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TERN-701

Investor Educational Webinar

NASDAQ: TERN

September 3, 2025

Forward-Looking Statements and Disclaimers

This presentation contains forward-looking statements about Terns Pharmaceuticals, Inc. (the "Company," "we," "us," or "our") and its industry within the meaning of the federal securities laws that involve substantial risks and uncertainties. All statements other than statements of historical facts contained in this presentation, including any statements regarding the Company's strategy, future operations, future results, future financial condition, projected costs, prospects and plans, objectives of management, and expected industry and market trends, are forward-looking statements. Forward-looking statements in this presentation include statements related to or in connection with expectations, timing and potential results of the clinical trials and other development activities for TERN-701, including with respect to the CARDINAL trial; the therapeutic potential of TERN-701; the potential for the mechanism of action of TERN-701 to effectively target CML; the potential utility and progress of TERN-701 for the treatment of CML, including the clinical utility of the data from and the endpoints used in the Company's clinical trials; the applicability of expected parameters and benchmarks on which to assess clinical trial results for TERN-701; the Company's clinical development plans and activities for TERN-701, including potential future trial designs, milestones and the results of any interactions with regulatory authorities; the Company's expectations regarding the profile and potential beneficial characteristics and therapeutic effects of TERN-701, including with respect to efficacy, tolerability, safety, pharmacokinetic profile and convenience and potential differentiation as compared to competitive products or product candidates; the best-in-class potential of TERN-701; the Company's plans for and ability to continue to execute on its current development strategy for TERN-701; the market potential for TERN-701; the anticipated regulatory pathway for TERN-701; and possible future regulatory approval and commercialization of TERN-701. In some cases, you can identify forward-looking statements by terminology such as "aim," "anticipate," "assume," "believe," "contemplate," "continue," "could," "design," "due," "estimate," "expect," "goal," "intend," "may," "objective," "plan," "position," "potential," "predict," "seek," "should," "target," "will," "would" and other similar expressions that are predictions of or indicate future events and future trends, or the negative of these terms or other comparable terminology. The Company has based these forward-looking statements largely on its current expectations, estimates, forecasts and projections about future events and financial trends that it believes may affect its financial condition, results of operations, business strategy and financial needs.

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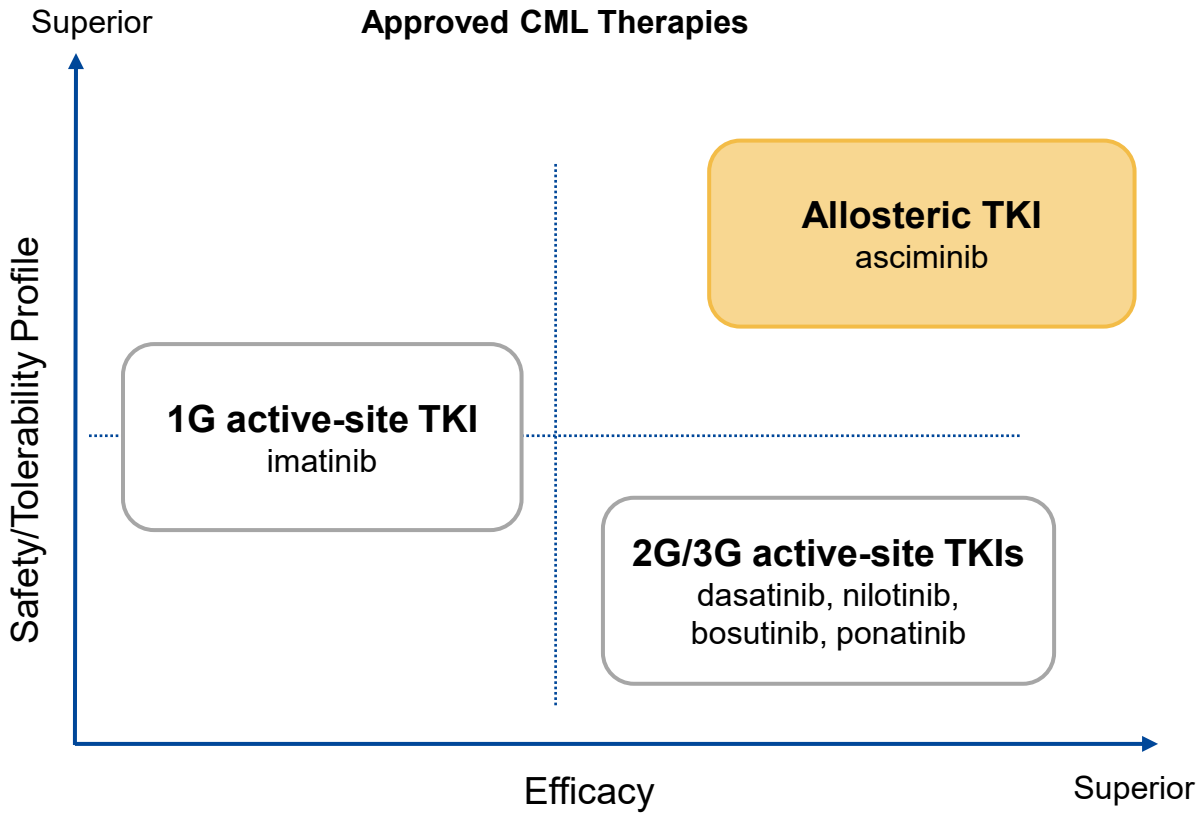
This presentation discusses product candidates that are investigational only and have not yet been approved for marketing by the FDA. No representation is made as to the safety or effectiveness of these product candidates for the use for which such product candidates are being studied. Data presented for our product candidates and other agents 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 information.

Agenda and Presenters

- **Opening Remarks** / Amy Burroughs, CEO
- **Recapping TERN-701's Best-in-Class Potential** / Emil Kuriakose, MD, CMO
- **Calibrating and Assessing CML Phase 1 Data** / Emil Kuriakose, MD, CMO
- **Future of CML and Development Path Ahead** / Scott Harris, CDO
- **Closing** / Amy Burroughs, CEO

The First Allosteric Inhibitor is Rapidly Becoming the Preferred Therapy of Choice in CML

Allosteric Inhibitors Represent a Significant Advancement Over All Active-site TKIs[1]



Asciminib Use is Rapidly Increasing in 1L and Beyond[2]

15% Share of front-line

35% ‘New to brand’ Rx share across all lines

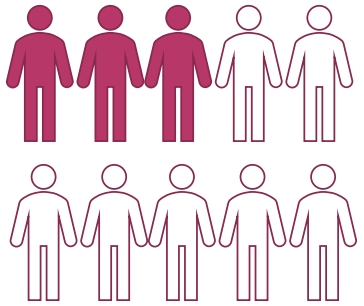
$3B+ Anticipated peak sales

1. Novartis ASCO Investor Event | June 2, 2024. 2. Novartis Q2 2025 Results Presentation | July 17, 2025

However, Asciminib Leaves Opportunities for Improvement Across Efficacy, Safety and Convenience

Asciminib Patients Who *Fail to Reach Efficacy* threshold ( )

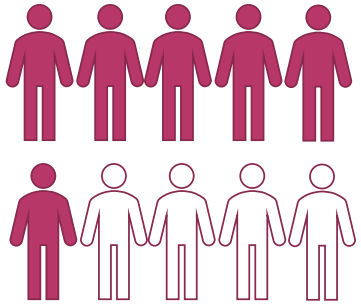
1L



- 32% -

do not reach **MMR** at 48 weeks[1]

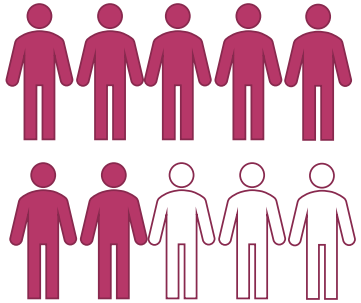
2L



- 57% -

do not reach **MMR** at 24 weeks[2]

3L+



- 75% -

do not reach **MMR** at 24 weeks[3]

Adverse Event Profile of Asciminib[4]

