Company Presentation

January 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; 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; 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.

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 limited operating history of Enliven; the ability to advance product candidates through preclinical and clinical development; the ability to obtain regulatory approval for, and ultimately commercialize or license, product candidates; the outcome of preclinical testing and early clinical trials for product candidates and the potential that the outcome of preclinical testing and early clinical trials may not be predictive of the success of later clinical trials; Enliven's limited resources; the risk of failing to demonstrate safety and efficacy of product candidates; Enliven's limited experience as a company in designing and conducting clinical trials; the potential for interim, topline and preliminary data from Enliven's preclinical studies and clinical trials to materially change from the final data; potential delays or difficulties in the enrollment or maintenance of patients in clinical trials; developments relating to Enliven's competitors and its industry, including competing product candidates and therapies; the potential market opportunity for any of Enliven's programs; the decision to develop or seek strategic collaborations to develop Enliven's current or future product candidates in combination with other therapies and the cost of combination therapies; 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; geopolitical developments, general market or macroeconomic conditions; and Enliven's ability to obtain additional capital to fund Enliven's general corporate activities and to fund Enliven's research and development. Information regarding the foregoing and additional risks may be found in the section entitled "Risk Factors" in documents that Enliven files from time to time with the Securities and Exchange Commission. 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. Data presented for ELVN-001 are not based on head-to-head trials and are based on publicly available data, which include cross-trial and/or cross-phase data and may not be directly comparable. 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.

Highly Distinguished & Industry-Leading Team

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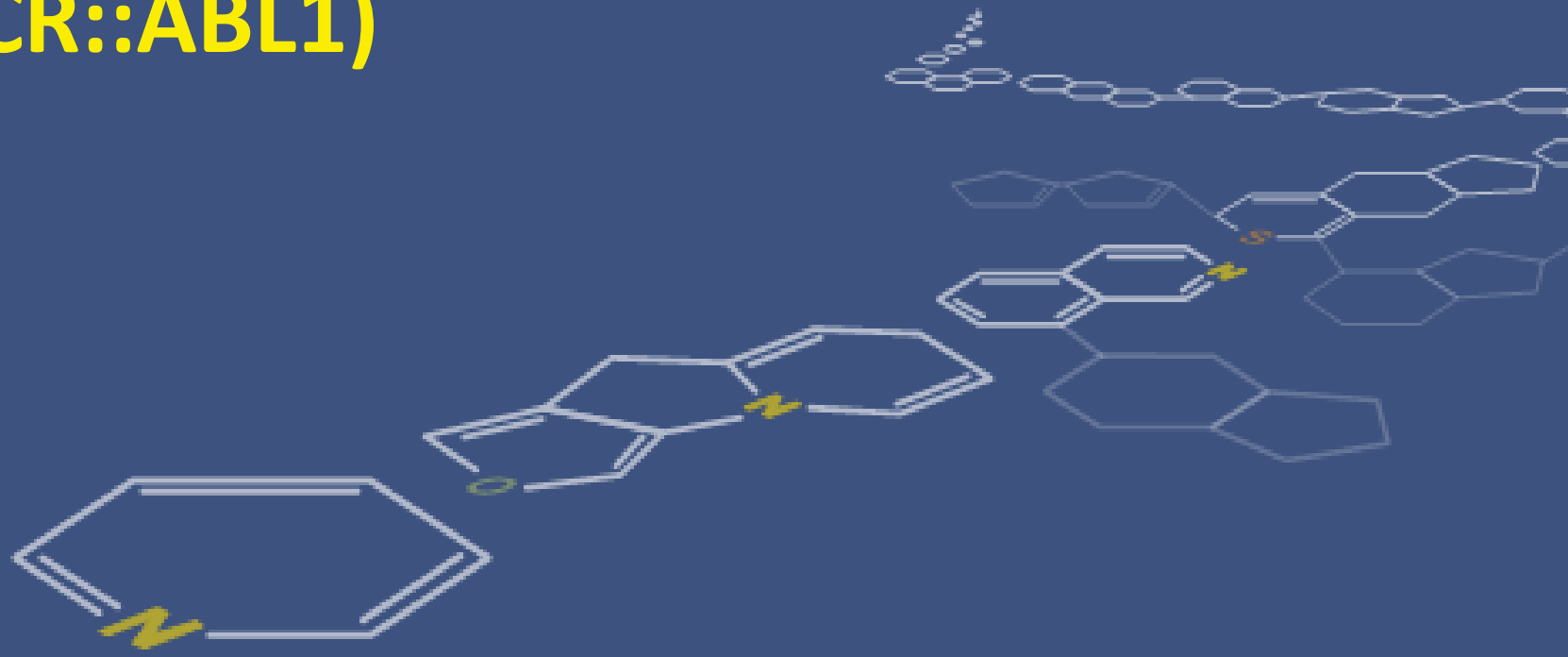
Pipeline & Discovery Programs Address New and Emerging Unmet Needs

Program	Target	Differentiation	Disease	Regimen	Discovery	IND-Enabling	Phase 1	Phase 2	Phase 3	Next Milestone	Milestone Expected
ELVN-001	BCR-ABL	Highly selective active site inhibitor w/activity against asciminib emergent mutations	CML	Monotherapy						Phase 3 Initiation	2H 2026

Multiple discovery stage efforts ongoing at various stages

BCR-ABL = Breakpoint cluster region-Abelson leukemia virus. CML = Chronic myeloid leukemia.

ELVN-001 (BCR::ABL1)



ELVN-001: Well-positioned to Compete in a Large CML Market

Large CML Market Opportunity

BCR::ABL1 TKIs have historically generated ~$6B of combined annual sales, despite generic options

Potential $9B opportunity for differentiated TKIs in the U.S. alone, validated by successful Scemblix launch

Unique and Complementary Design

ELVN-001 is the only selective ATP-competitive BCR::ABL1 TKI and has activity against asciminib-resistant mutations

PK supports QD dosing with or without food, and has low risk of DDIs

Differentiated Efficacy & Safety Profile

ELVN-001 efficacy, safety and tolerability profile in late line CML compare favorably to approved BCR::ABL1 inhibitors despite a more heavily pretreated patient population

Defined Regulatory Path

Phase 1 data predicted performance in precedent registrational trials; all CML TKIs have been approved based on a biomarker endpoint

Expected to initiate first ELVN-001 head-to-head pivotal trial in 2026

Enliven has a strong balance sheet expected to provide cash runway into 1H 2029

ATP = Adenosine triphosphate. BCR::ABL1 = Breakpoint cluster region-Abelson leukemia virus 1. CML = Chronic myeloid leukemia. DDI = Drug-drug interactions. QD = Once daily. PK = Pharmacokinetics. TKI = Tyrosine kinase inhibitor.
Note: U.S. CML market assumes branded pricing and is calculated based on historical sales while adjusting for current prevalence and pricing. References: public company filings and announcements.
Conclusions from cross-trial comparisons cannot be made, and no head-to-head clinical trials have been conducted. ELVN-001 data reported on June 13th, 2025.

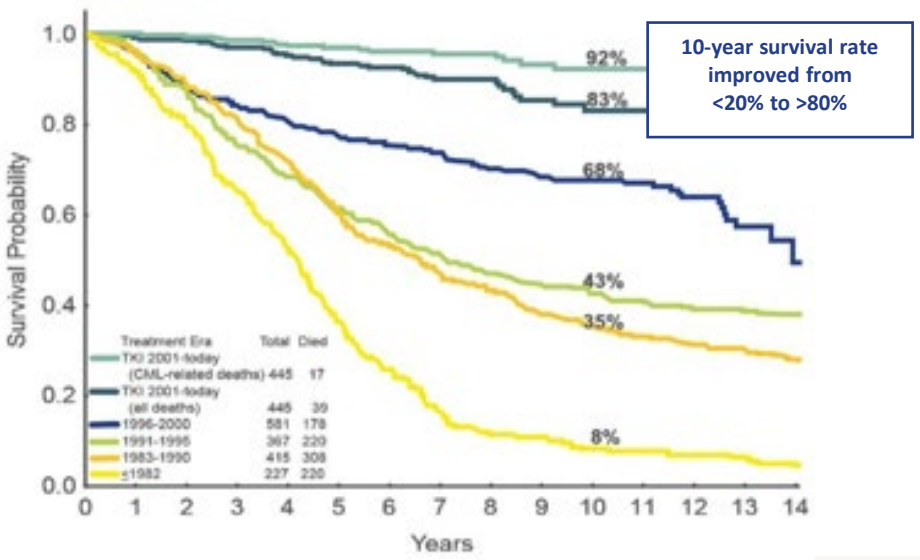
CML is Now a Long-Term Condition

As patients live longer on treatment, **quality of life** and **tolerability** have become important treatment goals

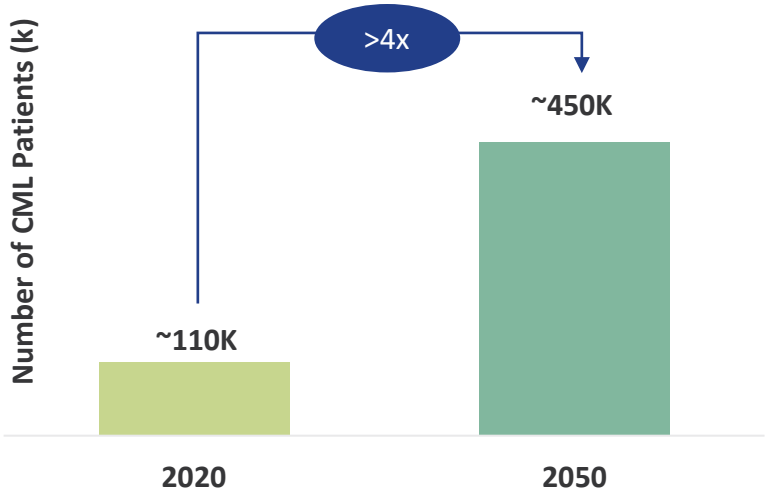
Prior to imatinib, the annual CML survival rate was

<20%

CML 10-Year Survival Rate Over Time



Estimated Prevalence of CML in the U.S. Over Time



- Prevalence is increasing globally with expected overall survival approaching age-matched controls
- CML has become a chronic disease that can require life-long TKI-treatment

Top Treatment Goals for Physicians and Patients*

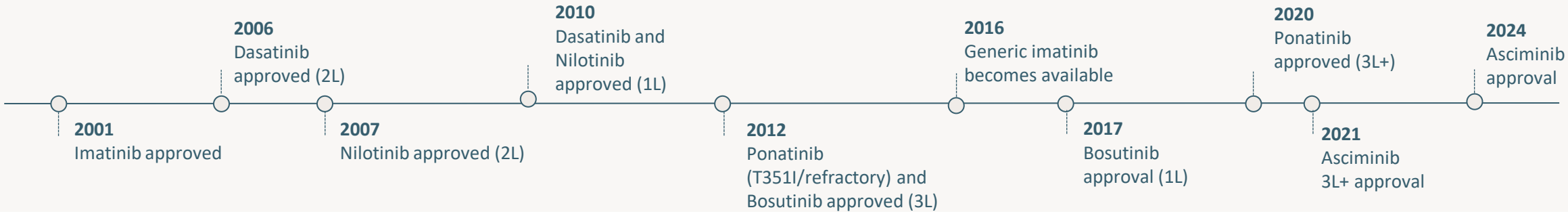
1st **Maintain or improve quality of life**

2nd **Manageable side effects**

CML = Chronic myeloid leukemia. FIH = First-in-human. k = Thousands. TKI = Tyrosine kinase inhibitors. *For patients who have received two prior therapies.

References: Huang X et al. Cancer. 2012;118:3213-3127. Kantarjian et al. Chronic Myeloid Leukemia, In: Harrison's Principles of Internal Medicine, 2014. Lang et al, EHA 2023; Jabbour E, Kantarjian H. Chronic myeloid leukemia: 2025 update on diagnosis, therapy, and monitoring. Am J Hematol. 2024 Nov;99(11):2191-2212

History of Chronic Myeloid Leukemia Treatment Advances



Key Pivotal Trials

2010 **Nilotinib 1L Approval**	2010 **Dasatinib 1L Approval**	2012 **Bosutinib 2L+ Approval**	2017 **Bosutinib 1L Approval**	2020 **Ponatinib 3L+ Approval**	2021 **Asciminib 3L+ Approval**	2024 **Asciminib 1L Approval**
ENESTnd Trial Nilotinib vs. Imatinib Primary Endpoint: MMR @ 12m	*DASISION Trial* Dasatinib vs. Imatinib Primary Endpoint: CCyR @ 12m	Bosutinib single arm Primary Endpoint: MCyR @ 6m	*BFORE Trial* Bosutinib vs. Imatinib Primary Endpoint: MMR @ 12m	*OPTIC Trial* Ponatinib single arm Primary Endpoint: ≤1% BCR::ABL1 @ 12m	*ASCEMBL Trial* Scemblix vs. Bosutinib Primary Endpoint: MMR @ 6m	*ASC4FIRST Trial* Scemblix vs. Imatinib / 2G TKIs Primary Endpoint: MMR @ 12m

~7 years after dasatinib and nilotinib were approved, bosutinib received 1L approval based on a H2H trial vs imatinib (trial began in 2014)

With asciminib moving to earlier lines of therapy, ***ELVN-001*** was developed to address the need for an **ATP-competitive TKI *with a better efficacy and optimized tolerability profile***, while addressing asciminib-emergent resistance mutations

1L = First line. 1L+ = First line and later. 2L = Second line. 3L = Third line. 3L+ = Third line and later. 2G = 2nd Generation TKIs = bosutinib, dasatinib, nilotinib. ATP = Adenosine triphosphate. BCR::ABL1 = Breakpoint cluster region-Abelson leukemia virus 1. CCyR = Complete cytogenic response. H2H = Head-to-head. M = Month. MCyR = Major cytogenic response. MMR = Major molecular response. TKI = Tyrosine kinase inhibitor.

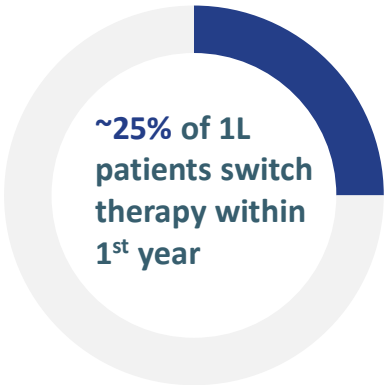
References: public company filings and announcements.

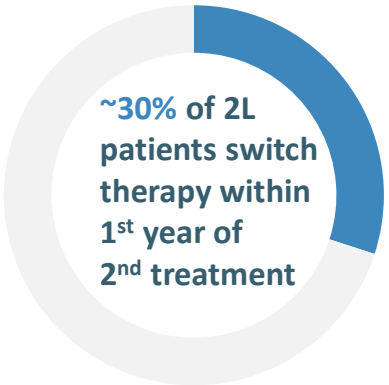
Significant Need Remains for Better Treatment Options

Challenges with Current Standard of Care

- **Growing 3L+** patient population (>25% of CP-CML) with **limited treatment options**
- All of the approved ATP-competitive TKIs have **poor kinase selectivity,** resulting in tolerability issues that can impact efficacy
- **Long-term use of 2nd generation TKIs is associated with adverse events** such as pleural effusions, GI and cardiovascular events
- Adverse events, comorbidities, restrictions with concomitant medications, and specific administration requirements may **impede long-term patient adherence**

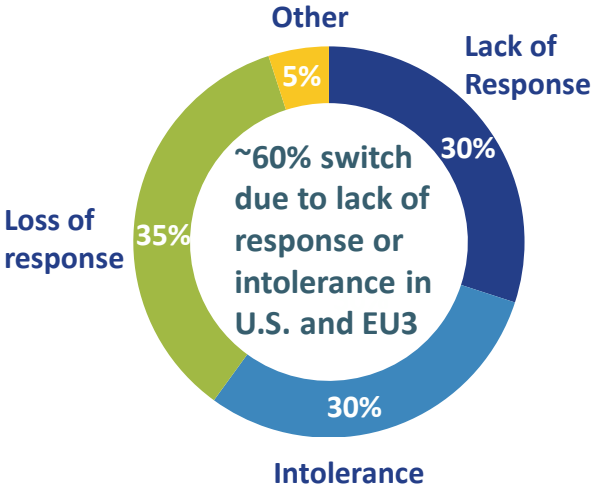
Switching Rates



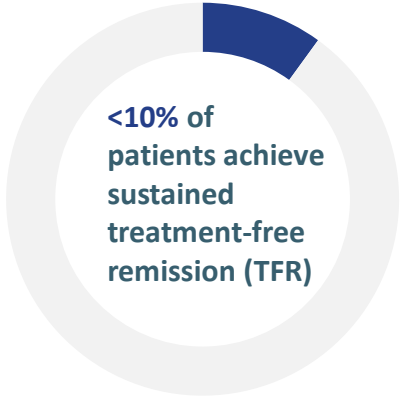


Switching Dynamics Demonstrate Unmet Need

Rationale for Switching Treatment



Need for Better Options



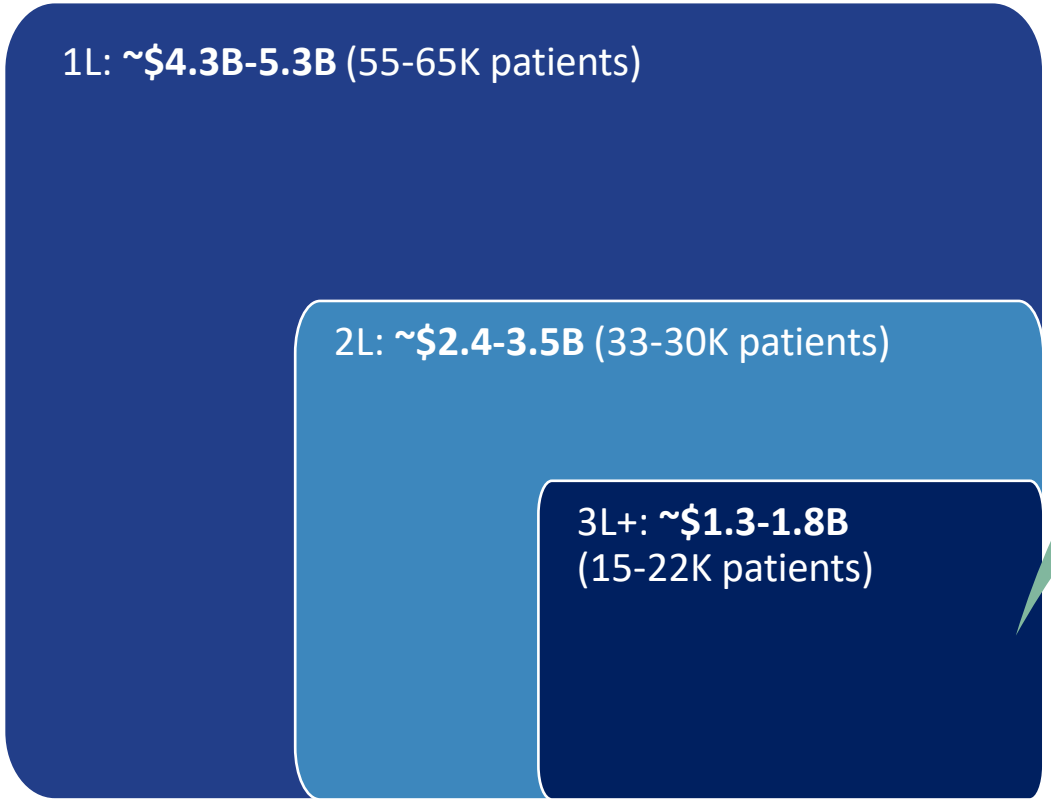
ATP = Adenosine triphosphate. 1L = First line. 2L = Second line. 3L+ = Third line or later. 2nd generation TKIs = Nilotinib, Dasatinib, Bosutinib. CML = Chronic myeloid leukemia. CP-CML = Chronic phase CML. GI = Gastrointestinal. HCP = Healthcare Professional TFR = Treatment-free reemission. TKI = Tyrosine kinase inhibitor. HCP = Healthcare professional. EU3 = France, UK, Germany.

