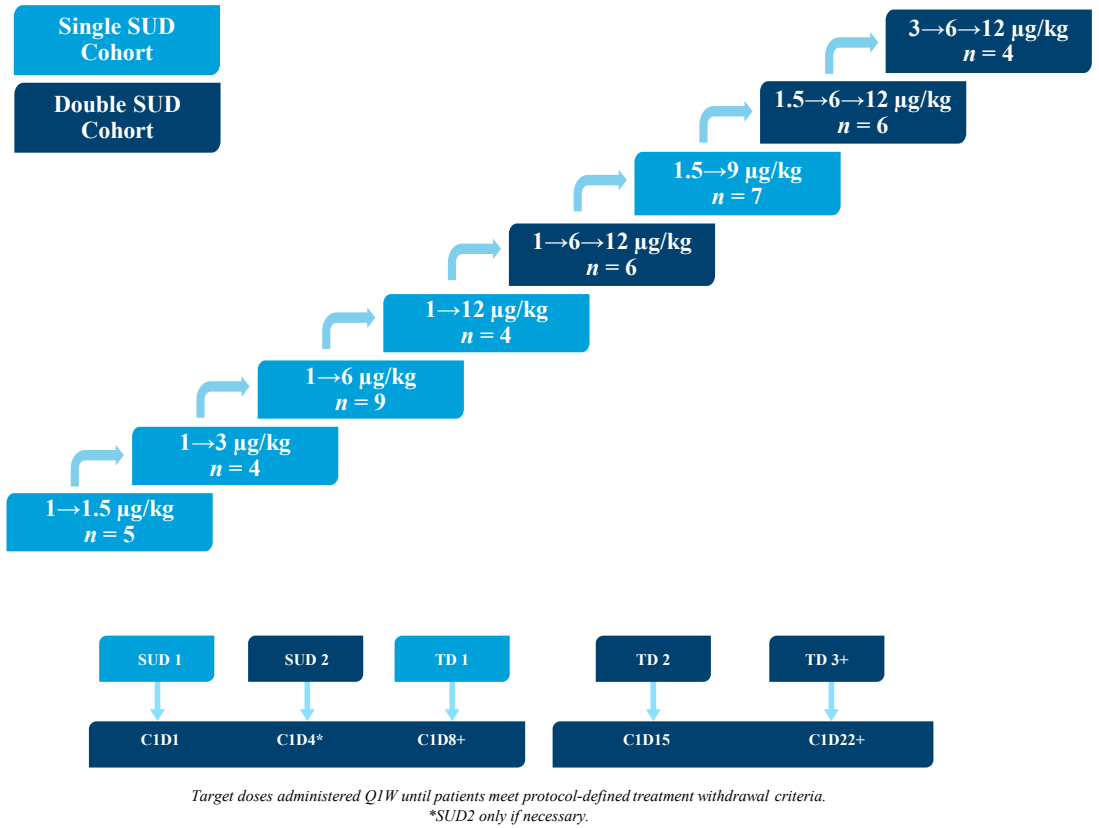
CLN-049 Dose Escalation in R/R AML

Results from Oral Presentation at 2025 ASH Annual Meeting

Jeff Jones, MD, MBA
Chief Medical Officer

CLN-049 Phase 1 study in patients with R/R AML or MDS: Initial results from the ongoing dose-escalation

Study Design – IV Dose Escalation Cohorts



Target doses administered Q1W until patients meet protocol-defined treatment withdrawal criteria.
**SUD2 only if necessary.*

Study Objective

- **To assess preliminary efficacy, safety, tolerability, PK, pharmacodynamics, and immunogenicity of IV-administered CLN-049 in patients with R/R AML or MDS**

Study Enrollment and Eligibility

- **45 patients ≥18 years with R/R AML or MDS (ECOG 0 to 2) enrolled as of August 2025 data cutoff**
  - 45 patients assessed for safety
  - 41 patients with available efficacy data
  - Efficacy assessments for 3→6→12 µg/kg cohort (*n*=4) not available at time of data cutoff
- **No requirement for baseline testing for FLT3 expression**

Study Efficacy Endpoints: Complete response (CR) rate; Composite complete response (CRc) rate: (CR/CRi/CRh in AML or CR/CRL/CRh in MDS); ORR: (CRc + MLFS + PR in AML or CRc + PR + HI in MDS); Response assessed using ELN 2022 (AML) or IWG 2023 (MDS) criteria
C, cycle; CRh, complete remission with partial hematologic recovery; CRi, complete remission with incomplete recovery; CRL, complete remission with limited response; D, day; ECOG, Eastern Cooperative Oncology Group; ELN, European LeukemiaNet; FIH, first in human; HI, hematologic improvement; IV, intravenous; IWG, International Working Group; MDS, myelodysplastic syndrome; MLFS, morphologic leukemia-free state; ORR, objective response rate; PK, pharmacokinetics; PR, partial response; Q1W, every week; R/R, relapsed/refractory; SUD, step-up dose; TD, target dose.

CLN-049 Phase 1: Enrolled patients are representative of the broad R/R AML population

Characteristic	All cohorts *n*=45	1→6 µg/kg cohort *n*=9	1.5→9 µg/kg cohort *n*=7	12 µg/kg cohorts[1] *n*=20
Diagnosis, n (%)				
AML	39 (87)	9 (100)	5 (71)	19 (95)
MDS/AML	3 (7)	0	2 (29)	0
MDS	3 (7)	0	0	1 (5)
ECOG at baseline, n (%)				
0	13 (29)	2 (22)	2 (29)	6 (30)
1	24 (53)	4 (44)	4 (57)	10 (50)
2	8 (18)	3 (33)	1 (14)	4 (20)
Prior therapies				
Median (range)	2 (1–8)	2 (1–7)	2 (1–5)	1.5 (1–8)
HMA/Venetoclax as last prior therapy, n (%)	27 (60)	7 (78)	2 (29)	12 (60)
Prior transplant, n (%)	10 (22)	2 (22)	3 (43)	4 (20)
BMA blasts[2] at screening, n (%)				
<30%	27 (60)	6 (67)	4 (57)	12 (60)
≥30–50%	6 (13)	0	2 (28)	3 (15)
>50%	7 (16)	0	1 (14)	4 (20)
Risk at time of diagnosis (AML), n (%)				
Favorable	2 (5)	0	1 (20)	0
Intermediate	6 (15)	1 (11)	2 (40)	1 (5)
Adverse	28 (72)	8 (89)	1 (20)	6 (84)
Cytogenetics/molecular annotation, n (%)				
Any abnormality	39 (87)	9 (100)	6 (86)	18 (90)
Complex cytogenetics	7 (16)	3 (33)	0	3 (30)
–5; −7; −17/abn(17p)	6 (13)	2 (22)	0	4 (20)
FLT3-ITD mutation[3]	6 (13)	2 (22)	0	1 (5)
***TP53* mutation[4]**	**16 (36)**	**3 (33)**	**0**	**11 (55)**

