TERNS
PHARMACEUTICALS

Company Overview

NASDAQ: TERN

December 2024

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Developing small molecule medicines, with clinically validated mechanisms of action, to address oncology and metabolic diseases with large unmet medical need

Terns Investment Highlights and Strategic Approach

Each of Terns' molecules meet the following strategic criteria:

- ✓ Oral, small molecule compounds
- ✓ Clinically validated mechanisms with higher PTS
- ✓ Indications with high unmet needs

Oncology

De-risked and accelerated development pathways

Optionality for in-house full development

Complementary with other assets

Metabolic

Large markets with multiple ways to win (e.g., combinations)

Opportunity to create significant value before seeking partnership

Strong Balance Sheet

Cash of $373M[1] expected to provide runway into 2028

1. As of September 30, 2024; includes marketable securities

PTS: probability of technical success

Terns Pipeline: Broad Rights to Multiple Wholly-owned Opportunities Targeting Serious Diseases

PROGRAM	MECHANISM	INDICATION	PRECLINICAL	EARLY-STAGE CLINICAL DEVELOPMENT	LATE-STAGE CLINICAL DEVELOPMENT	STATUS / NEXT MILESTONE
Oncology						
TERN-701	Allosteric BCR-ABL Inhibitor	CML	Phase 1 CARDINAL		*Anticipated registrational trial following Ph 1 trial*	**Ph1 CARDINAL ongoing** Positive initial data in Dec '24; dose expansion start in 1H25; additional efficacy data in 4Q25
Metabolic						
TERN-601	Oral GLP-1R Agonist	Obesity	Phase 2 Ready			**Positive top-line Ph1 data (28-day PoC) Sept '24** Phase 2 initiation early 2Q25, initial 12-week data in 2H25
TERN-501 Combination	THR-β Agonist + Metabolic Agent	Obesity	Phase 2 Ready			**Positive Ph2a NASH data** Preclinical data in combo with GLP-1 (enhanced and higher quality weight)
TERN-800 Series	GIPR Modulators	Obesity	GIPR Antagonist Lead Opt.			**GIPR antagonist lead optimization underway**

CML: chronic myeloid leukemia, GIPR: glucose-dependent insulinotropic polypeptide receptor, GLP-1R: glucagon-like peptide-1 receptor, NASH: non-alcoholic steatohepatitis, opt: optimization, PoC: proof of concept, THR-β: thyroid hormone receptor beta

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TERN-701

Allosteric BCR-ABL TKI for Chronic Myeloid Leukemia

- Allosteric TKIs have significant efficacy improvement over active-site TKIs
- CML is a ~$5B orphan indication with need for multiple agents and limited allosteric competition
- Ph 1 CARDINAL study ongoing; dose expansion expected to start in 1H25 with additional efficacy data in 4Q25

Chronic Myeloid Leukemia (CML) is a Chronic and Well-Established Indication, Yet an Unmet Need Still Exists

In 2024, CML therapies represented a ~$5B market opportunity

- ~10K new cases diagnosed in the United States, annually[1]
- U.S. prevalence is expected to triple by 2040[2]
- Majority of patients will take TKI therapy for life[3]

Approximately 40% switch therapy by five years due to intolerance and/or resistance[4]

- Chronic use of 1G, 2G active-site TKIs are associated with multiple AEs due to off target effects[4]
- First approved allosteric, asciminib, is superior to prior generation TKIs[5,6] and has opened up a new class
- There remains opportunity to continue to improve on efficacy, safety, tolerability and ease of use for these patients who are on lifelong therapy

1. American Cancer Society. (Jan 2024) Key Statistics for Chronic Myeloid Leukemia, (Aug 2024). 2. Jabbour E, Kantarjian H. Am J Hematol. (Sep 2022);97(9):1236-1256. 3. Bower H., et al. Journal of Clinical Oncology (Aug 2016);34(24):2851-7. 4. Novartis ASCO 2024 Investor Event. 5. Hughes TP et al. N Engl J Med. 2019;381(24):2315-2326. 6. Hochhaus A, et al. N Engl J Med. Published online 2024 May 31.
1G: first generation; 2G: second generation; AE: adverse events; TKI: tyrosine kinase inhibitors

Allosteric TKIs Represents the Next Generation of CML Medicines, with Superior Therapeutic Potential Over Active-Site TKIs

CML Drug Development by Decade

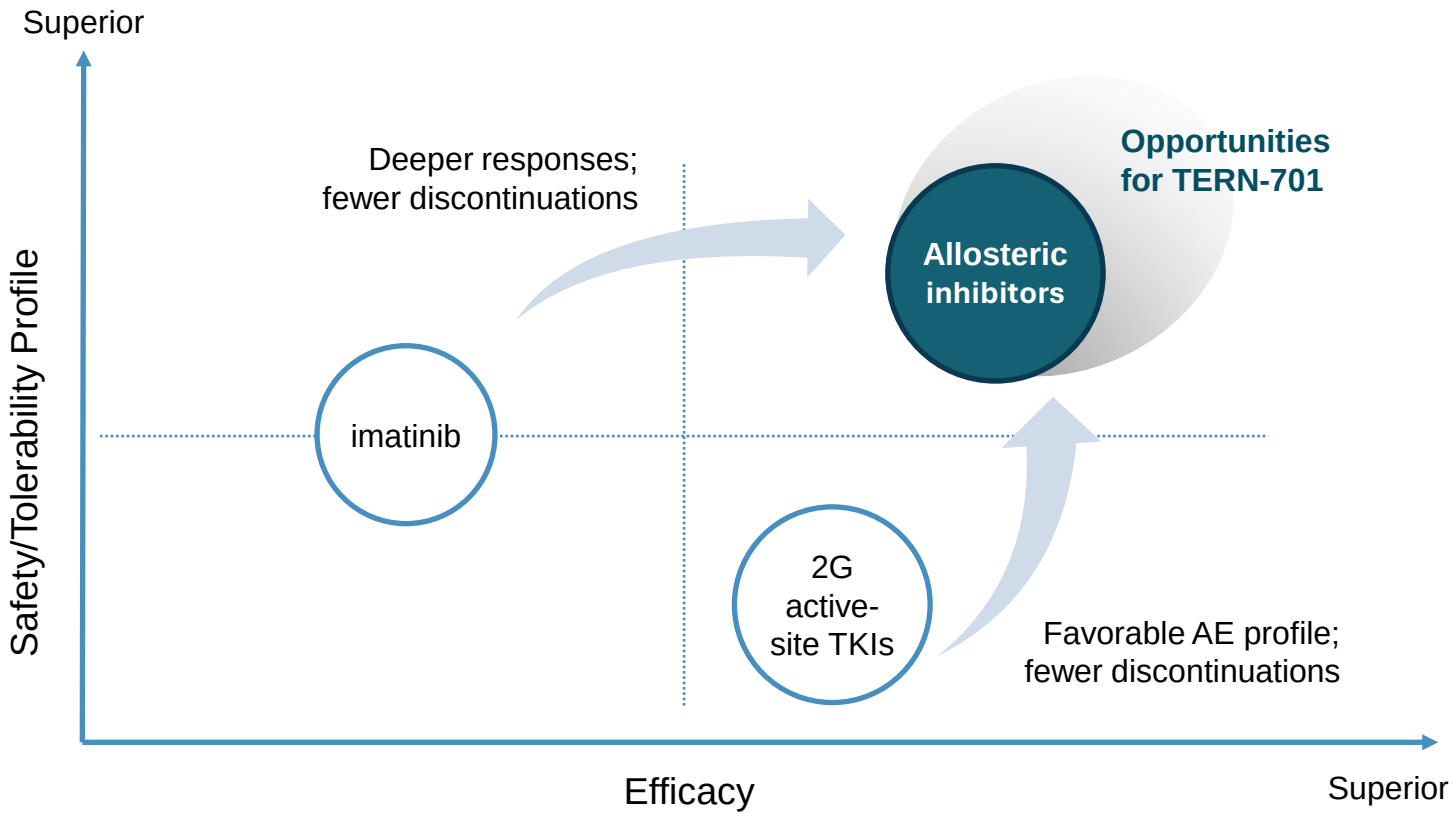
2000s Imatinib (1G active-site TKI) transforms CML from a fatal disease into a chronic, lifelong condition

2010s 2G active-site TKIs improve upon efficacy but with less favorable safety/tolerability

2020s Asciminib is the first approved allosteric BCR-ABL inhibitor, a novel, superior MoA for CML[2,3]

TERN-701 has the **potential to be the best-in-class** allosteric inhibitor

Opportunity for Next Generation, Allosteric BCR-ABL Inhibitors[1]



1. Per Novartis ASCO Investor Event | June 2, 2024. 2. Hughes TP et al. N Engl J Med. 2019;381(24):2315-2326. 3. Hochhaus A, et al. N Engl J Med. Published online 2024 May 31.
1G: 1st generation; 2G: 2nd generation; TKI: dasatinib, nilotinib, bosutinib; AE: adverse event; MoA: mechanism of action; TKI: tyrosine kinase inhibitor

TERN-701 has Early Signs of Differentiation from Asciminib and Opportunity to Achieve a Best-in-Class Profile

	TERN-701 Differentiation Matrix		
	Preclinical[1,2]	Early Clinical (Ph1)[3]	Late Clinical (Pivotal)[#]
Potency ≥ asciminib	✓	— N/A —	— N/A —
Once-daily (QD) dosing	✓	✓	✓
Lack of food effect	✓	✓	✓
Potential for improved efficacy & safety		**Early, encouraging data from** CARDINAL	✓
Potential for simplified label (QD across mutations, improved DDI)			✓