References: HCP Qualitative & Quantitative Interviews (ClearView); Hochhaus A et al. ASH 2015; Hochhaus A et al. Leukemia. 2017; Kota V, et al. Presented at: ASH 2023; 31(7):1525-1531; Osorio S et al. Ann Hematol. 2018; 97(11):2089-2098; Rea et al. Blood. 2021; blood.2020009984; Baccarani M and Gale RP. Leukemia. 2021; 35:2199-2204; Iclusig® (ponatinib) USPI; Sprycel® (dasatinib) USPI; Tasigna® (nilotinib) USPI.; Bosulif® (bosutinib) USPI.

The CML Market is Large and Open to New Entrants as Demonstrated by the Strong Launch of Scemblix

U.S. Branded CML Market has the Potential to be ~$9B Based on Historical Sales

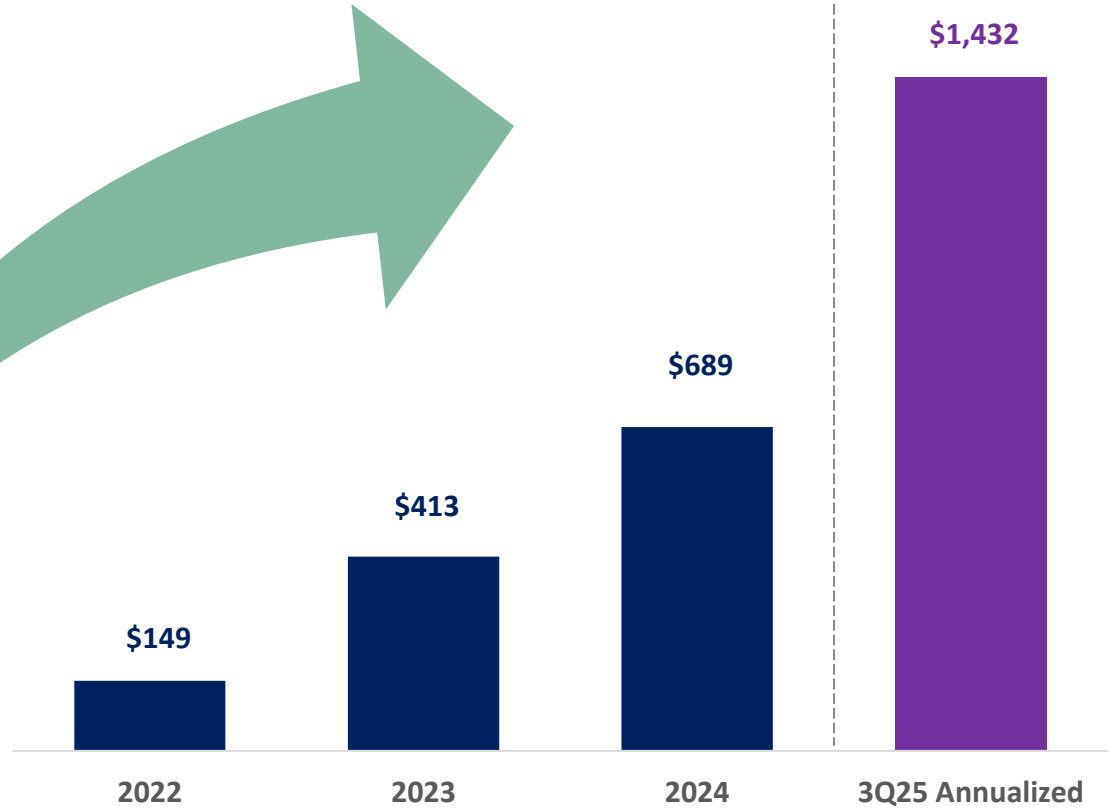
U.S. Patient Population: ~110K



U.S. Weighted Average WAC for Branded CML Drugs: ~$240K

Q4 2024 1L+ Approval Expanded Scemblix's Addressable Market by 4-5x and is Expected to Drive Significant Growth

Majority of Scemblix revenue generated to-date is from initial 3L+ approval



1L = First line. 2L = Second line. 3L+ = Third line and later. B = Billion. CML = Chronic myeloid leukemia. K = Thousand. WAC = Wholesale acquisition cost.

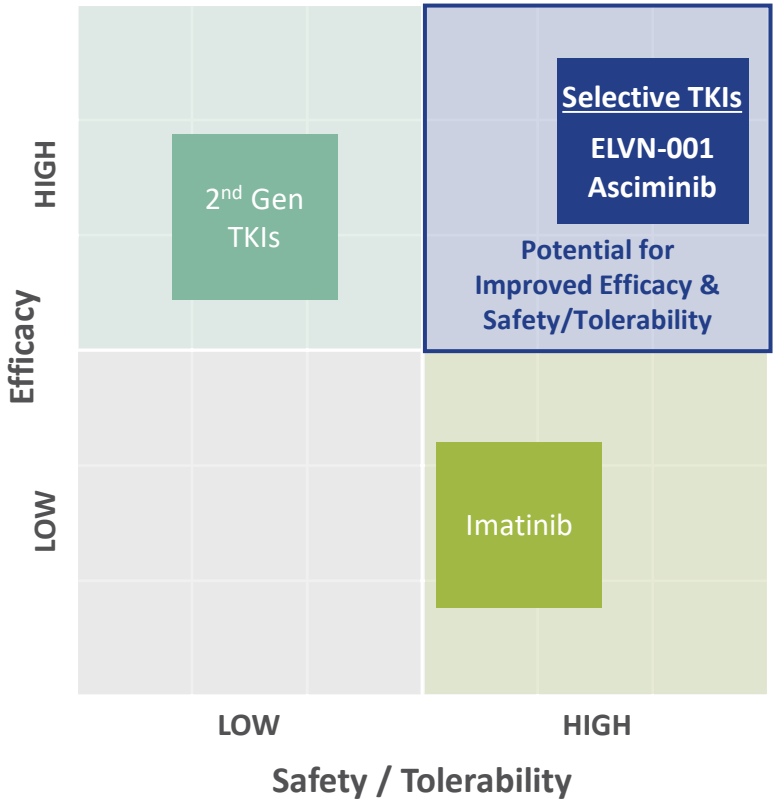
Notes: Percent of patient breakdown by line of therapy is based on HCP Qualitative & Quantitative Interviews (ClearView) and extrapolated from the November 2023 Novartis R&D Investor Event; U.S. branded CML market calculated using total U.S. 2015 branded sales and adjusting those figures for the pricing of CML drugs today and today's increased prevalence. $ in millions. The Scemblix launch may not be indicative of the potential success of any launch of ELVN-001.

References: : Public company filings, announcements and research reports; Huang X et al. Cancer. 2012;118:3213-3127

ELVN-001 is Well Positioned to Follow Scemblix in the Future CML Treatment Paradigm and Has Potential to Compete in 1L+

Limitations of Prior Generation TKIs

(if data supports)



Future Treatment Paradigm

(if data supports)

1L	Generic ATP-competitive TKI	Allosteric TKI	ELVN-001	• Asciminib (allosteric TKI) was recently approved in 1L+ based on improved efficacy/tolerability • **Opportunity for ELVN-001 to compete for 1L share** based on potentially differentiated efficacy, tolerability or convenience
2L	Allosteric TKI	ELVN-001	Allosteric TKI	• **ELVN-001 is potentially well positioned** to follow asciminib given its **unique binding mode and complementary MoA** (ATP-site/active form vs. allosteric/inactive form)
3L+	ELVN-001	ATP-competitive or Allosteric, based on patient need		• Launch of asciminib has recently demonstrated the **multi-billion-dollar opportunity in 3L+** for a a drug with improved efficacy & tolerability in late line CML

Market Insights & Assumptions

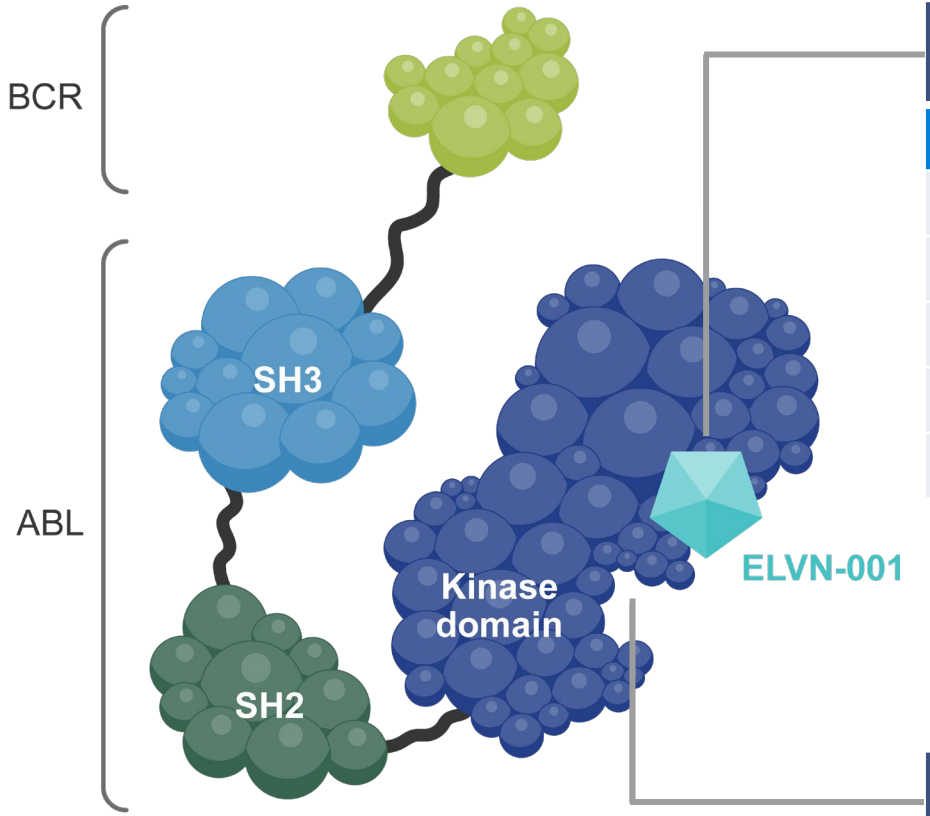
In 3Q 2025 Novartis **reported Scemblix NBRx of 52% in 2L and 22% in 1L in the U.S.**, further highlighting the **need for improved treatment options** across all lines of therapy

1L = First line. 2L = Second line. 2L+ = Second line or later. 3L+ = Third line or later. 2nd Gen TKIs = Nilotinib, Dasatinib, Bosutinib. ATP = Adenosine triphosphate. CML = Chronic myeloid leukemia. Gen = Generation. NBRx = New to brand prescription. MoA = Mechanism of action. 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.

ELVN-001 is a Selective Active Site, Active Form Inhibitor of BCR::ABL1



ATP-BINDING SITE

DRUG	BINDING CONFORMATION
ELVN-001	**Active**
BOSUTINIB	Active
DASATINIB	Active
IMATINIB	Inactive
NILOTINIB	Inactive
PONATINIB	Inactive

MYRISTOYL POCKET

DRUG	BINDING CONFORMATION
ASCIMINIB	**Inactive**

Key Attributes of ELVN-001:

- Type 1 small molecule inhibitor of BCR::ABL1 targeting the ATP-binding site of the ABL1 kinase domain that binds to a unique P-loop "folded-in" active conformation of ABL1 creating a narrow selectivity tunnel
- Broad activity against multiple clinically important BCR::ABL1 mutations, including T315I, and those that confer resistance to asciminib
- Unlike all the approved TKIs, ELVN-001 is not a substrate for the common drug efflux transporters, P-gp and BCRP, which may play a role in resistance to TKIs in CML
- PK supports once daily dosing with or without food, and has low risk of DDIs

ATP = Adenosine triphosphate. BCR::ABL1 = Breakpoint cluster region-Abelson leukemia virus 1. BCRP = Breast cancer resistance protein. CML = Chronic myeloid leukemia. DDI = Drug-drug interaction. P-gp = P-glycoprotein. PK = Pharmacokinetics. TKI = Tyrosine kinase inhibitor.

References: Braun T. et al. Cancer Cell 2020 2020 Apr 13;37(4):530-542; Qiang W et al., Mechanisms of Resistance to the BCR-ABL1 Allosteric Inhibitor Asciminib; Leukemia 2017.

ELVN-001 is Highly Selective and Active Against Asciminib Emergent Mutations

ELVN-001 selectively inhibits ABL with low off-target activity against other kinases

Cellular Phosphorylation IC_{50} (nM)

	cKIT	FLT3wt	PDGFRb	VEGFR2	cSRC
ELVN-001	>10,000	>10,000	>10,000	>10,000	>10,000
Ponatinib	30	3.8	89	4.8	630
Nilotinib	200	>10,000	720	2,900	>10,000
Dasatinib	0.6	>1,000	7.1	>1,000	10
Bosutinib	1,000	4,700	7,900	>10,000	16
Imatinib	82	>10,000	230	9,600	>10,000
Asciminib	>10,000	>10,000	>10,000	>10,000	>10,000

Off-target kinase inhibition (IC_{50}) by ELVN-001 vs. approved ABL TKIs in cell-based assays

ELVN-001 maintains activity against T315I and other BCR::ABL1 mutations known to confer resistance to asciminib

Fold-Shift from Native BCR::ABL1

	T315I	M244V	A337T	E355G	F359C	F359V	P465S
Asciminib	96	611	173	>2380	>2380	>2380	>2380
ELVN-001	**4**	**2**	**1**	**4**	**3**	**2**	**2**
Dasatinib	2935	2	1	3	4	2	2
Bosutinib	113	3	1	4	5	5	4
Ponatinib	3	2	1	3	5	5	2
Imatinib	>20	3	1	8	18	10	4
Nilotinib	>341	2	1	5	33	21	3

Antiproliferative activity of ELVN-001 vs. approved ABL TKIs in Ba/F3 cells harboring various BCR::ABL1 mutations

A337T and M244V were the most frequent emergent mutations to asciminib and F359C/V were the most frequent mutations at baseline in patients resistant to asciminib in ASCEMBL

BCR::ABL1 = Breakpoint cluster region-Abelson leukemia virus 1. IC_{50} = Half-maximal inhibitory concentration. nM = Nanomolar. TKI = Tyrosine kinase inhibitor. WT = Wildtype.
Cell viability measured with CellTiter-Glo luminescent assay. Values expressed as fold-shift in IC_{50} from $BCR::ABL1^{WT}$.

References: Réa D and Hughes TP. Crit Rev Oncol Hematol. 2022;171:103580.; Qiang W et al. Leukemia. 2017;31(12):2844-2847.; Réa D et al. Blood. 2021;138:2031-2041.

