Company Presentation

June 2026

Enliven THERAPEUTICS

Disclaimer

This presentation contains forward-looking statements that involve substantial risks and uncertainties of Enliven Therapeutics, Inc. (“Enliven” or the “Company”). All statements other than statements of historical facts contained in this presentation, including statements regarding our future financial condition, results of operations, business strategy and plans, and objectives of management for future operations, as well as statements regarding industry trends, are forward-looking statements. Such forward-looking statements include, among other things; the potential of, potential market opportunities for, and expectations regarding ELVN-001; expectations regarding the positioning of ELVN-001 with respect to other therapies for chronic myeloid leukemia (CML) and across all lines of therapy, including the potential for ELVN-001 to be a best-in-class ATP-competitive tyrosine kinase inhibitor for the treatment of CML; the expected milestones and timing of such milestones for ELVN-001, including the timing of the initiation of, and expectations regarding the design of and enrollment for, a Phase 3 head-to-head trial of ELVN-001, the Phase 3 ENABLE-2 trial, a Phase 3 first line trial and Phase 2 first line trial; the potential for receiving broad first line approval; the timing for meeting with the Food and Drug Administration (FDA) regarding the end of Phase 2 of our ELVN-001 trial; and statements regarding Enliven’s financial position, including its liquidity, cash runway and the sufficiency of its cash resources. In some cases, you can identify forward-looking statements by terminology such as “estimate,” “intend,” “may,” “plan,” “potentially” “will” or the negative of these terms or other similar expressions.

ELVN-001 is an investigational drug candidate and has not been approved by the FDA or any other regulatory authority. The safety and efficacy of ELVN-001 have not been established, and this presentation is not intended to be, and should not be construed as, medical advice or a recommendation, offer or solicitation relating to ELVN-001 or any other product candidate. We have based these forward-looking statements largely on our current expectations and projections about future events and trends that we believe may affect our financial condition, results of operations, business strategy and financial needs. These forward-looking statements are subject to a number of risks, uncertainties and assumptions, including, among other things the potential for interim, topline and preliminary or case study results from Enliven’s clinical trials to materially change as additional patient data become available or following more comprehensive review; the potential for results from the ongoing or any future clinical trial of ELVN-001 to differ from the results of earlier trials of ELVN-001; ELVN-001 failing to demonstrate sufficient safety, efficacy, tolerability, durability, differentiated attributes or therapeutic benefit in current or future clinical trials; risks associated with unexpected events in trials of ELVN-001 (including the ENABLE trial and ENABLE-2 trial) including serious adverse events, toxicities, dose reductions, discontinuations or other undesirable side effects; delays or difficulties in recruiting, enrolling or maintaining patients in ELVN-001 clinical trials; the risks of delays in initiating or completing the ongoing or future trials of ELVN-001 (including the ENABLE trial and ENABLE-2 trial); Enliven failing to complete the ongoing ENABLE trial or to initiate or complete the ENABLE-2 trial, to present additional data, or to advance ELVN-001 through clinical development; regulatory authorities disagreeing with Enliven's clinical trial design, dose selection, endpoints or interpretation of data, or requiring additional studies or diagnostics; lack of reliability of cross-trial comparisons because the referenced data are derived from different clinical trials at different points in time, with differences in trial design and patient populations, and results may differ in head-to-head studies; developments relating to Enliven's competitors and industry which may affect the development or potential market opportunity for ELVN-001; and the potential inability of Enliven to obtain regulatory approval for, or ultimately commercialize or license, ELVN-001 or other product candidates; Enliven's limited resources; the ability to attract, hire, and retain highly skilled executive officers and employees; the ability of Enliven to protect its intellectual property and proprietary technologies; the scope of any patent protection Enliven obtains or the loss of any of Enliven's patent protection; reliance on third parties, including medical institutions, contract manufacturing organizations, contract research organizations and strategic partners; geo-political developments, general market or macroeconomic conditions; Enliven's ability to obtain additional capital to fund Enliven's general corporate activities and to fund Enliven's research and development; and other risks and uncertainties are more fully described in Enliven's filings with the Securities and Exchange Commission (SEC), including under the heading "Risk Factors" in Enliven's Annual and Quarterly Reports on Form 10-K and Form 10-Q filed with the SEC and in Enliven's future SEC filings. These risks are not exhaustive. New risk factors emerge from time to time, and it is not possible for our management to predict all risk factors, nor can we assess the impact of all factors on our business or the extent to which any factor, or combination of factors, may cause actual results to differ materially from those contained in, or implied by, any forward-looking statements. You should not rely upon forward-looking statements as predictions of future events. Although we believe that the expectations reflected in the forward-looking statements are reasonable, we cannot guarantee future results, levels of activity, performance or achievements. Except as required by law, we undertake no obligation to update publicly any forward-looking statements for any reason after the date of this presentation.

This presentation also contains estimates and other statistical data made by independent parties and by us relating to market size and growth and other data about our industry. This data involves a number of assumptions and limitations, and you are cautioned not to give undue weight to such estimates. The Company has not performed any head-to-head trials for ELVN-001. As a result, the data referenced in this presentation is derived from different clinical trials at different points in time, with differences in trial design and patient populations. As a result, conclusions from cross-trial comparisons cannot be made. In addition, projections, assumptions, and estimates of our future performance and the future performance of the markets in which we operate are necessarily subject to a high degree of uncertainty and risk.

ELVN-001 Has the Potential to Be the Best-in-Class TKI in CML

Unique & Complementary Design	Novel ATP-competitive BCR::ABL1 TKI with distinct binding and selectivity compared to the approved ATP-competitive inhibitors and differentiated mechanism compared to allosteric inhibitors
Built for Long Term Treatment	Designed specifically for long-term use in CML, including convenient once daily dosing with or without food and reduced DDI potential, to help patients not only live longer, but live better
Promising Efficacy	Promising efficacy observed across lines of therapy that appear favorable compared to approved BCR::ABL1 inhibitors
Favorable Safety and Tolerability	Demonstrated a favorable safety and tolerability profile compared to the ATP-competitive inhibitors with a potentially differentiated profile compared to asciminib
Opportunity Across Lines of Therapy	Potentially best-in-class ATP-competitive inhibitor with differentiated and complementary mechanism relative to allosteric inhibitors positions it to compete across all lines of therapy
Near-term catalysts	On track to initiate 2L+ pivotal trial and 1L Phase 2 IST in 2H 2026
Balance Sheet	Strong balance sheet with $452M of cash[1], providing runway into 1H 2029

1L = First line. ATP = Adenosine triphosphate. CML = Chronic myeloid leukemia. DDI – Drug-drug interactions.. TKI = Tyrosine kinase inhibitor. BCR::ABL1 = Breakpoint cluster region-Abelson leukemia virus. 1. 2L+ = Second line and later. IST = Investigator-Sponsored Trial. Note: DDI potential refers to the potential for concomitant administration with CYP3A4 substrates or inhibitors and proton pump inhibitors. [1] Comprised of cash, cash equivalents and marketable securities as of March 31, 2026 (unaudited). Conclusions from cross-trial comparisons cannot be made, and no head-to-head clinical trials have been conducted.

CML Landscape & ELVN-001 Introduction

CML Is Now a Chronic Disease for Most Patients

BCR-ABL TKIs have profoundly extended survival, but unmet needs remain

Unmet needs in CML…

Poor Tolerability Negatively Affects Quality of Life

- Approved TKIs are associated with toxicities such as pleural effusions, edema, and cardiovascular events that can meaningfully impact quality of life and long-term treatment
- Some carry boxed warnings noting sudden death and heart failure

Suboptimal Efficacy

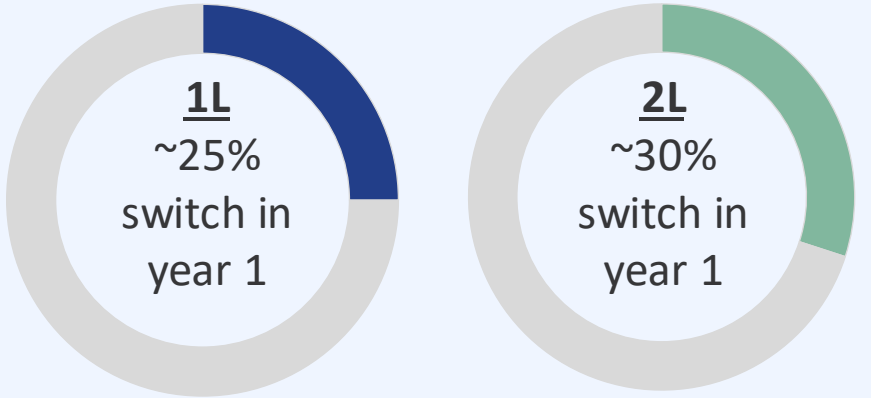
- **32%** of 1L patients do not reach MMR at 48 weeks
- **47%** of 2L patients do not reach MMR at 24 weeks
- **75%** of 3L+ patients do not reach MMR at 24 weeks