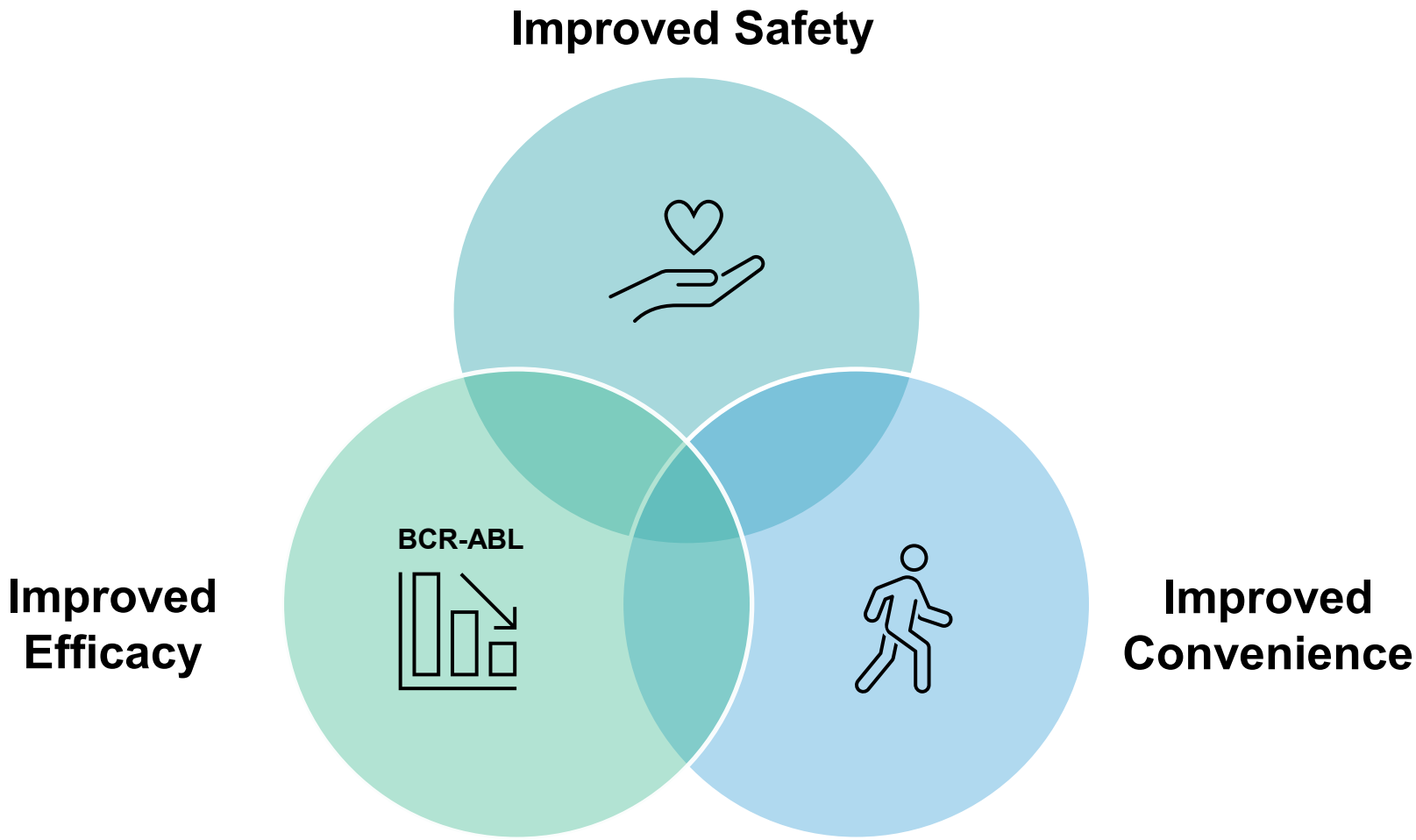
Pancreatic Toxicity	19%
Hypertension	16%

Patient Adherence[4]

Can’t be taken with food

1. Hochhaus A, et al. N Engl J Med 2024;391:885-898. 2. Atallah E. et al. Blood 2024; 144 (Supplement 1): 479. 3. Rea D et al. Blood 2021; 138 (21): 2031–2041.
4. SCEMBLIX® (asciminib). Prescribing information, 2024. Accessed August 2025.

Our Goal for TERN-701 is to be the Best Allosteric TKI in CML



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Recapping TERN-701's Best-in-Class Potential

Emil Kuriakose, MD
Chief Medical Officer

CARDINAL Phase 1 Study of TERN-701 in Chronic Myeloid Leukemia

Upcoming 4Q25 data will include patients from dose escalation and expansion cohorts

CARDINAL

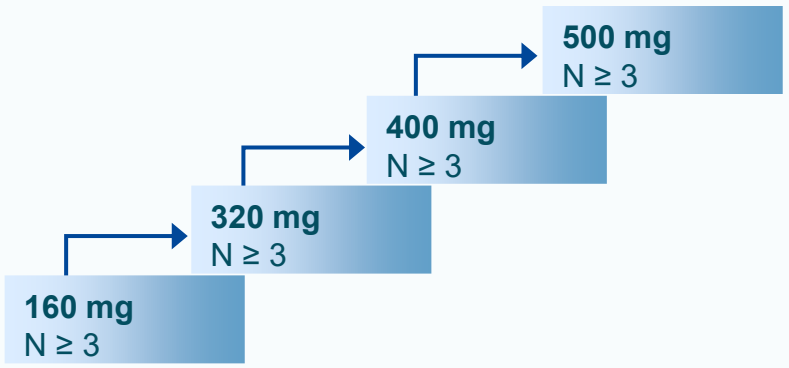
Study Population

Chronic phase **2L+** CML patients w/wo BCR-ABL1 mutations who have had:

- Treatment failure/suboptimal response OR intolerance to ≥1 TKI
- Prior asciminib treatment failure/intolerance allowed
- Myristate pocket resistance mutations excluded

Part 1 Dose Escalation

TERN-701 Once-daily Monotherapy (N= up to 80 via backfill)

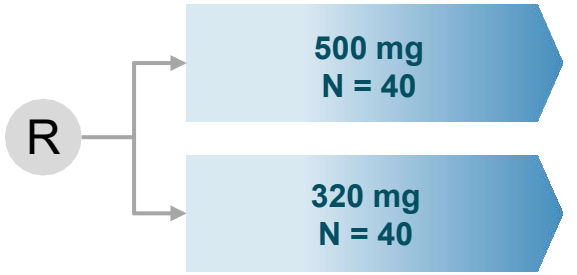


Endpoints For Part 1
- Primary: Safety/tolerability
- Secondary: PK, Efficacy

Part 2 Dose Expansion

TERN-701 Once-daily Monotherapy (N≈80)

RDE Selection



Endpoints For Part 2
- Primary: Efficacy
- Secondary: Safety/tolerability, PK

ClinicalTrials.gov identifier: NCT06163430
PK: pharmacokinetics; TKI: tyrosine kinase inhibitor

Early Data Showed TERN-701 has Potential to Achieve Best-in-Class Efficacy and Safety

Emerging Potential Differentiators for TERN-701 Based on Early Data

1. High target **selectivity** with greater **potency** than asciminib

+

2. Better in vivo **target coverage** than asciminib

+

3. Wider **therapeutic index**

=

Potential for **best-in-class MMR** achievement rates and safety

Observed in early CARDINAL data

- ✓ **Rescue of response in asciminib failure**
- ✓ **Rapid deep molecular response in asciminib naïve patient**
- ✓ **Trends towards improved safety over asciminib at higher doses**

Note: No head-to-head clinical studies have been conducted comparing TERN-701 with asciminib. Differences exist in study designs and conditions, and caution should be exercised when comparing data across studies.