ELVN-001 Clinical Focus and Target Product Profile

Our Opportunity

Drive Deeper Responses | **Improve Tolerability** | **Enhance Safety & Convenience**

Target Product Profile

- Activity against native BCR::ABL1, T315I and asciminib-resistant mutations
- **Highly selective:** No/minimal clinically relevant off-target toxicity
- **Efficacy**: MMR greater than approved active-site TKIs driven by an enhanced therapeutic window
- **Tolerability**: Fewer dose reductions & discontinuations
- **Safety**: No black box warnings; no edema, effusions, reduced GI toxicity
- **No restrictions** with concomitant medications

Phase 1a/b: Dose Escalation in Late Line

- Patients with CML who have received at least one prior second generation TKI or asciminib
- Seek to demonstrate improved therapeutic window & efficacy (BCR::ABL1 transcript level reductions) in highly resistant/intolerant disease

Status

- Completed enrollment of initial 3 Phase 1b cohorts

Initial 2L+ Pivotal Phase 3 Trial

- Superiority to imatinib / 2G TKIs based on MMR at 24 weeks
- Better overall tolerability, fewer dose reductions & discontinuations vs. approved active-site agents

Status

- Initiation of ENABLE-2 in 2H 2026

Future: 1L Pivotal Trial

- Superiority to imatinib / 2G TKIs based on MMR at 48 weeks
- Better overall tolerability, fewer dose reductions & discontinuations vs. approved active-site TKIs

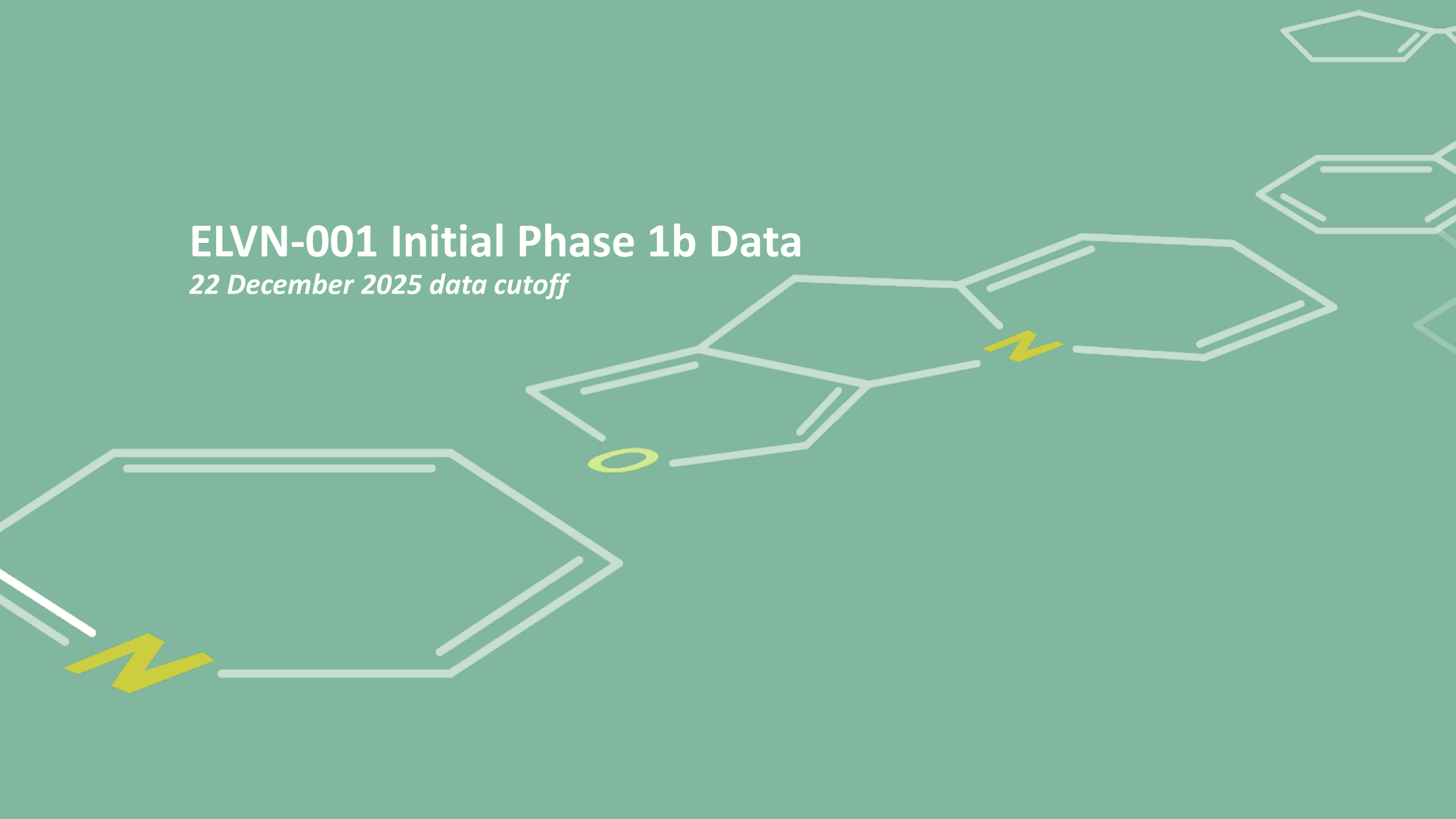
Status

- FDA interaction planned in 2026

1L = First line. 2L+ = Second line or later. 2G = Second generation. BCR::ABL1 = Breakpoint cluster region-Abelson leukemia virus 1. CML = Chronic myeloid leukemia. CP-CML = Chronic phase CML. MMR = Major molecular response. TKI = Tyrosine kinase inhibitor. 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 Initial Phase 1b Data

22 December 2025 data cutoff

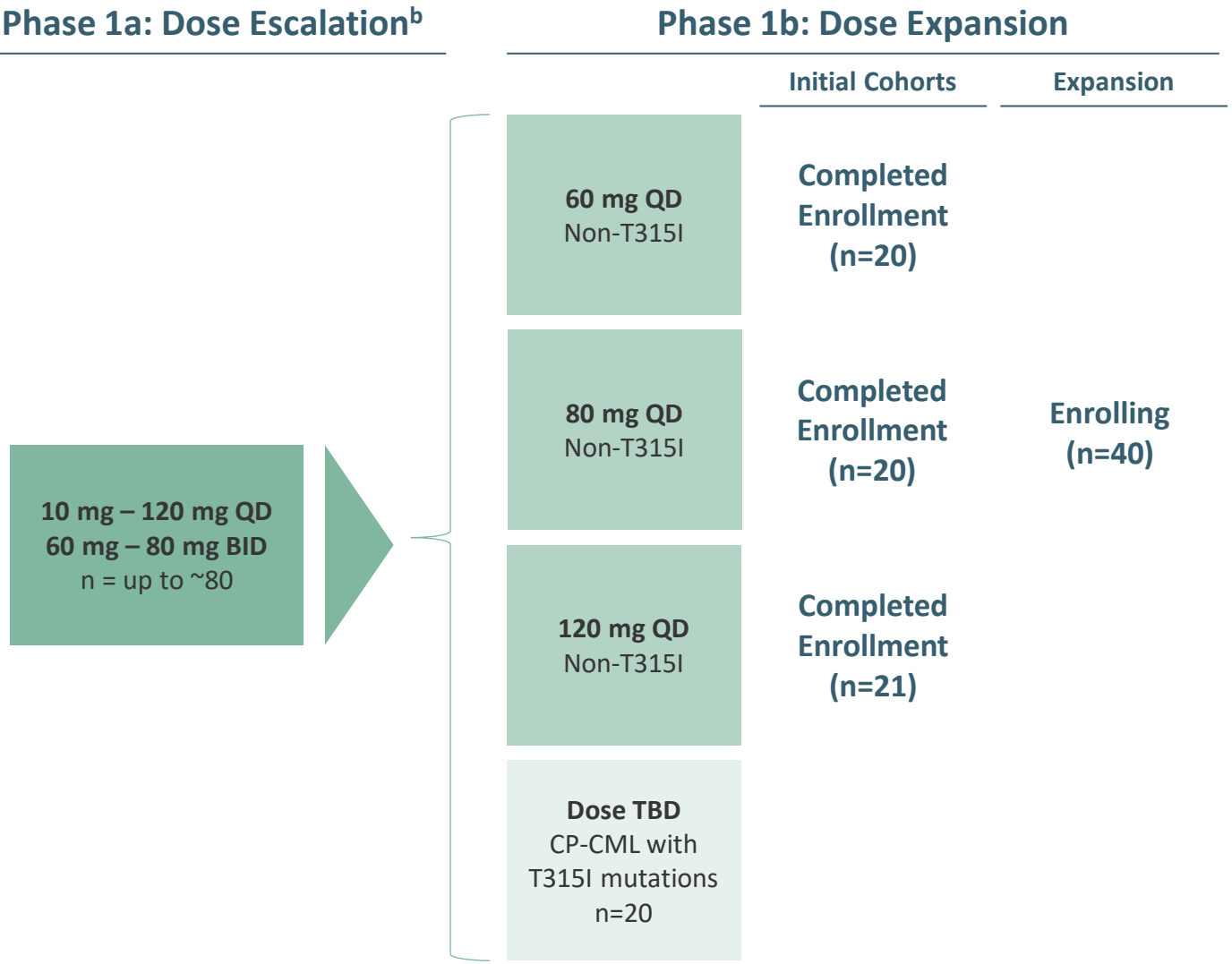


ENABLE (ELVN-001 Phase 1) Trial Design

ENABLE

Key eligibility criteria:

- Chronic Phase CML (CP-CML)
- Failed, intolerant to, or not a candidate for, available therapies known to be active for treatment of their CML[a]



Primary endpoints

- Incidence of dose limiting toxicities, adverse events, clinically significant laboratory and ECG abnormalities

Key Secondary endpoints

- Molecular response (MR) by central qPCR
- PK parameters

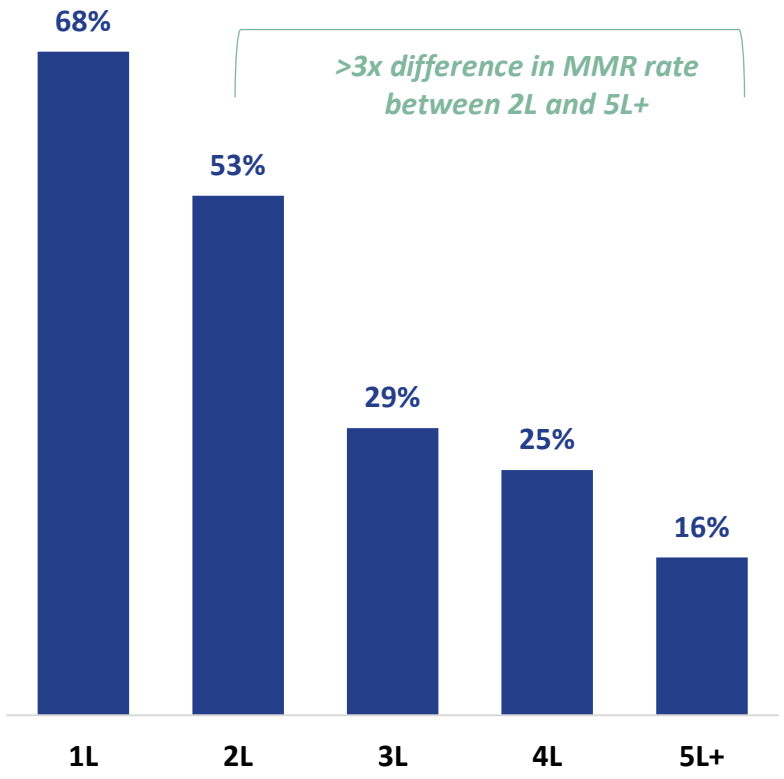
BID = Twice daily. CML = Chronic myeloid leukemia. CP = Chronic phase. ECG = Electrocardiogram. MR = Molecular response. n = Number of patients. PK = Pharmacokinetics. QD = Once daily. qPCR = Quantitative reverse transcriptase polymerase chain reaction. TBD = To be determined

Notes: Clinicaltrials.gov Identifier: NCT05304377. a. In the United States, S. Korea, Australia, EU; at least 2 prior therapies known to be active for treatment of their CML are required in Canada. b. Re-enrollment and intra-subject dose escalation allowed if meeting specific criteria.

Key Considerations for Evaluating MMR in Phase 1 CML Studies

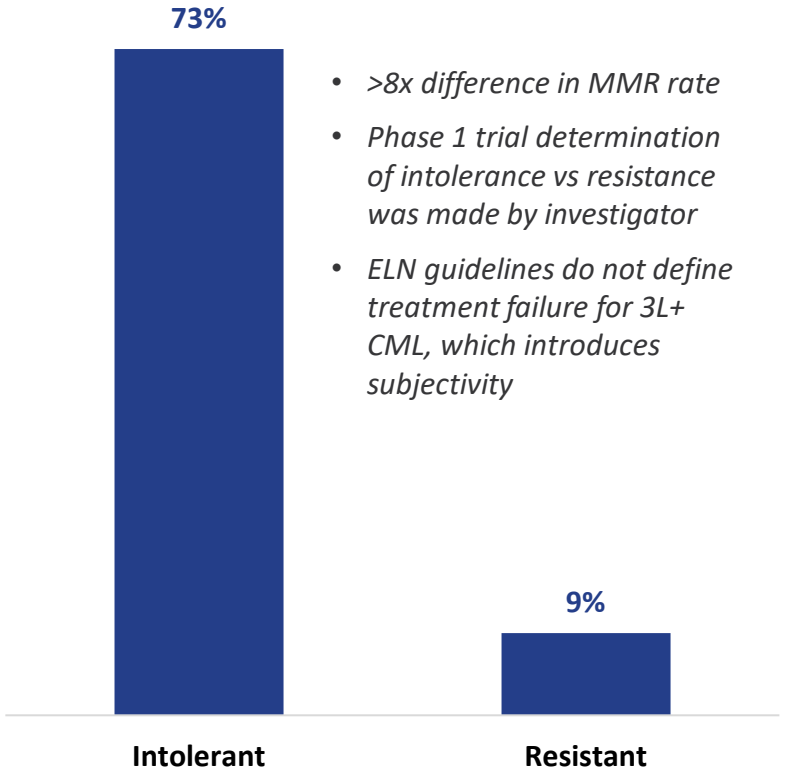
Chance of MMR Decreases Materially by Line of Therapy

Achieved MMR rates based on precedent asciminib studies



Reason for Discontinuation of Last Prior Therapy Significantly Drives MMR Rates

Cumulative MMR rates based on asciminib Phase 1



- *>8x difference in MMR rate*
- *Phase 1 trial determination of intolerance vs resistance was made by investigator*
- *ELN guidelines do not define treatment failure for 3L+ CML, which introduces subjectivity*

Calculation of Achieved MMR

<u>Criteria for Patients Evaluable for MMR Achievement calculation:</u>

- On study for at least 24 weeks;
- Achieved MMR or better prior to 24 weeks (if no MMR at baseline); or
- Discontinued treatment prior to 24 weeks

Any patient still on study who has been treated for <24 weeks and has not achieved MMR is NOT considered efficacy evaluable for MMR achievement

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Median time to MMR in ASCEMBL was 12.7 weeks for asciminib

1L = First line. 2L = Second Line. 3L = Third line. 4L = Fourth line. 5L+ = Fifth line and later. CML = Chronic myeloid leukemia. ELN = European LeukemiaNet. MMR = Major molecular response. 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. Hochhaus A, ASCEMBL, ASH 2020 Oral Presentation

Note: 1L MMR data at 48 weeks. 2L-5L+ MMR data at 24 weeks.

ELVN-001 Updated Ph1 Profile Remains Highly Encouraging

Summary of Patient Demographics and Efficacy Results

	Ph1b 80mg QD (n=19)	Randomized Ph1b 60/120mg QD (n=41)
Median Exposure (weeks)	55 (5-78)	24 (8-50)
Evaluable for MMR by Week 24	19 (100%)	26 (63%)
Patients w/ ≥ 4 Unique Prior TKIs (%)	8 (42%)	24 (59%)
Prior asciminib	9 (47%)	31 (76%)
Prior ponatinib	7 (37%)	12 (29%)
Lack of efficacy to last TKI	11 (58%)	22 (54%)
Cumulative MMR by 24 weeks	9/19 (47%)	18/26 (69%)
MMR Achieved	**6/16 (38%)**	**9/17 (53%)**
MMR Maintained	3/3 (100%)	9/9 (100%)
Deep Molecular Response	**3/19 (16%)**	**9/26 (35%)**

Key Commentary

- Key factors for interpreting interim CML Phase 1 efficacy:
  - # of prior TKIs
  - Reason for discontinuation of last prior TKI (lack of efficacy vs intolerance)
  - Data maturity, particularly the proportion of patients who have been on study ≥24 weeks
- Efficacy continues to compare favorably to precedent Phase 1 trials of approved BCR::ABL1 TKIs despite more heavily pretreated patients
- As expected, no clear evidence of dose response (safety or efficacy) across the 1b doses of 60mg-120mg QD
- Favorable safety and tolerability observed consistent with previous reports

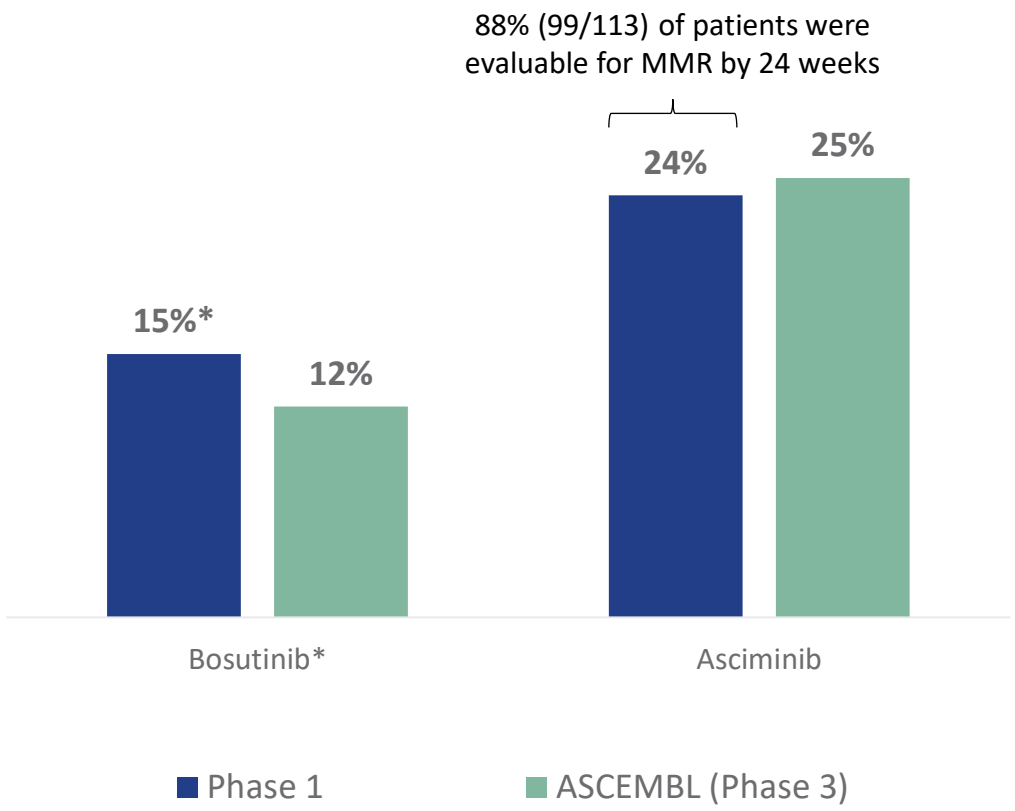
BCR::ABL1 = Breakpoint cluster region-Abelson leukemia virus 1. CML = Chronic myeloid leukemia. MMR = Major molecular response. n = number of patients. Ph1 = Phase 1. QD = Once daily. TKI = Tyrosine kinase inhibitor.
Notes: Data snapshot December 22, 2025; Evaluable patients had baseline typical BCR::ABL1 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. Conclusions from cross-trial comparisons cannot be made, and no head-to-head clinical trials have been conducted.