August 2025 data cutoff

[1]12 µg/kg cohorts include 1 → 12 µg/kg, 1 → 6 → 12 µg/kg, 1.5 → 6 → 12 µg/kg, and 3 → 6 → 12 µg/kg dose levels.
[2]Bone marrow biopsy data used where bone marrow aspirate data was not available.
[3]FLT3-ITD identified through cytogenic/molecular annotation in EDC and eligibility packets, or prior treatment with an approved FLT3 inhibitor
[4]TP53 mutation identified through cytogenetic/molecular annotation in EDC and eligibility packets
BMA, bone marrow aspirate; Unknown or not-specified values not shown

CLN-049 Phase 1: Treatment-emergent adverse events demonstrate a favorable safety profile

	Single step-up cohorts					Double step-up cohorts			Total *n*=45
TEAEs by preferred term, >15% of patients, n (%)	**1→1.5 µg/kg *n*=5**	**1→3 µg/kg *n*=4**	**1→6 µg/kg *n*=9**	**1.5→9 µg/kg *n*=7**	**1→12 µg/kg *n*=4**	**1→6→12 µg/kg *n*=6**	**1.5→6→12 µg/kg *n*=6**	**3→6→12 µg/kg *n*=4**	
Patients with ≥1 TEAE	5 (100.0)	4 (100.0)	8 (88.9)	7 (100.0)	4 (100.0)	6 (100.0)	6 (100.0)	2 (50.0)	**42 (93.3)**
Cytokine release syndrome (CRS)	0	1 (25.0)	2 (22.2)	3 (42.9)	4 (100.0)	3 (50.0)	2 (33.3)	1 (25.0)	**16 (35.6)**
Infusion-related reaction	1 (20.0)	1 (25.0)	4 (44.4)	3 (42.9)	0	1 (16.7)	3 (50.0)	2 (50.0)	**15 (33.3)**
Febrile neutropenia	1 (20.0)	1 (25.0)	3 (33.3)	0	1 (25.0)	2 (33.3)	1 (16.7)	0	**9 (20.0)**
White blood cells decreased	1 (20.0)	1 (25.0)	1 (11.1)	1 (14.3)	2 (50.0)	1 (16.7)	0	1 (25.0)	**8 (17.8)**
Pneumonia	0	1 (25.0)	2 (22.2)	1 (14.3)	0	2 (33.3)	2 (33.3)	0	**8 (17.8)**
Diarrhea	0	1 (25.0)	2 (22.2)	0	2 (50.0)	1 (16.7)	1 (16.7)	0	**7 (15.6)**
Hypomagnesemia	0	1 (25.0)	2 (22.2)	1 (14.3)	2 (50.0)	0	0	1 (25.0)	**7 (15.6)**
Stomatitis	2 (40.0)	1 (25.0)	1 (11.1)	0	1 (25.0)	2 (33.3)	0	0	**7 (15.6)**
Hypokalemia	1 (20.0)	1 (25.0)	3 (33.3)	2 (28.6)	0	0	0	0	**7 (15.6)**

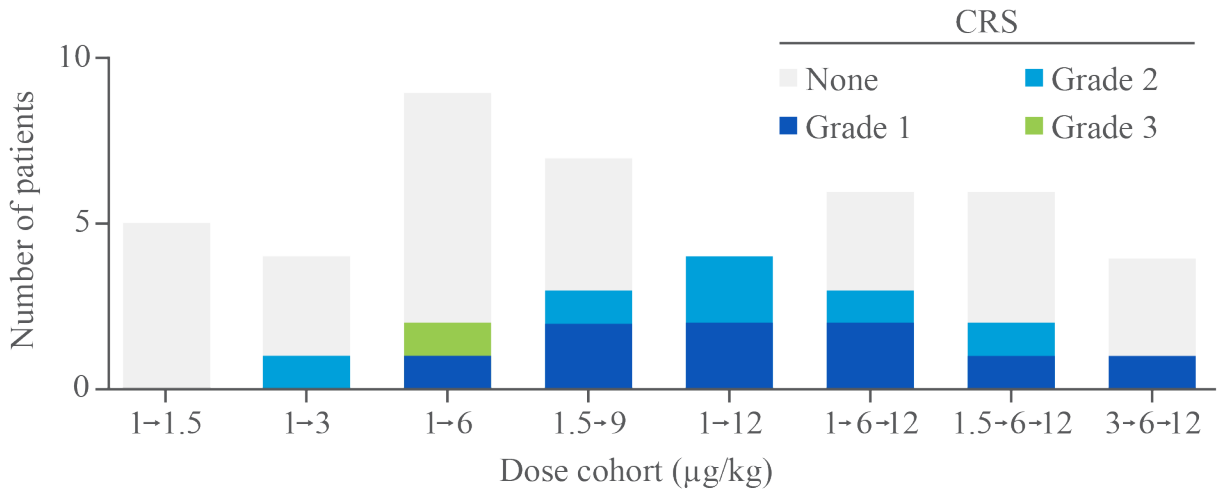
Frequency and severity of CRS can be mitigated through step-up dosing

August 2025 data cutoff

Cytokine release syndrome (CRS) and associated adverse events can be mitigated through an optimized step-up dosing scheme

Nearly all CRS events limited to grade 1 or 2

- Not treatment limiting
- Onset usually after a SUD or the first TD
- No grade 3 events in regimens utilizing 2 SUDs
- Frequency of CRS reduced at the highest 12 µg/kg TD with use of a second SUD



Other less common cytokine-associated adverse events can also be mitigated through step-up dosing

- **ICANS:** Two patients with grade 1 events that occurred at SUD2 or TD1
  - Both events were preceded by grade 2 CRS, were transient and reversible, and were not treatment limiting
- **Transaminitis:** 3 patients with grade 4 events that occurred after TD1 or TD2 in association with grade 1-3 CRS
  - All events were asymptomatic, transient and reversible, and mitigated by use of a second SUD

CRS, cytokine release syndrome; ICANS, immune effector cell-associated neurotoxicity syndrome.