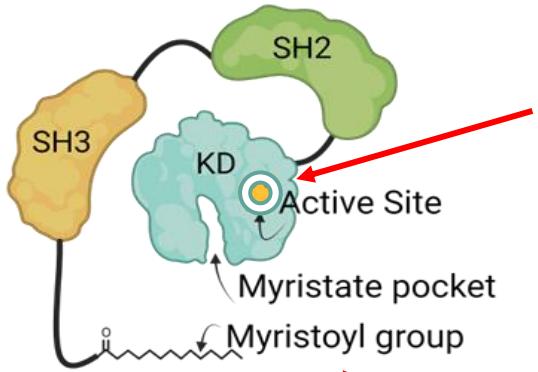
① Allosteric BCR-ABL Inhibitors are Inherently More Selective than Active Site Targeting Inhibitors

Kinase domain (KD) consists of:

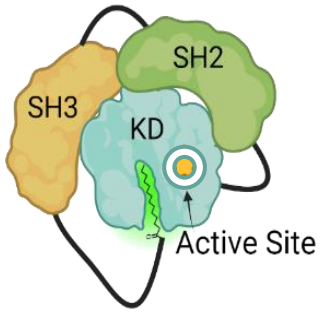
1. active site (shared with other kinases)
2. myristate pocket
3. myristoyl group (unique to ABL kinases)

Wild-type (WT) ABL

Active



Inactive

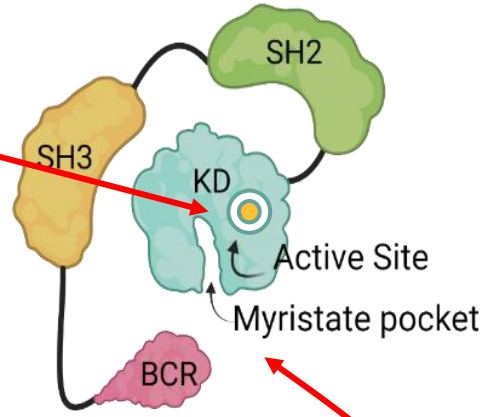


Myristoyl binding to the myristate pocket in WT ABL **turns off kinase activity**

Active site inhibitors have **low selectivity for BCR-ABL** because of its shared active site with WT ABL and other kinases[1,2,3]

BCR-ABL fusion in CML

Active



BCR replaces myristoyl group of ABL leaving myristate pocket open → **kinase stays on**

Allosteric inhibitors (bind to the myristate pocket) have

1. **high selectivity for BCR-ABL** due to its open myristate pocket that is unique to BCR-ABL[4] ; and
2. **low risk of WT ABL** inhibition due to myristoyl group blocking access to the pocket

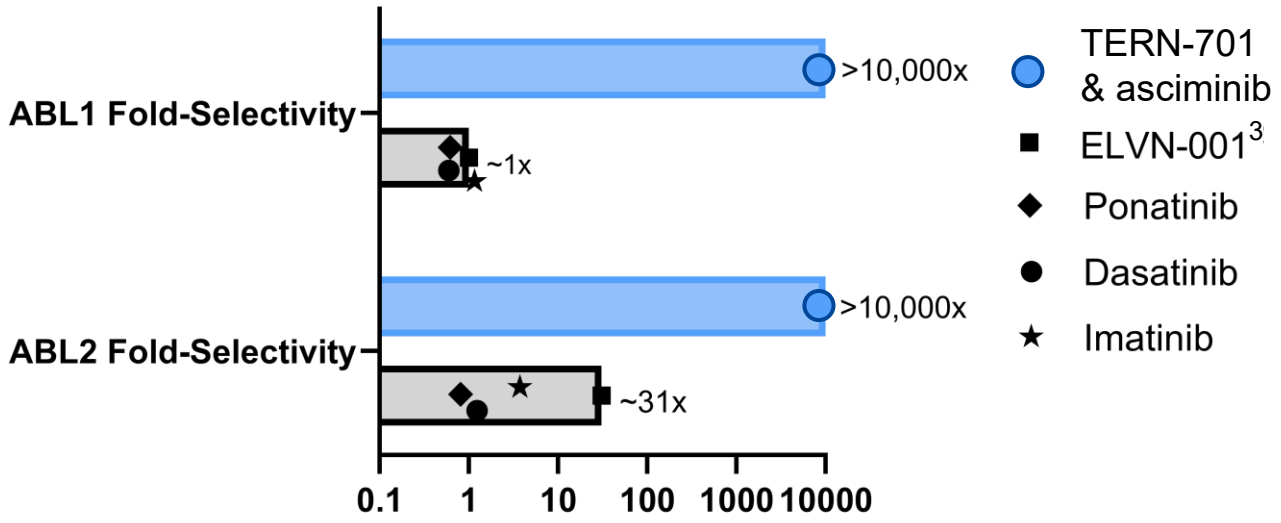
1. Rix U, et al. Blood 2007;110:4055–63. 2. Remsing Rix LL, et al. Leukemia 2009;23:477–85. 3. O'Hare T, et al. Cancer Cell 2009;16:401–12. 4. Adrián FJ, et al. Nat Chem Biol. 2006 Feb;2(2):95-102.

① High Selectivity of Allosteric Inhibitors Allows Targeted, Potent BCR-ABL Inhibition Without Interfering with Activity of Normal Kinases

Less off-target kinase inhibition allows improved safety compared to non-selective active-site TKIs

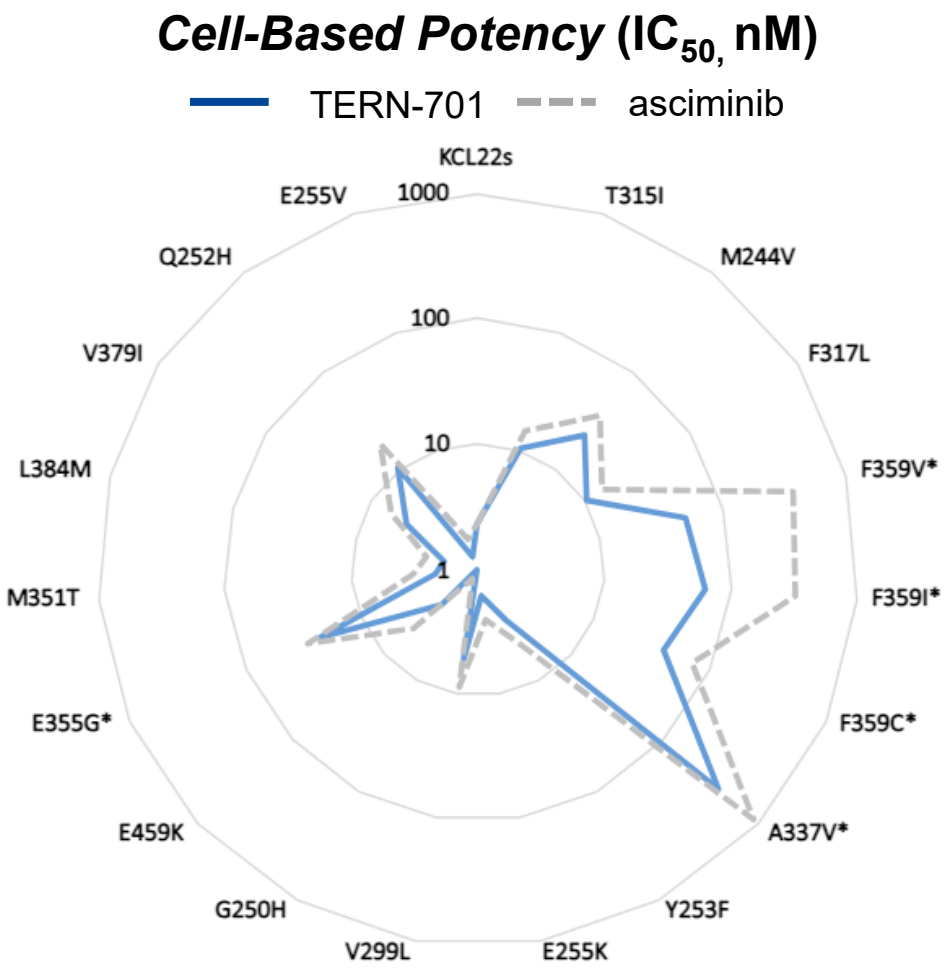
- Off-target inhibition of wild-type kinases including ABL1, ABL2, SRC, VEGF is linked to **cardiovascular and other toxicities**[1]
- **>10,000X better selectivity for BCR-ABL** with allosterics over the closest related wild-type kinases (ABL1, ABL2) and over 450 other kinases compared to active-site TKIs
- Higher target selectivity of allosterics drives the improved safety profile of asciminib vs. active-site TKIs in randomized clinical trials

Selectivity of Allosteric TKIs (TERN-701 & Asciminib) vs Active-Site TKIs[2]



1. Moslehi Javid J. Journal of Clinical Oncology, 2015, Vol 33 (35), 4210-4218. 2. BCR-ABL1 potency derived from KCL22-s cytotoxicity assay (n=3);
3. ELVN-001 selectivity data derived from Enliven Company Overview, April 2024.