Next Steps for ELVN-001

Phase 1 Data in Late Line CML has Historically Predicted Regulatory Success

In precedent Phase 1 trials, achieved MMR rates have predicted performance in late-line pivotal trials

(Cumulative Achieved MMR Rates by 24 weeks)



Clear Translation from Phase 1 to Pivotal Trials

- Achieved MMR rates in late line CML Phase 1 trial for asciminib and bosutinib predicted MMR rates at 24 weeks, the **primary endpoint** in ASCEMBL

ELVN-001's Phase 1 Data Highly Encouraging

- ELVN-001's data in late line CML has consistently compared favorably to precedent Phase 1 trials, despite enrolling more heavily pretreated patients

On Track to Initiate First Pivotal Trial in 2H 2026

CML = Chronic myeloid leukemia. MMR = Major molecular response.

Notes: *Phase 1 bosutinib reported overall MMR with a median follow up of 28.5 months.MMR at 24 weeks in ASCEMBL was 25% and 13% for asciminib and bosutinib, respectively. Conclusions from cross-trial comparisons cannot be made, and no head-to-head clinical trials have been conducted.

References: Rea et al. Blood. 2021; blood.2020009984; Khoury HJ et al. Blood. 2012;119(15):3403-3412; Hughes TP, et al. N Engl J Med. 2019;381(24):2315-2326; Asciminib USPI.

Precedent CML Pivotal Trials Establish Potential Path for ELVN-001

ASCEMBL: Resulted in 3L+ Approval

Key Enrollment Criteria

- Adults with CML-CP previously treated with ≥ 2 TKIs
- Stratified by MCyR vs no MCyR at baseline

R 2:1
n = 233

- Asciminib 40 mg twice daily n = 157
- Bosutinib 500 mg once daily n = 76

MMR evaluation

- Primary endpoint: MMR at 24 weeks
- Key secondary endpoint: MMR at 96 weeks
- Protocol allowed for a crossover if a patient experienced a lack of efficacy

Enrollment start	Enrollment ends	Topline data	Approval
Nov 2017	Dec 2019	May 2020	Oct 2021

3L+ Market Opportunity

14-20% of patients

~$1.3-1.8B U.S. market size

ASC4FIRST: Resulted in 1L+ Approval

Key Enrollment Criteria

- Newly diagnosed patients with CML-CP
- Stratified by pre-randomization selection of TKI (imatinib/2G TKI) and ELTS (high/ intermediate/low)

R 1:1
n = 402

- Asciminib 80 mg once daily n = 200
- Investigator selected (IS) TKI: imatinib (n = 102) 2G TKI (n = 102)

MMR evaluation

- Primary endpoint: MMR at 48 weeks vs imatinib
- Primary endpoint: MMR at 48 weeks vs IS TKI
- Key secondary endpoint: MMR at 96 weeks

Enrollment start	Enrollment ends	Topline data	Approval
Nov 2021	Dec 2022	Nov 2023	Oct 2024

1L+ Market Opportunity

~4-5x larger patient population than just 3L+

~$9B total U.S. market size

2G = 2nd generation TKIs - bosutinib, dasatinib or nilotinib. 1L+ = First line and later. 3L+ = Third line and later. B = Billion. CML-CP = Chronic myeloid leukemia in chronic phase. ELTS = EUTOS long-term survival. EUTOS = European Treatment and Outcome Study. MCyR = Major and complete cytogenic response. MMR = Major molecular response (*BCR::ABL1*^{IS} ≤ 0.1%). n = Number of patients. R = Randomized. TKI = Tyrosine kinase inhibitor.

References: Publicly available filings, announcements and research reports; Percent of patient breakdown by line of therapy is based on HCP Qualitative & Quantitative Interviews (ClearView) and the November 2023 Novartis R&D Investor Event; Huang X et al. Cancer. 2012;118:3213-3127; PriceRx; Assumes 2024 weighted average U.S. WAC of ~$240,000. Assumes current U.S. prevalence of ~110,000 based on American Journal of Hematology: Chronic myeloid leukemia: 2025 update on diagnosis, therapy, and monitoring.

Initial Pivotal Trial Could Provide Access to 2L+ CML Patients, with a Roadmap for a Broad Approval in CML

Initial 2L+ Trial

ELVN-001 vs. Physician's Choice (2G TKI, imatinib)

- ~50% of patients with CML are 2L+
- By including all 2G TKIs and imatinib, the comparator arm reflects real-world clinical practice when a physician has determined that an active-site TKI is appropriate
- Valuable post-asciminib data
- Attractive comparator and large switching patient population to drive enrollment

- Potential to conduct the 1L pivotal trial with a staggered start to the initial 2L+ trial or sequentially
- Timing of the 1L trial will depend on feedback from regulatory agencies

1L Trial

ELVN-001 vs. 2G TKI or imatinib

- Modeled after successful ASC4FIRST trial
- Broad label: provides full access to growing ~110K U.S. patient population
- Potential to demonstrate best-in-class active-site TKI profile in homogeneous 1L patient population

Potential to Demonstrate ELVN-001's best-in-class active-site TKI profile | **Expected predictable and attractive timelines** | **Success of recent launches demonstrates need for improved treatment options**

1L = First line. 2L+ = Second line and later. 2G = 2nd generation TKIs - bosutinib, dasatinib or nilotinib. 3L+ = Third line and later. CML = Chronic myeloid leukemia. TKI = Tyrosine kinase inhibitor.
Note: "Predictable timelines" refers to the length of the recent CML pivotal trials (ASCEMBL and ASC4FIRST).
References: Publicly available filings, announcements and research reports. Huang X et al. Cancer. 2012;118:3213-3127

ELVN-001 has Potential to Become Preferred TKI for Patients with CML

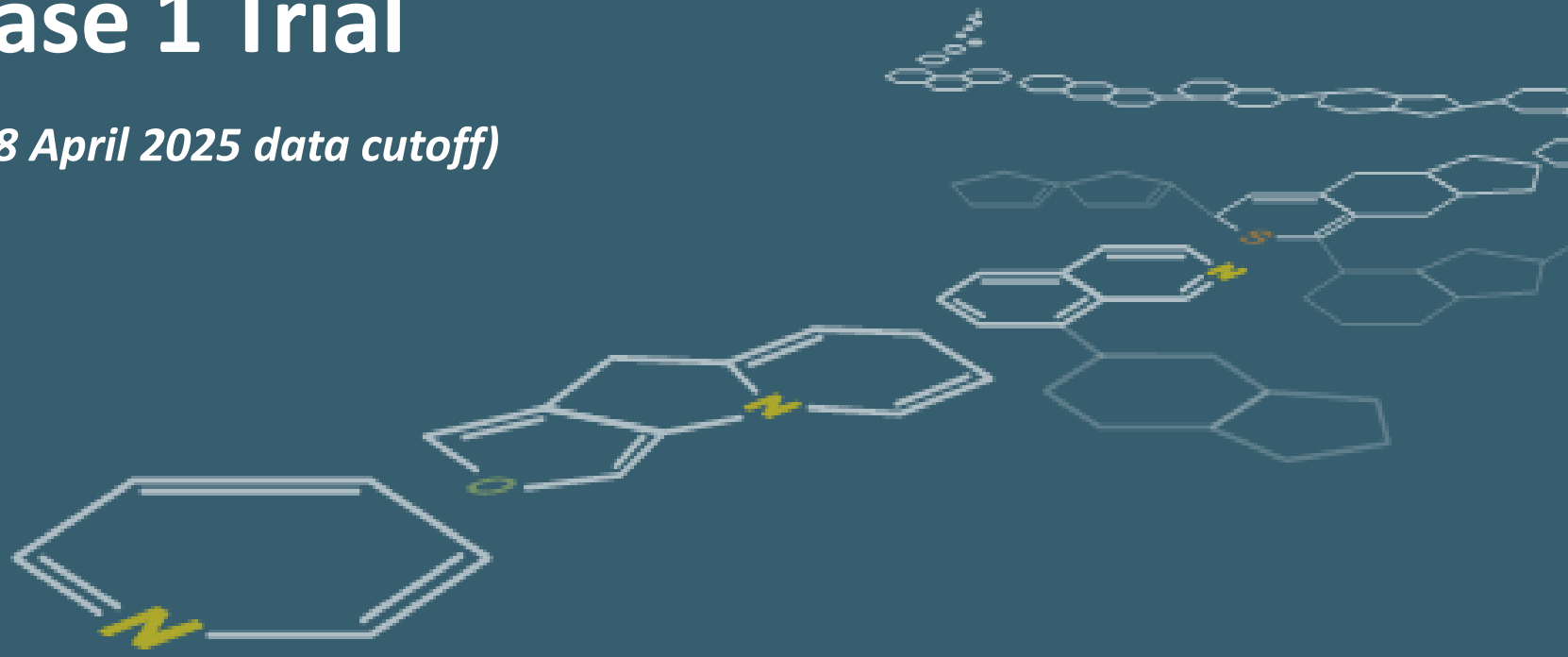
Large Market Opportunity	Potential ~$9B opportunity for differentiated TKIs in the U.S. alone
Evolving SoC	Recent successful Scemblix launch validates the need for better treatment options and with its adoption in earlier lines of therapy, creates a need for a selective ATP-competitive TKI
Potentially Best-in-Class	ELVN-001 efficacy, safety and tolerability Phase 1 data compare favorably to approved inhibitors, and PK supports QD dosing with or without food, and has low risk of DDIs
Defined Regulatory Path	Phase 1 data predicted performance in precedent registrational trials; all CML TKIs have been approved based on a biomarker endpoint
Next Steps	On track to initiate first ELVN-001 head-to-head pivotal trial in 2026; opportunity to move to 1L & compete across lines of therapy based on differentiated efficacy, tolerability or convenience

1L = First line. ATP = Adenosine triphosphate. B = Billion. CML = Chronic myeloid leukemia. DDI – Drug-drug interactions. PK = Pharmacokinetic. QD = once daily. SoC = Standard of care. TKI = Tyrosine kinase inhibitor.

Appendix

ELVN-001 Phase 1 Trial

EHA 2025 Data Update (*28 April 2025 data cutoff)*



Baseline Characteristics: Heavily Pre-treated Patient Population Enrolled

Patient Demographics and Baseline Characteristics

Parameter	All Patients[a] (N = 90)
Age, years, median (range)	58 (19–79)
Male / female, n (%)	52/38 (57.8%/42.2%)
Race	
White	63 (70.0%)
Asian	16 (17.8%)
Black or African American	1 (1.1%)
Other or not reported	10 (11.1%)
ECOG performance status 0/1 (%)	74%/26%
Median time since diagnosis, months (range)	58.1 (2.6–281.9)
Typical BCR::ABL1 transcript (e13a2 and e14a2)	84 (93.3%)
Baseline BCR::ABL1 Transcript Level[b]	
<=0.1%	15 (17.9%)
>0.1%	63 (75.0%)
BCR::ABL1 mutation at baseline (central)[c], n (%)	
No mutation	49 (54.4%)
T315I mutation	8 (8.9%)[d]
Other mutation	6 (6.7%)
Not available	27 (30.0%)

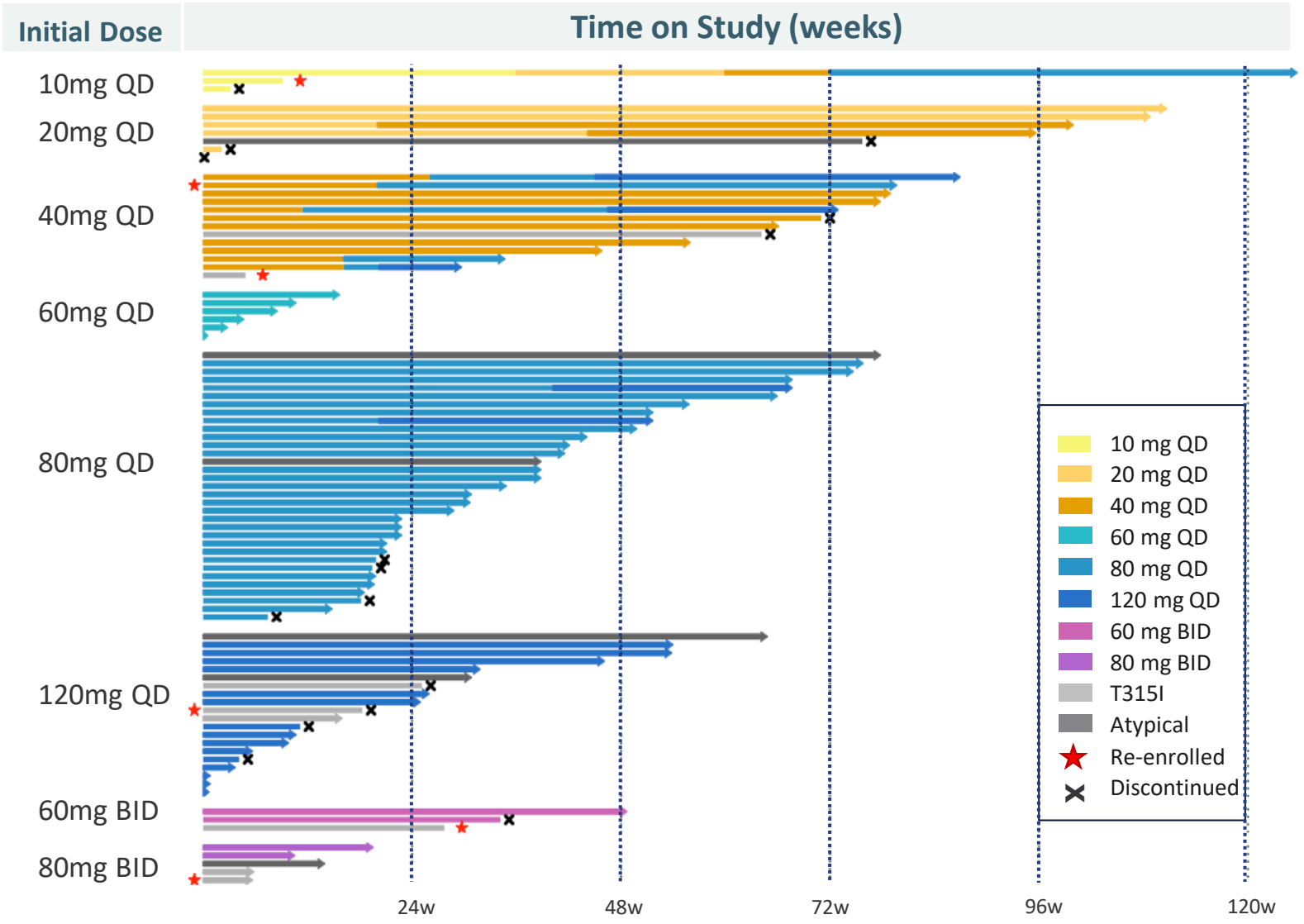
Parameter	All Patients (N = 90)
Median number of prior unique TKIs[e], n (range)	3 (1–7)
1 prior TKI, n (%)	7 (7.8%)
2 prior TKIs, n (%)	22 (24.4%)
3 prior TKIs, n (%)	16 (17.8%)
4 prior TKIs, n (%)	21 (23.3%)
≥ 5 prior TKIs, n (%)	23 (25.6%)
Prior TKI, n (%)	
Dasatinib	66 (73.3%)
Imatinib	60 (66.7%)
Asciminib	52 (57.8%)
Nilotinib	49 (54.4%)
Ponatinib	39 (43.3%)
Bosutinib	34 (37.8%)
Reason for discontinuation of last TKI, n (%)	
Lack of efficacy	65 (72.2%)
Lack of tolerability	21 (23.3%)
Other	3 (3.3%)

[a] Includes 3 re-enrolled patients (87 individual patients). [b] Percentages based on 84 patients with typical transcript. [c] Only available for patients with typical transcripts. Other mutations include: E255V, F359V, H375Y, M244V, P465L, L387M/M244V. [d] Includes 2 re-enrolled patients (6 individual patients with T315I). [e] Median lines of prior TKIs is 4 (range 1-9).

BCR::ABL1 = Breakpoint cluster region-Abelson leukemia virus 1. ECOG status = Eastern Cooperative Oncology Group performance status. N= number. TKI = Tyrosine kinase inhibitor.
Data cutoff: 28 Apr 2025.