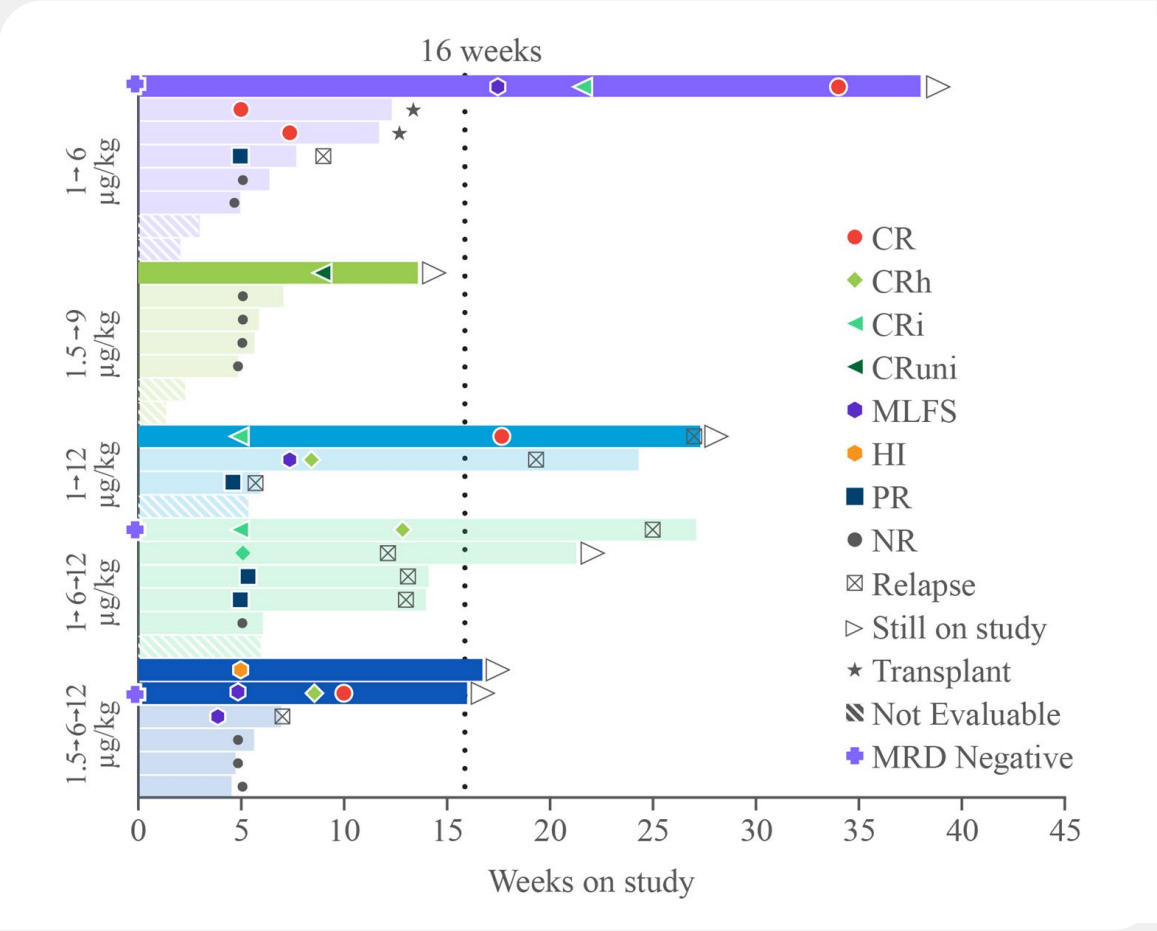
CLN-049 Phase 1: Preliminary efficacy data highlights potential to achieve deep responses in a heavily pre-treated population

Response rate (best response), n (%)	Single step-up cohorts					Double step-up cohorts		All cohorts *n* = 41*	≥6 µg/kg cohorts *n* = 32	12 µg/kg cohorts *n* = 16*
	1→1.5 µg/kg *n* = 5	1→3 µg/kg *n* = 4	1→6 µg/kg *n* = 9	1.5→9 µg/kg *n* = 7	1→12 µg/kg *n* = 4	1→6→12 µg/kg *n* = 6	1.5→6→12 µg/kg *n* = 6			
CR	0	0	3 (33)	0	1 (25)	0	1 (17)	5 (12)	5 (16)	2 (13)
CR+CRh	0	0	3 (33)	0	2 (50)	2 (33)	1 (17)	8 (20)	8 (25)	5 (31)
CRc	0	1 (25)	3 (33)	1 (14)	2 (50)	2 (33)	1 (17)	10 (24)	9 (28)	5 (31)
ORR	0	1 (25)	4 (44)	1 (14)	3 (75)	4 (67)	3 (50)	16 (39)	15 (47)	10 (63)

*Enrollment into 3→6→12 µg/kg cohort (n = 4) ongoing at time of data cutoff; efficacy data not available for this cohort.

31% CR+CRh rate at the 12µg/kg dose in this heavily pre-treated R/R AML population

CLN-049 Phase 1: Promising initial response durability data



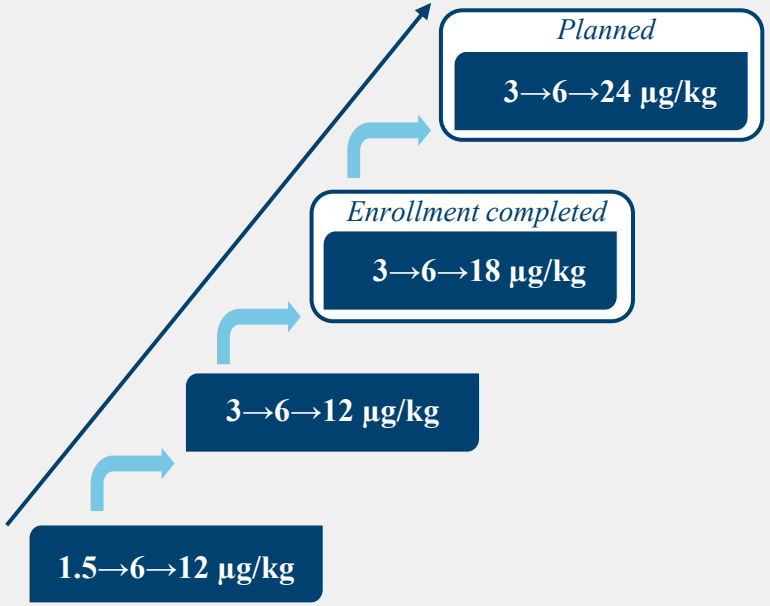
BM, bone marrow; CRuni, complete remission unilineage; MRD, measurable residual disease.; DoR, duration of response; HSCT, hematopoietic stem cell transplant; NR, no response.