① TERN-701 has Numerically Greater Potency than Asciminib Against Multiple BCR-ABL Variants in Preclinical Assays



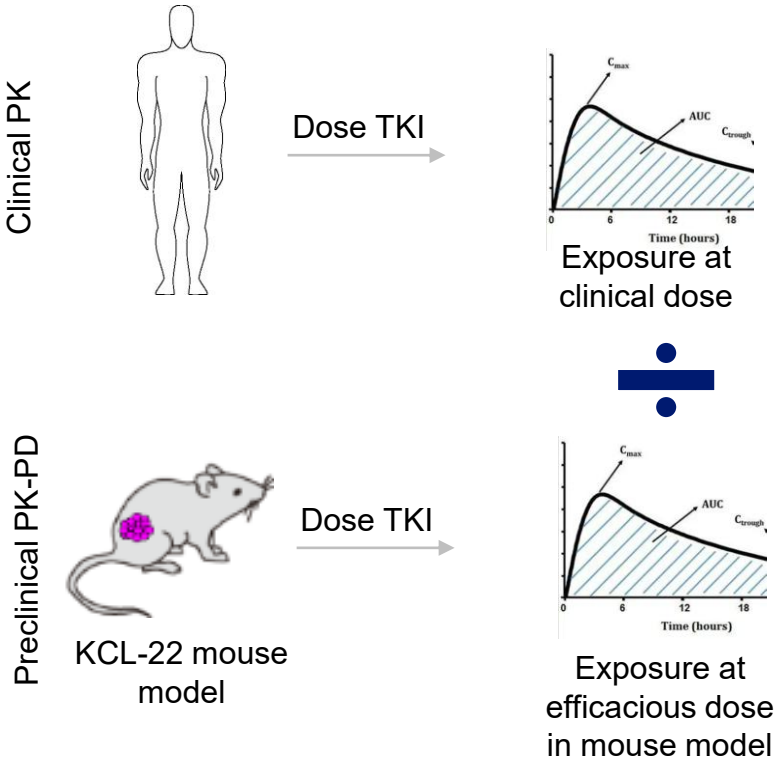
In cell proliferation assays, TERN-701 demonstrated **numerically greater potency vs. asciminib** against several BCR-ABL variants, including active site and myristoyl site mutations

In vitro IC_{50} values determined via cytotoxicity assay (BaF3 cell line for mutations)
* denotes myristoyl mutations or mutations indicated in resistance to allosteric inhibition of BCR-ABL1

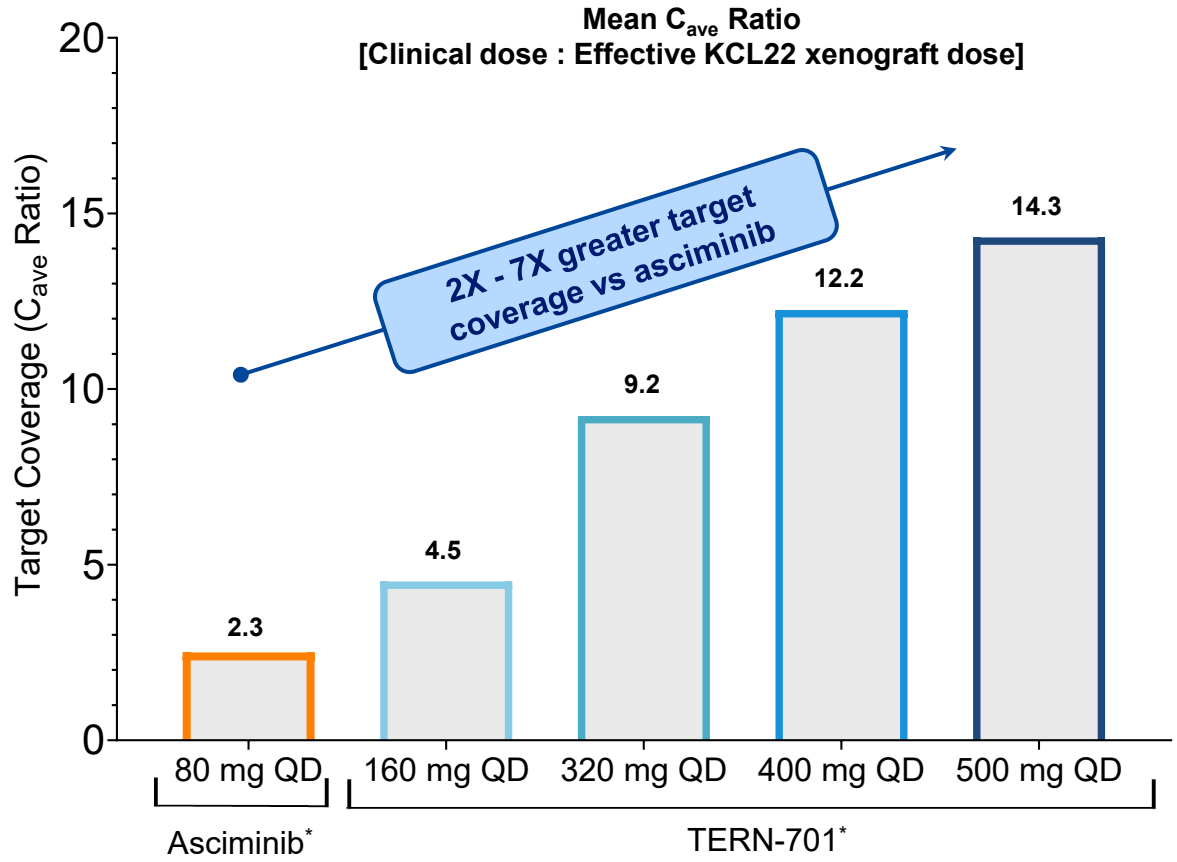
② TERN-701 Clinical Doses Appear to Achieve Multiple Times Higher Target Coverage Than Approved Dose of Asciminib

Asciminib 80mg Dose Informed by Clinical Exposures Relative to Efficacious Exposures in KCL-22 Mouse Model[1]

Target coverage is the **ratio** of exposure at clinical doses in patients to maximally efficacious exposures in a reference mouse model



TERN-701 Clinical Doses Likely Achieve 2X - 7X Greater Target Coverage than Asciminib 80mg

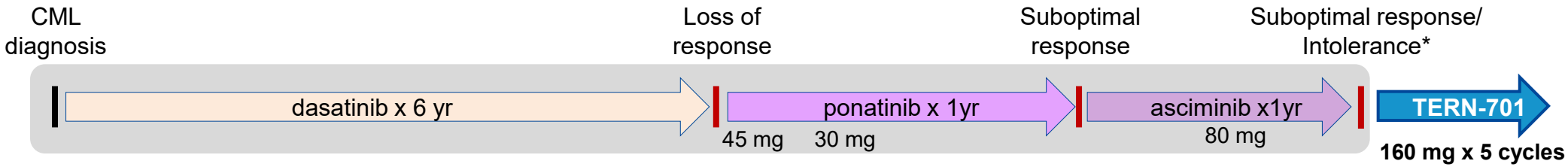


* For TERN-701, the C_{ave} ratio compares C_{ave} in humans (steady state PK from CARDINAL Ph1 study) to C_{ave} in KCL22 mouse xenograft at which >90% inhibition of tumor growth and downregulation of BCR-ABL signaling was seen. Asciminib C_{ave} ratio is C_{ave} in humans at 80mg QD to efficacious exposure (121ng/ml) in KCL-22 mouse xenograft as previously reported[1].