Notes: Patients who had gone through intra-patient dose escalation as per protocol were counted under their initial treatment group only. 3 patients who were re-enrolled were counted under their initial treatment group and their re-enrolled treatment group.

ELVN-001 Median Duration of Exposure 29 Weeks



- 80% of patients remain on study
- 56% of patients have been on study > 24 weeks with the longest 126 weeks (~2.5 years) ongoing
- ≥ 40mg QD anticipated to have improved anti-CML activity compared to second generation TKIs based on target coverage

BID = Twice daily. CML = Chronic myeloid leukemia. QD = Once daily. TKI = Tyrosine kinase inhibitor. W = Weeks.
Data cutoff: 28 Apr 2025.

Notes: The protocol allows re-enrollment and intrasubject dose escalation, as shown by color. The swimmer plot does not include 2 patients that received their first ELVN-001 dose in June (these patients were included in the safety analysis as it was confirmed they received at least one dose of ELVN-001, but daily dosing information had yet to be provided at cutoff date).

ELVN-001 24-Week Efficacy Data Continue to be Highly Encouraging

Overall MMR (BCR::ABL1 ≤ 0.1%) by 24 weeks	
Overall MMR by 24 weeks	25/53 (47%)
Achieved (not in MMR at baseline)	13/41 (32%)
Maintained (in MMR at baseline)	12/12 (100%)
Key subgroups	
Post asciminib	9/28 (32%)
Post ponatinib	7/20 (35%)
Lack of efficacy to last TKI	14/34 (41%)
Intolerant to last TKI	9/17 (53%)

Overall MR2 (BCR::ABL1 ≤ 1%) by 24 weeks	
Overall MR2 by 24 weeks	43/56 (77%)
Achieved (not in MR2 at baseline)	14/27 (52%)
Maintained (in MR2 at baseline)	29/29 (100%)

Robust efficacy profile despite heavily pretreated patient population, including in patients exposed to prior asciminib or ponatinib

BCR::ABL1 = Breakpoint cluster region-Abelson leukemia virus 1. MMR = Major molecular response. MR2 – Molecular response 2. TKI = Tyrosine kinase inhibitor.
Data cutoff: 28 Apr 2025.
BCR::ABL1 = Breakpoint cluster region-Abelson leukemia virus 1. MMR = Major molecular response. MR = Molecular response. TKI = Tyrosine kinase inhibitor.
Notes: Subjects who had gone through intra-subject dose escalation as per protocol were counted under their initial treatment group only. Subjects who were re-enrolled were summarized under the treatment groups they enrolled to with the corresponding data collected during the treatment episode, respectively. Subjects are included if they had baseline 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 subjects with MMR /≤1% at baseline, only postbaseline assessments beyond 70 days will be included in the analysis.

ELVN-001 Data Compares Favorably to Precedent Phase 1 Trials

		Asciminib Phase 1 (2019)[1]	Bosutinib Phase 1 (2012)[2]	ELVN-001 Phase 1[a]
Demographics (Prior TKIs)	2	30 (27%)	115 (97%)	22 (24%)
	3	41 (36%)	3 (3%)[b]	16 (18%)
	4	32 (28%)		21 (23%)
	≥ 5	9 (8%)		23 (26%)
Efficacy (Non-T315I)	Cumulative MMR	37/99 (37%)	16/105 (15%)	25/53 (47%)
	MMR Achieved[c]	19/80 (24%)		13/41 (32%)
	MMR Maintained[d]	18/19 (95%)		12/12 (100%)
	MMR in TKI-resistant patients	3/32 (9%)	3/54 (6%)	14/34 (41%)
	Time Frame	by 24 weeks	median follow-up 28.5 mo.	by 24 weeks

More heavily pre-treated patients

12/34 (35%) who had prior asciminib or ponatinib were in MMR by 24 weeks

Asciminib's Phase 1 rate of MMR Achieved by 24 weeks (24%) predicted successful Phase 3 approval endpoint (25%)[3]

CML = Chronic myeloid leukemia. MMR = Major molecular response. Mo. = Month. TKI = Tyrosine kinase inhibitor.
Data cutoff: 28 Apr 2025
Notes: 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: 1. Hughes et al., NEJM 2019. 2. Khoury HJ et al. Blood. 2012. 3. Asciminib USPI.

98% (52/53) Patients with Improved or Stable MR Category by 24 Weeks

Change in BCR::ABL1 Transcript in Patients Evaluable for MMR by 24 Weeks (n=53)

Improvement in MR Category
No Category change
Worsening in MR category

BCR::ABL1 transcript by 24-weeks	Baseline *BCR::ABL1* transcript						
	>MR4.5 ≤ 0.0016 (n = 1)	**MR4.5** > 0.0016 to 0.0032 (n = 0)	**MR4** > 0.0032 to 0.01 (n = 3)	**MR3** > 0.01 to 0.1 (n = 8)	> 0.1 to 1 (n = 16)	> 1 to 10 (n = 9)	> 10 (n = 16)
>MR4.5 ≤ 0.0016	1		1	2			
MR4.5 > 0.0016 to 0.0032							
MR4 > 0.0032 to 0.01			2		1	1	
MR3 > 0.01 to 0.1				6	5	4	2
> 0.1 to 1					10	3	2
> 1 to 10							1
> 10						1[a]	11

- Improvement in transcript category was observed in patients independent of baseline transcript

BCR::ABL1 = Breakpoint cluster region-Abelson leukemia virus 1. CML = Chronic myeloid leukemia. MMR = Major molecular response. MR = Molecular response.
Data cutoff: 28 Apr 2025. a. Worsening of transcript level from 6.3% at baseline to 13% after 4 weeks in patient with E255V mutation who previously discontinued asciminib and ponatinib due to lack of efficacy.
Notes: >MR4.5 category assigned based on transcript level < limit of quantitation. 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.

ELVN-001 Well Tolerated with No Observed Dose-Toxicity Relationship

- **Well-tolerated** across all evaluated doses (10 mg QD to 80 mg BID)
- **The majority of treatment emergent adverse events (TEAEs) were low grade**
  - Hematologic TEAE profile similar or better than other TKIs
  - Low frequency of non-hematologic TEAEs
- **Low number of dose adjustments due to TEAEs**
  - 14 patients (16.1%) with dose interruptions
  - 3 patients (3.4%) with dose reductions[b]
  - 4 patients (4.6%) with discontinuations[c]
- **Maximum tolerated dose has not been identified**
- **No exposure-toxicity relationship observed**

TEAEs (regardless of attribution) in ≥ 10% of patients

Preferred term n (%)	Total (N = 87)	
	Any	Grade 3-4
Lipase increased	16 (18.4%)	1 (1.1%)
Diarrhea	13 (14.9%)	0
Thrombocytopenia[a]	12 (13.8%)	6 (6.9%)
Arthralgia	11 (12.6%)	1 (1.1%)
Headache	11 (12.6%)	0
Fatigue	9 (10.3%)	0
Myalgia	9 (10.3%)	0

AE = Adverse event. BID = Twice daily. G = Grade. QD = Once daily. TEAE = Treatment-emergent adverse event. TKI = Tyrosine kinase inhibitor.
Data cutoff: 28 Apr 2025.
Notes: a. Combined term: platelet count decreased/thrombocytopenia. b. Dose reductions due to AE were: G3 Musculoskeletal pain in patient who discontinued 6 prior TKIs due to musculoskeletal or neuropathic pain (80 mg QD); G3 Arthralgia in patient who discontinued 2 prior TKIs due to intolerance (60 mg QD), G2 lumbar radicular pain in patient who discontinued 2 prior TKIs due to intolerance, including arthralgia and myalgia (60 mg QD). c. G2 alcoholic pancreatitis (10 mg QD), G3/4 platelet count decreased/thrombocytopenia (20 mg QD and 80 mg QD; both in patients who discontinued prior TKIs due to hematologic toxicities), dyspnea (80 mg QD; confounded by pulmonary comorbidities including sleep apnea, diffuse interstitial pneumonitis, pulmonary hypertension and obesity).

Patient Disposition: Majority of Patients Remain on Study

Disposition	Total (N = 90)
Median Duration of Exposure, weeks (range)	29 (0.1–126)
Ongoing, n (%)	72 (80.0%)
Discontinued, Total n (%)	18 (20.0%)
Lack of efficacy	11 (12.2%)*
Adverse Event	4 (4.4%)
Death	1 (1.1%)
Protocol violation	1 (1.1%)
Withdrawal of consent	1 (1.1%)

*3 of 11 patients discontinued at lower doses, subsequently re-enrolled at higher dose levels; no patients progressed to blast crisis or acute leukemia

- 80% of patients remain on study with a median duration of exposure of 29 weeks
- Four patients discontinued due to adverse events:
  - Alcoholic pancreatitis (10 mg QD)
  - Thrombocytopenia (20 mg QD and 80 mg QD)
  - Dyspnea (80 mg QD; confounded by pulmonary comorbidities)
- One patient died of a post-operative complication (after hip surgery; not related to study drug)

QD = Once daily. N = Number.
Data cutoff: 28 Apr 2025.
Notes: Patients who had gone through intra-patient dose escalation as per protocol were counted under their initial treatment group only. 3 patients who were re-enrolled were counted under their initial treatment group and their re-enrolled treatment group.

ELVN-001 Safety Profile Consistent with High Selectivity for ABL1

Treatment Related Adverse Events in ≥ 5% of patients

Preferred term n (%)	ELVN-001 Dose Group										Total (N = 87)	
	10 mg to 40 mg QD (n = 23)		60 mg QD (n = 6)		80 mg QD (n = 33)		120 mg QD (n = 20)		60 and 80 mg BID (n=8)			
	Any Gr	Gr 3+	Any Gr	Gr 3+	Any Gr	Gr 3+	Any Gr	Gr 3+	Any Gr	Gr 3+	Any Gr	Gr 3+
Subjects with Any Treatment Related Adverse Event	14 (60.9%)	3 (13.0%)	3 (50.0%)	1 (16.7%)	21 (63.6%)	4 (12.1%)	12 (60.0%)	1 (5.0%)	6 (75.0%)	0	56 (64.4%)	9 (10.3%)
Lipase increased	3 (13.0%)	0	0	0	7 (21.2%)	0	3 (15.0%)	1 (5.0%)	2 (25.0%)	0	15 (17.2%)	1 (1.1%)
Arthralgia	1 (4.3%)	0	1 (16.7%)	1 (16.7%)	3 (9.1%)	0	3 (15.0%)	0	0	0	8 (9.2%)	1 (1.1%)
Thrombocytopenia*	3 (13.0%)	2 (8.7%)	0	0	3 (9.1%)	2 (6.1%)	0	0	1 (12.5%)	0	7 (8.0%)	4 (4.6%)
Amylase increased	1 (4.3%)	0	0	0	3 (9.1%)	0	1 (5.0%)	0	2 (25.0%)	0	7 (8.0%)	0
Fatigue	0	0	1 (16.7%)	0	2 (6.1%)	0	2 (10.0%)	0	2 (25.0%)	0	7 (8.0%)	0
Myalgia	1 (4.3%)	0	0	0	2 (6.1%)	0	3 (15.0%)	0	0	0	6 (6.9%)	0
Neutropenia*	3 (13.0%)	3 (13.0%)	0	0	1 (3.0%)	0	0	0	1 (12.5%)	0	5 (5.7%)	3 (3.4%)
Headache	1 (4.3%)	0	0	0	3 (9.1%)	0	1 (5.0%)	0	0	0	5 (5.7%)	0

AE = Adverse events. BID = Twice daily. Gr = Grade. QD = Once daily. N = number.
Data cutoff: 28 Apr 2025.
*combined terms: platelet count decreased/thrombocytopenia, neutrophil count decreased/ neutropenia
Notes: Subjects who had gone through intra-subject dose escalation as per protocol were counted under their initial treatment group only. Subjects who were re-enrolled were summarized under the treatment groups they enrolled to with the corresponding data collected during the treatment episode, and counted as one subject in total respectively

Grade 3/4 TEAEs are Uncommon and Not Dose-Dependent

Grade 3/4 TEAEs (regardless of attribution) by Dose Level reported in ≥ 5% of patients

Preferred term n (%)	10 - 40 mg QD (n = 23)	60 mg QD (n = 6)	80 mg QD (n = 33)	120 mg QD (n = 20)	60 - 80 mg BID (n=8)	Total (n=87[a])
Subjects with any G3/4	5 (21.7%)	1 (16.7%)	8 (24.2%)	4 (20.0%)	2 (25.0%)	20 (23.0%)
Thrombocytopenia	2 (8.7%)	0	3 (9.1%)	0	1 (12.5%)	6 (6.9%)
Neutropenia	4 (17.4%)	0	0	0	1 (12.5%)	5 (5.7%)

- Only 2 patients (2.3%) reported Grade 3 AOEs[b] (no Grade 4 AOEs were reported); both patients had prior ponatinib and nilotinib and both events were considered unrelated to ELVN-001 per investigator. In addition, both patients remain on study.
  - One patient with a history of pericarditis, transient ischemic attacks, and cardiac chest pain reported angina pectoris; of note, the patient also discontinued prior nilotinib, ponatinib and asciminib due to persistent cardiovascular events (80 mg QD)
  - One patient with a history of high blood pressure and high cholesterol reported coronary artery disease (120 mg QD)

AOE = Arterial occlusion event. BID = Twice daily. CPK = Creatine phosphokinase. G = Grade. SMQ = Standardized MeDRA queries. TEAE = Treatment-emergent adverse event. QD = Once daily.
Data cutoff: 28 Apr 2025
Notes: a. Patients with intra-subject dose escalation were counted under their initial treatment group only. Re-enrolled subjects were summarized at both dose levels with the corresponding data collected during each period, and once in the total column. b. AOEs were defined by SMQ search terms.

ELVN-001 Enrolled More Heavily Pretreated Patients than ASCEMBL

Prior TKIs	ELVN-001 (N=90)	Asciminib (N=157)
Median number of prior unique TKIs, n (range)	3 (1–7)	
1 prior TKI, n (%)	7 (7.8%)	
2 prior TKIs, n (%)	22 (24.4%)	89 (56.7%)
3 prior TKIs, n (%)	16 (17.8%)	53 (33.8%)
≥ 4 prior TKIs, n (%)	44 (48.9%)	15 (9.6%)

Prior Lines of TKI Therapy	ELVN-001 (N=90)	Asciminib (N=157)
Median number of lines of TKI therapy, n (range)	4 (1–9)	
1 prior line, n (%)	6 (6.7%)	
2 prior lines, n (%)	21 (23.3%)	82 (52.2%)
3 prior lines, n (%)	13 (14.4%)	44 (28.0%)
4 prior lines, n (%)	18 (20.0%)	24 (15.3%)
≥ 5 prior lines, n (%)	31 (34.4%)	7 (4.5%)

Notable Prior TKIs	ELVN-001 (N=90)	Asciminib (N=157)
Asciminib	52 (57.8%)	-
Ponatinib	39 (43.3%)	23 (14.6)
Reason for discontinuation of last TKI, n (%)		
Lack of efficacy	65 (72.2%)	95 (60.5)

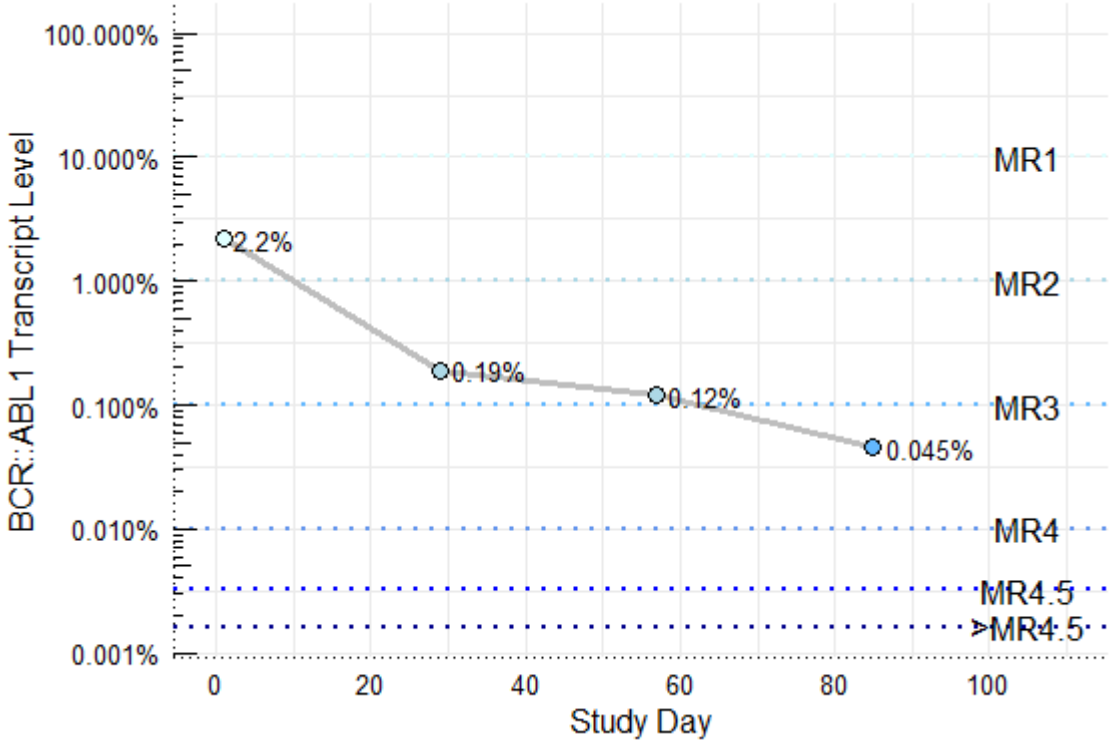
More heavily pretreated patient population in ELVN-001 in ENABLE vs. asciminib in ASCEMBL

N= Number. TKI = Tyrosine kinase inhibitor. Data cutoff date: 28 Apr 2025

References: Rea D et al., Blood. 2021. Hochhaus et al, Leukemia. 2023

MMR in Patient with Resistant CML with F359V Mutation

MMR by Day 90 @ 80 mg QD ELVN-001



Patient Background

Relevant past medical history	None
Prior TKI therapy (reason for switch)	Nilotinib (discontinued due to lack of efficacy)
Mutations	F359V
Safety	No adverse events reported
Efficacy	Major Molecular Response

F359C/V were the most frequent mutations at baseline in patients resistant to asciminib in ASCEMBL

AE = Adverse event. CML = Chronic myeloid leukemia. LOE = Lack of efficacy. MMR = Major molecular response. MR = Molecular response. NA = Not applicable. QD = Once daily. TKI = Tyrosine kinase inhibitor.
Data cutoff: 28 Apr 2025.