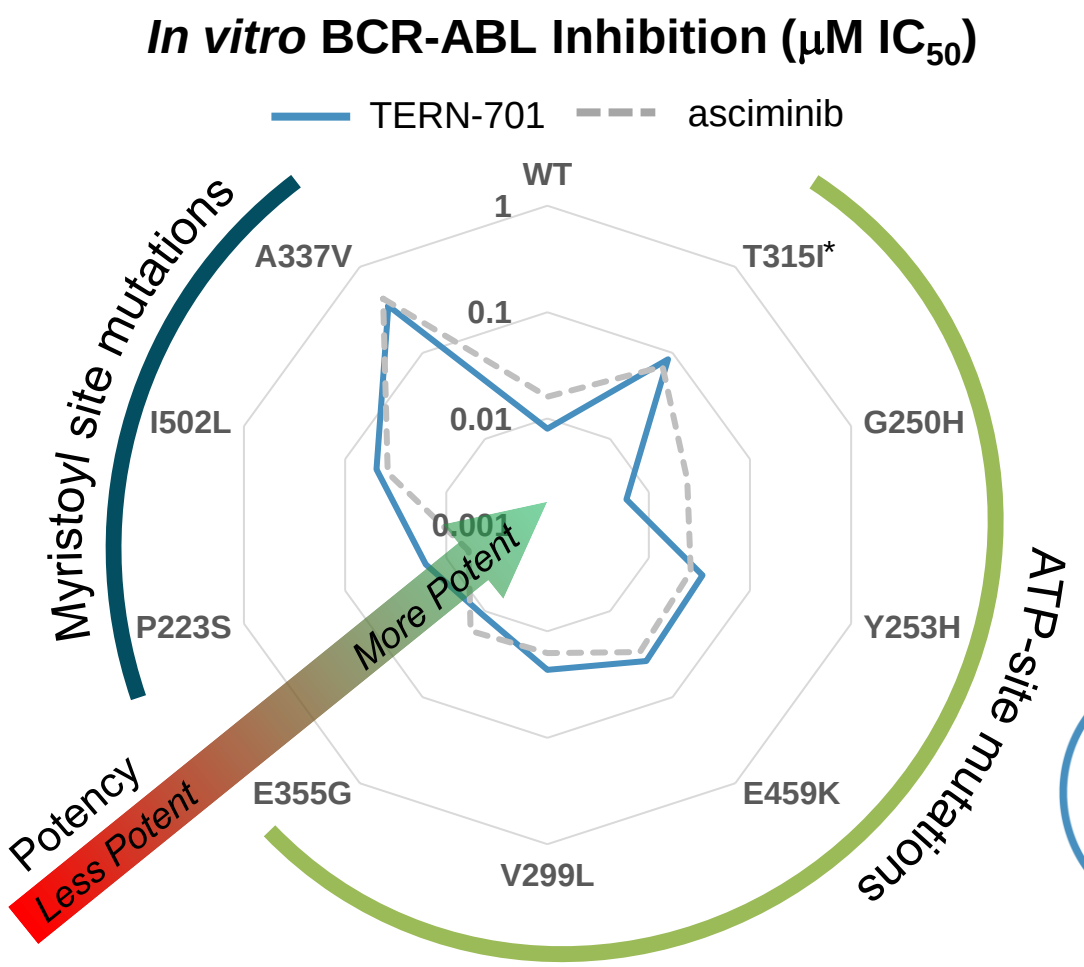
DDI: drug-drug interactions; N/A: not applicable; Ph: phase

1. Zhou et al. ASPET 2023. TERN-701 Preclinical Poster.pdf. 2. Data on File. 3. Anderson et al. SOHO 2024. TERN-701_FE_Poster.pdf

[#] Featured opportunities for TERN-701 are not based on late-stage clinical data and are potential differentiation points that Terns is exploring.

Note: No head-to-head study has been conducted with TERN-701 against asciminib or any other drug or product candidate. Differences exist in study designs and conditions, and caution should be exercised when comparing data across studies. Data and comparisons are shown for illustrative purposes only.

TERN-701 Potency Suggests Anti-Tumor Activity Comparable to asciminib; With Opportunities to Differentiate



In non-clinical assays, **TERN-701 demonstrated a similar profile to asciminib** including high potency against:

- wild type BCR-ABL, and
- most-common mutations occurring in patients treated with active-site TKIs

TERN-701 could have optimized dosing and easier use vs asciminib

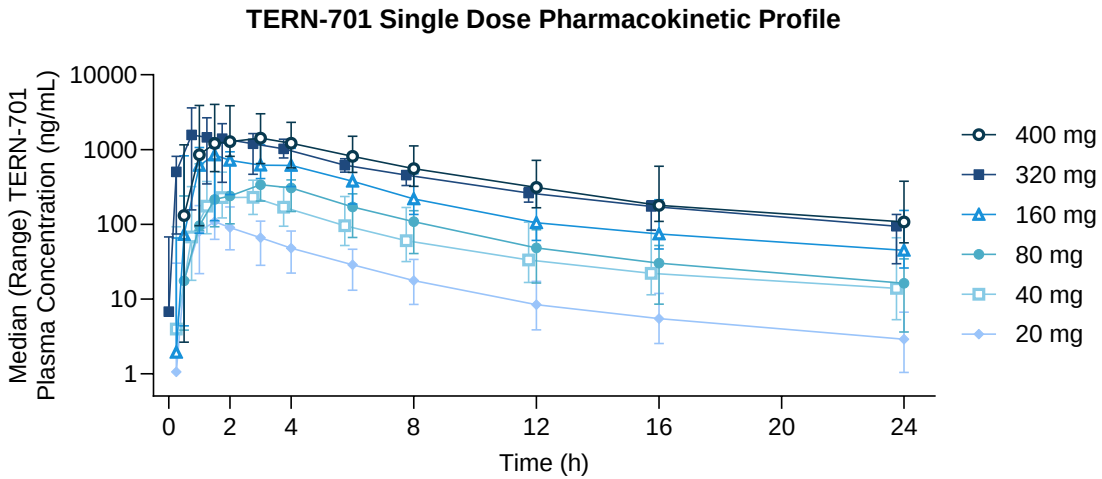
Note: WT (wild-type) and BCR-ABL mutations were evaluated in an ABL auto-phosphorylation assay
* T315i mutation was evaluated in a cell proliferation assay

PK Data from Adult Healthy Volunteer Study Supports Once-daily Dosing Without Regard to Food

Dosing with or without food is a key differentiator within the allosteric BCR-ABL class

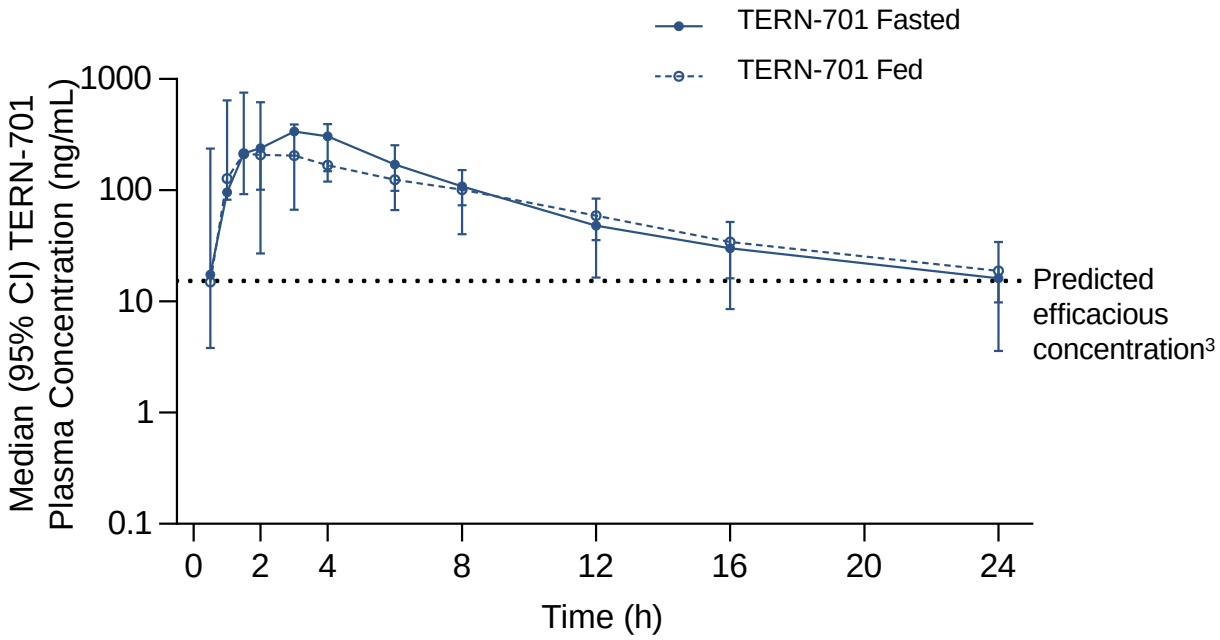
Favorable TERN-701 PK Profile

- Linear PK with approximately **dose proportional increase in exposure** from 40-400mg[1]
- Median half-life of **8-14 hours** supporting QD dosing



No TERN-701 Food Effect

- **No clinically significant difference in exposure** (AUC) when dosed fasted or with a high-fat meal[2]



1. Across single dose TERN-701 range of 20 mg to 400 mg
2. TERN-701 80 mg dose; asciminib (40mg) change in exposure (ΔAUC_{inf}) from fed relative to fasted was (62%)
3. Effective plasma IC90 for the native BCR-ABL KCL-22 cell line

CARDINAL is a Multicenter Global Phase 1 Study of TERN-701 in Patients with Relapsed/Refractory Chronic Phase CML

Dose escalation has enrolled rapidly and is near completion

CARDINAL

Study Population

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

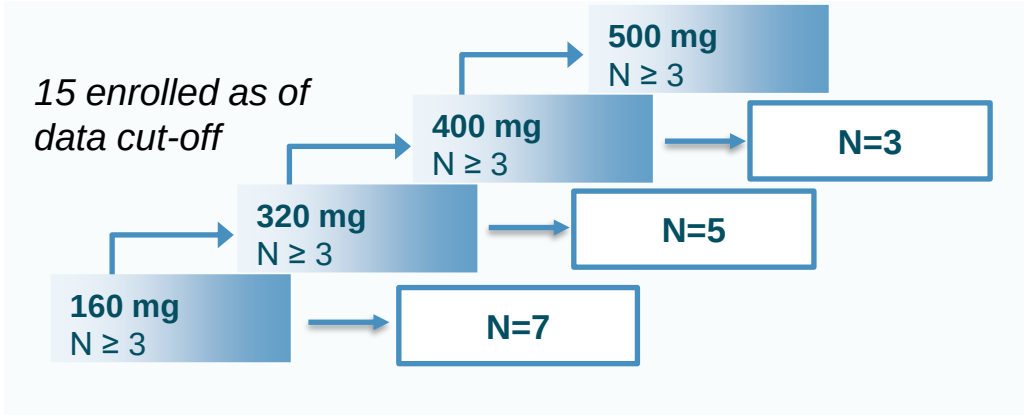
- Treatment failure / suboptimal response to ≥1 2G-TKI

OR

- Treatment failure / suboptimal response / intolerance to any ≥2 active-site TKIs
- Prior asciminib allowed

Part 1 Dose Escalation

TERN-701 Once-daily Monotherapy (N= up to 60)



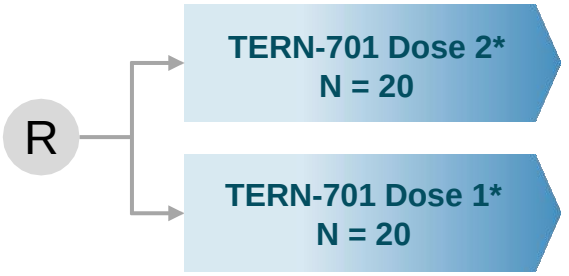
RDE Selection‡

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

Part 2 Dose Expansion

TERN-701 Once-daily Monotherapy (N≈40)