8 patients achieved CR or CRh at a TD of ≥6 µg/kg:

- 5 patients had DoR >16 weeks
  - 3 MRD-negative patients all achieved DoR >16 weeks, including 1 patient with an ongoing response for >36 weeks
- 2 additional patients attained CR and proceeded to HSCT

Next steps: Dose expansion cohorts in parallel AML subpopulations

Phase 1 IV Dose Escalation



53 patients enrolled to date*

Phase 1 Dose Expansion/RP2D Determination

R/R AML
- *TP53* mutation negative
- ≤ 3 prior lines of therapy

Alternate Assignment

- **Dose Level 1** *n* = 20
- **Dose Level 2** *n* = 20

TP53m AML
- *TP53* mutation
- Frontline or R/R AML
- ≤ 3 prior lines of therapy

- **Dose Leve1 1** *n* = 20

Target completion by year-end 2026

**As of December 8, 2025; RP2D = recommended phase 2 dose*

Summary of results shared at ASH 2025 Annual Meeting

- **CLN-049 monotherapy demonstrated compelling efficacy in a broad, heavily pretreated population of patients with R/R AML and MDS**
- **At the highest target dose tested of 12 µg/kg, CR+CRh rate was 31%**
- **The majority of responses were durable beyond 16 weeks**
- **Safety was favorable with no G3 CRS and no dose-limiting adverse events observed at the highest target dose in regimens utilizing 2 step-up doses**
- **CLN-049 development will proceed under FDA Fast Track designation, with expansion cohorts in R/R AML and *TP53*m AML planned to begin in early 2026**

August 2025 data cutoff

Risk-adapted therapy in AML

Overview and Patient Case

David Sallman, MD
Associate Member, Myeloid Section Head,
Moffitt Cancer Center & Research Institute

Significant unmet need in adult AML

- The only curative therapy is intensive chemotherapy +/- stem cell transplantation
- Curative therapy remains out of reach for most AML patients: 85% patients >60 years old are ineligible for intensive chemotherapy
- Recent treatment advancements have not significantly improved the likelihood of cure for the majority of AML patients
- **A significant unmet need remains for –**
  - a broadly applicable therapy that can produce high rates of durable response
  - eradication of measurable residual disease (MRD) that portends relapse even when patients meet clinical criteria for complete remission

US AML incidence **22,010**[1]

Average age at diagnosis **69**[1]

5-year survival **10% or less** in relapsed setting[2]

1. American Cancer Society (2025) 2. DeWolf and Tallman. Blood 2020.

Summary treatment guidelines for newly diagnosed and R/R AML

Newly diagnosed AML

Intensive chemotherapy eligible	**CBF-AML**	IC+-gemtuzumab ozogamacin
	Secondary AML	CPX-351
	FLT3 ITD or TKD	IC+midostaurin or quizartinib
	CD33+	IC+gemtuzumab ozogamicin
	All others	IC
All	***TP53m* or other high risk**	Clinical trial
Ineligible for intensive chemotherapy	**IDH1m**	HMA+VEN or ivosidenib
	IDH2m	HMA+VEN or enasidenib
	FLT3 ITD or TKD	HMA+gilteritinib
	CD33+	gemtuzumab ozogamicin
	All others	HMA+VEN

Consolidation therapy (various) and maintenance therapy (various)

R/R AML

IDH1m	ivosidenib or olutasidenib or BSC
IDH2m	enasidenib or BSC
FLT3 ITD	• gilteritinib • HMA+sorafenib • quizartinib • BSC
FLT3 TKD	gilteritinib or BSC
NPM1	revumenib or ziftomenib or BSC
KMT2A	revumenib or BSC
CD33+	gemtuzumab ozogamacin or BSC
Other intensive and less intensive options	• IC or LDAC • HMA+-VEN • Clinical trial or BSC

1. NCCN Guidelines Version 3.2026

In AML, genetic features at diagnosis determine outcome – for now

Favorable

- t(8;21)(q22;q22.1)/RUNX1::RUNX1T1
- inv (16)(p13.1q22) or t(16;16)(p13.1;q22)/CBFB::MYH11
- Mutated NPM1 without FLT3-ITD
- bZIP in-frame mutated CEBPA

~34% to ~55%
5-year survival rates

Intermediate

- Mutated NPM1 with FLT3-ITD
- Wild-type NPM1 with FLT3-ITD (without adverse-risk genetic lesions)
- t(9;11)(p21.3;q23.3)/MLLT3::KMT2A
- Cytogenetic and/or molecular abnormalities not classified as favorable or adverse

~13% to ~38%
5-year survival rates

Poor/Adverse

- **Mutated *TP53* (variant allele frequency ≥ 10%)**
- t(6;9)(p23;q34.1)/DEK::NUP214
- t(v;11q23.3)/KMT2A rearranged (excluding KMT2A-PTD)
- t(9;22)(q34.1;q11.2)/BCR::ABL1
- (8;16)(p11;p13)/KAT6A::CREBBP
- inv (3)(q21.3q26.2) or t(3;3)(q21.3;q26.2)/GATA2, MECOM(EVI1)
- t(3q26.2;v)/MECOM(EVI1)-rearranged
- −5 or del(5q); −7; −17/abn(17p)
- Complex karyotype; Monosomal Karyotype
- Mutated ASXL1, BCOR, EZH2, RUNX1, SF3B1, SRSF2, STAG2, U2AF1, or ZRSR2

~2% to ~11%
5-year survival rates

1. NCCN Guidelines Version 3.2026

TP53 mutations in AML portend a dismal prognosis

- *TP53* mutations have been reported in approximately 12% to 13% of AML cases, and estimated median survival is less than 6 months, regardless of age or fitness
- *TP53* mutations are most common in AML with complex karyotype and present more frequently in patients ≥60 years old (approximately 27%)
- In therapy-related AML, the frequency of *TP53* mutations is approximately 23%
- Higher rates of relapse are seen after initial therapy, including treatment with allogeneic stem cell transplant