References: Rea et al., Blood (2021) 138 (21): 2031–2041.

ELVN-001 in Patient with asciminib-resistant CML with A337T mutation

MR5 post-Asciminib @ 40mg QD ELVN-001

Patient Background

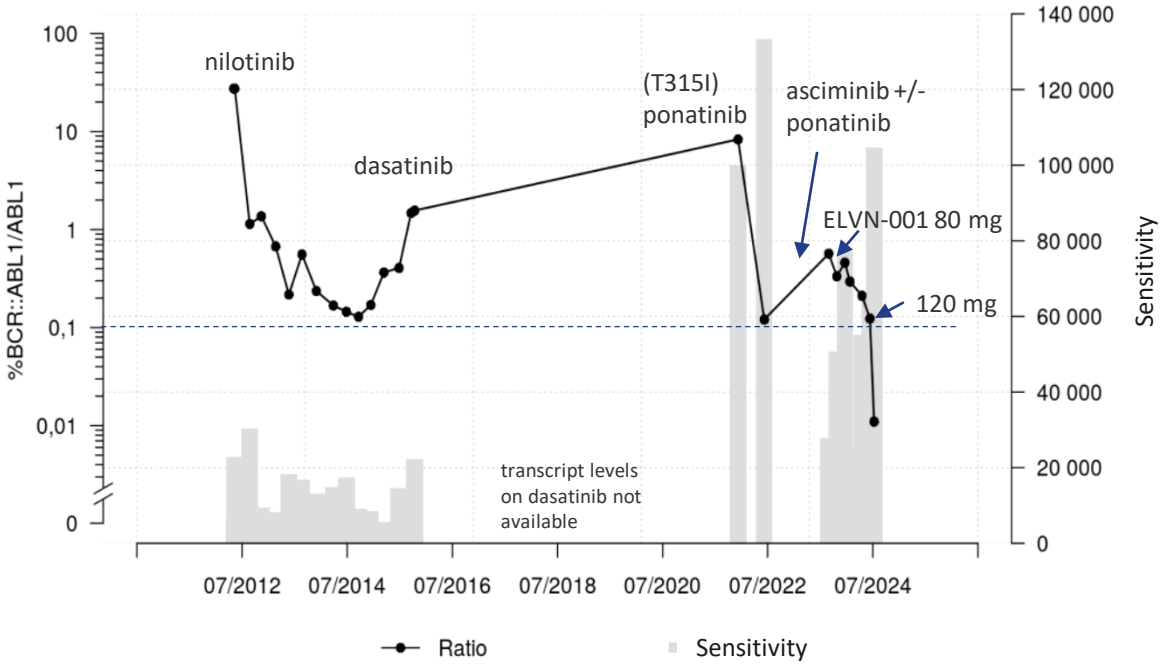
Relevant past medical history	Hyperlipidemia
Prior therapy (reason for switch)	Asciminib (LOE), ponatinib (LOE)
Mutations	A337T and V506M (mutation detected locally)
Safety	G1 rash (R) resolved by day 28 on study
Efficacy	Molecular response = MR5

A337T was the most common clinically emergent mutation that conferred resistance to asciminib on ASCEMBL

CML = Chronic myeloid leukemia. 1L = First line. G1 = Grade 1. QD = Once daily. LOE = Lack of efficacy. MR = Molecular response. R = Related to ELVN-001 per investigator.
Notes: Mutation data not confirmed by central lab (transcript level too low at baseline). Data extracted as of 18 March 2024; data from ongoing study-may change.
References: Rea et al., Blood (2021) 138 (21): 2031–2041.

Patient with BCR::ABL1 (atypical, e19a2), T315I (post-Ponatinib): Deep Response

Achieved Deep Response in T315I 80 mg → 120 mg QD ELVN-001



Patient Background

Prior therapy (reason for switch)	nilotinib (LOE), dasatinib (LOE), ponatinib (LOE), asciminib (LOE) and ponatinib + asciminib combination (LOE)
Mutations	T315I
Safety	G1 dry skin
Efficacy	>1-log decrease

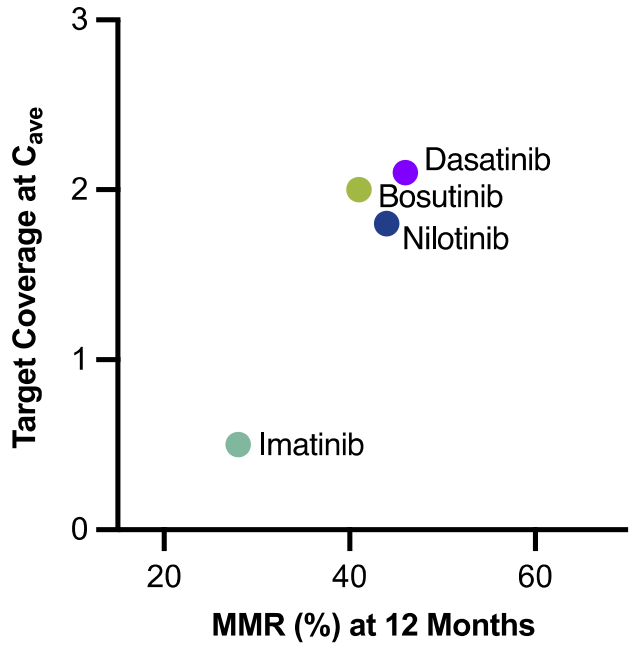
Resistant to 4 prior TKIs, deep response on ELVN-001

BCR::ABL1 = Breakpoint cluster region-Abelson leukemia virus 1. G1 = Grade 1. LOE = Lack of efficacy. MR = Molecular response. QD = Once daily. TKI = Tyrosine kinase inhibitor.
Notes: A deep response is defined as a 4-log-reduction. Data as of 12 July 2024, based on a local laboratory (non-International System) as reported by the study investigator.

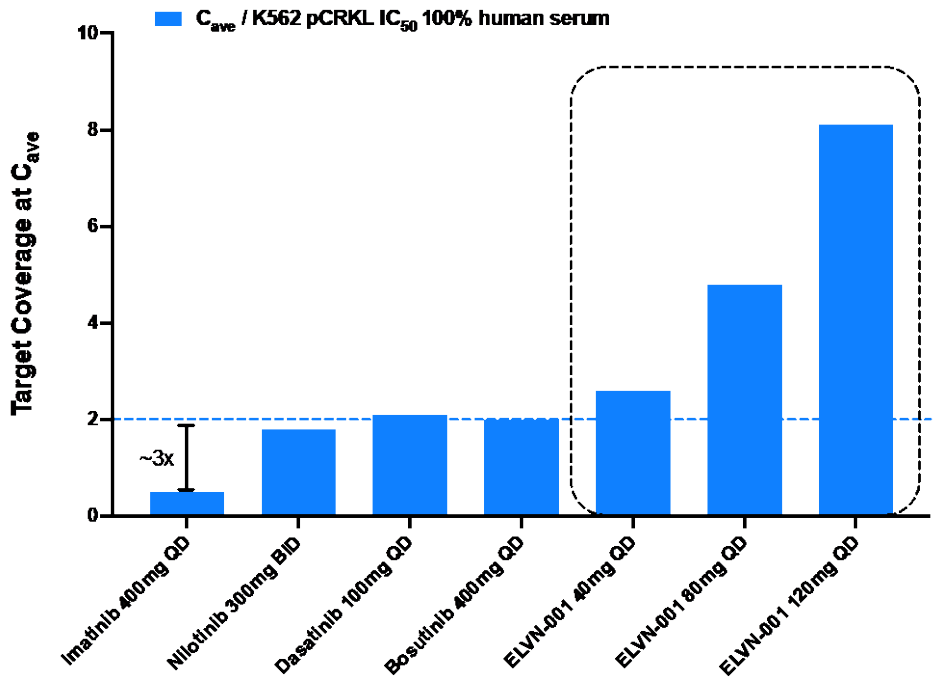
ELVN-001 Additional Information

ELVN-001 Achieved Superior Target Coverage Compared to 2nd Gen TKIs

C_{ave} Target Coverage vs. 1L MMR at 12 mo.



C_{ave} Target Coverage vs. Active Site TKIs (1L)



At doses ≥ 40mg QD, **ELVN-001 achieved better target coverage** compared to 2nd Generation TKIs

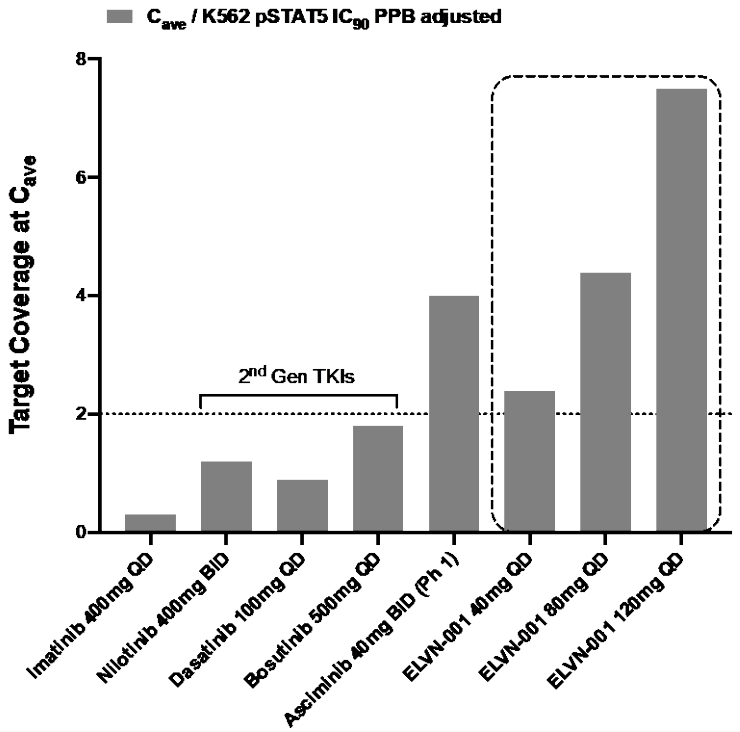
1L = First line. 2nd Gen TKIs = bosutinib, dasatinib, nilotinib. C_{ave} = Average concentration. CRKL = Crk-like protein. IC_{50} = Half-maximal inhibitory concentration. MMR = Major molecular response. QD = Once daily. TKI = Tyrosine kinase inhibitor.

References: 1. Imatinib clin pharm in CML pts: Peng et al, Clin Pharmacokinet 2005. 2. Imatinib NDA. 3. Nilotinib USPI. 4. Dasatinib USPI. 5. Bosutinib USPI.
MMR References: (Bosutinib) Cortes JE et al. J Clin Oncol. 2012; 30(28):3486-92; (Nilotinib and Imatinib) Saglio G et al. NEJM. 2010; 362(24):2251-9; (Dasatinib) Kantarjian H et al. NEJM, 2010; 362(24):226.

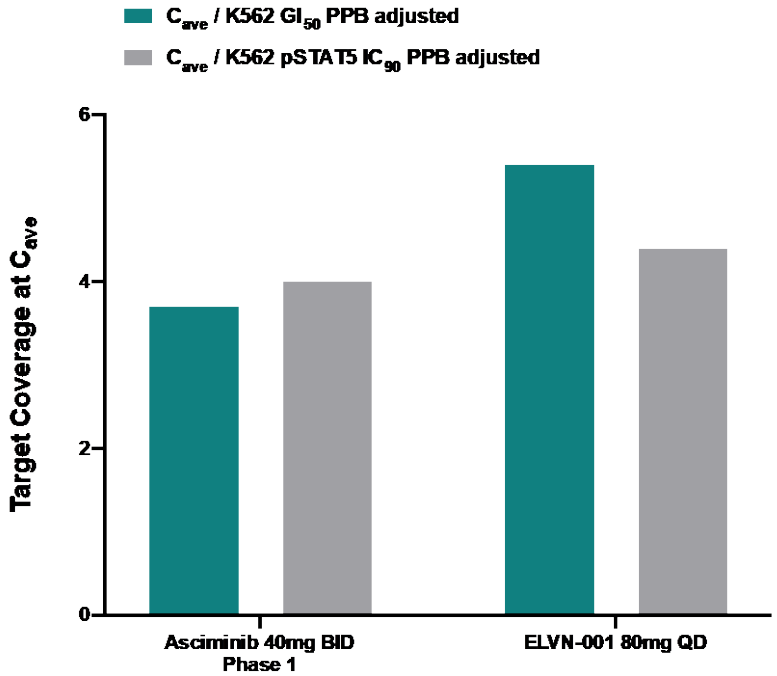
Notes: C_{ave} = Area under the curve (AUC) divided by 24 hours. For the approved drugs, human pharmacokinetic (PK) values were obtained from population PK (popPK) simulation data reported in respective USPIs or from Ref 1 (imatinib). ELVN-001 human PK values are the mean values from a preliminary popPK simulation based on PK from 78 healthy volunteer subjects; to date, there has been no significant difference between ELVN-001 PK in cancer patients and healthy subjects. *In vitro* cell pharmacodynamic measurements were performed head-to-head and represent the average value from multiple experiments (n≥3). K562 cells were employed for these experiments. pCRKL IC_{50} measurements were performed in the presence of 100% human serum.

ELVN-001 Achieved Superior Target Coverage Compared to 2nd Gen TKIs and Similar Target Coverage Compared to Asciminib

C_{ave} Target Coverage vs. All TKIs (Late Line)



C_{ave} Target Coverage vs. Asciminib (Phase 1)



- ELVN-001 had better target coverage based on plasma protein binding adjusted pSTAT5 IC_{90} at ≥ 40 mg QD compared to 2nd Gen TKIs, and similar target coverage as asciminib at 80 mg QD

- Novartis referenced preclinical 90% inhibitory concentration for phosphorylated STAT5 or pSTAT5 IC_{90} and anti-proliferation GI_{50} as the key target coverage metrics supporting an optimal asciminib dose of 40 mg BID or 80 mg QD for CML patients without T315I mutations

2nd Gen TKIs = bosutinib, dasatinib, nilotinib. BID = Twice daily. C_{ave} = Average concentration. GI_{50} = 50% growth inhibition. IC_{90} = 90% inhibitory concentration. QD = Once daily. CML = Chronic myeloid leukemia. CRKL = CRK-like protein. PPB = Plasma protein binding. STAT5 = Signal transducer and activator of transcription 5. TKI = Tyrosine kinase inhibitor.

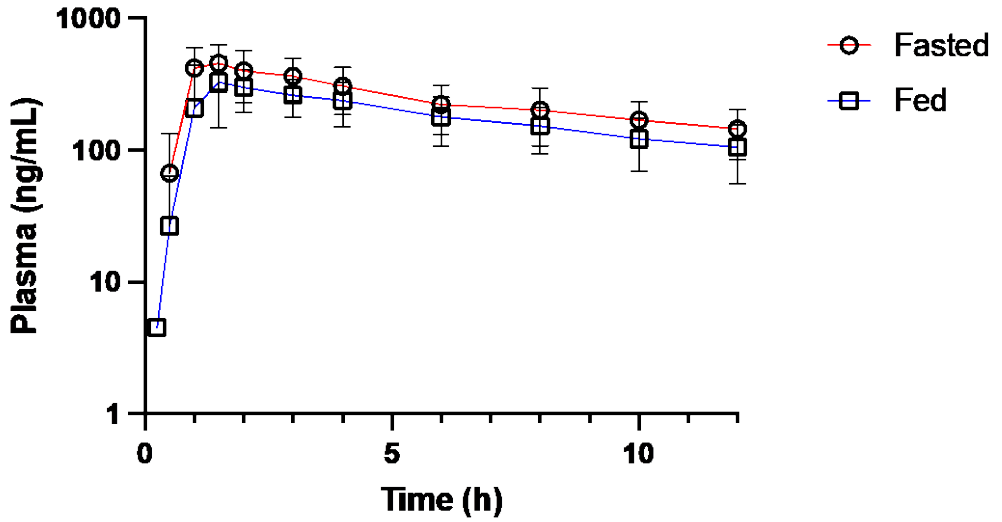
References: 1. Imatinib clin pharm in CML pts: Peng et al, Clin Pharmacokinet 2005. 2. Imatinib NDA. 3. Nilotinib USPI. 4. Dasatinib USPI. 5. Bosutinib USPI. 6. Hughes TP et al. NEJM. 2019;381(24):2315-2326. 7. Asciminb NDA.

Notes: Cave = Area under the curve (AUC) divided by 24 hours. For the approved drugs, human pharmacokinetic (PK) values were obtained from population PK (popPK) simulation data reported in respective USPIs or from Ref 1 (imatinib) and Ref 6 (asciminib Phase 1). ELVN-001 human PK values are the mean values from a preliminary popPK simulation based on PK from 78 healthy volunteer subjects; to date, there has been no significant difference between ELVN-001 PK in cancer patients and healthy subjects. Human plasma protein binding values were obtained from the respective NDAs or measured in house (ELVN-001). *In vitro* cell pharmacodynamic measurements were performed head-to-head and represent the average value from multiple experiments (n≥3). K562 cells were employed for these experiments. pSTAT5 IC_{90} and GI_{50} measurements were performed in 10% FBS and the values were adjusted to account for human plasma protein binding by dividing by the unbound fraction for each drug.