At least 2 dose levels will be selected



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

‡RDE: recommended dose for expansion will be selected following a Part 1 interim analysis
*Dose 1 expected to be ≥ 160mg. Dose 2 targeted be a dose level > 160 mg QD with sufficiently non-overlapping exposures and comparable safety to Dose 1
2G-TKI: dasatinib, nilotinib or bosutinib; PK: pharmacokinetics; TKI: tyrosine kinase inhibitor

Dose Escalation Interim Data Show Compelling Clinical Activity and Encouraging Safety

- **Early, promising safety and efficacy profile in a small number of difficult to treat patients (n=15)[1]**
  - **Compelling molecular responses** in heavily pre-treated patients with high baseline transcripts, and decreases in BCR::ABL1 in the majority of response evaluable patients
  - **Highly encouraging cumulative MMR rate of 50%[2]**
  - **No DLTs, AE-related treatment discontinuations, or dose reductions**
  - **Robust and continuous coverage** over target efficacious exposures at all dose levels
- As of December 3, 2024, 19 patients enrolled in the study with at least three patients enrolled in all escalation cohorts
- Plan to initiate dose expansion in 1H25

1. N=15 as of October 28, 2024 data cut-off
2. 5 of 10 non-T315i mutation patients with 3 or more months of treatment and/or MMR or better at baseline

AE: adverse event; DLT: dose limiting toxicities; MMR: major molecular response

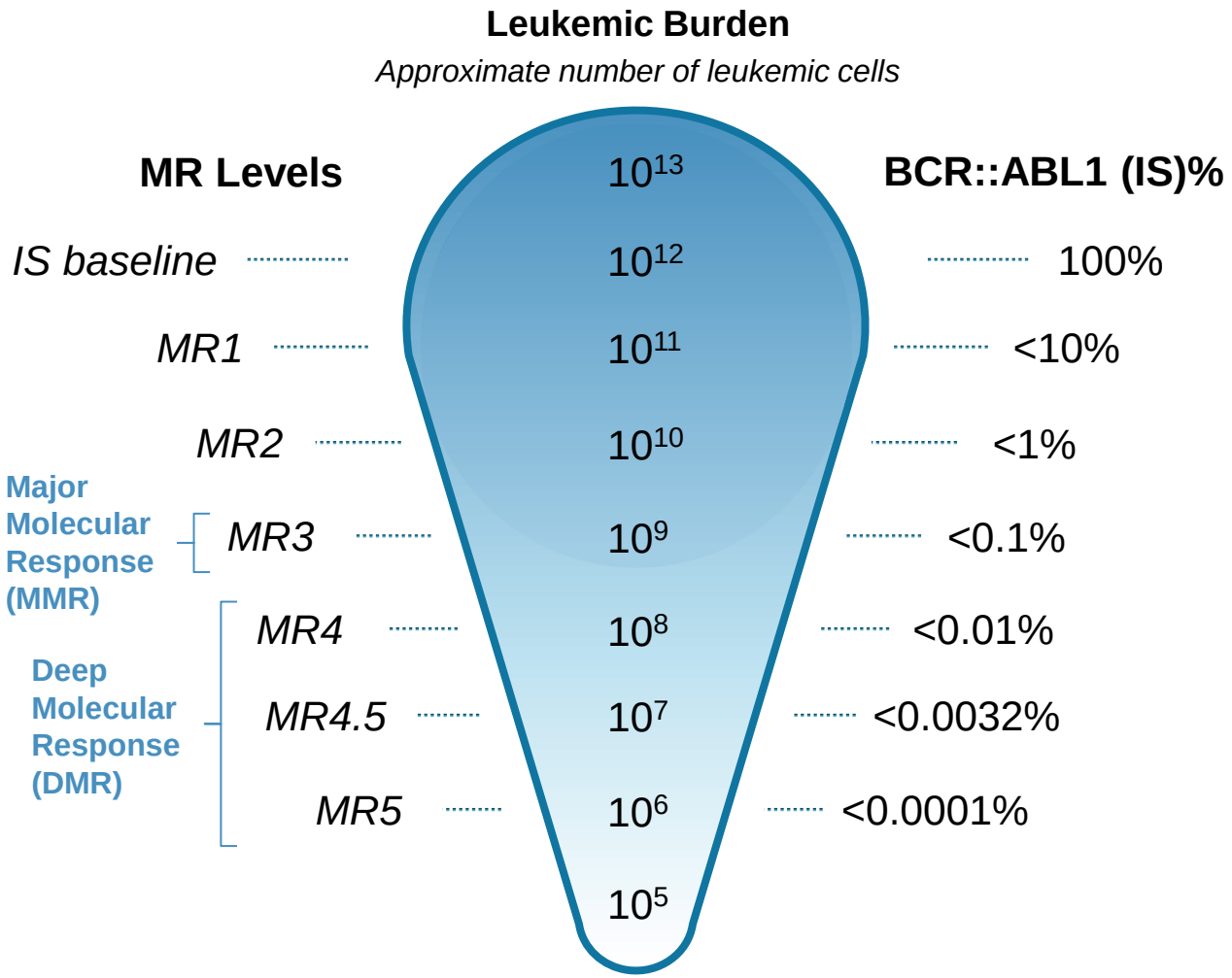
Efficacy and Safety Assessments in the CARDINAL Study

Efficacy Measures

- Molecular response assessed centrally evaluating change in BCR-ABL (IS) transcript levels from baseline
- Hematologic response in patients with hematologic relapse at baseline

Safety Assessments

- Dose limiting toxicities
- Treatment emergent hematologic and non-hematologic AEs
- Serious adverse events
- Dose discontinuations and reductions



1. Wang R et al. Medicine (Baltimore). 2019 Apr;98(15):e15222. 2. Saussele S et al. Leukemia. 2018 May;32(5):1222-1228. 3. Shah NP et al. Journal of the National Comprehensive Cancer Network 2024, 22(1), 43-69.
4. Talpaz M et al. Cancer. 2018 Apr 15;124(8):1660-1672. AEs: adverse events; IS: international standard; MR: molecular response

Enrolled Patients Have Heavily Pretreated Relapsed/Refractory CML with High Disease Burden

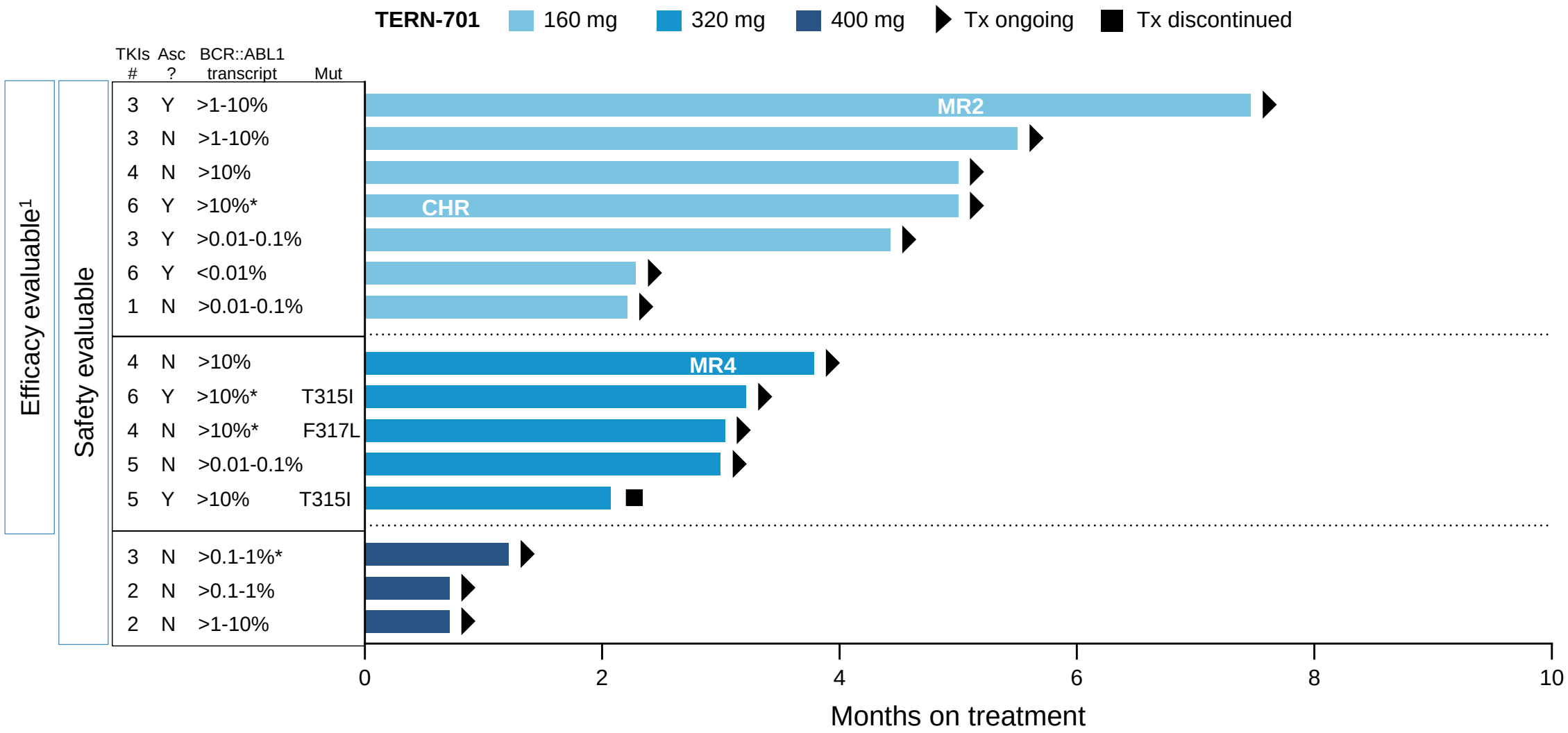
- High baseline disease burden
  - 60% with baseline BCR::ABL1 >1%
  - 73% without baseline MMR
  - 20% with BCR::ABL1 resistance mutation
- Heavily pre-treated population
  - Median 4 prior TKIs
  - 80% had ≥ 3 therapies
  - 47% had prior ponatinib
  - 40% had prior asciminib
- Of asciminib pre-treated patients
  - 1 treatment failure in a remote prior line
  - 5 had asciminib immediately before TERN-701
    - 1 treatment failure
    - 1 suboptimal response with intolerance*
    - 3 intolerant*