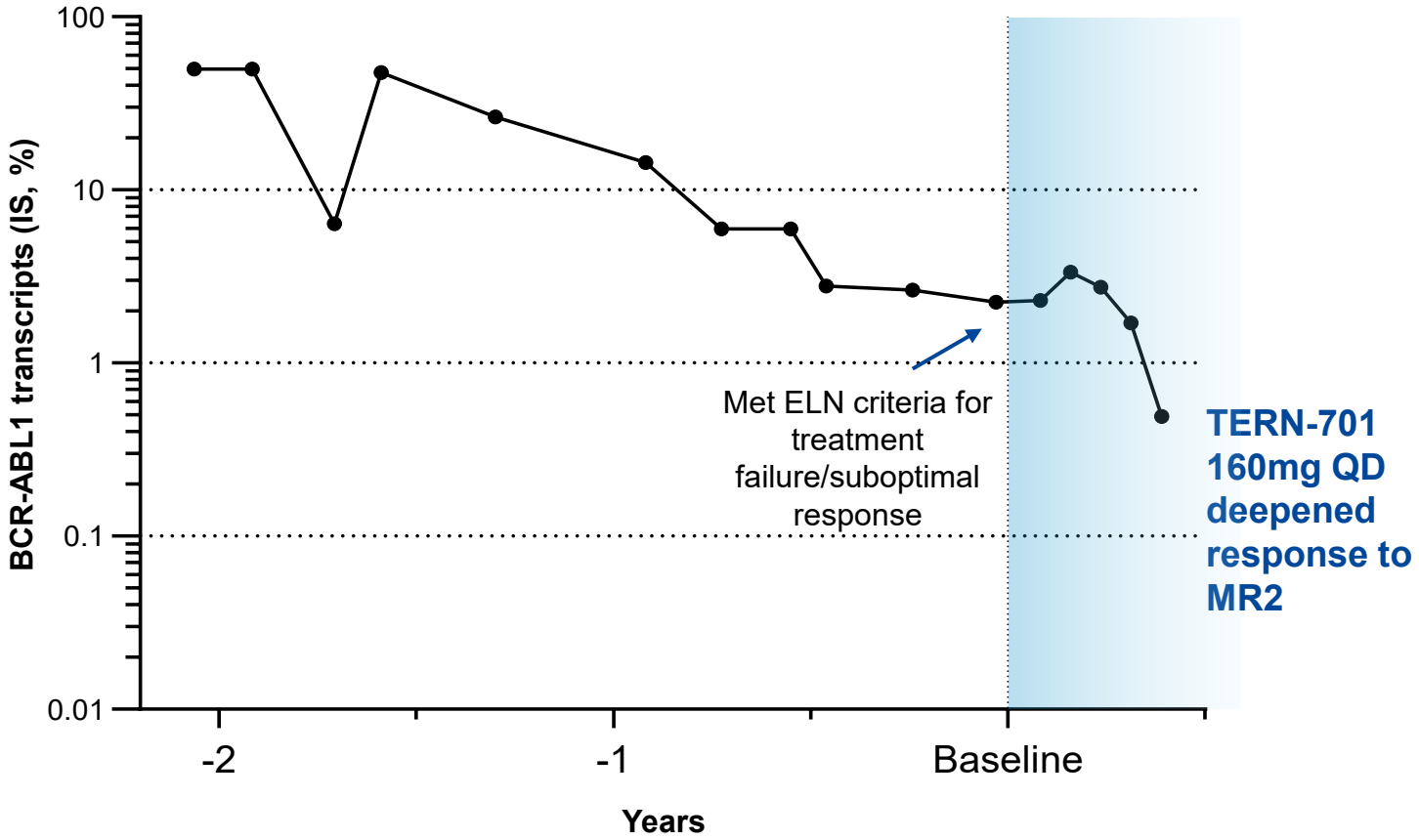
1. Hughes TP et al. NEJM. 2019;381(24):2315-2326.

② Improved Target Coverage Hypothesis is Supported by Rescue of Response in Asciminib Failure by the *Lowest Dose* of '701

MR2 in 4L patient treated with 2G TKI, 3G TKI and asciminib with baseline BCR-ABL1 >1%



Patient Characteristics	
Age	35 years
Gender	Male
# of prior TKIs	3
BCR-ABL1 Mutations	None
Efficacy	MR1 to MR2



Note: Data previously presented in December 2024 (October 2024 cutoff)
* Hypertriglyceridemia/elevated liver function tests
MR1: at least 1-log reduction; MR2: at least a 2-log reduction (i.e., BCR-ABL1 ≤ 1%); cycle = 28 days

③ Trend Towards Improved Safety Relative to Asciminib in Dose Escalation Supports Potential Wider Therapeutic Index (TI)

Wider TI enables administration of higher doses that achieve maximal target coverage and potentially improved efficacy

Incidence of Dose Limiting Toxicities (DLTs) During First 28 Days of Treatment

TERN-701 Dose	Dose Limiting Toxicities
160 mg QD	No DLTs
320 mg QD	No DLTs
400 mg QD	No DLTs
500 mg QD	No DLTs

Asciminib Dose	Dose Limiting Toxicities
40 mg BID	Grade 3 lipase elevation (n=2)
80 mg BID	Grade 2 myalgia & arthralgia (n=1)
150 mg BID	Grade 3 acute coronary syndrome (n=1)
200 mg QD	Grade 3 clinical pancreatitis (n=1)
	Grade 3 lipase elevation (n=1)
	Grade 3 abdominal pain (n=1)
200 mg BID	Grade 3 bronchospasm (n=1)

Hughes TP, et al. N Engl J Med 2019;381:2315-2326.
Note: No head-to-head study has been conducted with TERN-701 against asciminib. Differences exist in study designs and conditions, and caution should be exercised when comparing data across studies. Data previously presented in December 2024.

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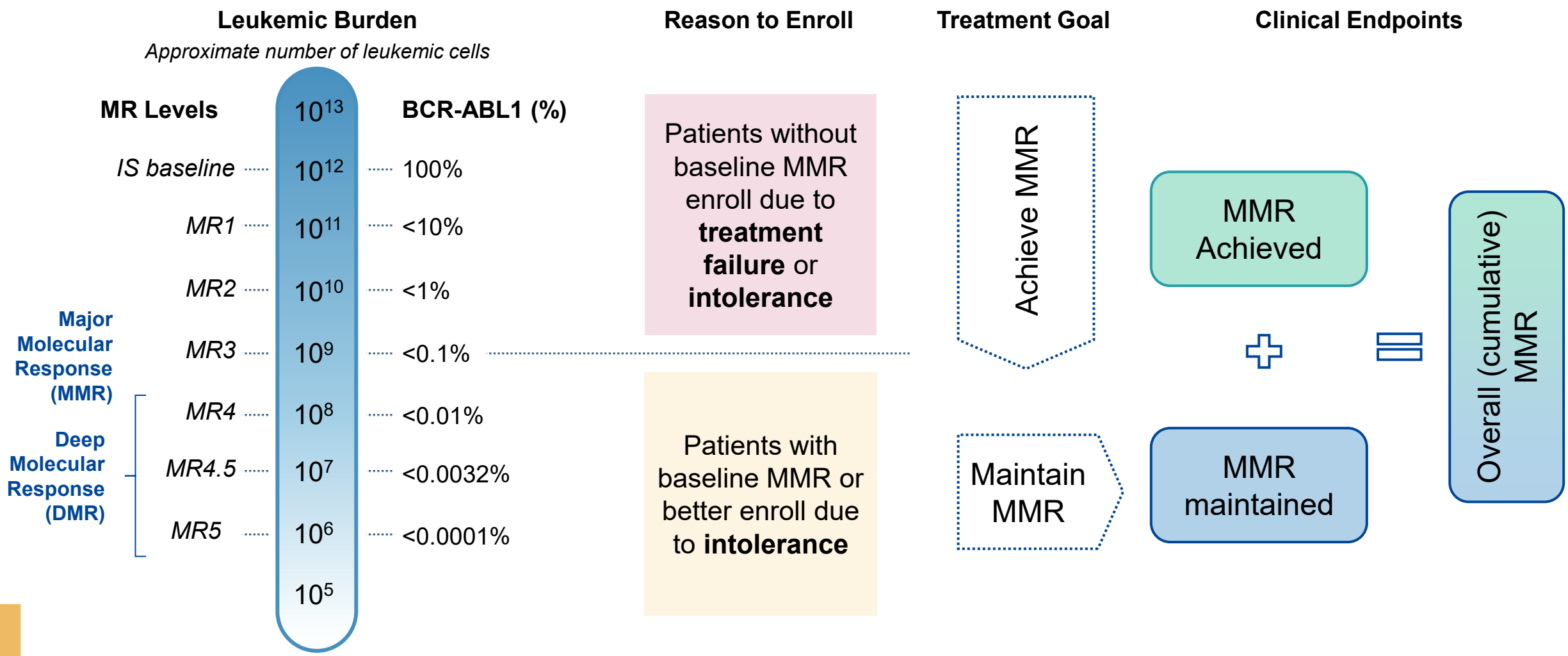
Calibrating and Assessing CML Phase 1 Data