Treatment Burden and Adherence Issues

- Clinically meaningful drug–drug interactions
- Requirements to take with/without food

…drive real-world treatment changes

Switching Rates

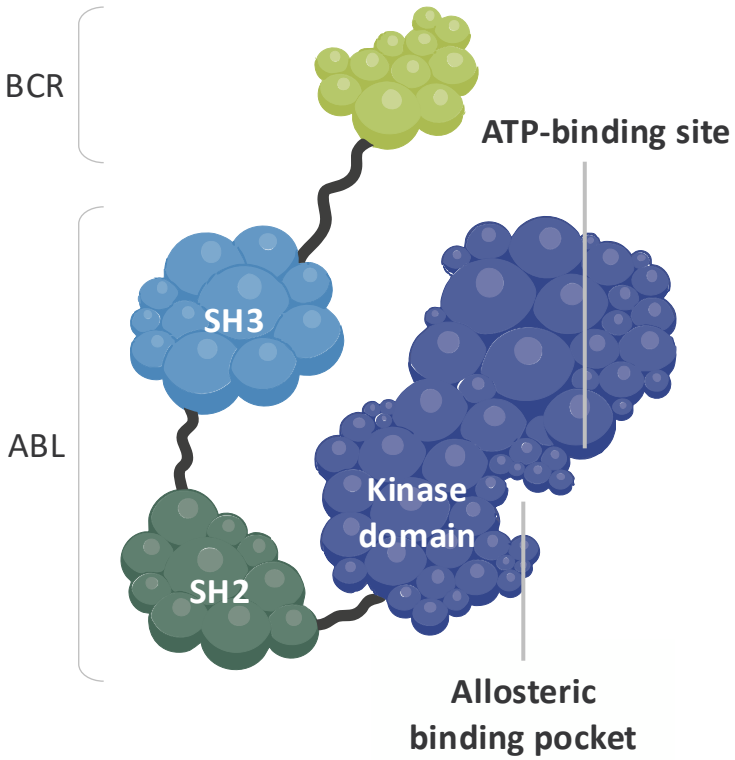


Despite multiple options, patients with CML regularly switch therapy seeking the optimal treatment for long-term use

BCR::ABL1 = Breakpoint cluster region-Abelson leukemia virus 1. CML = Chronic myeloid leukemia. MMR = Major molecular response. TKI = Tyrosine kinase inhibitors. 1L = First line. 2L = Second line. 3L+ = Third line or later. Note: suboptimal efficacy percentages refer to the rates of patients that did not reach MMR in asciminib clinical trials. References: Hochhaus A, et al. N Engl J Med 2024;391:885-898. 2024 Cortes JE, ASC2ESCLATE. Rea D et al. Blood 2021; 138 (21): 2031–2041. Kota V, et al. Presented at: ASH 2023; 31(7):1525-1531. USPIs of Approved Drugs for CML

Two Distinct Mechanisms to Treat CML – Yet Meaningful Gaps Remain

CML is a BCR-ABL driven disease, with two mechanistic approaches that offer different efficacy, tolerability, and resistance profiles



ATP-Competitive TKIs

Established the foundation for modern CML treatment

- Dramatically improved overall survival in CML
- 2nd and 3rd generation treatments enhanced molecular response and addressed resistance to imatinib

Key Limitations Remain

- Off-target toxicities can impair adherence and quality of life
- Safety and tolerability challenges can limit response, durability and persistence

Allosteric TKIs

Improved selectivity

- Resulting in improved tolerability profile and dose intensity
- Myristoyl pocket binding circumvents select ATP-site resistance mutations

Current and Emerging Challenges

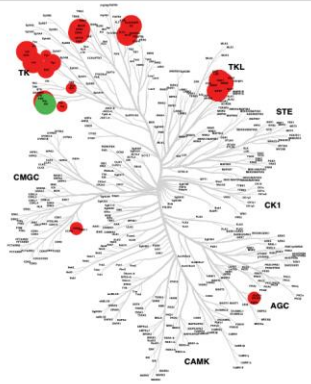
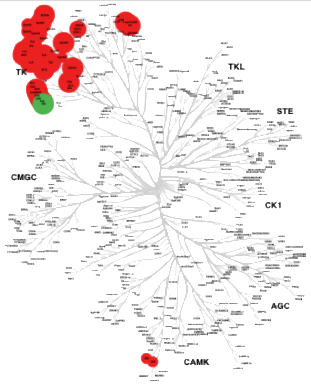
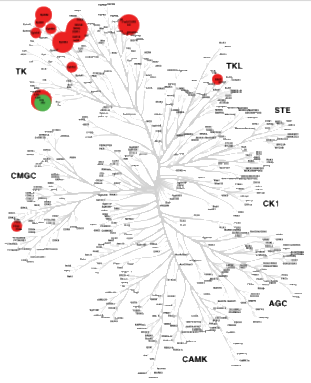
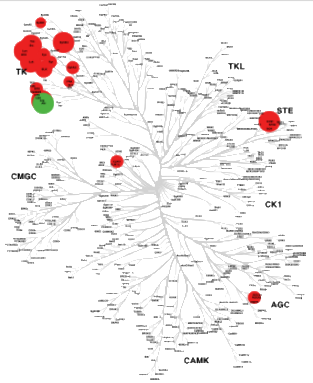
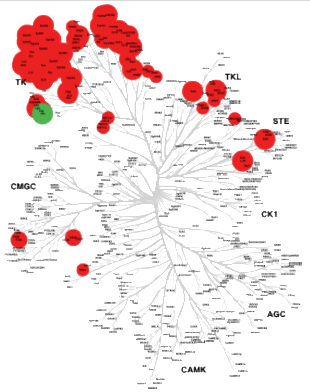
- Growing number and incidence of allosteric resistance mutations
- Treatment burden, including clinically meaningful DDIs and fasting requirements

ATP = Adenosine triphosphate. BCR::ABL1 = Breakpoint cluster region-Abelson leukemia virus 1. CML = Chronic myeloid leukemia. DDI = Drug-drug interaction. TKI = Tyrosine kinase inhibitor. References: Huang X et al. Cancer. 2012;118:3213-3127, Braun T. et al. Cancer Cell 2020 2020 Apr 13;37(4):530-542; Schoenbeck K et al., Blood 144 (2024) 7913–7914, CML NCCN Guidelines V1.2026, Collins JB et al., J Oncol Pharm Practice 2024, Vol. 30(2) 385–396, Hughes et al, NEJM 2019, Cortes J et al, ASCO 2026, USPIs of Approved Drugs for CML.

Approved ATP-Competitive TKIs Are Limited by Off-target Effects

Broad kinome inhibition by all approved ATP-competitive CML TKIs can lead to clinically meaningful toxicities

Previously Approved ATP-Competitive TKIs

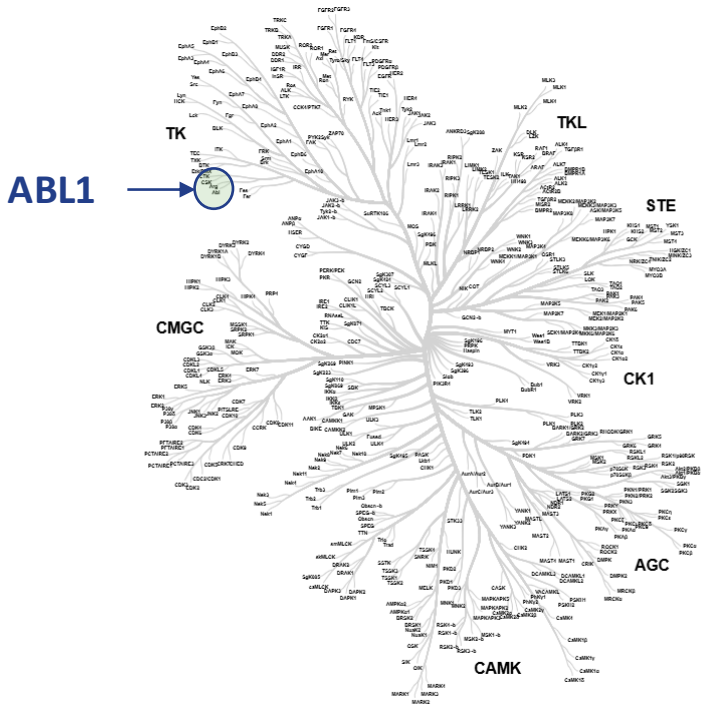
	Imatinib (Gleevec®)	Dasatinib (Spyrcel®)	Nilotinib (Tasigna®)	Bosutinib (Bosulif®)	Ponatinib (Iclusig®)
Off-target kinase activity	KIT, CSFR-1, PDGFR	SRC family, KIT, PDGFR	KIT, PDGFR, CSFR-1, DDR-1, (hERG Channel)	SRC family	KDR, FGFR, KIT, RET, FLT3, PDGFR
Chronic safety & tolerability burden	Peripheral Edema and Nausea	Fluid Retention, Pleural Effusions, Diarrhea	Rash, Headache, GI Toxicity, **Boxed Warning**: QT Prolongation (Sudden Death) Risk	Hepatic Dysfunction, Diarrhea, Abdominal Pain	**Boxed Warning**: Arterial Occlusive Events, Heart Failure, VTE, Hepatotoxicity
Broad kinome inhibition					