	Baseline Disease Status	CARDINAL (N=15)	
	Baseline BCR::ABL1		
No MMR	> 10%	40%	
No MMR	> 1% to 10%	20%	
No MMR	> 0.1% to 1%	13%	
≥ MMR	> 0.01% to 0.1%	20%	
≥ MMR	< 0.01%	7%	
	Median prior TKIs (range)	4 (1-6)	
	≥ 3 prior lines	80%	
	Prior ponatinib	47%	
	Prior asciminib	40%	
	BCR::ABL1 mutations	T315I	13%
		F317L	7%

* Reasons for asciminib intolerance: headache, skin rash & joint pain, hypertriglyceridemia & elevated liver function tests, edema and itching, ocular toxicity (right central retinal vein thrombosis)
MMR: major molecular response; TKI: tyrosine kinase inhibitor

Meaningful Activity in Refractory Patients with High BCR::ABL1

3-month median treatment duration; 14 of 15 patients remain on treatment



1. Defined as having a baseline BCR::ABL1 transcript and at least two post-baseline BCR-ABL transcript levels (centrally assessed)

* hematologic relapse

Asc?: prior asciminib; CHR: complete hematologic response; Mut: mutation; MR2: at least a 2-log reduction (i.e., BCR::ABL1^{IS} ≤ 1%); MR4: at least 4-log reduction (i.e., BCR::ABL1^{IS} ≤ 0.01%); Tx: treatment; TKI #: number of prior TKIs

Highly Encouraging Cumulative MMR Rate of 50% (5/10)

TERN-701 improved or maintained categorical response in all patients without T315I mutation

Categorical BCR::ABL1 (IS) response shift in non-T315Im patients with ≥ 3 months of treatment and/or ≥ MMR at baseline

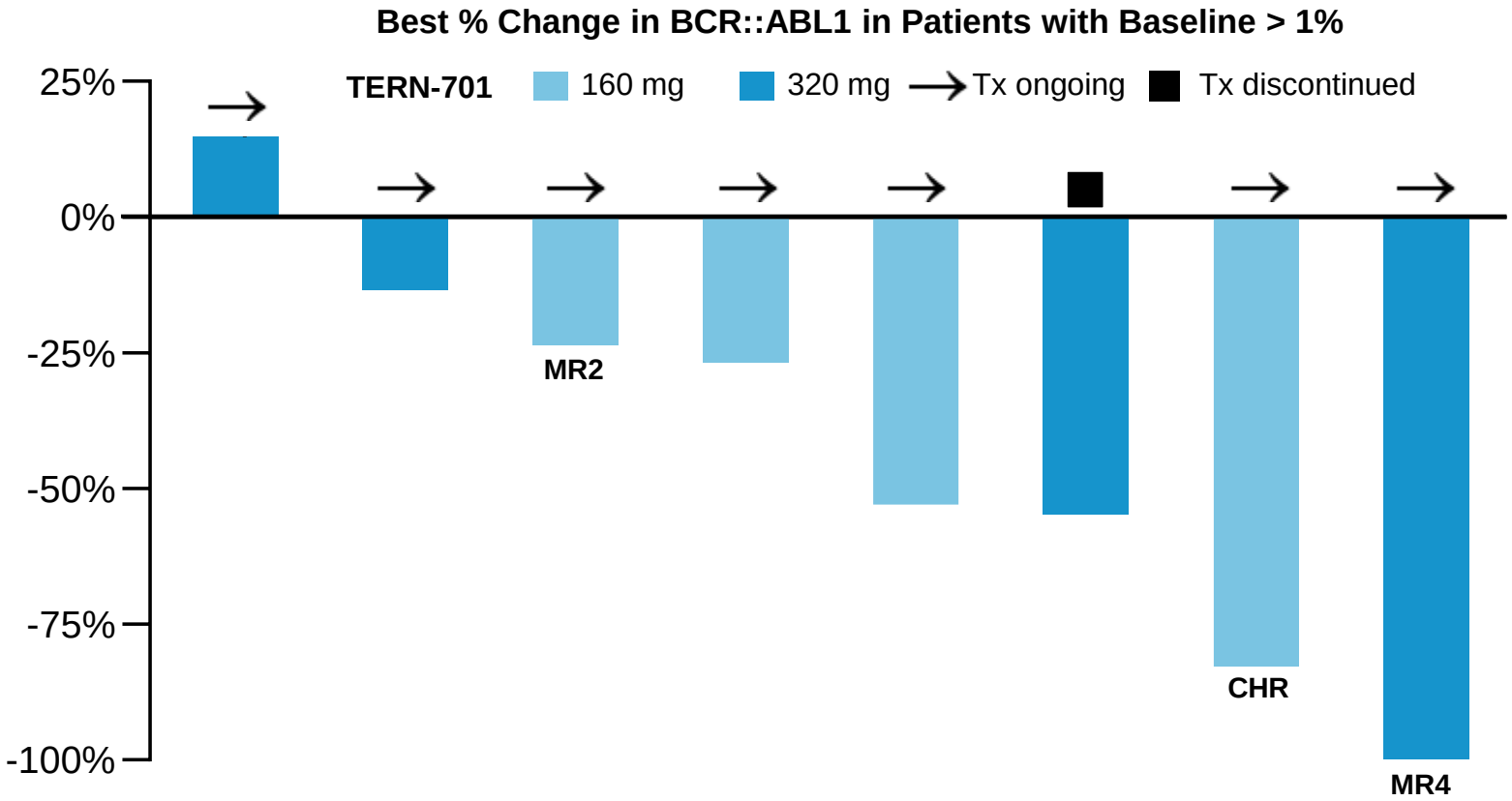
Post-treatment BCR::ABL1	Baseline BCR::ABL1						
	MR5 ≤0.001 (n=0)	**MR4.5** >0.001 to 0.0032 (n=0)	**MR4** >0.0032 to 0.01% (n=1)	**MR3 (MMR)** >0.01 to 0.1% (n=3)	**MR2** >0.1 to 1% (n=0)	**MR1** >1 to 10% (n=2)	>10% (n=4)
MR5 ≤0.001							
MR4.5 >0.001 to 0.0032							
MR4 >0.0032 to 0.01%			1				1
MR3 (MMR) >0.01 to 0.1%				3			
MR2 >0.1 to 1%						1	
MR1 >1 to 10%						1	
>10%							3

Table includes response evaluable patients without T315Im with ≥ 3 months of treatment with corresponding 3-month transcript level reported at visit cutoff, ≥ MMR at baseline, or treatment discontinuation at any time

Improvement in MR category | Stable | Lack of Efficacy | Molecular response shift

88% of Patients with Baseline Transcript > 1% Have Decreases in BCR::ABL1 Levels on Treatment

- 8 response evaluable patients had baseline transcript >1%
  - 6 had baseline transcript >10%
  - 4 had prior asciminib and 3G TKI*
- 88% (7/8) have decrease in BCR::ABL1 and continue treatment as of data cut-off
- One discontinuation due to loss of response after >50% decline in BCR::ABL1 in 6L patient with T315I mutation

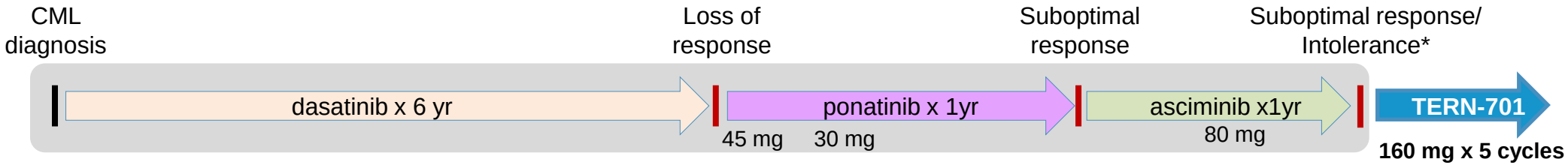


Baseline transcript	> 10%#	> 10%#	>1-10%	> 10%#	> 1-10%	> 10%#	> 10%#	> 10%
Mutation	F317L	T315I				T315I		
# prior TKIs	4	6	3	4	3	5	6	4
Prior asciminib	N	Y	Y	N	N	Y	Y	N
Prior 3G TKI*	N	Y	Y	N	N	Y	Y	N

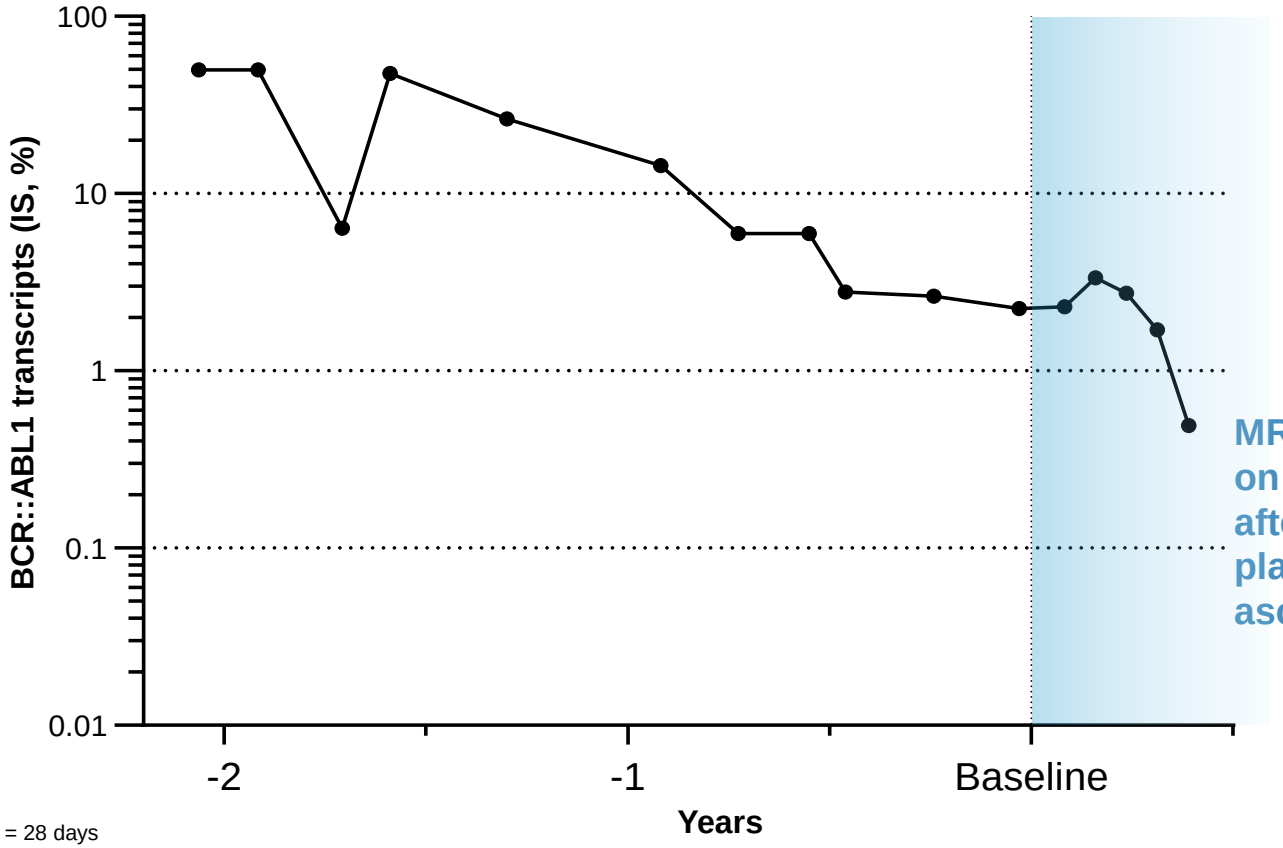
*3G TKI= ponatinib/olverembatinib/ELVN-001; # Baseline transcript >50%