ELVN-001's PK Profile Supports Once Daily Dosing with Flexible Administration Requirements

- Linear PK observed in healthy volunteers (HV) and patients
  - No time-dependent PK observed in either HVs or cancer patients
  - Both C_{max} and AUC increased dose-proportionally
  - High concordance between HV and patient PK based on current data
- Fast and complete absorption with no significant food effect
- Mean terminal $t_{1/2}$ is ~12 hours in healthy volunteers
  - Similar effective $t_{1/2}$ observed in patients (10-20 hours)
  - Suitable for QD regimen
- Minimal risk of drug-drug interactions (DDIs)
  - Not an inhibitor (competitive or time-dependent) or inducer of major CYP enzymes, or of UGT1A1
  - Not a substrate for major CYP enzymes
  - Not a substrate of BCRP or P-gp
- No correlation between AEs and PK parameters in patients

120 mg ELVN-001 (single dose)

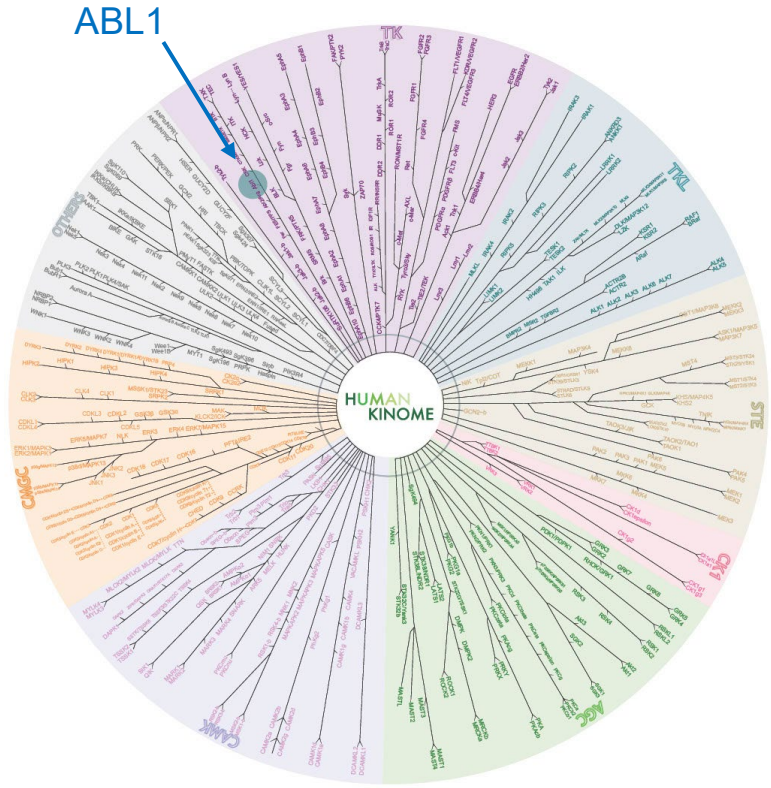


- Food effect study at 120mg single dose in HVs showed that:
  - AUC_{inf} under fasting conditions were similar to that under fed conditions, with a fed/fasted AUC ratio of 1.2.
  - C_{max} under fasting conditions were similar to that under fed conditions, with a fed/fasted C_{max} ratio of 0.8.

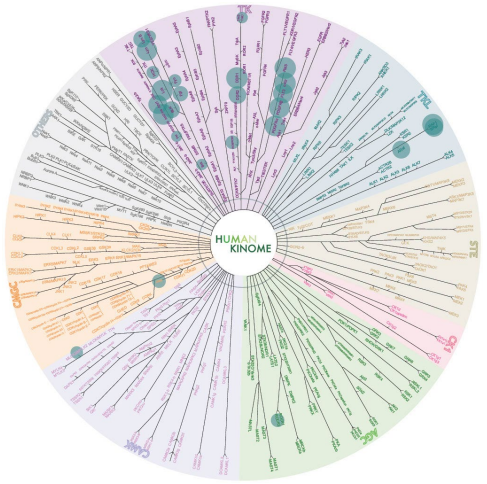
AEs = Adverse events. AUC = Area under the curve. AUC_{inf} = AUC extrapolated to infinity. BCRP = Breast cancer resistance protein. C_{max} = Maximum serum concentration. CYP = Cytochromes P450. DDI = Drug-drug interactions. HV = Healthy volunteers. P-gp = P-glycoprotein. PK = Pharmacokinetics. QD = Once daily. $t_{1/2}$ = Half life. UGT1A1 = UDP Glucuronosyltransferase Family 1 Member A1.

ABL TKI Kinome Profiles [30x ABL1 IC_{50}]

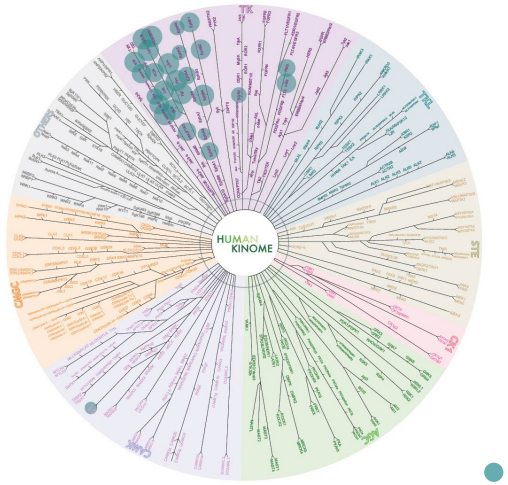
- Unlike other active site TKIs, ELVN-001 did not inhibit any kinases other than ABL1 >50% at 30x IC_{50} (~IC_{95})[1]



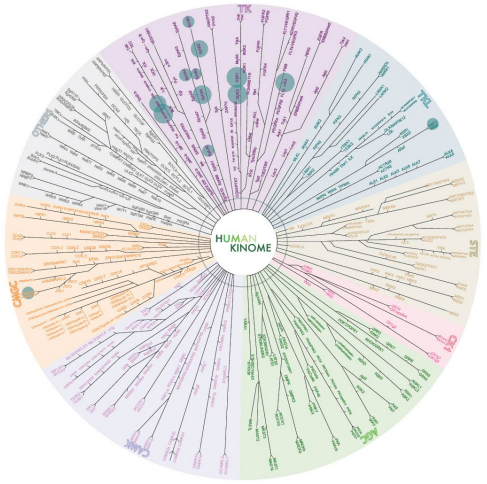
ELVN-001



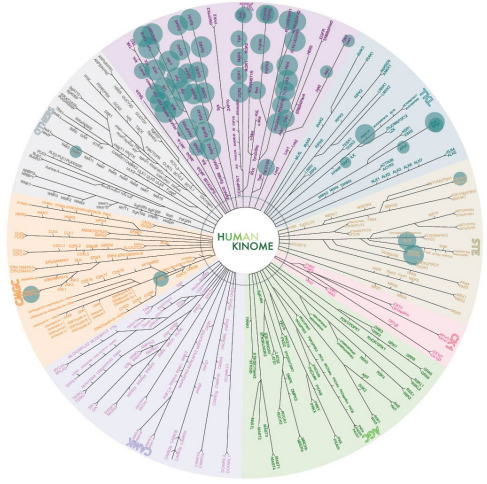
Imatinib



Dasatinib



Nilotinib



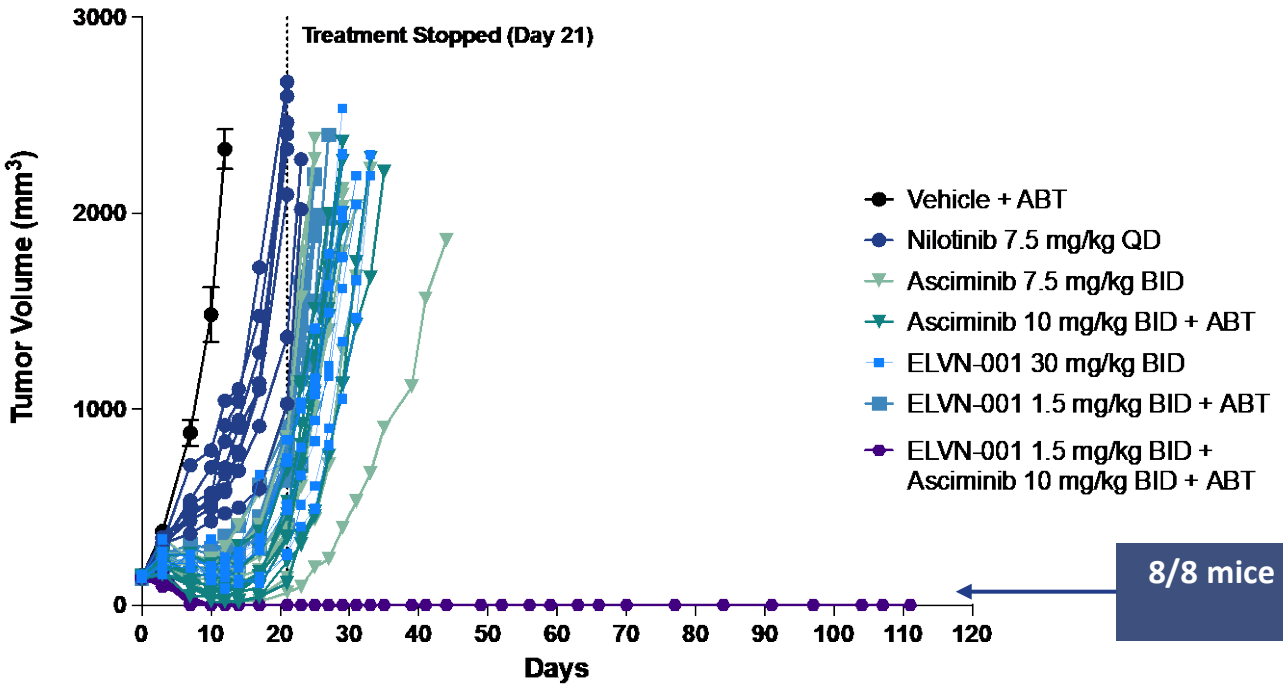
Ponatinib

- 80-100%
- 70-80%
- 60-70%
- 50-60%

ABL1= Abelson murine leukemia viral oncogene homolog 1. IC_{50} = Half-maximal inhibitory concentration. IC_{95} = 95% inhibitory concentration. TKI= Tyrosine kinase inhibitor.
Notes: 1. Kinase inhibition assay with: ABL1 enzyme concentration 0.2 nM, 100 uM ATP.

ELVN-001 + Asciminib Combination Elicits Cures at Physiologically Relevant (Human-Matched) Drug Exposures in Mice

K562 Xenograft



8/8 mice cured in combination arm @ Day 111 (90 days after last dose)

Drug Exposure: Mouse vs. Human

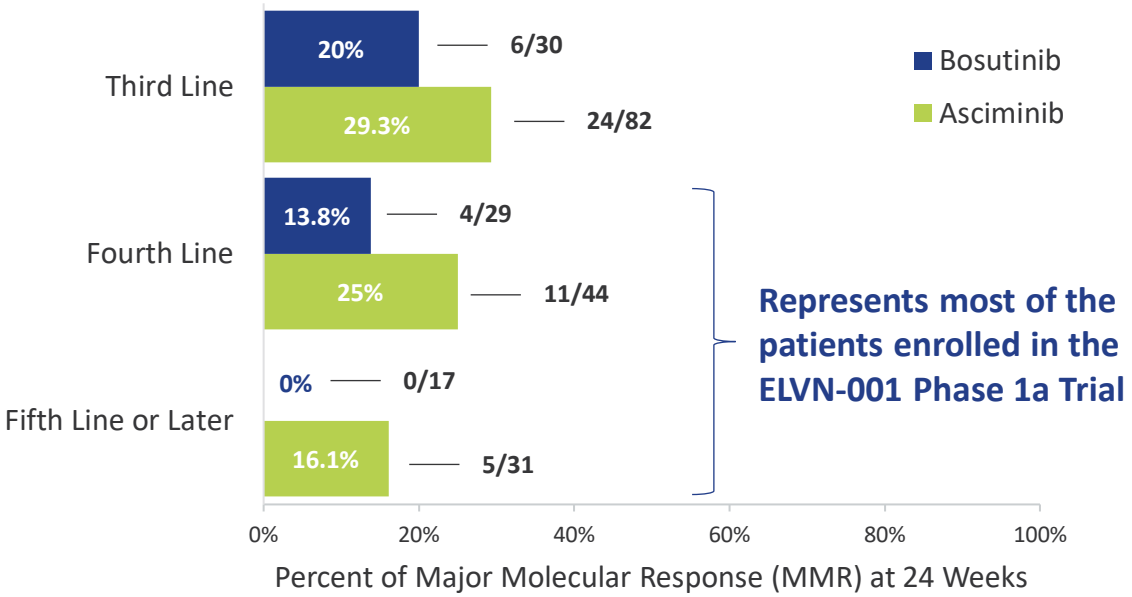
	Mouse Dose (mg/kg)	Human Dose (mg)	Ratio of mouse to human exposure
Nilotinib	7.5 QD	400 BID	**1.9**
Asciminib	7.5 BID	40 BID	**1.2**
Asciminib	10 BID*	40 BID	**1.7**
ELVN-001	30 BID	80 QD	**1.1**
ELVN-001	1.5 BID*	80 QD	**1.0**

- ELVN-001 elicits anti-tumor activity in this model comparable to asciminib and superior to nilotinib at their respective human-matched drug exposures
- **Combination treatment with ELVN-001 + asciminib** for 21 days at their respective human-matched drug exposures **resulted in 8/8 cures** in this model as of Day 111 (90 days after treatment discontinuation); no cures observed in the monotherapy arms

BID = Twice daily. QD = Once daily. Cure = No evidence of recurrent disease 90 days after last dose.
Notes: *Co-dosed with ABT, a CYP inhibitor that increased the exposure of ELVN-001 in mouse PK studies to better mimic its human PK profile. PK studies were performed to confirm no significant drug-drug-interactions in combination; in fact, the combination resulted in slightly lower exposures compared to the respective monotherapy PK at the doses described.
Exposure: unbound fraction area under the curve (AUC); mouse exposure represents Day 1 PK values for nilotinib and asciminib and Day 5 (steady state) exposure for ELVN-001 and asciminib + ABT to take into account potential induction related to ABT administration. ELVN-001 human PK values are the mean values from a preliminary popPK simulation based on PK from 78 healthy volunteer subjects; to date, there has been no significant difference between ELVN-001 PK in cancer patients and healthy subjects.
References: Nilotinib NDA & USPI.; Hughes TP et al. NEJM. 2019;381(24):2315-2326.

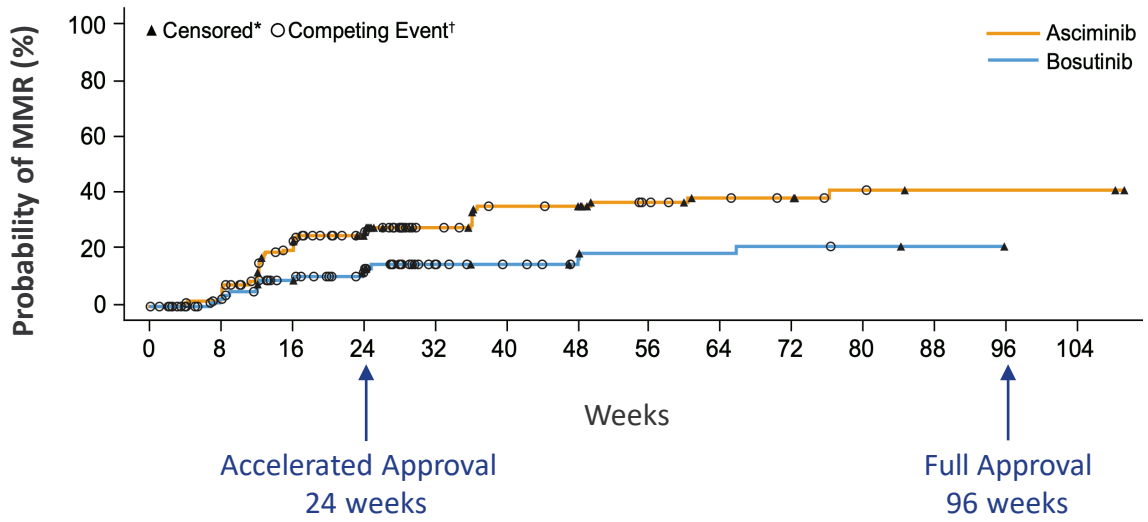
Phase 1 Data Predicted Pivotal Trial Data
Asciminib vs. Bosutinib in Late-Line CML (ASCEMBL)

Percent MMR at 24 Weeks



MMR rate decreased with increasing number of prior TKIs

Probability of MMR Over Time



MMR rate increased over time for both drugs

- Cumulative MMR at 24 weeks for asciminib vs. bosutinib was 25% vs. 12%
- Dose reductions due to adverse events: 21% asciminib vs. 42% bosutinib*

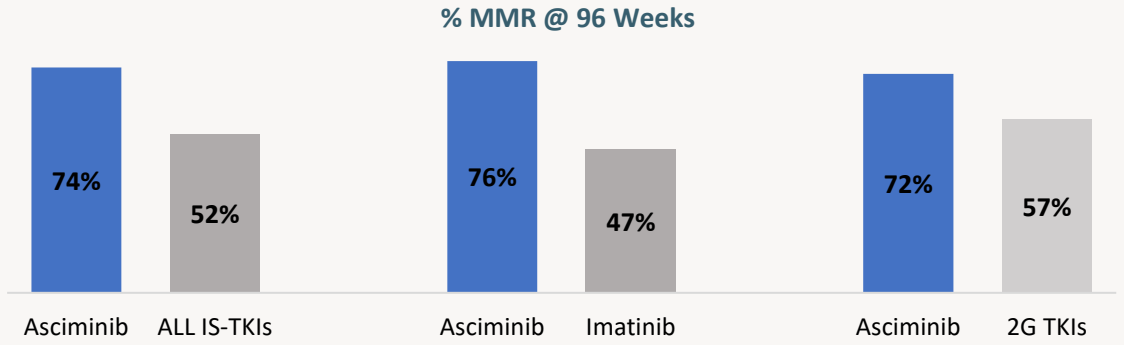
CML = Chronic myeloid leukemia. MMR = Major molecular response. TKI = Tyrosine kinase inhibitor.
Notes: *Median duration of exposure of 23.7 months for asciminib and 7 months for bosutinib. Note: overall MMR by 24 weeks was 25.5% for asciminib and 13.2% for bosutinib.
References: Rea D et al., Blood. 2021.