ABL1 = Abelson murine leukemia viral oncogene homolog 1. ATP= Adenosine triphosphate. IC_{50} = Half-maximal inhibitory concentration. C_{95} = 95% inhibitory concentration. CML = Chronic myeloid leukemia. GI = gastrointestinal. TKI= Tyrosine kinase inhibitor. VTE = Venous thromboembolism Notes: 1. Kinase inhibition assay with: ABL1 enzyme concentration 0.2 nM, 100 uM ATP Off-target kinases activity and selected safety/tolerability findings derived from published kinases selectivity analyses, pivotal clinical studies, and US prescribing information for approved CML TKIs. References: Pophali PA and Patnaik MM. Cancer J. 2016 ; 22(1): 40–50, Lipton et al. Science Direct Blood Reviews 2022 100968

ELVN-001: Purpose-Built for Best-in-Class Potential in CML

Selectivity is the foundation of a molecule designed for lifelong CML treatment

High Specificity



Differentiated Design

Novel binding mode and enhanced selectivity

- Uniquely binds the P-loop "folded-in" active conformation of ABL1
- High specificity for BCR::ABL1 reduces off-target toxicities
- Enables efficacy-optimized dosing through improved tolerability profile

Broad mutational coverage

- Designed to be active against clinically important mutations that can confer resistance to other ATP-competitive inhibitors**,** including T315I
- Retained activity against emerging allosteric resistance mutations, including those known to drive resistance to asciminib

Differentiated PK and dosing profile

- Potential for concomitant administration with CYP3A4 substrates, inhibitors, inducers and PPIs
- Not a substrate for common efflux transporters (P-gp, BCRP), avoiding resistance mediated by these transporters
- Once-daily dosing with no food restrictions

Unlike other active site TKIs, ELVN-001 did not inhibit any kinases other than ABL1 >50% at 30x IC50 (~IC95) ABL1= Abelson murine leukemia viral oncogene homolog 1. IC_{50} = Half-maximal inhibitory concentration. IC_{95} = 95% inhibitory concentration. ATP = Adenosine triphosphate. BCR::ABL1 = Breakpoint cluster region-Abelson leukemia virus 1. TKI= Tyrosine kinase inhibitor. BCRP = Breast Cancer Resistance Protein. CML = Chronic myeloid leukemia. P-gp = P-glycoprotein. PK = Pharmacokinetics. Notes: 1. Kinase inhibition assay with: ABL1 enzyme concentration 0.2 nM, 100 uM ATP. Aspirational information: ELVN-001 is an investigational drug and is not approved by any regulatory agency, and its safety and efficacy have not been established.

ELVN-001 Has the Potential to Compete Across All Lines of Therapy

Future treatment paradigm expected to sequence ATP-competitive inhibitors and allosteric inhibitors

Treatment Pathways				Market Insights and Assumptions
1L	Generic ATP-competitive TKI	Allosteric TKI	**ELVN-001**	• ELVN-001 data suggest **highly competitive efficacy, safety and ease of use profile** relative to currently approved therapies • Later-line efficacy and safety have **historically predicted earlier line outcomes** for CML TKIs
2L	Allosteric TKI	**ELVN-001**	Allosteric TKI	• **Switching mechanisms of action** is common in oncology to address acquired resistance or intolerance to certain adverse events • We believe ELVN-001's ATP-competitive, active site mechanism of action **complements allosteric TKIs, potentially supporting sequencing** and **combination strategies**
3L+	**ELVN-001**	ATP-competitive or Allosteric, based on patient need		• Asciminib's successful 3L+ launch **highlights a multibillion-dollar late-line** market opportunity for improved CML therapies • **The high frequency of late-line switching highlights the unmet need** for well–tolerated, effective therapies

1L = First line. 2L = Second line. 2L+ = Second line or later. 3L+ = Third line or later. ATP = Adenosine triphosphate. CML = Chronic myeloid leukemia. TKI = Tyrosine kinase inhibitor. Note: Illustrative current and future treatment paradigm. Conclusions from cross-trial comparisons cannot be made, and no head-to-head clinical trials have been conducted.
References: HCP Qualitative & Quantitative Interviews (ClearView). Public company filings and announcements.

Initial Pivotal Trial Creates Path to 2L+ CML Access; Future Pivotal Trial Could Double the Opportunity

U.S. Branded CML Market has the Potential to be ~$10B

U.S. Patient Population: ~110K

2L+

~$5B market
55K patients

Phase 3 ENABLE-2
2L+ Patients with CML
Expected initiation in 2H **2026**

1L

~$5B market
55K patients

Phase 3 1L
1L Patients with CML
Potential initiation in **2028**

ELVN-001 2026 Key Milestones

- ✓ EHA Phase 1b data presentation
- ✓ End-of-Phase 1 meeting
- Q3 2026: End-of-Phase 2 meeting planned
- 2H 2026: Planned Initiation of Phase 3 ENABLE-2

- Initiate 1L Phase 2 IST
- Solicit health authority input on requirements to initiate 1L Phase 3

IST = Investigator-Sponsored Trial. 1L = First line. 2L+ = Second line and later. B = Billion. 2H – second half. Q3 = third quarter. K = thousands. CML = Chronic myeloid leukemia. EHA = European Hematology Association. Notes: Percent of patient breakdown by line of therapy is based on HCP Qualitative & Quantitative Interviews (ClearView); U.S. branded CML market calculated using total U.S. 2015 branded sales and adjusting those figures for the pricing of CML drugs today and today's increased prevalence. References: public company filings and announcements and research reports; Huang X et al. Cancer. 2012;118:3213-3127. Am J Hematol. 2024 Nov;99(11):2191-2212.

ELVN-001 Is a Differentiated CML Inhibitor Expected to Begin a Phase 3 2L+ Pivotal Trial this Year

Successful EOP1 Meeting

Key outcomes of FDA meeting:

- ✓ 80 mg as recommended Phase 3 dose
- ✓ 2L+ patient population for Phase 3 trial
- ✓ Comparator arm of physician's choice of ATP-competitive TKIs

Completed key step to initiating 2L+ pivotal trial

Encouraging Efficacy

- ✓ 48% MMR achieved by 24 weeks at 80 mg QD
- ✓ 55% MMR achieved by 24 weeks in 2L/3L patients
- ✓ Consistent MMR rates in patients with or without prior asciminib, across all lines of therapy