TERN-701 Deepens Response in Patient with Suboptimal Response to Asciminib

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

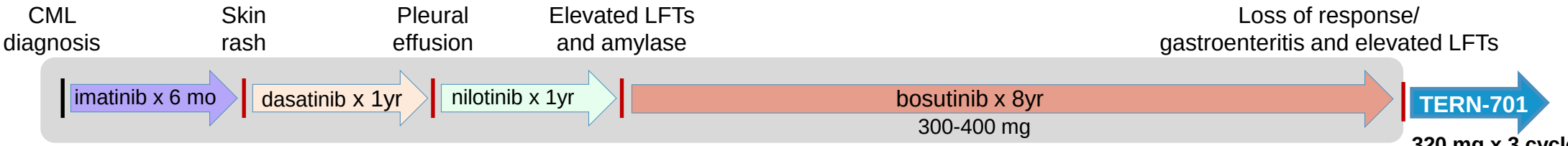


MR2 attained on TERN-701 after response plateau on asciminib

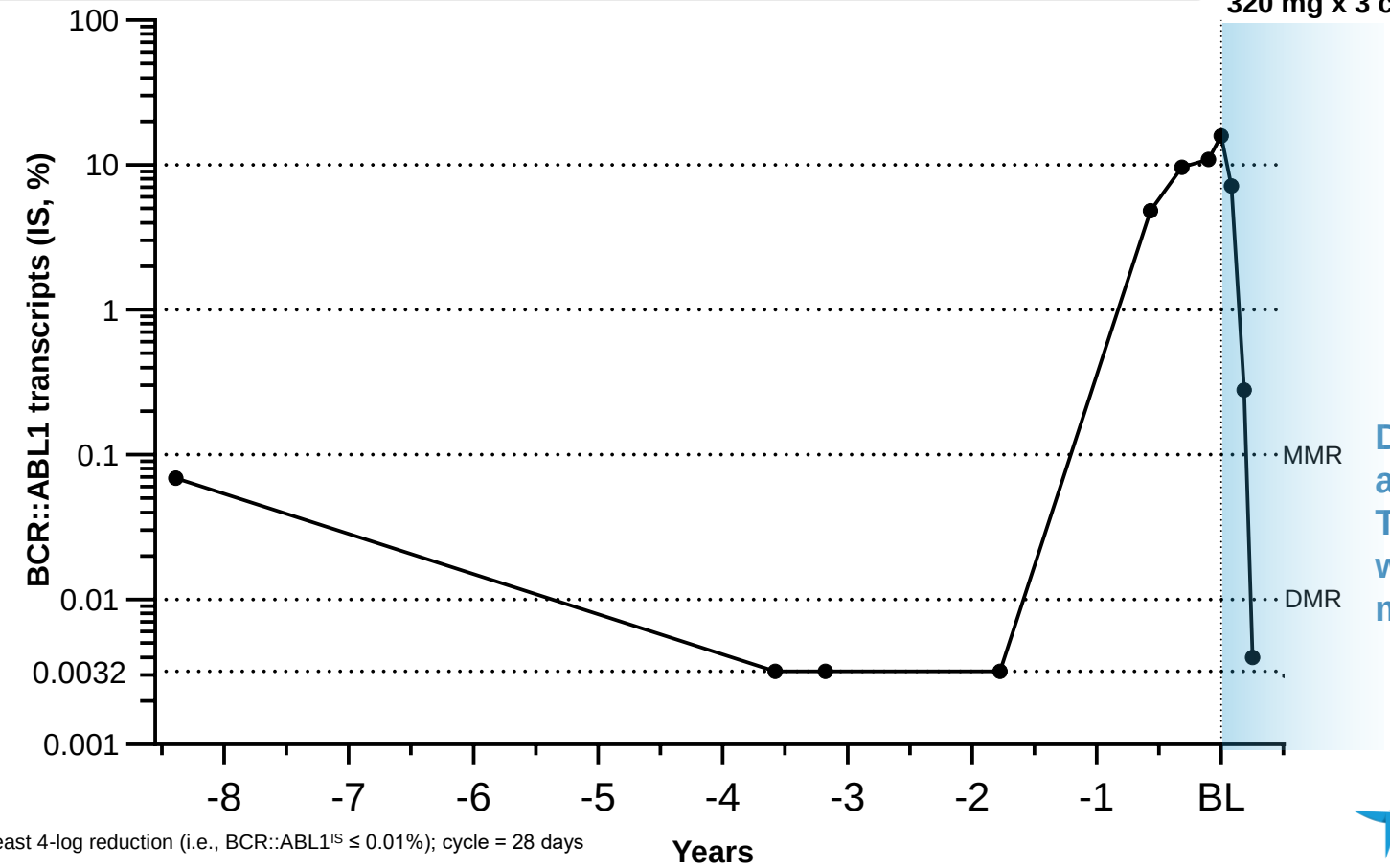
Hypertriglyceridemia/elevated liver function tests
MR1: at least 1-log reduction; MR2: at least a 2-log reduction (i.e., BCR::ABL1^{IS} ≤ 1%); cycle = 28 days

TERN-701 Achieves Rapid Deep Molecular Response in 5L Refractory Patient

MR4 in patient treated with imatinib and all 2G TKIs with loss of response to bosutinib and baseline transcript >10%



Patient Characteristics	
Age	52 years
Gender	Female
# of prior TKIs	4
BCR::ABL1 Mutations	None
Efficacy	>10% to MR4

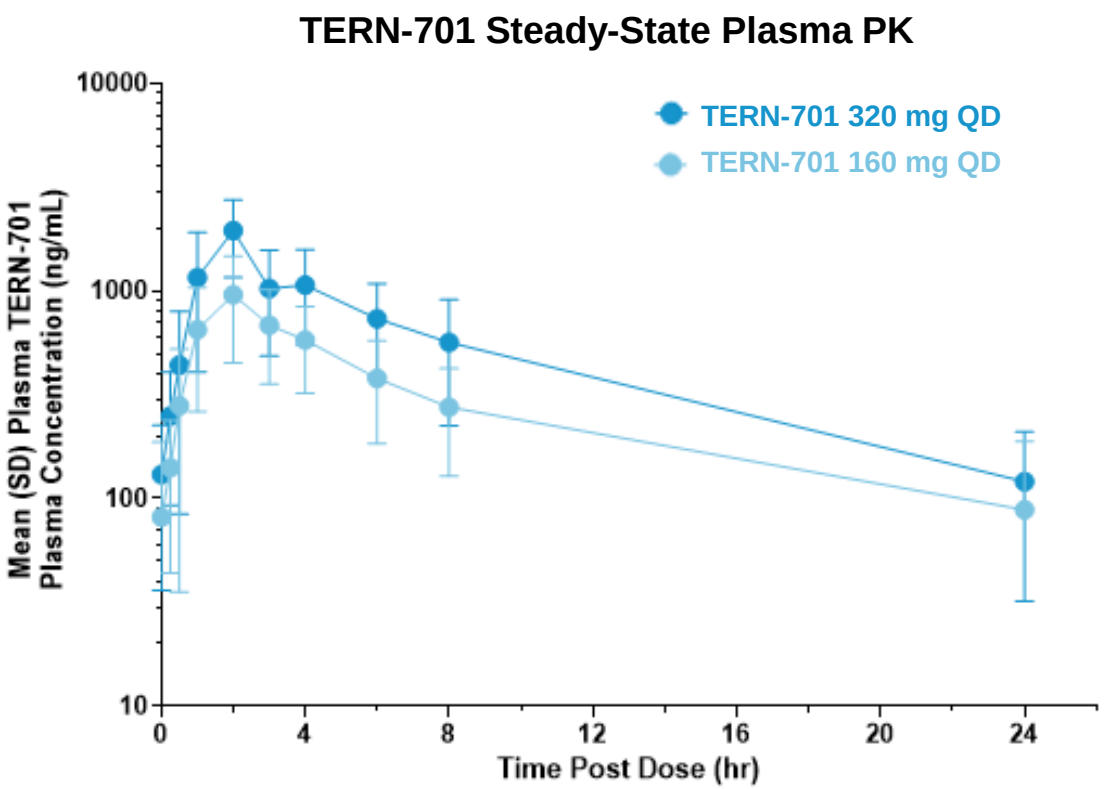


DMR achieved on TERN-701 within 3 months

BL: baseline; DMR: deep molecular response; LFTs: liver function tests; MR4: at least 4-log reduction (i.e., BCR::ABL1^{IS} ≤ 0.01%); cycle = 28 days