Scemblix Updates Continue to Demonstrate the Need for Better Treatment Options

ASC4FIRST 96-week Data Update

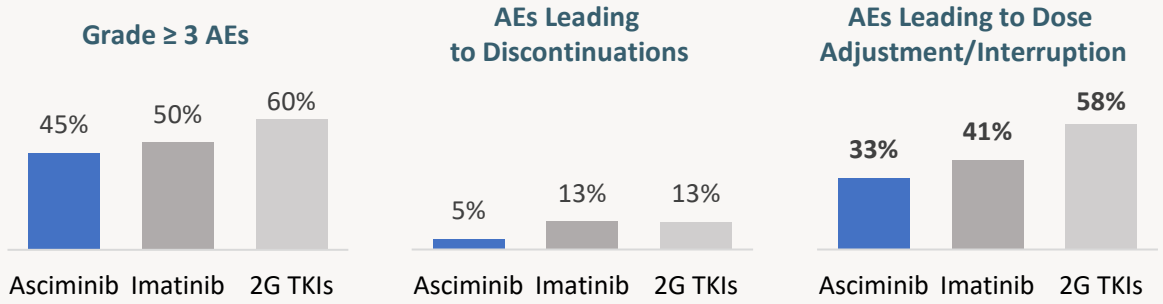
Efficacy

- Sustained MMR advantage vs all investigator selected TKIs
- Clinically relevant 15% higher MMR rate vs 2G TKIs (95% CI, 2.3-28)



Safety / Tolerability

- Favorable safety/tolerability profile compared to imatinib and 2G TKIs



Review of the Recent Scemblix Updates

Recent Updates	
Oct. 2024	• Accelerated Approval for 1L and received broad approval for any patient with CML
Nov. 2024	• NCCN Category 1 Preferred recommendation received
Dec. 2024 (ASH)	• ASC4FIRST 96-week update shows continued efficacy and safety advantage

1Q 2025 Report	• $238M in sales, implying annualized revenue of **$952M**
	• High adoption rate shown by patients making switch decisions:
	  • **40% NBRx** share in 2L U.S.
	  • **10% NBRx** share in 1L U.S.

1L = First line. 2L = Second line. 3L+ = Third line or later. 2G TKIs = Second generation TKIs - bosutinib, dasatinib, nilotinib. AE = Adverse event. ASH = American Society of Hematology. CML = Chronic myeloid leukemia. IS = Investigator-selected. MMR = Major molecular response. NBRx = New to brand prescription. NCCN = National comprehensive cancer network. TKI = Tyrosine kinase inhibitor.

References: Cortes et al. ASH 2024; Scemblix (Asciminib) USPI; Publicly available filings and press releases

Only Myristoyl Pocket Mutations Emerge Post 1L Asciminib (ASC4FRIST)

Patients	Post-baseline mutations[a]	Discontinuation reason	Postprotocol therapy (2L+)	Last disease/survival status
Asciminib	**Myristoyl pocket**			
1	A433D	Treatment failure per ELN	Bosutinib, dasatinib	CP/alive
2	A337V, V506M[b]		Dasatinib	CP/alive
3	A337T, A344P,[b] P465Q,[b] I502N[b]		Dasatinib	AP/alive
4	A433D		Dasatinib, olverembatinib	AP/alive
5	A337T, V506M[b]		Ponatinib	Discontinued study
6	L340Q		Not available	Discontinued study
7[c]	A337T	Confirmed loss of MMR	Dasatinib	Discontinued study
8	A337T, L340Q	Unsatisfactory therapeutic effect (other)	Dasatinib	CP/alive
9	A337T,[b] F497L[b]	Progressive disease (BP)	Ponatinib	CP/death post HSCT
10[c]	A337V	Ongoing on study	Not applicable	
Imatinib	**ATP-binding domain**			
1	L248V, E255V,[b] G250E[b]	Treatment failure per ELN	Flumatinib, olverembatinib	BP/death post HSCT
2[c]	F317L[b]		Imatinib	CP/alive
3	L248V, E450G[b]		Nilotinib	CP/alive
4[c]	E459K	Confirmed loss of MMR	Dasatinib	CP/alive
Nilotinib	**ATP-binding domain**			
5[c]	Y253H	Treatment failure per ELN	Dasatinib	CP/alive
6	Y253H		Dasatinib, ponatinib	CP/alive
7	Y253H[b]	Ongoing on study	Not applicable	

2L+= Second line and beyond. AP = Accelerated phase. ATP = Adenosine triphosphate. BP - Blast phase. CP=Chronic phase. ELN = Electronic laboratory notebook. HSCT=Hematopoietic stem cell transplant. NGS=Next-generation sequencing.
[a] A patient with multiple mutations is only counted once. [b] Variant allele frequency was <20%. [c] Patients with new mutations since the week 48 data cutoff (November 28, 2023).
Reference: Cortes J et al, ASH 2024

Review of Asciminib ASCEMBL Study

Observations

- Asciminib's **strong launch** demonstrates the **large market** size and **need for better agents**
- However, unmet needs still exist. In ASCEMBL, only **1.2% of patients discontinued due to PD/death, but due to lack of efficacy/AE:**
  - ~30% of patients discontinue by week 48
  - **~50% of patients discontinue** by week 96
- Asciminib has limitations:
  - **Resistance mutations** in both the allosteric binding site and the ATP pocket result in loss of activity
  - **Drug-drug interactions** require avoiding drugs that are CYP2C9 substrates (up to 20% of commonly prescribed medications)
  - **Requires fasting** 2 hours before and 1 hour after each dose
  - **Substrate for efflux transporters** (P-gP & BCRP), which may contribute to **lack of efficacy**
  - Treatment of **T315I mutations requires 5x dose** resulting in more dose reductions (23%), increased pancreatic & liver enzyme elevation

Emerging BCR-ABL mutations upon discontinuation due to lack of efficacy or progressive disease

	Asciminib (n=39)	Bosutinib (n=30)
No mutations	22 (56%)	20 (67%)
ATP Binding Site	M244V (n=3), E355G, F359V, T315I	T315I, V299L
Myristol Binding Pocket	A337T (n=3), P465	None

Mutations at baseline & end of treatment

ATP Binding Site	F359C/V (n=3), F317L (n=2), Y253H	M244V (n=2), E255V, F317L, Q252H

1L = First line. 2L = Second line. 3L+ = Third line and later. AE = Adverse event. ATP = Adenosine triphosphate. BCR::ABL1 = Breakpoint cluster region-Abelson leukemia virus 1 B = Billions. BCRP = Breast cancer resistance protein. EU = European Union. M = Millions. PD = Progressive disease. P-gp = P-glycoprotein. Q4 = Fourth fiscal quarter. TKI = Tyrosine kinase inhibitor. ASCEMBL: A phase 3, open-label, randomized study of asciminib vs bosutinib in CML after 2 or more prior TKIs.

References: Publicly available filings, announcements and research reports; Hochhaus et al. ASH 2020; Cortes et al. ASH 2020; ASH 2021; Scemblix (Asciminib) USPI; ASCO 2022; Eadie et al Oncotarget 2018; Pharmaceogent Genomics, 2010 Apr; 20(4):277-281]; Novartis 2Q 2024 Investor Presentation.

Notes: 3L+ market dynamics based on Novartis 4Q 2024 investor presentation. Estimated 3L+ market size calculated using Scemblix Q2 24 U.S.annualized sales and 26% U.S. market penetrance.

Poor Selectivity Limits Tolerability & Efficacy of 1st, 2nd & 3rd Gen Agents

	Compound	Off Target(s) & Treatment-Emergent, Non-Hematologic Adverse Events (All Gr / Gr 3+)		1L Efficacy	Drug & Administration Requirements	Peak Sales (USD) *(US WAC in Peak Sales Year)*	
1st Gen	**Imatinib (Gleevec®)**	c-KIT, CSFR-1, PDGFR	Peripheral Edema (20% / 0%) Nausea (41% / 2%)	28% MMR 3% MR4.5	Avoid strong CYP3A inhibitors or inducers	**$4.7B**	**$120K** *(2014)*
2nd Gen	**Dasatinib (Spyrcel®)**	SRC family, c-KIT, PDGFR-αβ	Fluid Retention (38% / 5%) Pleural Effusions (28% / 3%) Diarrhea (22% / 1%)	46% MMR 5% MR4.5	Avoid strong CYP3A inhibitors or inducers, PPIs, antacids, and H2 blockers	**$2.2B**	**$190K** *(2022)*
2nd Gen	**Nilotinib (Tasigna®)**	c-KIT, PDGFR, CSFR-1, DDR-1 (hERG Channel)	Rash (38% / <1%) Headache (32% / 3%) Nausea (22% / 2%); Diarrhea (19% / 1%) **Black Box:** QT Prolongation/Sudden Deaths	44% MMR 11% MR4.5	Avoid strong CYP3A inhibitors or inducers and PPIs; avoid food 2 hours before and 1 hour after each dose	**$2.1B**	**$203K** *(2021)*
2nd Gen	**Bosutinib (Bosulif®)**	SRC family	Hepatic dysfunction (45% / 27%) Diarrhea (75% / 9%) Abdominal Pain (39% / 2%)	41% MMR 7.5% MR4.5	Avoid strong CYP3A inhibitors or inducers, PPIs, antacids, and H2 blockers	**$650M**	**$241K** *(2024)*
3rd Gen	**Ponatinib (Iclusig®)**	KDR, FGFR, c-KIT, RET, FLT3, PDGFR	**Black Box:** Arterial Occlusive Events, Heart Failure, VTE, Hepatoxicity	N/A	Avoid strong CYP3A inhibitors or inducers	**$640M**	**$256K** *(2024)*
STAMP	**Asciminib (Scemblix®)**	N/A	Hypersensitivity (32% / 2%) Hypertension (19% / 9%) Cardiovascular (13% / 3.4%)	68% MMR 17% MR4.5	Avoid CYP2C9 substrates and certain statins; avoid food 2 hours before and 1 hour after each dose	**$690M**	**$261K** *(2024)*

A selective BCR-ABL inhibitor could yield enhanced target coverage, leading to greater efficacy and better long-term tolerability

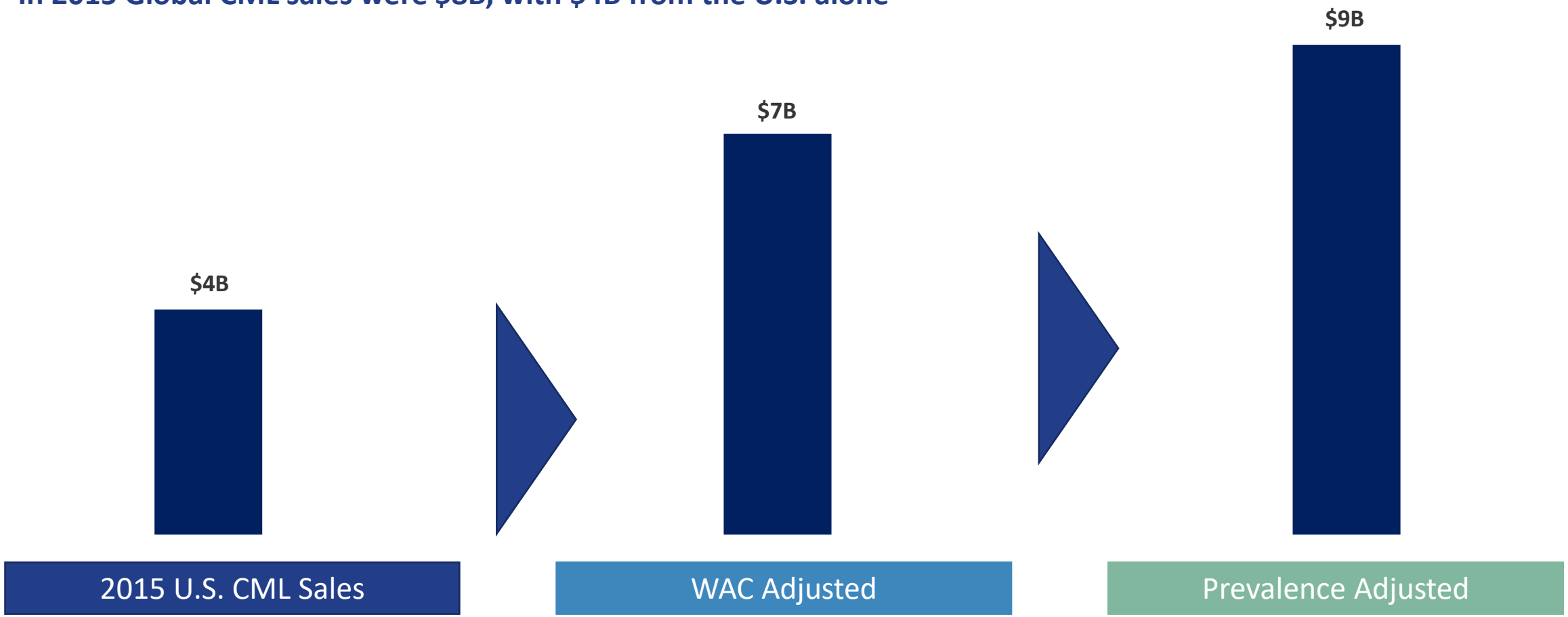
1L = Front line. B = billion. Gen = Generation. GI = Gastrointestinal. Gr = Grade. FY = Fiscal Year. K = thousands. . M = millions. MMR = Major Molecular Response. MR4.5 = Deep Molecular Response. PPI = Proton pump inhibitors. STAMP = specifically targeting the ABL myristoyl pocket. MMR and MR4.5 at 12 months. VTE = Venous thromboembolism. WAC = Wholesale acquisition cost.

Notes: Numbers in the billions have been rounded to the 1/10th of a billion and sales numbers in the millions have been rounded to the nearest $10 million increment from Company Investor Reports. Iclusig sales calculated using the latest available information by region for the 4 trialing quarters; Iclusig Japan sales reported by Otsuka are as of 2020.

References: Publicly available filings, announcements and research reports; Gleevec® (imatinib) USPI; Sprycel® (dasatinib) USPI; Kantarjian H et al. NEJM, 2010; 362(24):2260-70; Cortes JE et al. J Clin Oncol. 2016; 34(20):2333-40; Tasigna® (nilotinib) USPI; Saglio G et al. NEJM 2010; 362(24):2251-9; Hochhaus A et al. Leukemia. 2016; 30(5):1044-54; Bosulif® (bosutinib) USPI. Cortes JE et al. J Clin Oncol, 2012; 30(28):3486-92; Iclusig® (ponatinib) USPI; Scemblix® (asciminib) USPI. Hochhaus A et al. NEJM, 2024; 391(10):885-898.

The U.S. CML Market has Historically Been Large and has the Potential to be Larger

In 2015 Global CML sales were $8B, with $4B from the U.S. alone



Since 2015, the U.S. CML sales have on average been ~50% of global sales

Since 2015, the weighted average WAC has increased by 78%

Since 2015, prevalence has increased ~22%

B = Billions. CML = Chronic myeloid leukemia. WAC = Wholesale acquisition cost.

References: Publicly available filings, announcements and research reports; Huang X et al. Cancer. 2012;118:3213-3127; PriceRx; Assumes 2024 weighted average U.S. WAC of ~$240,000. Assumes current U.S. prevalence of ~110,000 based on American Journal of Hematology: Chronic myeloid leukemia: 2025 update on diagnosis, therapy, and monitoring.

Molecular Response Milestones

≥ 1 log reduction in BCR::ABL1 transcript levels is a meaningful indication of efficacy

BCR::ABL1 Transcript Level	Molecular Response	Relevance
≤ 10%	MR1	• Even in 3L+ setting insufficient for optimal survival
≤ 1%	MR2	• Equivalent to complete cytogenetic remission (absence of Philadelphia chromosome) by bone marrow biopsy
≤ 0.1%	MR3	• ≥ MR3 is also known as a **major molecular response (MMR)** • Has become a key regulatory endpoint as this predicts close to 100% CML-specific survival
≤ 0.01%	MR4	• MR4/4.5 lasting ≥ 2 years has been used as a benchmark to stop treatment (typically in earlier line treatment), which is the ultimate goal
≤ 0.0032%	MR4.5	
≤ 0.001%	MR5	

There is no standard definition of an acceptable response to third, fourth or fifth-line treatment

3L+ = Third line or later. BCR::ABL1 = Breakpoint cluster region-Abelson leukemia virus 1. CML = Chronic myeloid leukemia. MMR = Major molecular response. MR = Molecular response.
Molecular response in CML is measured by the ratio of BCR::ABL1/ABL1.
References: Hochhaus, et al. Leukemia 2020; NCCN Guidelines 2.2024.