Further derisks pivotal trial and market positioning

Favorable Safety and Tolerability Profile

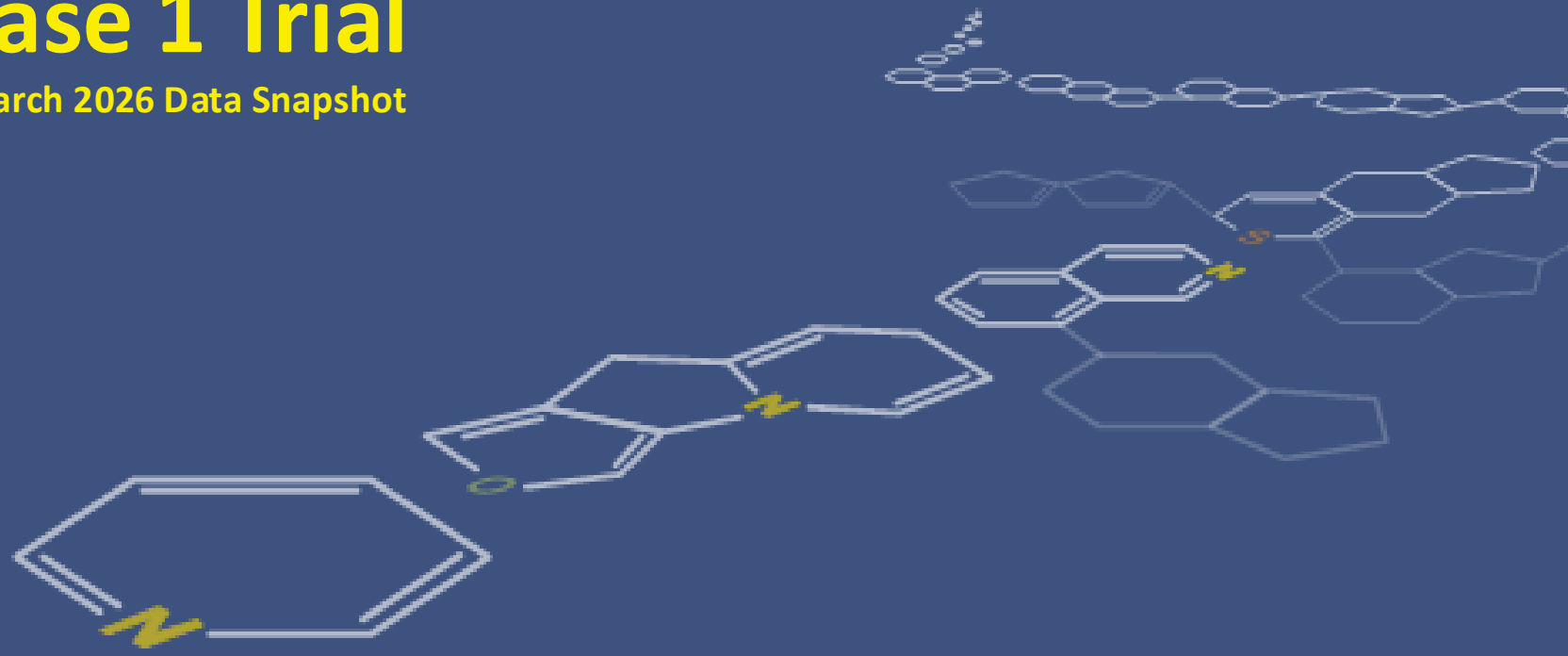
- ✓ AE profile compares favorably to precedent trials of other ATP-competitive TKIs
- ✓ Early evidence of potentially differentiated hematologic AE profile
- ✓ Well tolerated: only 6% discontinuations due to AE

Emerging data support selectivity and favorable safety profile

AE = adverse event. ATP = adenosine triphosphate. CML = Chronic myeloid leukemia. MMR = Major molecular response. QD = Once daily. TKI = Tyrosine kinase inhibitor. 1L = First line. 2L+ = Second line or later. 3L = Third line. EOP1= End of Phase One. FDA = Federal Drug Administration. Conclusions from cross-trial comparisons cannot be made, and no head-to-head clinical trials have been conducted.

ELVN-001 Phase 1 Trial

EHA 2026 Presentation || 10 March 2026 Data Snapshot



ELVN-001: Phase 1 Trial Design and Status

Key Eligibility Criteria:

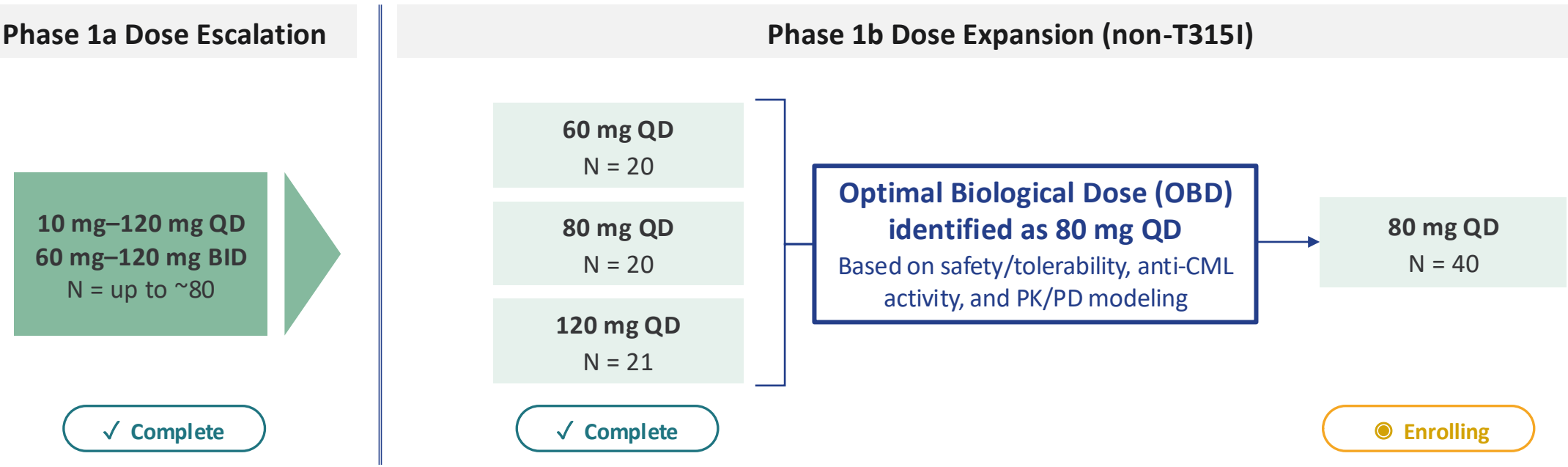
- Chronic phase CML
- Failed, intolerant to, or not a candidate for available therapies known to be active for treatment of their CML
- Typical or atypical transcripts

Primary Endpoints

- Incidence of DLTs, AEs, clinically significant laboratory and ECG abnormalities

Key Secondary Endpoints

- Molecular response by central qPCR
- PK parameters



Phase 1 is ongoing (NCT05304377). AE, adverse event; CML, chronic myeloid leukemia; DLT, dose-limiting toxicity; ECG, electrocardiogram; BID, twice daily; OBD, optimal biological dose; CML, chronic myeloid leukemia; PD, pharmacodynamics; PK, pharmacokinetics; QD, once daily; qPCR, quantitative polymerase chain reaction.

Patient Demographics and Baseline Characteristics

Parameter	All Patients (N = 161[a])	80 mg QD (Phase 1b) (N = 49)
Median age in years (range)	59 (19–82)	57 (19–82)
Typical BCR::ABL1 transcript (e13a2/e14a2), n (%)	150 (93%)	47 (96%)
Baseline BCR::ABL1 transcript[b] ≤0.1%	26 (17%)	8 (17%)
>0.1% – 1%	37 (25%)	11 (23%)
>1% – 10%	28 (19%)	10 (21%)
>10%	50 (33%)	10 (21%)
Baseline BCR::ABL1 mutation (Sanger), n (%)	39 (24%)	5 (10%)
T315I mutation	12 (7%)	n/a
Mutations associated with resistance to allosteric inhibition	13 (8%)	2 (4%)
Other mutations	9 (6%)	3 (6%)

[a] Includes 3 re-enrolled patients (158 individual patients)

Parameter	All Patients (N = 161[a])	80 mg QD (Phase 1b) (N = 49)
Median **lines** of prior TKI therapy, n (range)	4 (1–10)	3 (1–9)
Median number of prior **unique** TKIs, n (range)	3 (1–7)	3 (1–6)
1–2 prior, n (%)	46 (29%)	19 (39%)
3 prior, n (%)	39 (24%)	11 (22%)
4 prior, n (%)	37 (23%)	11 (22%)
≥ 5 prior, n (%)	37 (23%)	7 (14%)
Prior asciminib or ponatinib at any time, n (%)		
Asciminib[b]	100 (62%)	27 (55%)
Ponatinib	60 (37%)	13 (27%)
Reason for discontinuation of last TKI, n (%)[c]		
Lack of efficacy	99 (61%)	25 (51%)
Lack of tolerability	51 (32%)	20 (41%)

[b] Most received asciminib in late line and the majority discontinued asciminib due to lack of efficacy
[c] Unknown/other in 9 patients (all patients) and 3 patients (80 mg QD)

Majority of patients received ≥ 3 prior TKIs (70%) and prior ponatinib and/or asciminib (72%)

Data cutoff: 10 March 2026.
QD, once daily; TKI, tyrosine kinase inhibitor; BCR::ABL1, Breakpoint cluster region-Abelson leukemia virus 1.

Patient Disposition: Majority of Patients Remain on Study

Disposition	All Patients (N = 161)	80 mg QD (Phase 1b) (N = 49)
Median duration of exposure, weeks (range)	35 (0.1–156)	16.1 (0.3–86.7)
Ongoing, n (%)	123 (76%)	40 (82%)
Discontinued, total n (%)	38 (24%)	9 (18%)
Lack of efficacy	21 (13%)	1 (2.0%)
Adverse event	10 (6.2%)	3 (6.1%)
Death	1 (0.6%)	1 (2.0%)
Other[a]	6 (3.7%)	4 (8.2%)

[a] Protocol violation, investigator decision, and consent withdrawal

Overall, 146 person-years of exposure and 118 patients treated at doses ≥ 80 mg QD

At 80 mg QD in phase 1b:

- 82% of patients remain on study
- Current median duration of exposure of 16 weeks (cohort actively enrolling)
- Low rate of discontinuation due to adverse events

- No patients progressed to blast phase
- Death was due to post-operative complication (not related to ELVN-001)

Data cutoff: 10 March 2026.
QD, once daily. Note: death was previously reported at EHA 2025.