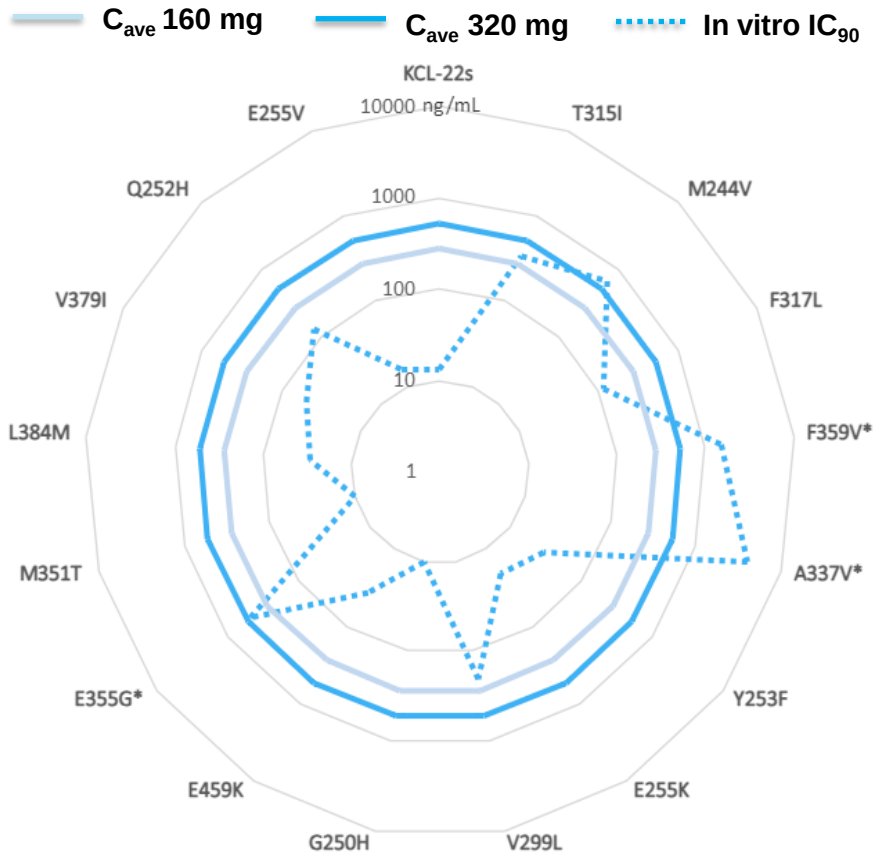
TERN-701 Achieves Robust Target Coverage Over Mutated and Non-Mutated BCR::ABL1 Variants with Once Daily Dosing

Linear PK with approximately dose proportional increases in exposure



Steady state PK for 400 mg not available as of data cut-off date
C_{ave} = $C_{average}$; PK: pharmacokinetics

Starting doses attain exposures exceeding in vitro IC_{90} for multiple BCR::ABL1 variants



In vitro IC_{90} values corrected for plasma protein binding
* denotes myristoyl mutations or mutations indicated in resistance to allosteric inhibition of BCR::ABL1

Emerging Safety Data for TERN-701 are Highly Encouraging

- ✓ No dose limiting toxicities (DLTs)
- ✓ No AE-related treatment discontinuations or dose reductions
- ✓ No ≥ Grade 3 treatment-related AEs
- ✓ No treatment-related SAEs
- ✓ No clinically meaningful changes in LFTs, amylase, or lipase
- ✓ No clinically meaningful changes in blood pressure, ECG, or other vitals

AEs: adverse events; ECG: electrocardiogram; LFTs: liver function tests; SAEs: serious adverse events

Robust Clinical Data Generated Across Multiple Clinical Studies of TERN-701 Supports Efficient Full Development

CARDINAL dose expansion start in 1H25; additional efficacy data in 4Q25

1 Hansoh Phase (Ph) 1 3L+ CML

Informed Terns' Ph1 starting dose; provides additional clinical data in CML to support full development

2 Adult SAD HV Study

Showed consistent PK and lack of food effect

3 Terns Ph1 2L+ CML

CARDINAL

Early, encouraging safety and efficacy profile in difficult to treat patients

Next steps: dose expansion start in 1H25; **additional efficacy data in 4Q25**

Phase 3 Registrational Trial

Evaluating multiple options for pivotal trial(s) including frontline and second line patients

Phase 3 Monotherapy
Frontline CML patients

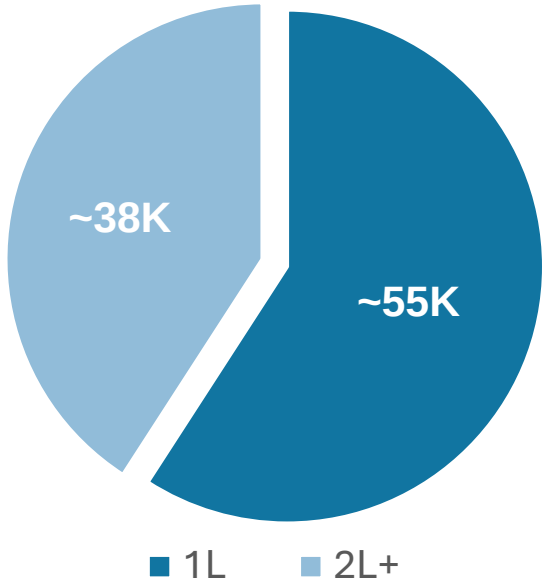
Phase 3 Monotherapy
2L+ CML patients

SAD: single ascending dose; HV: healthy volunteer; PK: pharmacokinetics

TERN-701 Has Broad Anticipated Opportunity Across 1L and 2L+

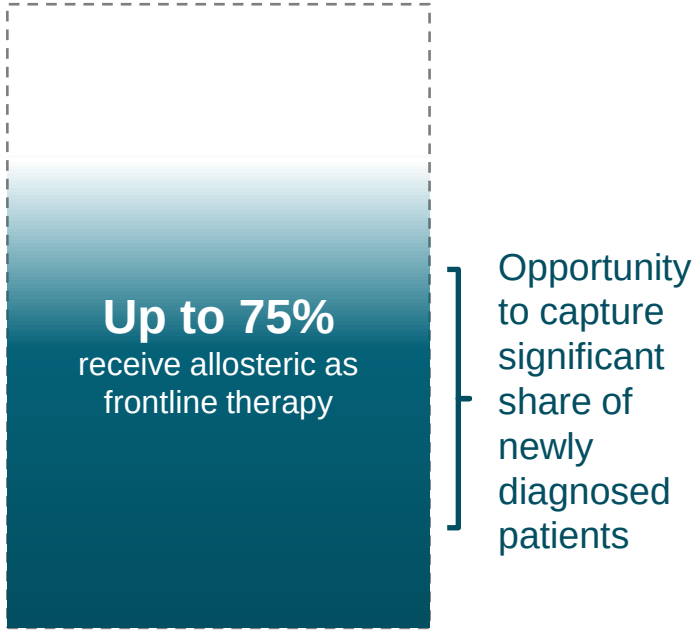
$5 billion current CML market opportunity poised for expansion with increasing addressable patient population

G7 Population with CML On Treatment[1]



1L Market Size
17K newly dx / year[1]

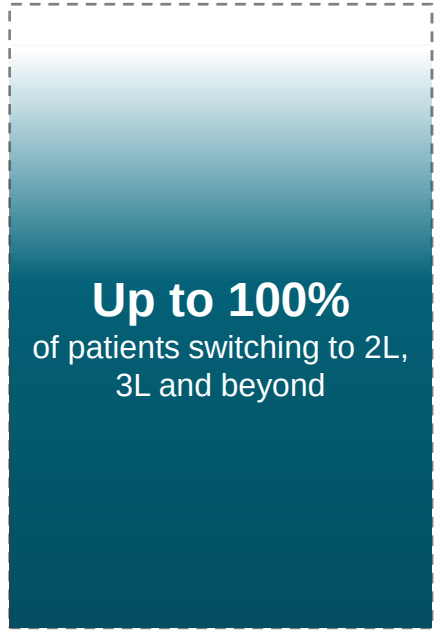
% of newly diagnosed patients addressable by TERN-701



Opportunity to capture significant share of newly diagnosed patients

2L+ Market Size
15K annual switches, ≥2L[2]

% of switching patients addressable by TERN-701



Opportunity to broadly address all lines of previously treated CML

Addressable market to expand as U.S. CML prevalence is expected to **triple by 2040**[3]

1. Novartis ASCO Investor Event | June 2, 2024; 2. Novartis R&D Investor Event | November 28, 2023; 3. Jabbour E, Kantarjian H. Am J Hematol. (Sep 2022);97(9):1236-1256
G7: Canada, France, Germany, Italy, Japan, the United Kingdom, and the United States; Dx: diagnosed

TERNS PHARMACEUTICALS

Our Approach for Metabolic

Focused on the discovery and development of oral, small-molecule candidates within established MoAs for building future, *best-in-class oral combination therapies* for the treatment of obesity

TERNS PHARMACEUTICALS

TERN-601

Oral GLP-1 Agonist with Differentiated Profile for Obesity

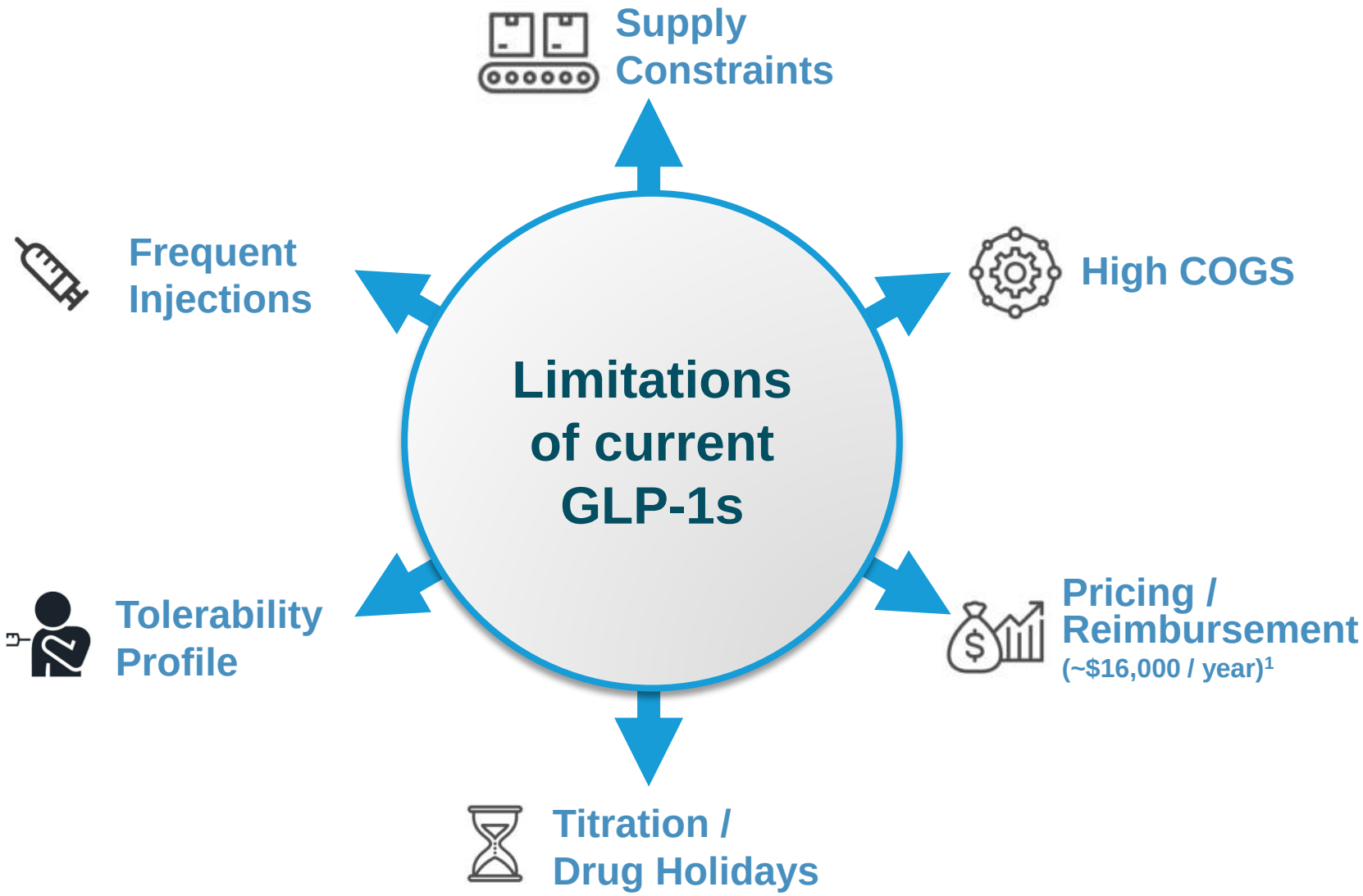
- Statistically significant and dose-dependent weight loss over 28 days with QD dosing
- Well-tolerated with unremarkable safety findings despite rapid titration to target doses
- Potential to be a leading GLP-1R agonist; Ph 2 initiation expected in early 2Q25 with initial 12-week data in 2H25