Emil Kuriakose, MD
Chief Medical Officer

Phase 1 CML Studies Enroll Patients With or Without Baseline MMR

MMR achievement is the regulatory endpoint in pivotal studies which only enroll patients without baseline MMR



Wang R et al. Medicine (Baltimore). 2019 Apr;98(15):e15222; Saussele S et al. Leukemia. 2018 May;32(5):1222-1228; Shah NP et al. Journal of the National Comprehensive Cancer Network 2024, 22(1), 43-69; Talpaz M et al. Cancer. 2018 Apr 15;124(8):1660-1672

IS: international standard; MR: molecular response

Interpreting CML Phase 1 Data: Key Parameters and Questions

Parameter	Key Questions
Baseline Demographics	
- # of prior TKIs; prior asciminib; prior ponatinib - Lack of efficacy vs intolerance to last TKI - Baseline BCR-ABL transcript distribution	- Is the CARDINAL population comparable to recent Ph1 studies in CML?
Key Measures of Molecular Response	

- MMR achievement:
  - at 6 months
  - across baseline transcripts
  - within key patient subgroups
	- Is MMR achievement at 6 months competitive with asciminib and other novel agents?
	- Are MRs occurring across the range of baseline BCR-ABL transcripts?
	- Meaningful MMR achievement within difficult to treat patient subsets?

Safety and Tolerability	
- Treatment discontinuation, dose reduction, etc. - Gr 3 or higher and all grade hematologic and non-hematologic AEs	- Are majority of patients staying on treatment? - Are safety differentiators seen early in dose escalation holding at higher doses with longer follow up?

TKI: tyrosine kinase inhibitor; MMR: major molecular response; MR: molecular response; AE: adverse events

Majority of Patients in Phase 1 CML Trials Have High Baseline Transcripts and Lack of Efficacy on Prior TKI

		Baseline characteristics in Ph1 CML studies over the last decade
Baseline transcript >1%		>50%
Baseline MMR or Better (%)		<20%
Lack of efficacy to last TKI		>60%
Resistance mutations (%)	**T315I**	7-25%
	Other	~10%

T315Im and non-T315I mutant patients are historically separated for efficacy reporting

Hughes TP, et al. N Engl J Med 2019;381:2315-2326. Hochhaus A et al. HemaSphere 2025; 9(S1), pg 155-156. Jiang Q et al. Blood 2023; 142, Supplement 1, pg 867.

CARDINAL Enrolls Patients Who Have Had Treatment Failure/Intolerance to Asciminib and Other TKIs

2014 — **Ph1 study of asciminib**

2017 — **Asciminib Ph3 study**

2021 — SCEMBLIX® (asciminib) 20 mg, 40 mg tablets approved in 3L+ CP-CML[3]

2022 — **Ph1 ENABLE study of ELVN-001** (active-site TKI)

2024 — **Ph1 CARDINAL study of TERN-701** (allosteric TKI)

Enrolled population

Asciminib naïve[1,2]	Asciminib failure / intolerant[4]
Imatinib / 2GTKI / ponatinib failure/ intolerant[1,2]	Imatinib / 2GTKI / ponatinib failure / intolerant[4]
Primarily 3L+ patients[1,2]	Primarily 3L+ patients[4]

CARDINAL enrolling a similar population as ENABLE[5]

1. Hughes TP, et al. N Engl J Med 2019;381:2315-2326. 2. Rea D et al. Blood 2021; 138 (21): 2031–2041. 3. Pamuk et al. Clin Cancer Res. 2024 Oct 1;30(19):4266-4271.
4. Hochhaus A et al. HemaSphere 2025; 9(S1), pg 155-156. 5. ClinicalTrials.gov identifier: NCT06163430.

MMR Rates from Completed Asciminib Trials and Ongoing Ph1 Trials Provide Informative Efficacy Benchmarks

6-month timepoint	Asciminib		ELVN-001
	Ph1 ’X2101 (N=99)	Ph3 ASCEMBL (N=157)	Ph1 ENABLE (N=53)
MMR Achievement	24%	25%	32%
Overall (cumulative) MMR	37%	N/A*	47%
DMR Achievement	14%	10%	Not disclosed

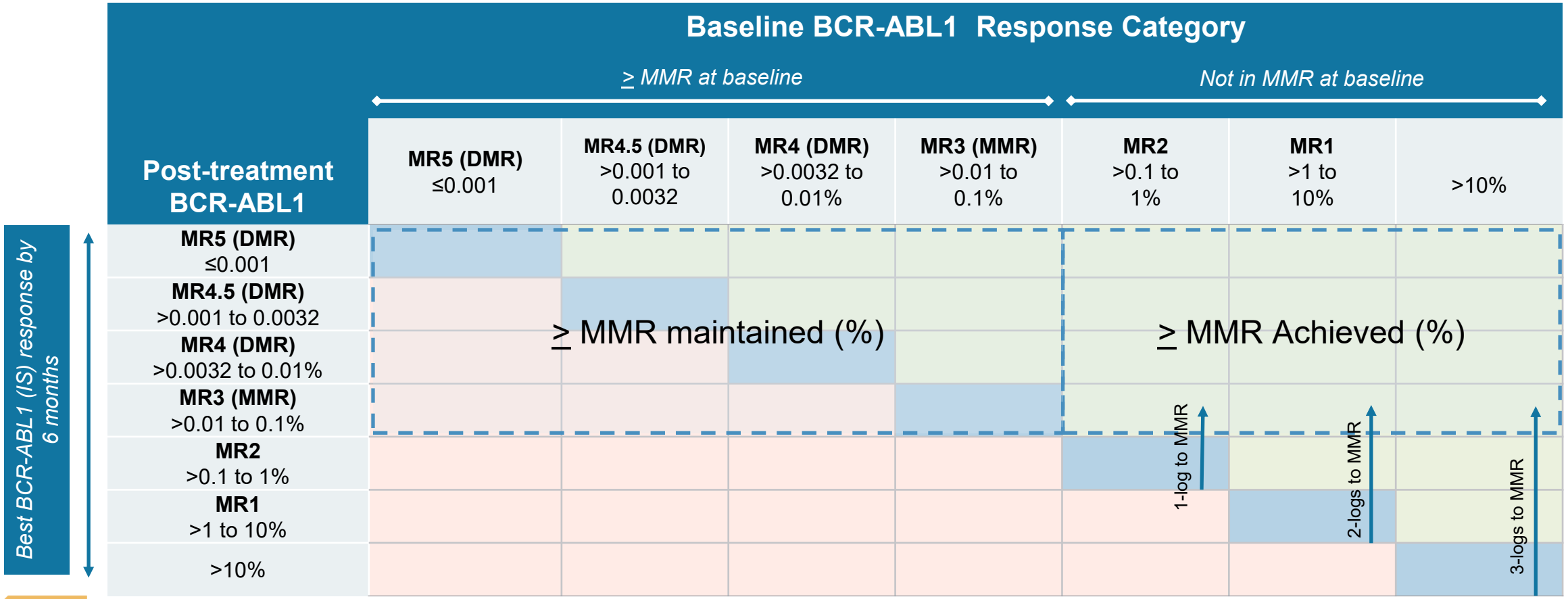
- Additionally, it is important to look at MMR achievement and cumulative MMR in key patient subgroups which reflect more refractory patients (responses are strong indicators of efficacy):
  - Patients with prior asciminib
  - Patients with prior ponatinib
  - Patients with lack of efficacy to last TKI

Response rates of non-T315Im patients who achieved at or by 6 months. *BCR ABL1 > 0.1% was required at screening.
DMR= deep molecular response. Included patients achieving MR4, BCR-ABL1IS ≤0.01%; MR4.5, BCR-ABL1IS ≤0.0032%; and MR5, BCR-ABL1IS ≤0.001; N/A = not applicable
Hughes TP, et al. N Engl J Med 2019;381:2315-2326. Mauro M et al. Leukemia 2023; 37:1048-1059. Rea D et al. Blood 2021; 138 (21): 2031–2041. Hochhaus A et al. HemaSphere 2025; 9(S1), pg 155-156.