ELVN-001 had Favorable Safety and Tolerability

- **Wide therapeutic window**
  - 80 mg BID determined to be MTD:
    - 2 patients with DLT at 120 mg BID (one Grade 3 hallucination in patient with history of hallucinations and one Grade 3 myalgia)
  - Low incidence of AOEs*:
    - 7 patients (4.4%) with any grade; 3 (1.9%) with Grade 3, all with CV disease/risk factors incl. prior nilotinib and/or ponatinib
- **Safety of 80 mg QD similar to overall safety; determination of optimal biological dose based on PK/PD modeling**

Most Common TEAEs (by Preferred Term), n (%)	All Patients (N = 158 Safety Analysis Set)		80 mg QD (N = 62 Safety Analysis Set)	
	Any	≥ Grade 3	Any	≥ Grade 3
Any TEAE	142 (90%)	53 (34%)	51 (82%)	15 (24%)
Hematologic TEAEs ≥ 5%				
Thrombocytopenia[a]	23 (15%)	10 (6.3%)	10 (16%)	4 (6.5%)
Neutropenia[a]	11 (7.0%)	10 (6.3%)	1 (1.6%)	0
Non-hematologic TEAEs ≥ 10%				
Lipase increased	35 (22%)	9 (5.7%)[b]	13 (21%)	3 (4.8%)[b]
Fatigue	28 (18%)	2 (1.3%)[b]	7 (11%)	1 (1.6%)[b]
Headache	24 (15%)	1 (0.6%)[b]	11 (18%)	1 (1.6%)[b]
Arthralgia	22 (14%)	2 (1.3%)[b]	10 (16%)	1 (1.6%)[b]
Myalgia	22 (14%)	1 (0.6%)[b]	7 (11%)	0
Nausea	19 (12%)	0	8 (13%)	0
Diarrhea	18 (11%)	0	6 (9.7%)	0
Amylase increased	16 (10%)	0	5 (8.1%)	0

[a] Grouped terms: platelet count decreased/thrombocytopenia and neutrophil count decreased/neutropenia
[b] Grade 3 only

Data cutoff: 10 Mar 2026.

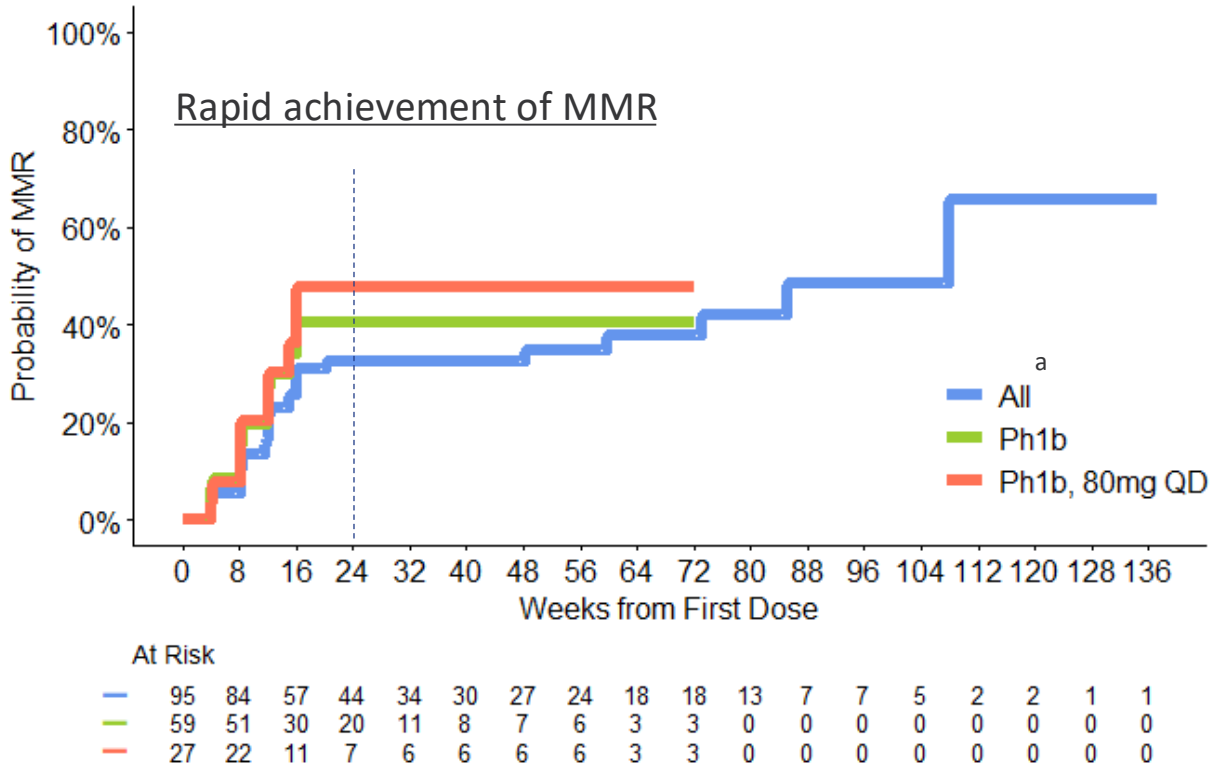
AOE, arterial occlusive event; BID, twice daily; CNS, central nervous system; CV, cardiovascular; DLT, dose-limiting toxicity; MTD, maximum tolerated dose; PD, pharmacodynamics; PK, pharmacokinetics; QD, once daily; TEAE, treatment-emergent adverse event. *AOEs identified by MedDRA Queries (embolic, thrombotic, arterial; ischemic CNS vascular; other ischemic heart disease; myocardial infarction). TEAE of hypertension, any grade, reported in 5.7% of patients, 1.9% Grade 3.

Encouraging Anti-CML Activity in Phase 1b

Key Efficacy Milestones by Week 24 for Phase 1b

	Total QD (Phase 1b) (N = 90)	80 mg QD (Phase 1b) (N = 49)
Median exposure (weeks)	28.0 (0.3-86.7)	16.1 (0.3-86.7)
MMR (*BCR::ABL1* ≤ 0.1%)		
Overall MMR	37/69 (54%)	17/28 (61%)
MMR achieved	21/53 (40%)	10/21 (48%)
MMR maintained	16/16 (100%)	7/7 (100%)
MR2 (*BCR::ABL1* ≤ 1%)		
MR2 achieved	19/33 (58%)	9/13 (69%)
DMR (*BCR::ABL1* ≤ 0.01%)		
DMR achieved	14/64 (22%)	8/27 (30%)

Cumulative Incidence of MMR in Patients Not in MMR at Baseline



[a]Patients enrolled to phase 1a were allowed to increase the dose if certain criteria were met

NOTE: Patients were included if they had baseline typical BCR::ABL1 transcript, and postbaseline assessment of BCR::ABL1 transcript at 24 weeks or achieved MMR/≤1% within 24 weeks or discontinued treatment before 24 weeks without achieving MMR/≤1%. For patients with MMR/≤1% at baseline, only postbaseline assessments beyond 70 days were included in the analysis.

Data cutoff: 10 Mar 2026.

DMR, deep molecular response; QD, once daily; MMR, major molecular response; MR, molecular response; BCR::ABL1, Breakpoint cluster region-Abelson leukemia virus 1.

All Patients in Phase 1b had Improved or Stable MR Category

Change in *BCR::ABL1* Transcript in Patients Evaluable for MMR by Week 24 (N = 69)