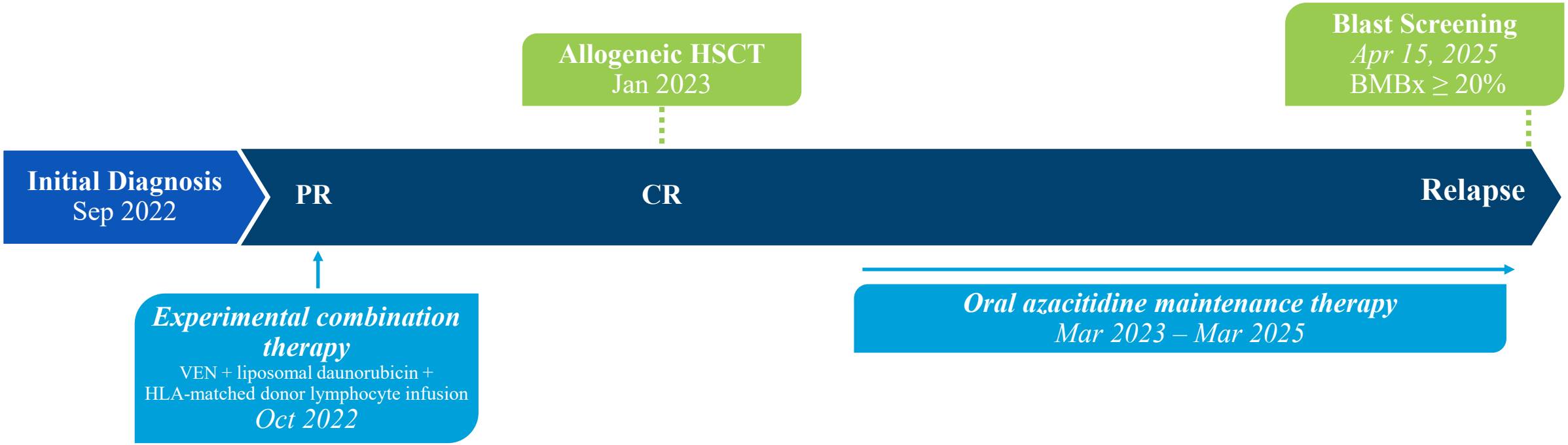
1. NCCN Guidelines Version 3.2026
2. Bataller, A., DiNardo, C.D., Bazinet, A., Daver, N.G., Maiti, A., Borthakur, G., Short, N., Sasaki, K., Jabbour, E.J., Issa, G.C., Pemmaraju, N., Yilmaz, M., Montalban-Bravo, G., Loghavi, S., Garcia-Manero, G., Ravandi, F., Kantarjian, H.M. and Kadia, T.M. (2024), Targetable genetic abnormalities in patients with acute myeloblastic leukemia across age groups. Am J Hematol, 99: 792-796. https://doi.org/10.1002/ajh.27236

Case Study: 70-year-old patient with *TP53*m AML – *before the study*

Patient demographics:

Age: 70 **Sex**: F **Race**: White

- **Diagnosis**: AML with MDS-related changes
- **Diagnosis Date**: Sep 2022
- **Genetics**: Complex karyotype, *TP53*m



**Illustrative only; patient case studies may not be representative of overall data set*

Case Study A: 70-year-old patient with *TP53*m AML – *on-study*

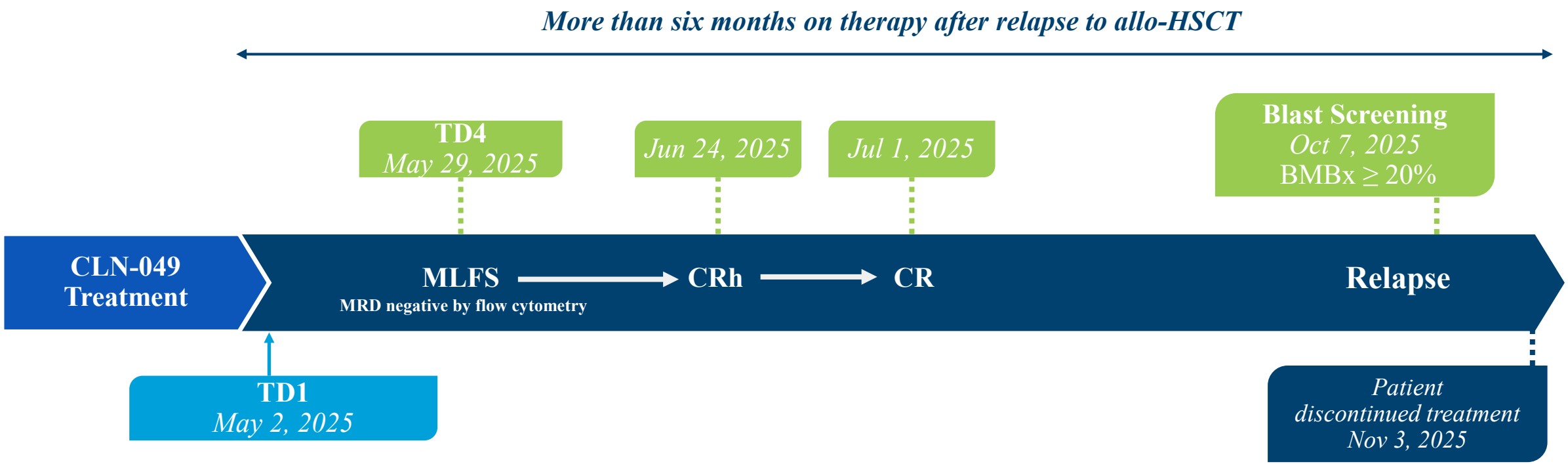
Patient Demographics:

Age: 70 **Sex**: F **Race**: White

- **Diagnosis**: AML with MDS-related changes
- **Diagnosis Date**: Sep 2022
- **Genetics**: Complex karyotype, *TP53*m
- **CLN-049 dose:** 1.5→6→12 µg/kg