Positive Phase 1 Results Demonstrate TERN-601 is Well Positioned for Phase 2 and Long-Term Differentiation

- **Statistically significant** and **dose-dependent** weight loss over 28 days with QD dosing
- **Well tolerated** with unremarkable safety findings despite **rapid titration to target doses**
- **Distinct drug properties** enabled sustained target coverage and a flat PK curve, and may lead to a differentiated clinical profile in subsequent studies
- Potential to be a leading GLP-1R agonist with promising **efficacy, tolerability and manufacturing scalability**
- **Plan to initiate Phase 2** trial in early 2Q25

GLP-1R: glucagon-like peptide-1 receptor, PK: pharmacokinetic, QD: once-daily

Oral, Small-Molecule GLP-1s May Address Limitations of Current Injectable GLP-1s



1. Novocare: Wegovy has a list price of $1,349 / package * 12 pkgs/year

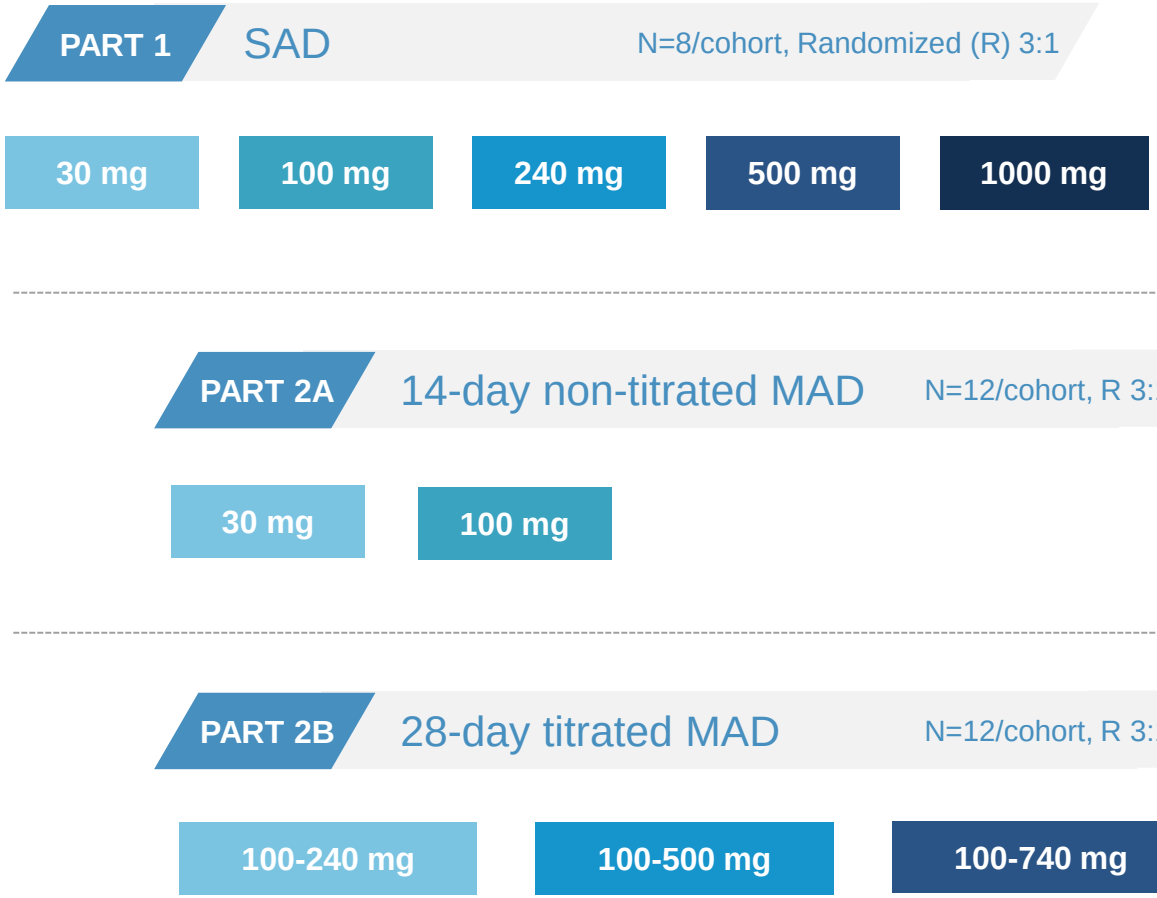
TERN-601 First-In-Human Study Leveraged an Efficient Design to Explore a Wide Dose Range

Phase 1 Trial Design

Population
- Healthy adults with obesity or overweight
- Non-diabetic
- BMI ≥ 27 to < 40 kg/m^2 (Part 2)

Endpoints
- Primary: safety and tolerability
- Secondary / exploratory: PK, change in body weight over 28 days, etc.

Location
- U.S. inpatient Phase 1 center



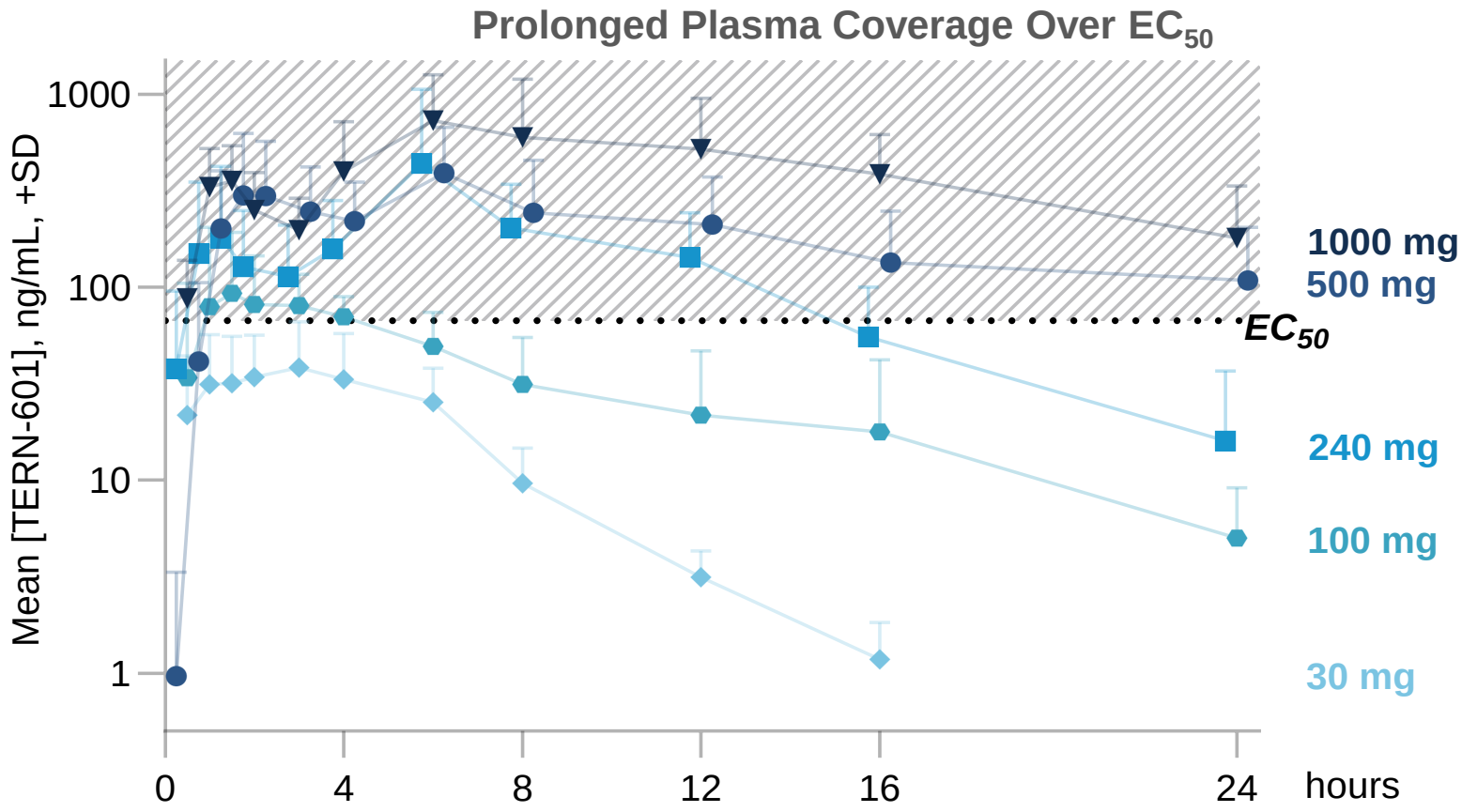
Study objectives

- Use PK data to identify once-daily target doses for 28-day titration
- Use safety/tolerability and PD data to identify optimal starting dose for 28-day titration
- Assess safety / tolerability of fast titration to target doses and weight loss over 28 days

BMI: body mass index, MAD: multiple ascending dose, PD: pharmacodynamic, PK: pharmacokinetic, SAD: single ascending dose

Prolonged Absorption of TERN-601 at Target Doses Drove Sustained Target Coverage with Once-Daily Dosing

- Prolonged absorption at ≥240 mg led to sustained 16-24 hour target coverage in plasma despite ~4-6 hour elimination half-life
- SAD PK identified 240 mg and above as potentially efficacious target doses for 28-day MAD cohorts