Improvement in MR category
No category change
Worsening in MR category

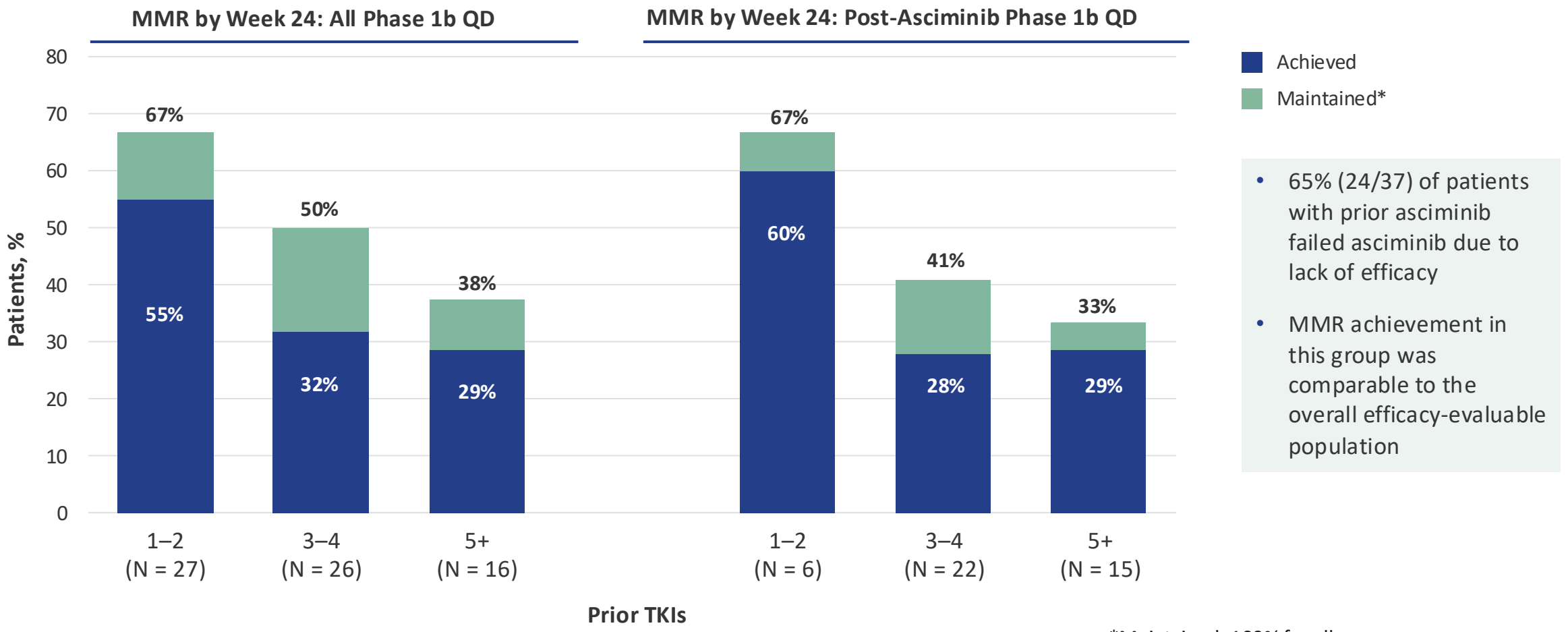
BCR::ABL1 transcript (%) by Week 24	*BCR::ABL1* transcript (%) at baseline					
	≥MR4.5 ≤0.0032 (N = 4)	**MR4** > 0.0032 to 0.01 (N = 1)	**MR3** > 0.01 to 0.1 (N = 11)	> 0.1 to 1 (N = 21)	> 1 to 10 (N = 15)	> 10 (N = 17)
≥MR4.5 ≤0.0032	4	1	4	5	2	
MR4 > 0.0032 to 0.01			1	1	1	
MR3 > 0.01 to 0.1			6	6	4	2
> 0.1 to 1				9	4	5
> 1 to 10					4	3
> 10						7

In subgroup with baseline transcript >10%:

59% (10/17) had improved MR category

Data cutoff: 10 Mar 2026.
MMR, major molecular response; MR, molecular response; BCR::ABL1, Breakpoint cluster region-Abelson leukemia virus 1.
NOTE: Evaluable patients had baseline typical BCR::ABL1 transcript without T315I mutation and post-baseline assessment of BCR::ABL1 transcript at 24 weeks or achieved MMR within 24 weeks or discontinued treatment before 24 weeks without achieving MMR. For patients with MMR at baseline, only post-baseline assessments beyond 70 days were included in the analysis.

Encouraging MMR Rates Across Lines of Therapy and After Asciminib Failure



- 65% (24/37) of patients with prior asciminib failed asciminib due to lack of efficacy
- MMR achievement in this group was comparable to the overall efficacy-evaluable population

*Maintained: 100% for all groups

Data cutoff: 10 Mar 2026.
MMR, major molecular response; TKI, tyrosine kinase inhibitor; QD, once daily.
NOTE: Analysis by number of prior TKIs done with prior *unique* TKIs. Evaluable patients had baseline typical BCR::ABL1 transcript without T315I mutation and post-baseline assessment of BCR::ABL1 transcript at 24 weeks or achieved MMR within 24 weeks or discontinued treatment before 24 weeks without achieving MMR. For patients with MMR at baseline, only post-baseline assessments beyond 70 days were included in the analysis.

Rapid Deep Molecular Response in Patient with Myristoyl Pocket Mutation

Patient Information

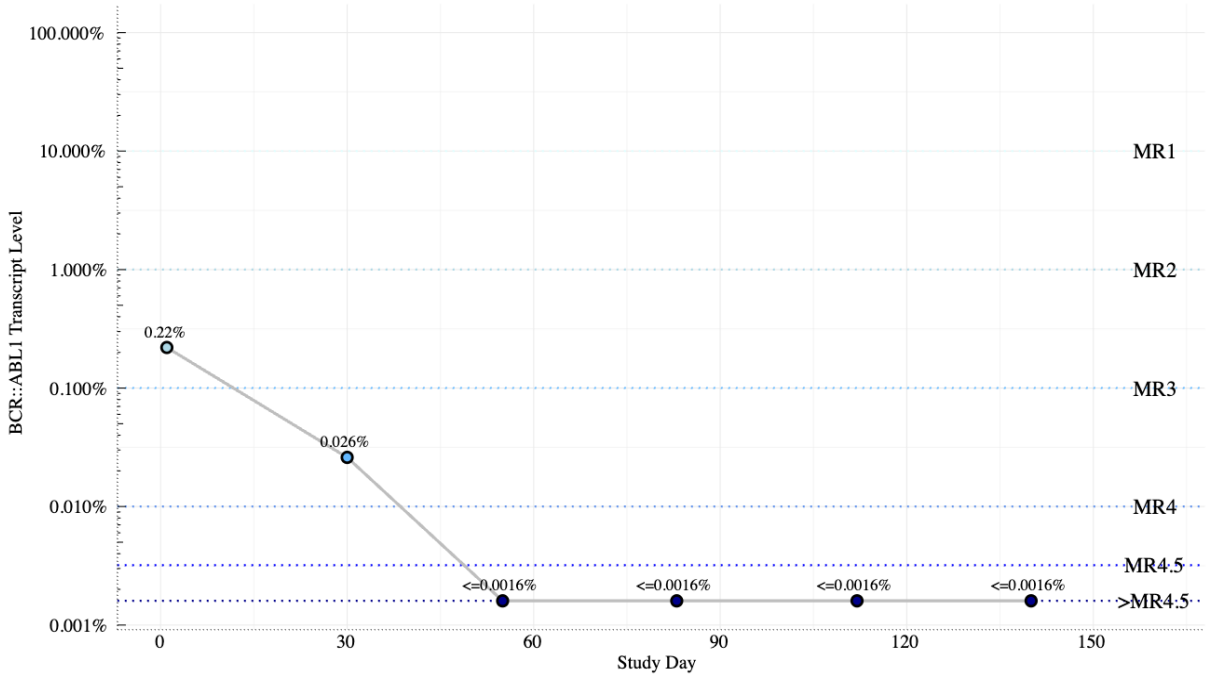
- 57-year-old female
- Primary diagnosis of CML in 2021
- Pertinent medical history: pericardial effusion, fibromyalgia, venous insufficiency, sciatica
- Central mutation[a]: A344D

Prior TKI	Reason for Discontinuation	Time on Treatment
Imatinib	Lack of efficacy	268 days
Bosutinib	Lack of efficacy	256 days
Asciminib	Lack of efficacy (A344D)	1034 days

Treatment-Emergent Adverse Events on ELVN-001 80 mg QD

- Grade 1 diarrhea (R), Grade 1 dry skin (NR), Grade 2 sciatica (NR), Grade 1 headache (NR), Grade 1 bronchitis (NR), Grade 1 foot fracture (NR)

BCR::ABL1 Transcript on ELVN-001 80 mg QD



NR, not related to ELVN-001 per investigator; R, potentially related to ELVN-001 per investigator.

[a]100% by Sanger Sequencing; 99.7% by Next Generation Sequencing

Data cutoff: 27 May 2026. BCR::ABL1 = Breakpoint cluster region-Abelson leukemia virus. 1. CML = chronic myeloid leukemia. TKI = tyrosine kinase inhibitor. QD = Once Daily.