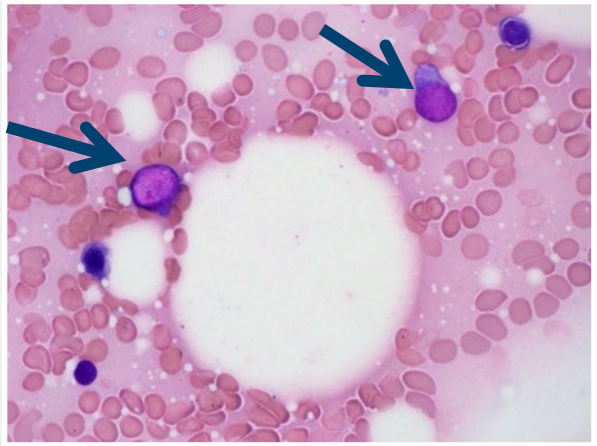
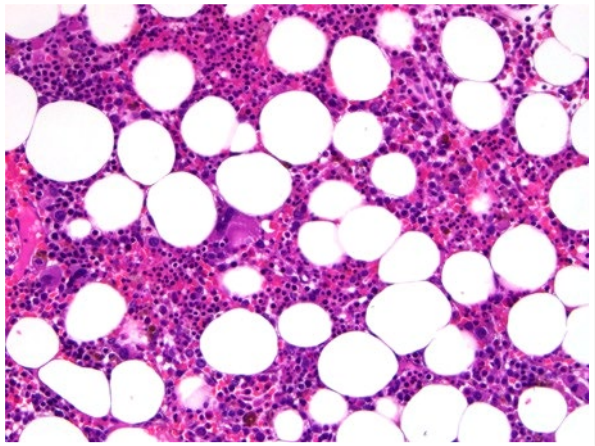
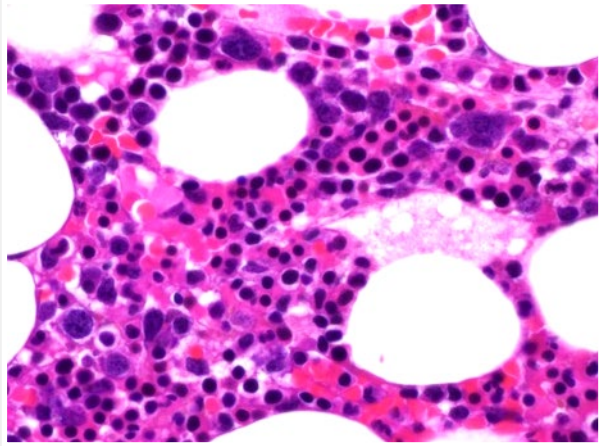
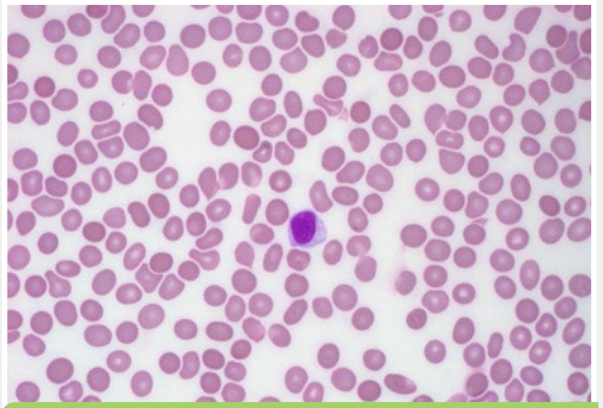
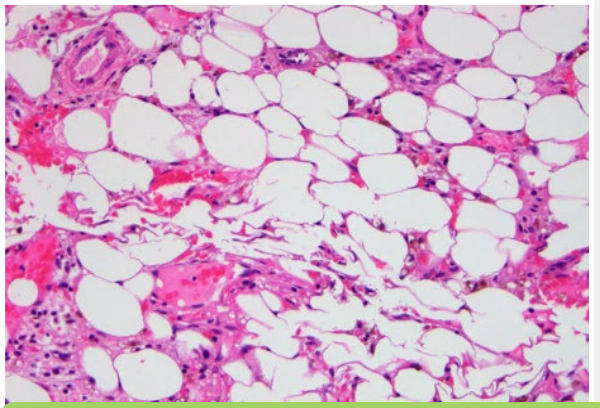
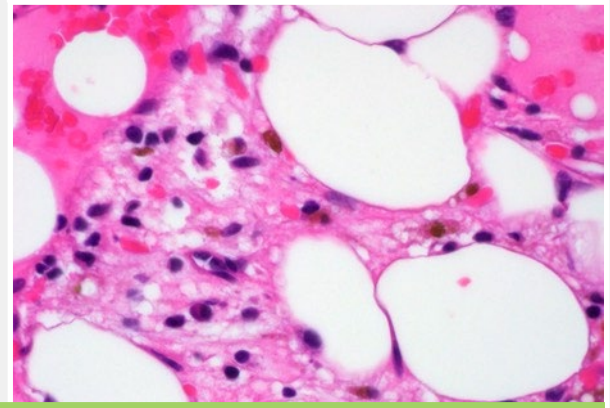
Prior Therapies

- VEN + liposomal daunorubicin + HLA-matched DLI
- Allogeneic HSCT
- Oral azacitidine



*Illustrative only; patient case studies may not be representative of overall data set

Case study: Bone marrow samples at baseline vs. post-therapy

	Bone marrow aspirate	Bone marrow core 1 (H&E x 200)	Bone marrow core 2 (H&E x 600)
Baseline prior to treatment – April 15, 2025			
MLFS response after TD4 - May 29, 2025			
	Elimination of AML blasts	**Eradication of AML blasts in hypercellular bone marrow**	

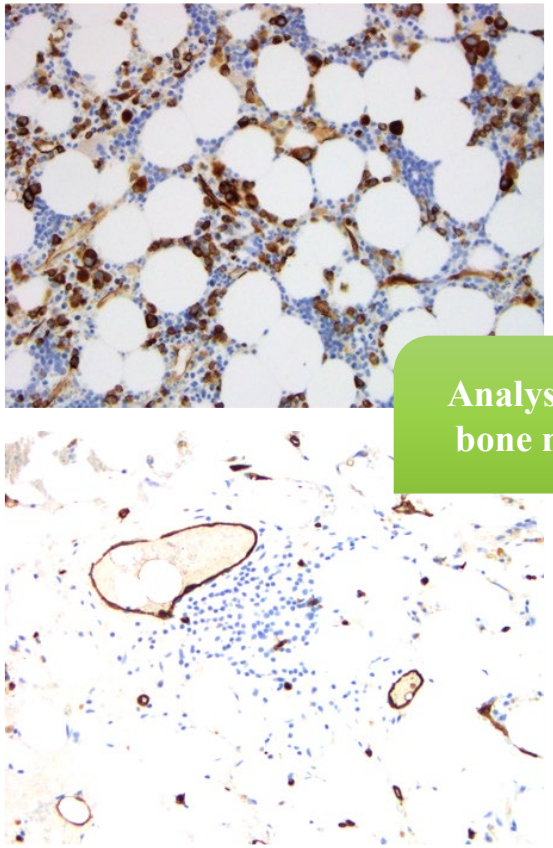
*Illustrative only; patient case studies may not be representative of overall data set

Case study: CD34 staining at baseline vs. post-therapy

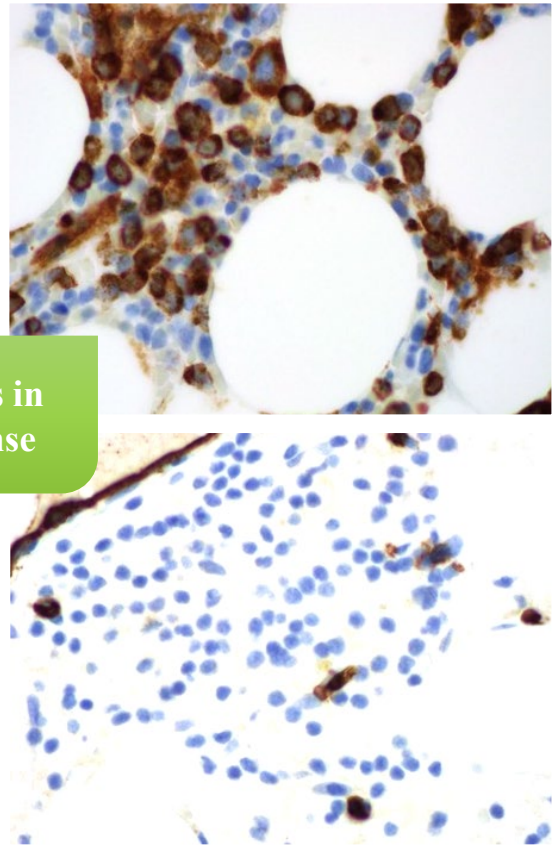
Baseline prior to treatment – April 15, 2025