The Response Shift Table Can Be Used to Comprehensively Assess Depth and Quality of Molecular Responses

In general, the goal is to shift patients 'upwards' into lower BCR-ABL1 MR categories

- 6-month MMR achievement is the percent of patients with baseline BCR-ABL1> 0.1% who achieve ≤ 0.1% by 6 months
- Achieving 6-month MMR is more challenging with increasing baseline transcript categories from 0.1-1% to >10%
- Seeing shifts to MMR within all baseline categories >0.1% indicates robust efficacy



Hughes TP, et al. N Engl J Med 2019;381:2315-2326.

Upcoming Data for TERN-701 will Build on 4Q24 Interim Data and Provide Benchmarkable Ph1 Efficacy and Safety Data

Q4 2024: Proof of Clinical Activity and Safety (n=15)

- ✓ No DLTs in dose escalation; no MTD
- ✓ Linear PK with target coverage > asciminib 80mg
- ✓ Compelling molecular responses in heavily pre-treated patients with high baseline transcripts
  - ✓ Rescue of response in asciminib treatment failure
  - ✓ Rapid DMR achievement in 2G TKI failure

Q4 2025: Benchmarkable Efficacy and Safety (n=50+)

- 6-month molecular response: MMR achievement, cumulative MMR
- Molecular responses across baseline transcript levels
- Population subset analysis, including MMR achievement in:
  - post-asciminib
  - post-ponatinib
  - failure to last TKI
- Longer term safety data with larger number of patients and longer follow up

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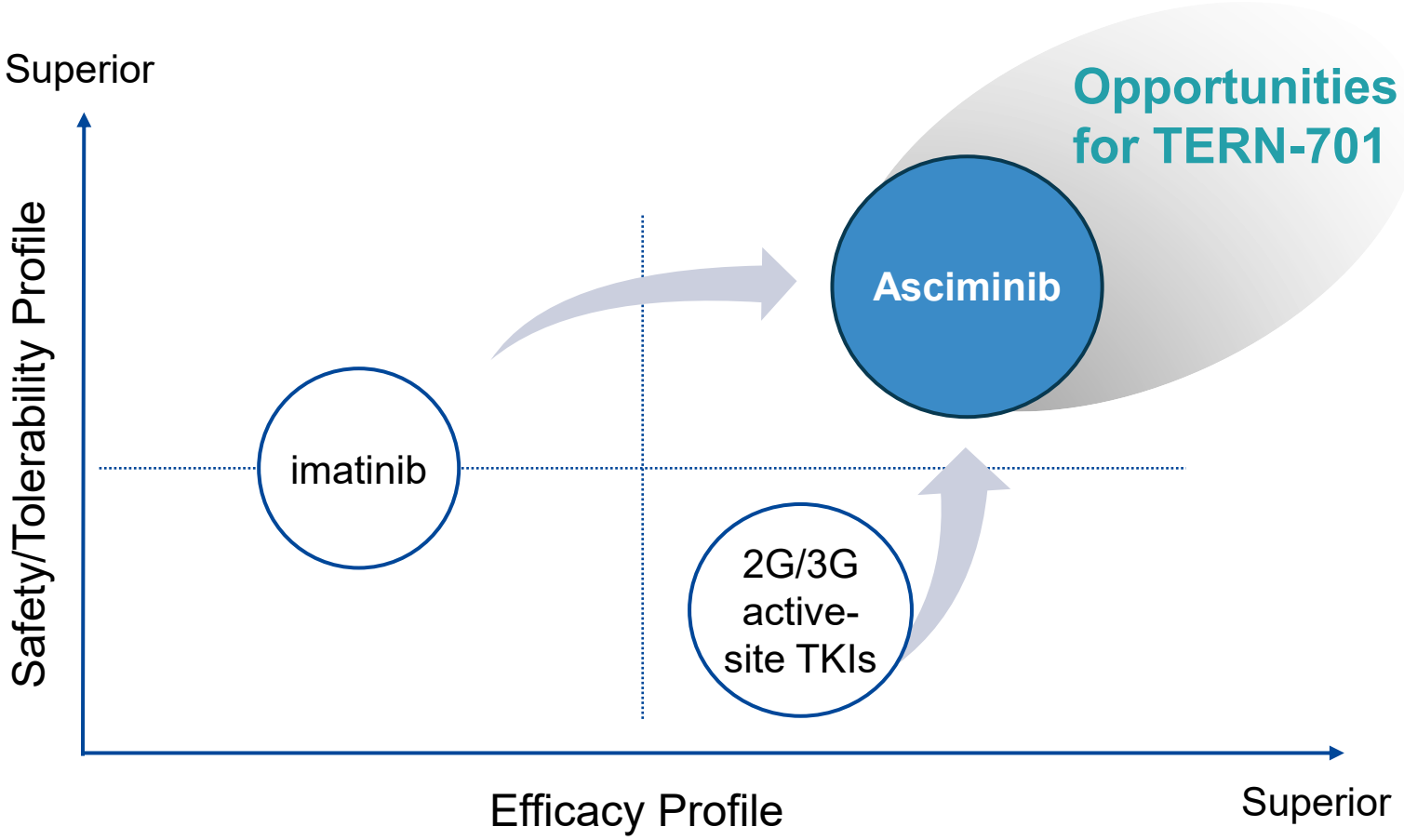
Future of CML and Development Path Ahead

Scott Harris
Chief Development Officer

TERN-701 has Potential to Further Improve Efficacy and/or Safety of the Allosteric Class in CML



In a Multi-Allosteric Treatment Paradigm, TERN-701 has Ample Opportunity to Capture Market Share in 1L and 2L

Anticipated Patient Flow and Market Share with Multiple Approved Allosteric TKIs[1]

Allosteric | Active-site

Majority of newly diagnosed patients will start on the best available therapy (i.e., allosteric)…

1L
31 months avg DoT[3]

17K[2] patients

… asciminib failures in 1L → switch to more effective allosteric
… generic active-site failures in 1L → switch to allosteric (lower barrier to access)

2L
26 months avg DoT[3]

7.4K[2] patients switching to 2L

… patients who progress beyond two allosteric TKIs will likely receive an active-site

3L+
22 months avg DoT[3]

5.5K[2] patients switching to 3L+

TERN-701 Potential Position

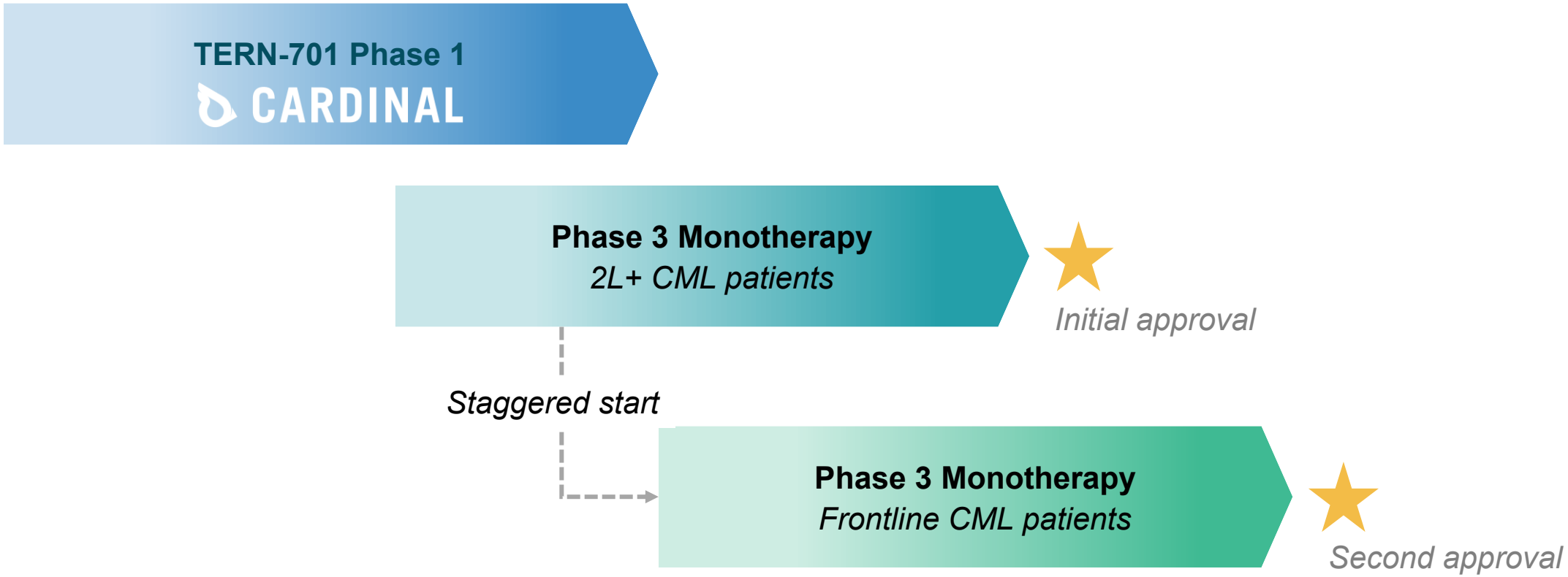
Compete for significant 1L share with potential best-in-class profile