Major Molecular Response in Patient with T315I Mutation

Patient Information

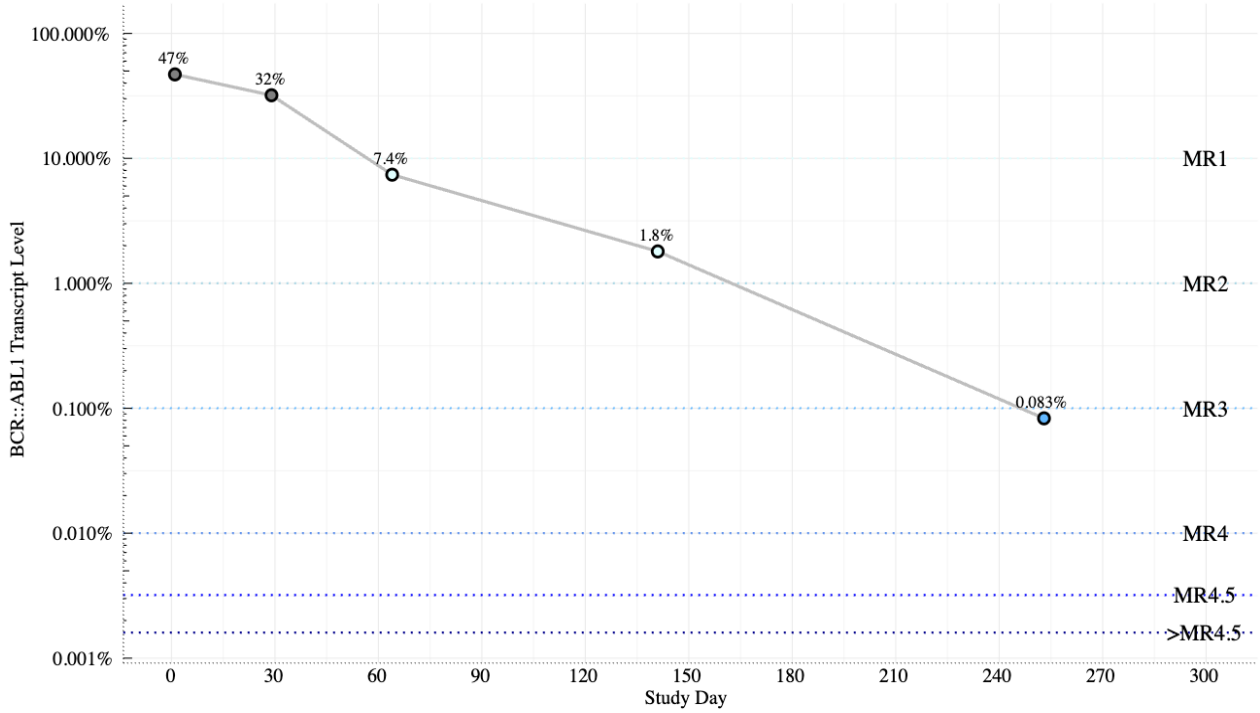
- 72-year-old male
- Primary diagnosis of CML in 2023
- Pertinent medical history: arterial hypertension
- Central mutation[a]: T315I

Prior TKI	Reason for Discontinuation	Time on Treatment
Nilotinib	Leg pain	164 days
Imatinib	Lack of efficacy	214 days
Asciminib	Lack of efficacy (T315I)	186 days

Treatment-Emergent Adverse Events on ELVN-001 80 mg BID

- Grade 1 peripheral edema (R), Grade 1 bronchitis (NR), Grade 1 dry skin (R), Grade 1 hypophosphatemia (R)

BCR::ABL1 Transcript on ELVN-001 80 mg BID



NR, not related to ELVN-001 per investigator; R, potentially related to ELVN-001 per investigator.

[a]100% by Sanger Sequencing; 99% by Next Generation Sequencing

Data cutoff: 27 May 2026. BCR::ABL1 = Breakpoint cluster region-Abelson leukemia virus. 1. CML = chronic myeloid leukemia. TKI = tyrosine kinase inhibitor. BID = twice daily.

Conclusions

Key take aways from the updated data

- ✓ The optimal biological dose (Phase 3 dose) was identified as 80 mg QD
- ✓ With longer follow-up and more patients enrolled, ELVN-001 showed a favorable safety profile, with low discontinuation rate due to adverse events, and most TEAEs being low grade
- ✓ Encouraging anti-CML activity was observed in a heavily pre-treated patient population, including in patients with prior asciminib exposure
- ✓ Case studies support mechanistic rationale in resistance mutations

We believe these data provide a strong foundation for the next stage of clinical development

TEAE = treatment-emergent adverse event. CML = chronic myeloid leukemia.

Key Takeaways and Next Steps for ELVN-001

Patients Enrolled Materially Affect MMR Achievement Rates

Cross-Trial MMR comparisons require careful interpretation

Differences in Enrollment Criteria Influence Patient Population Composition

	Asciminib	Tern-701 / MK-4208	ELVN-001
Initial Enrollment Criteria	3L+ patients	2L+ patients, excluding asciminib resistance	Not candidate for available therapies
1st / 2nd / 3rd Gen TKI resistance/ Intolerance	✓	✓	✓
Asciminib intolerant		✓	✓
Asciminib resistance			✓

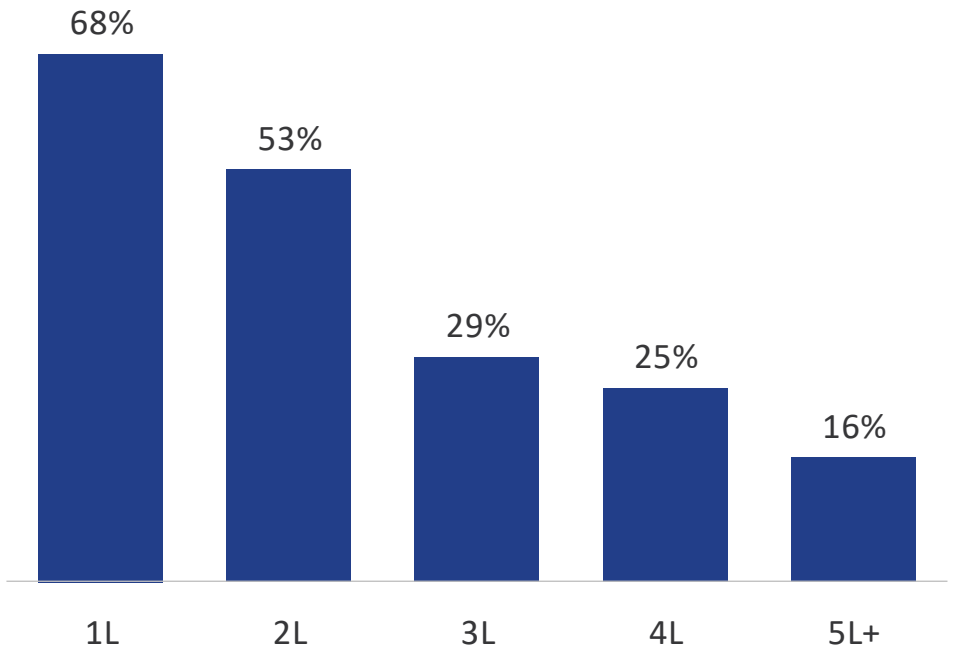
As a result of this criteria, the ENABLE trial enrolled a patient population that has a higher proportion of:

- **Patients with 4+ prior TKIs**
- **Patients with prior asciminib and/or ponatinib exposure**
- **Patients who discontinued asciminib due to resistance**

MMR Declines by Therapy Line

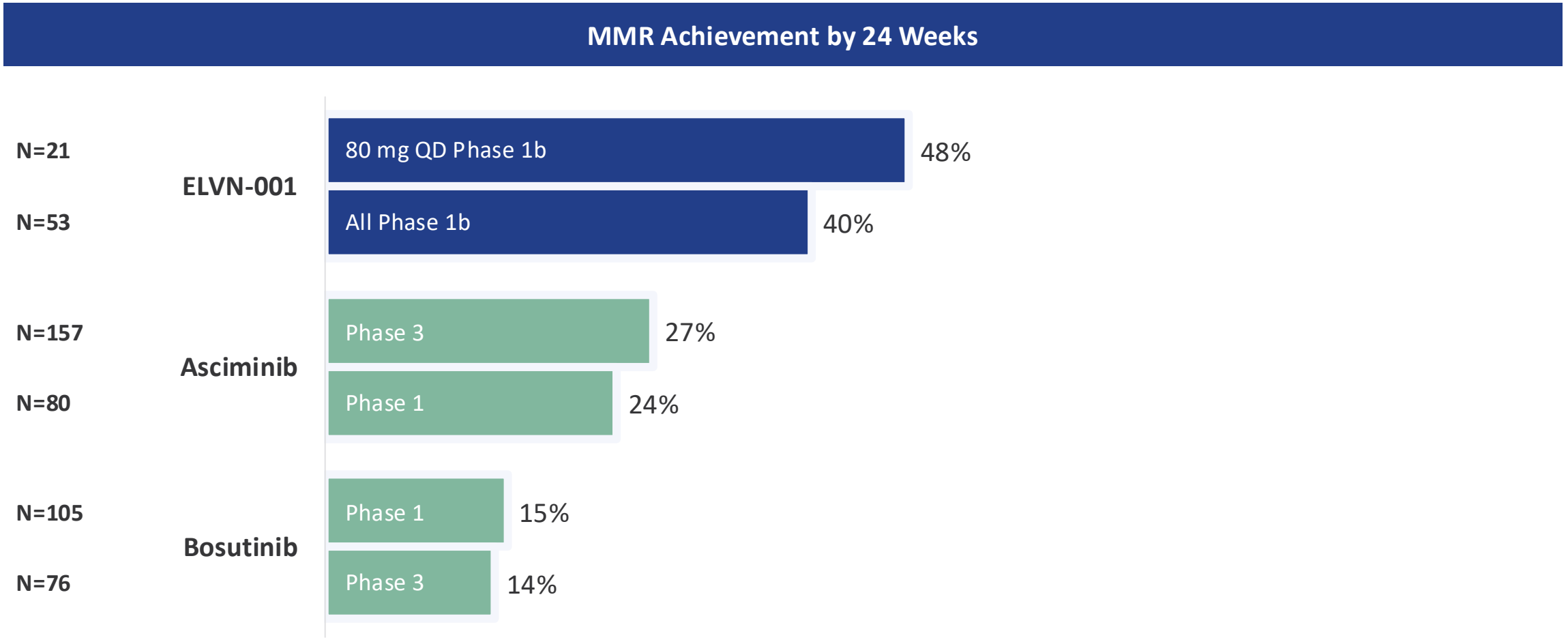
Achieved MMR rates based on precedent asciminib studies