MLFS response after TD4 - May 29, 2025

CD34 (immunoperoxidase x 200)



CD34 (immunoperoxidase x 600)

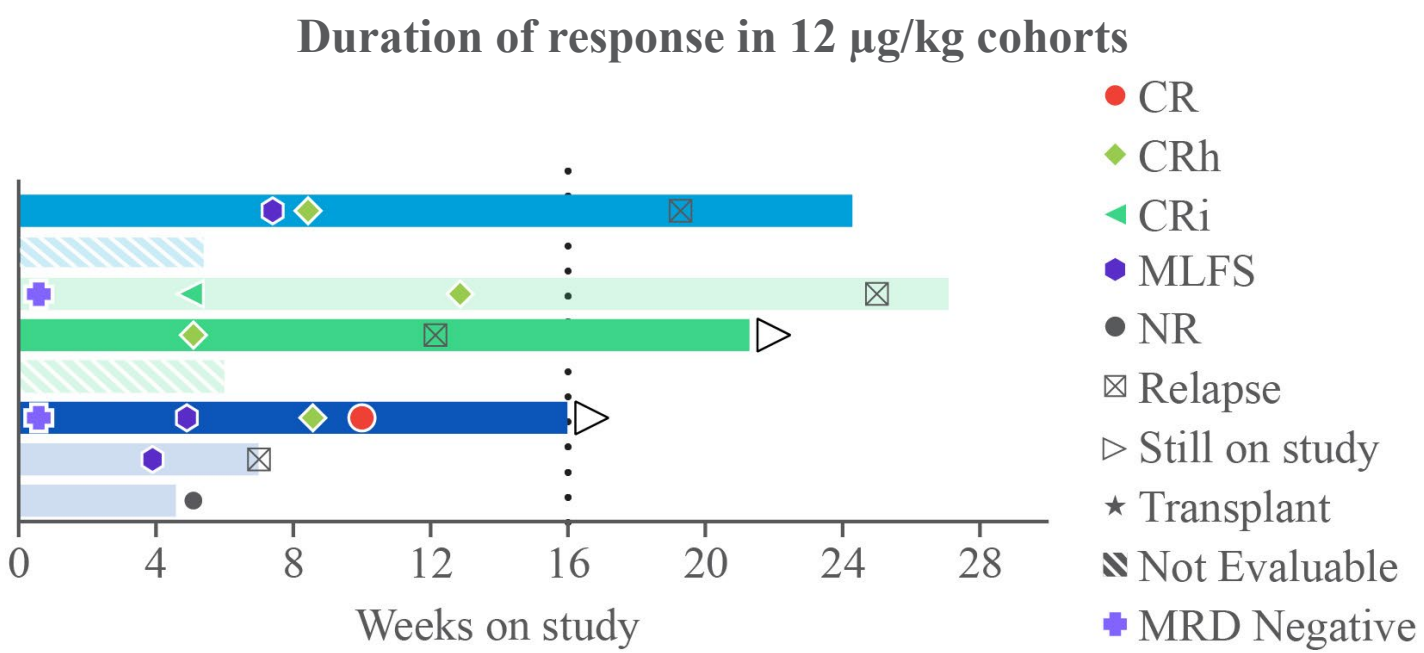


Analysis of CD34-positive cells in bone marrow confirms response

**Illustrative only; patient case studies may not be representative of overall data set*

Encouraging initial data suggests robust efficacy in TP53 mutated AML, among the hardest-to treat patient subsets

TP53-mutated AML response rate (best response), n (%)	All cohorts N=13	12 µg/kg cohorts N=8
CR	2 (15)	1 (13)
CR+CRh	5 (38)	4 (50)
CRC	5 (38)	4 (50)
ORR	6 (46)	5 (63)



- **4/8 patients with *TP53*-mutated AML treated at the highest TD of 12 µg/kg achieved CR or CRh**
- **CR+CRh responses were durable beyond 16 weeks in 3 of these patients**

August 2025 data cutoff

Strategic Perspective & Next Steps

Nadim Ahmed
Chief Executive Officer

Recent monotherapy approvals in R/R AML provide regulatory efficacy benchmarks for CLN-049 development

	Gilteritinib[1]	Enasidenib[2]	Ivosidenib[3]	Revumenib[4]	Revumenib[4]	Ziftomenib[5]
Year approved	2018	2017	2018	2024	2025	2025
Target population	FLT3	IDH2	IDH1	KMT2A	NPM1	NPM1
No. of patients	138	199	174	104	65	112
CR	11.6%	19%	24.7%	12.5%	18.5%	17.0%
CR+CRh	21%	23%	32.8%	21.2%	23.1%	21.4%
mDoCR+CRh	4.6 months	8.2 months	8.2 months	6.4 months	4.5 months	5.0 months