Initial target segments (2L+) as potential best option for rescue

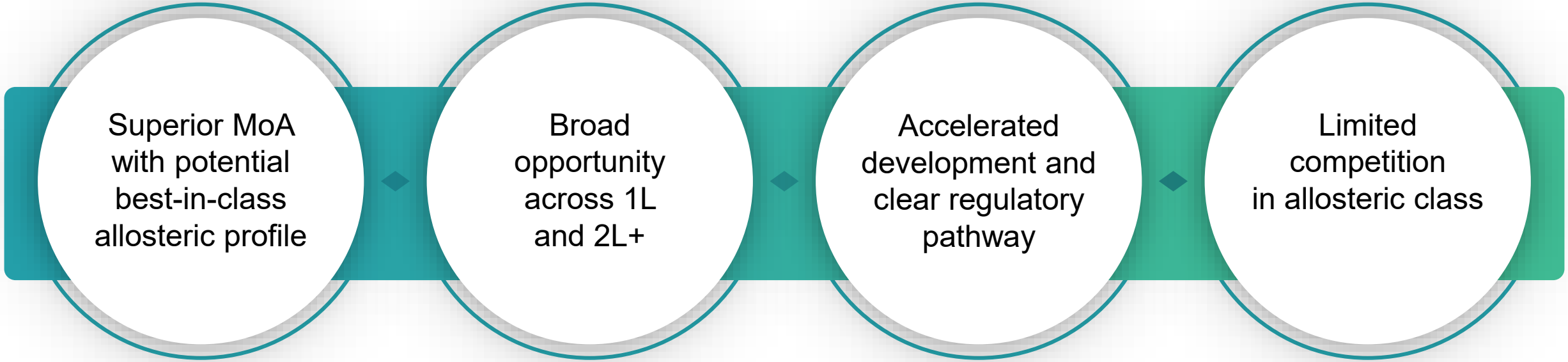
Note: Patient figures represent **new starts** by line of therapy in G7 nations (Canada, France, Germany, Italy, Japan, the United Kingdom and the United States)
1. Clearview Market Sizing 2025 and Terns market research; 2. Novartis ASCO Investor Event | June 2, 2024; 3. Average Duration of Treatment from CancerMPact® Treatment Architecture

Anticipated TERN-701 Registration Path in Early Line CML Patients

- Potential for initial approval as 2L+ therapy in patients failing frontline treatment with asciminib or active-site TKI
- Clinical development in newly diagnosed CML patients expected to run in parallel



Summarizing a Compelling Potential Opportunity for TERN-701 in CML



Mission. Vision. Identity.

mission

To advance transformative medicines that address serious diseases.

vision

To pioneer significant innovations across the lifecycle of drug development.

our name tells our story

Terns are remarkable birds whose instincts mirror our approach to medicine. Like these extraordinary navigators, we at Terns are:

dynamic and agile

We approach complex science with focused innovation and elegant solutions

instinctively adaptive

We pivot quickly when needed without losing our rigor and focus

natural pathfinders

We chart clear courses from validated science to meaningful therapeutic advances

built for the journey

We pursue breakthrough treatments with the endurance to overcome almost any obstacle

steadfast in purpose

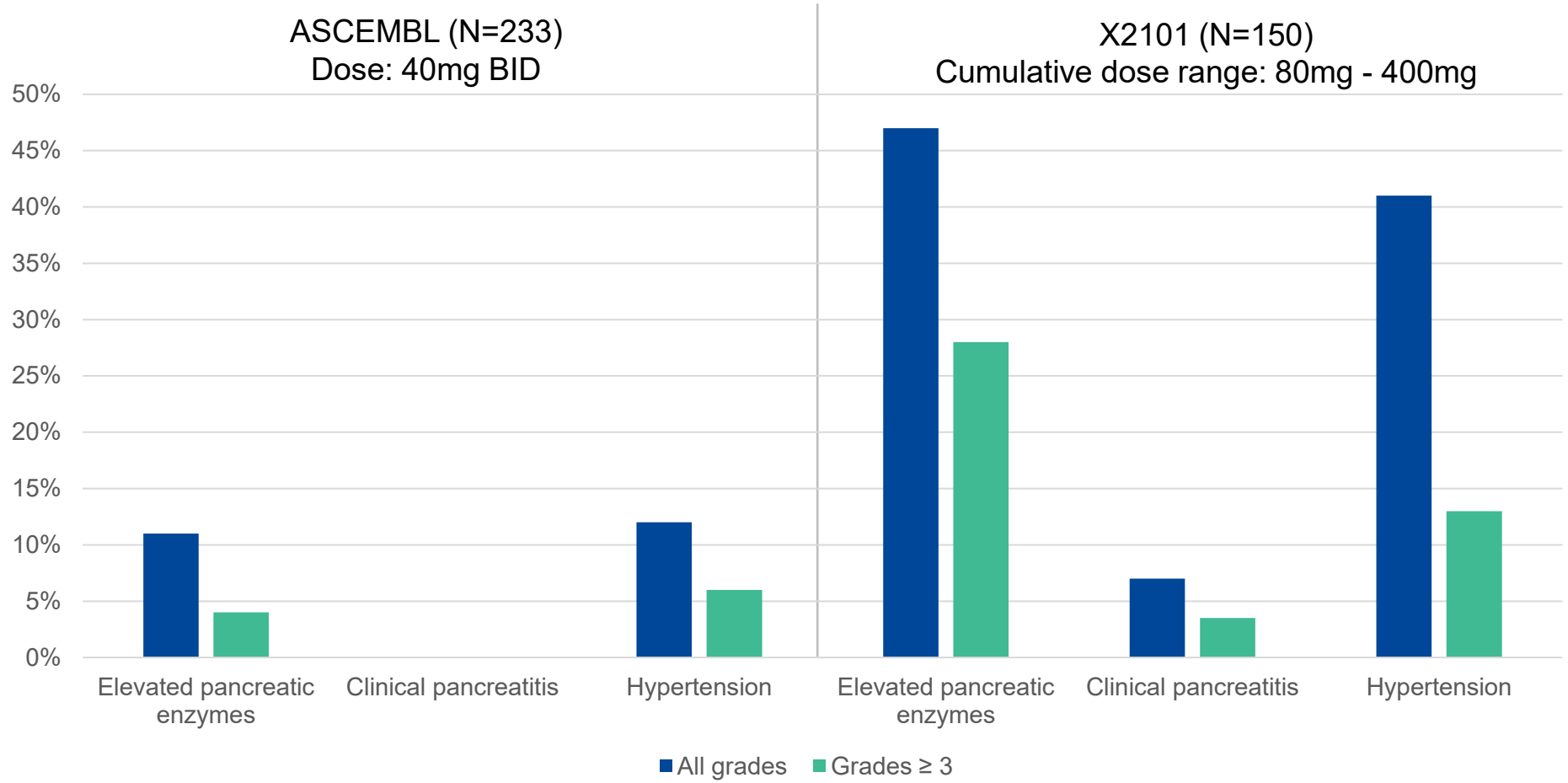
We maintain an unwavering commitment to people living with serious diseases

terns

Appendix

Asciminib Showed Dose Related Increases in Pancreatic Adverse Events and Hypertension

- TERN-701 early Ph1 data showed trend towards lower rates of pancreatic toxicity and hypertension compared to asciminib
- Additional safety data (more patients, longer follow up) would further support a differentiated safety profile if trends hold



Hughes TP, et al. N Engl J Med 2019;381:2315-2326; Rea D et al. Blood 2021; 138 (21): 2031–2041; Asciminib USPI; Pamuk et al. Clin Cancer Res 2024