>3x difference in MMR rate between 2L and 5L+



1L = First line. 2L = Second Line. 3L = Third line. 4L = Fourth line. 4L+ = Fourth line and later. 5L+ = Fifth line and later. Gen = generation. MMR = Major molecular response. TKI = Tyrosine kinase inhibitor. References: Scemblix (asciminib) USPI; Cortes JE, ASC2ESCLATE, ASH 2025 Oral Presentation; Réa D et al, Blood. 2021;138(21):2031-2041. (SUPPL); Hughes TP, et al. N Engl J Med. 2019;381(24):2315-2326. Jabbour E. et al, CARDINAL Study TiP, SOHO 2024. Note: 68% for 1L is the MMR at Week 48, while the other bars are MMR at Week 24. Conclusions from cross-trial comparisons cannot be made, and no head-to-head clinical trials have been conducted.

To date, ELVN-001 Data Compares Favorably to the Last Two Approved Drugs in CML



CML = Chronic myeloid leukemia. MMR = Major molecular response. Data cutoff: March 10, 2026.
Notes: ELVN-001 (80 mg) refers to the Phase 1b 80 mg QD patients in the ENABLE trial. MMR is defined as BCR::ABL1 ≤ 0.1%. a. MMR rates includes all evaluable patients treated who had typical BCR::ABL1 transcripts without T315I mutation b. Refers to ≥ 3 prior TKIs. c. MMR in patients with BCR::ABL1 transcript > 0.1% at baseline. d. MMR in patients with BCR::ABL1 transcript ≤ 0.1% at baseline. These data are derived from different clinical trials at different points in time, with differences in trial design and patient populations. As a result, conclusions from cross-trial comparisons cannot be made, and no head-to-head clinical trials have been conducted. References: Hughes et al., NEJM 2019. Khoury HJ et al. Blood. 2012. Asciminib USPI. Asciminib NDA Filing.

Potentially Differentiated Safety & Tolerability Profile

Hematologic Toxicity

	ELVN-001 Phase 1 Trial[1] (N = 158)		Scemblix USPI[3] Asciminib	
Laboratory Value	Any %	Grade 3-4 %	Any %	Grade 3-4 %
Thrombocytopenia	25	7	46	24
Neutropenia	18	8	43	22

Non-Hematologic Adverse Events

	ELVN-001 (N = 158)		Asciminib	
Treatment Emergent Adverse Events	Any %	Grade 3-4 %	Any %	Grade 3-4 %
Arterial Occlusive Events	4.4	1.9	12	2.5
Hypertension	5.7	1.9	14	7

Compared with asciminib label

- Lower rates of thrombocytopenia and neutropenia
- Lower rates of arterial occlusive events and hypertension

Differentiation is potentially a result of ELVN-001's selectivity

- ELVN-001 is selective for ABL1 vs ABL2, unlike allosteric inhibitors, which may contribute to lower rates of hematologic and vascular toxicity[2,4,5,6]

1 Local laboratories and adverse events reported in the ongoing ELVN-001 Phase 1; snapshot date 10 March 2026. 2 Gross et al, ASH 2022. 3 SCEMBLIX USPI; preferred terms from ASCEMBL and AOEs from Section 5.5. 4 ABL2 is involved in cytoskeletal dynamics, cell adhesion and signaling in marrow. (Greuber et al Nature Reviews 2013) 5 More complete ABL-family inhibition leads to weaker endothelial survival signaling and greater dysfunction which potentially causes more vascular toxicity (Chislock et al PNAS 2013). 6 Eadie et al Blood 2024. ABL1 = Abelson leukemia virus 1. ABL2= Abelson murine leukemia viral oncogene homolog 2 gene. These data are based on comparisons to published data from independent studies. Conclusions from cross-trial comparisons cannot be made, and no head-to-head clinical trials have been conducted.

Successful FDA Interaction Supports 2L+ Pivotal Trial Design

Key Outcomes from recent End-of-Phase 1 Meeting with the FDA

- ✓ 80 mg QD as recommended Phase 3 dose
- ✓ 2L+ patient population for Phase 3 trial
- ✓ Comparator arm of physician's choice of ATP-competitive TKIs (excludes asciminib and ponatinib)

End-of-Phase 2 meeting with the FDA expected in 3Q 2026 to finalize the Phase 3 protocol

Expect to initiate the 2L+ pivotal trial later this year

ATP = Adenosine triphosphate. FDA = Federal Drug Administration. TKI = Tyrosine kinase inhibitors. 2L+ = Second line or later. 3Q = Third Quarter. QD = Once Daily. FDA = Federal Drug Administration.

ELVN-001 Data Supports Broad Registrational Development in CML

24-week MMR has readthrough from Phase 1 to Phase 3 trials in relapsed/refractory CML

ENABLE Phase 1 Trial
Previously treated Patients with CML

ENABLE-2: Phase 3 2L+ CML Trial
Expected initiation in **2H 2026**

Potential initial approval in 2L+

Anticipated 2L+ trial design:

- ELVN-001 vs. physician's choice of ATP-competitive inhibitor
- Proposed primary endpoint of MMR achievement at 24 weeks
- Includes post-asciminib population
- Potential to demonstrate best-in-class ATP-competitive inhibitor

Phase 3 1L CML Trial
Potential initiation in **2028**

Potential broad CML approval

Potential 1L trial design:

- ELVN-001 vs physician's choice ATP-competitive inhibitor, potential to include asciminib
- Primary endpoint of MMR achievement at 48 weeks
- Potential to demonstrate best-in-disease TKI

ATP = Adenosine triphosphate. TKI = Tyrosine kinase inhibitors. 1L = First line. 2L+ = Second line or later. CML = Chronic Myeloid Leukemia . MMR = Major Molecular Response.

ELVN-001 Has the Potential to Be the Best-in-Class TKI in CML

Unique & Complementary Design	Novel ATP-competitive BCR::ABL1 TKI with distinct binding and selectivity compared to the approved ATP-competitive inhibitors and differentiated mechanism compared to allosteric inhibitors
Built for Long Term Treatment	Designed specifically for long-term use in CML, including convenient once daily dosing with or without food and reduced DDI potential, to help patients not only live longer, but live better
Promising Efficacy	Promising efficacy observed across lines of therapy that appear favorable compared to approved BCR::ABL1 inhibitors
Favorable Safety and Tolerability	Demonstrated a favorable safety and tolerability profile compared to the ATP-competitive inhibitors with a potentially differentiated profile compared to asciminib
Opportunity Across Lines of Therapy	Potentially best-in-class ATP-competitive inhibitor with differentiated and complementary mechanism relative to allosteric inhibitors positions it to compete across all lines of therapy
Near-term catalysts	On track to initiate 2L+ pivotal trial and 1L Phase 2 IST in 2H 2026
Balance Sheet	Strong balance sheet with $452M of cash[1], providing runway into 1H 2029

1L = First line. ATP = Adenosine triphosphate. CML = Chronic myeloid leukemia. DDI – Drug-drug interactions.. TKI = Tyrosine kinase inhibitor. BCR::ABL1 = Breakpoint cluster region-Abelson leukemia virus. 1. 2L+ = Second line and later. IST = Investigator-Sponsored Trial. M = million. 1H = first half. Note: DDI potential refers to the potential for concomitant administration with CYP3A4 substrates or inhibitors and proton pump inhibitors. [1] Comprised of cash, cash equivalents and marketable securities as of March 31, 2026 (unaudited). Conclusions from cross-trial comparisons cannot be made, and no head-to-head clinical trials have been conducted.

Thank you