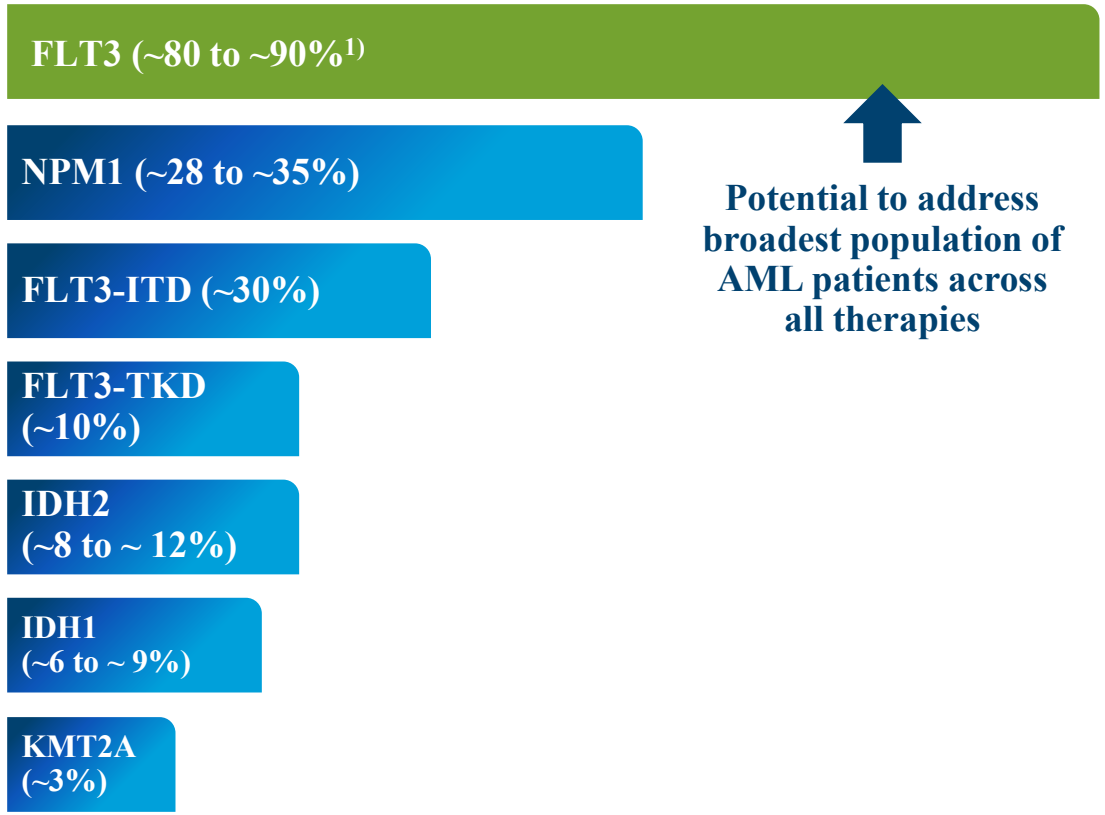
CR, complete remission; CRh, complete remission with partial hematologic recovery; FDA, US Food and Drug Administration; mDoCR+CRh, median duration of complete response/complete remission with partial hematologic recovery; R/R, relapsed/refractory.

- **Recent FDA approvals target molecularly defined subsets of R/R AML with single arm studies of ~ 100 patients**
- **Regulatory endpoints of relevance (CR+CRh and mDoCR+CRh) establish a reference benchmark -**
  - CR+CRh of 20% to 30% with response duration of approximately 4 to 6 months

1. https://www.accessdata.fda.gov/drugsatfda_docs/label/2019/211349s001lbl.pdf
2. https://www.accessdata.fda.gov/drugsatfda_docs/label/2025/209606s007lbl.pdf
3. https://www.accessdata.fda.gov/drugsatfda_docs/label/2021/211192_s008lbl.pdf
4. https://cms.syndax.com/wp-content/uploads/Revuforj-full-prescribing-info.pdf
5. https://kuraoncology.com/wp-content/uploads/prescribinginformation.pdf

CLN-049 has broad potential across all AML patient segments

Targeted therapies often apply to small subgroups, underscoring the fragmented and limited reach of current precision approaches



Reference: NCCN Guidelines Version 3.2026

The ability to address a **broad all comer patient population** with a potential **first-in-class therapy** represents a **sizeable commercial opportunity**

Due to the high rate of relapse in AML, the **R/R AML segment** alone represents a **$1B+ opportunity**[2] in estimated peak U.S. sales

Expansion to **1L setting** significantly **increases commercial opportunity** based on larger patient pool and increased treatment duration

1. Gebru, M.T., Wang, HG. Therapeutic targeting of FLT3 and associated drug resistance in acute myeloid leukemia. J Hematol Oncol 13, 155 (2020). https://doi.org/10.1186/s13045-020-00992-1
2. Internal company estimate - based on available historical sales data and consensus estimates for commercially approved drugs

CLN-049 AML development strategy: Clear and expeditious development pathway to regulatory approval

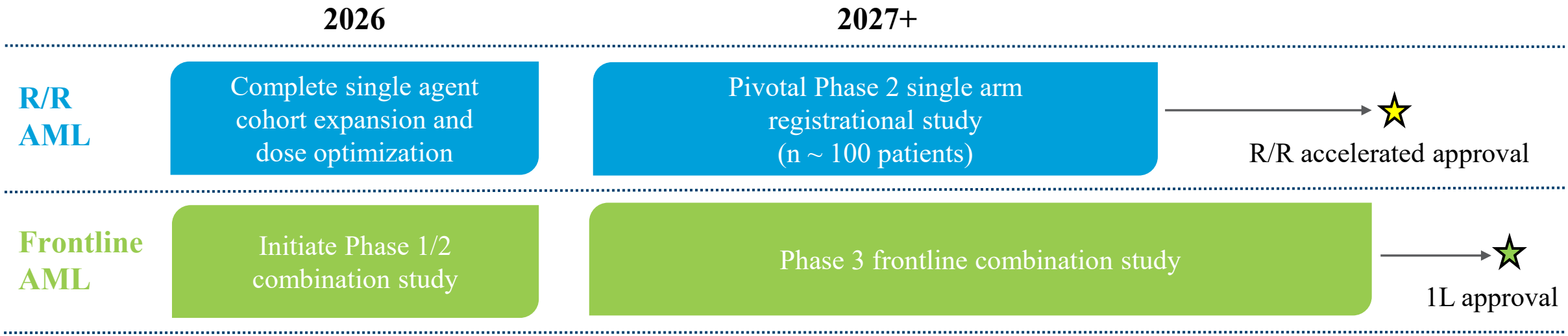
CLN-049 development strategy in AML

Monotherapy in R/R AML

- Execute single agent dose expansion/optimization study
- Identify RP2D and move to pivotal Phase 2 single arm registrational trial for accelerated approval

Combination therapy in 1L AML

- Generate initial POC data in combination with standard of care therapies in frontline AML
- Confirm efficacy and safety in randomized Phase 3 study for full approval and label expansion into frontline setting



CLN-049: Compelling efficacy enables accelerated approval pathway and drives attractive commercial opportunity

- **CLN-049 is a first-in-class differentiated molecule demonstrating compelling monotherapy efficacy with promising initial response durability and favorable safety in R/R AML; dose escalation is still ongoing**
- **CLN-049 can address a broad, all-comer population of AML patients with no biomarker testing required**
- **Rapid development under U.S. FDA Fast Track Designation for R/R AML**
- **Initial efficacy data for CLN-049 meets the benchmark for recently approved agents with clear pathway to accelerated approval in R/R AML**
  - Single-arm Phase 2 study likely sufficient to support initial registration via accelerated approval
- **Internal deep hematology expertise facilitates rapid development of CLN-049**
- **CLN-049 provides a commercially attractive opportunity in AML, with R/R segment alone representing a $1B+ opportunity[1]**

1. Internal company estimate - based on available historical sales data and consensus estimates for commercially approved drugs

Q&A

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THANK YOU!
cullinan
THERAPEUTICS