Note: Dotted line represents estimated protein-binding adjusted EC_{50} (concentration at which 50% of maximal activity is observed) in CHO-K1 cells (subclone of the Chinese hamster ovary cell line) expressing hGLP-1R (humanized GLP1 receptor)
MAD: multiple ascending dose, PK: pharmacokinetic, SAD: single ascending dose, SD: standard deviation

Baseline Characteristics Well-Balanced Across 28-Day MAD Cohorts

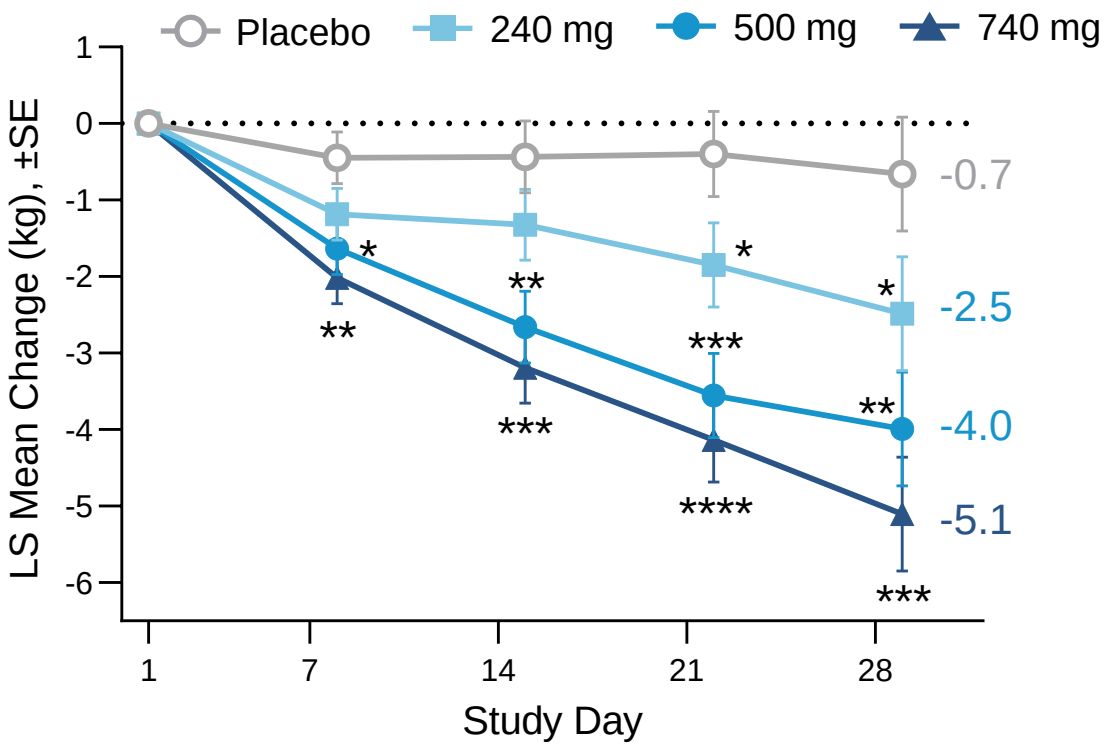
BMI consistent across groups (~30 kg/m^2), with predominantly male participants (≥70%)

Mean (SD) Median	Placebo pooled (N=9)	240 mg (N=10)	500 mg (N=9)	740 mg (N=9)
Age, year	41.4 (9.2) 40	44.7 (10.7) 49.5	46.7 (12.7) 45	46.7 (12.1) 50
Male, n (%)	7 (78%)	7 (70%)	8 (89%)	7 (78%)
Weight, kg	90.9 (7.8) 91.8	93.4 (14.2) 92.6	95.0 (10.6) 93.8	93.3 (13.7) 93.1
BMI, kg/m^2	29.7 (1.6) 28.8	30.6 (2.8) 30.3	31.2 (2.1) 30.4	30.1 (2.2) 29.4
HbA1c, %	5.6 (0.2) 5.5	5.5 (0.3) 5.7	5.6 (0.3) 5.6	5.5 (0.2) 5.5

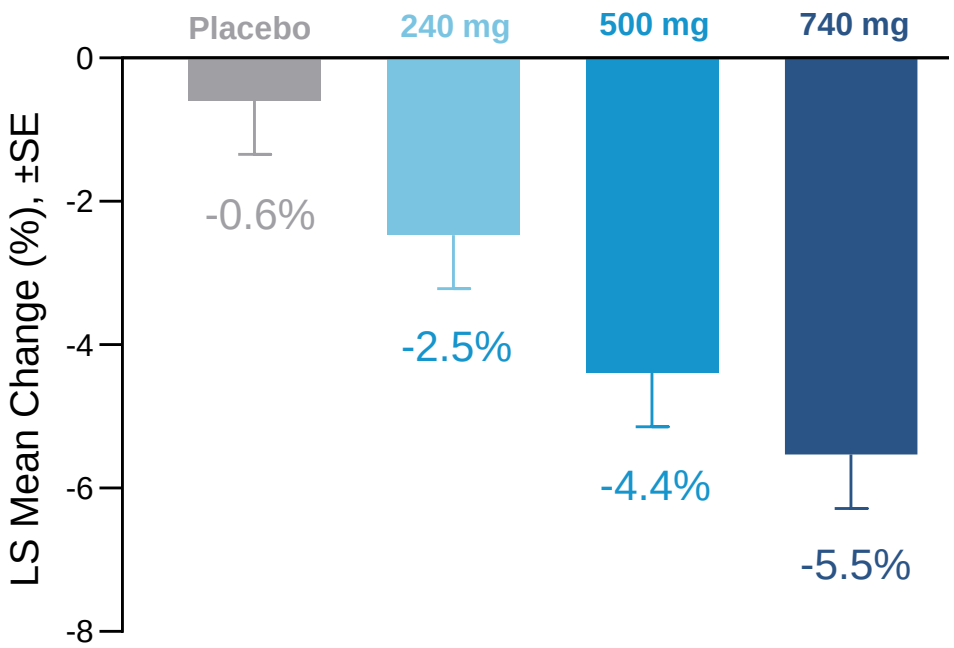
BMI: body mass index, HbA1c: hemoglobin A1c, MAD: multiple ascending dose, N: number of participants in analysis set, SD: standard deviation

TERN-601 Showed Dose-Dependent 28-Day Mean Weight Loss Up to 5.5%

Mean Body Weight Change from Baseline (kg)



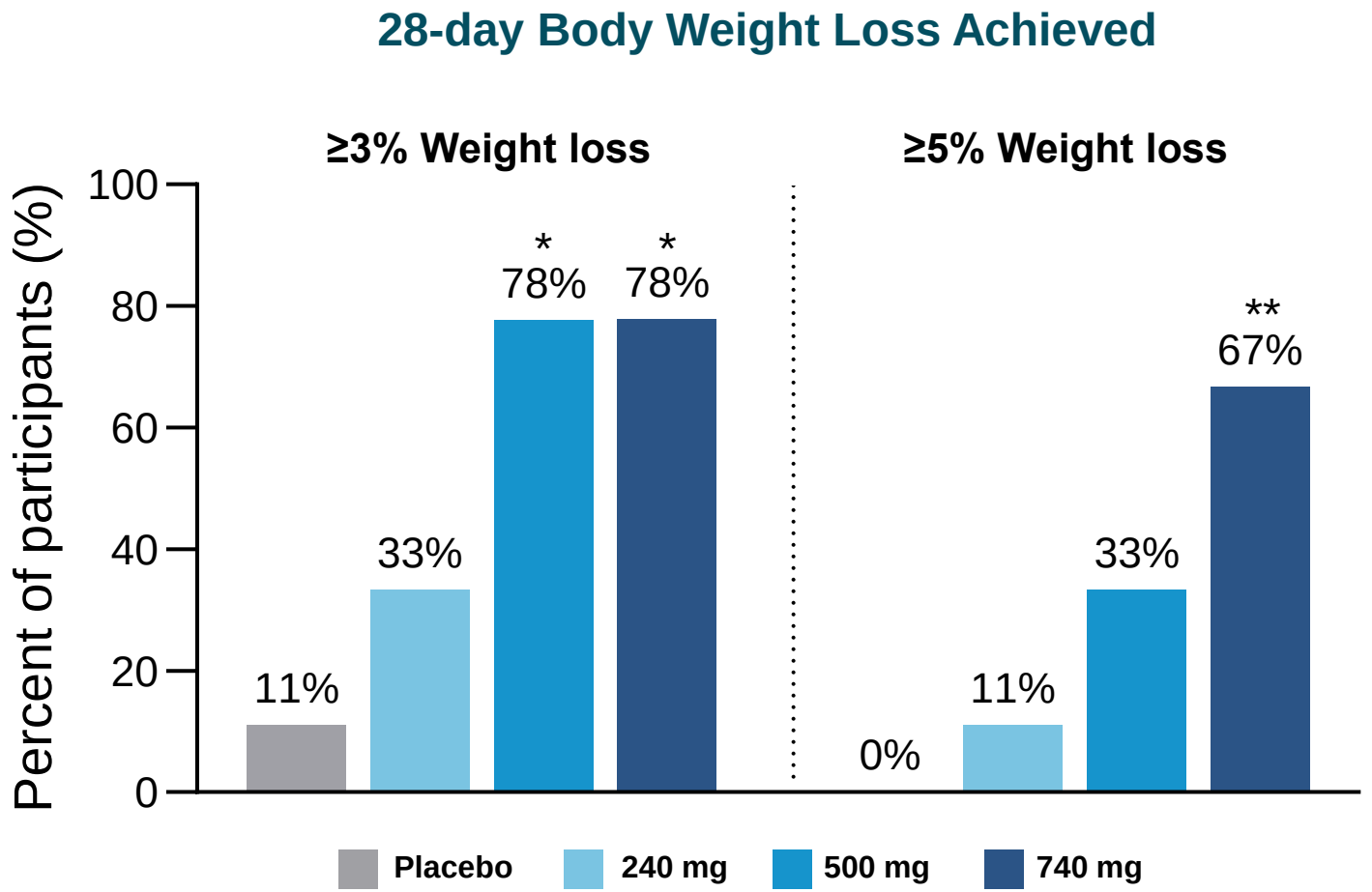
Mean Body Weight Change from Baseline (%)



N	9	9	9	9
PBO-adjusted	-	-1.9%	-3.8%	-4.9%
P-value	-	<0.1	<0.01	<0.0001

*p-value <0.1; **p-value <0.01; ***p-value <0.001, ****p <0.0001
LS: Least Squares, N: number of participants in analysis set, PBO: placebo, SE: standard error
Note: 1 participant (240mg) discontinued study early due to unrelated Grade 1 AE (menstrual bleeding determined to be unrelated to study drug); participant was replaced

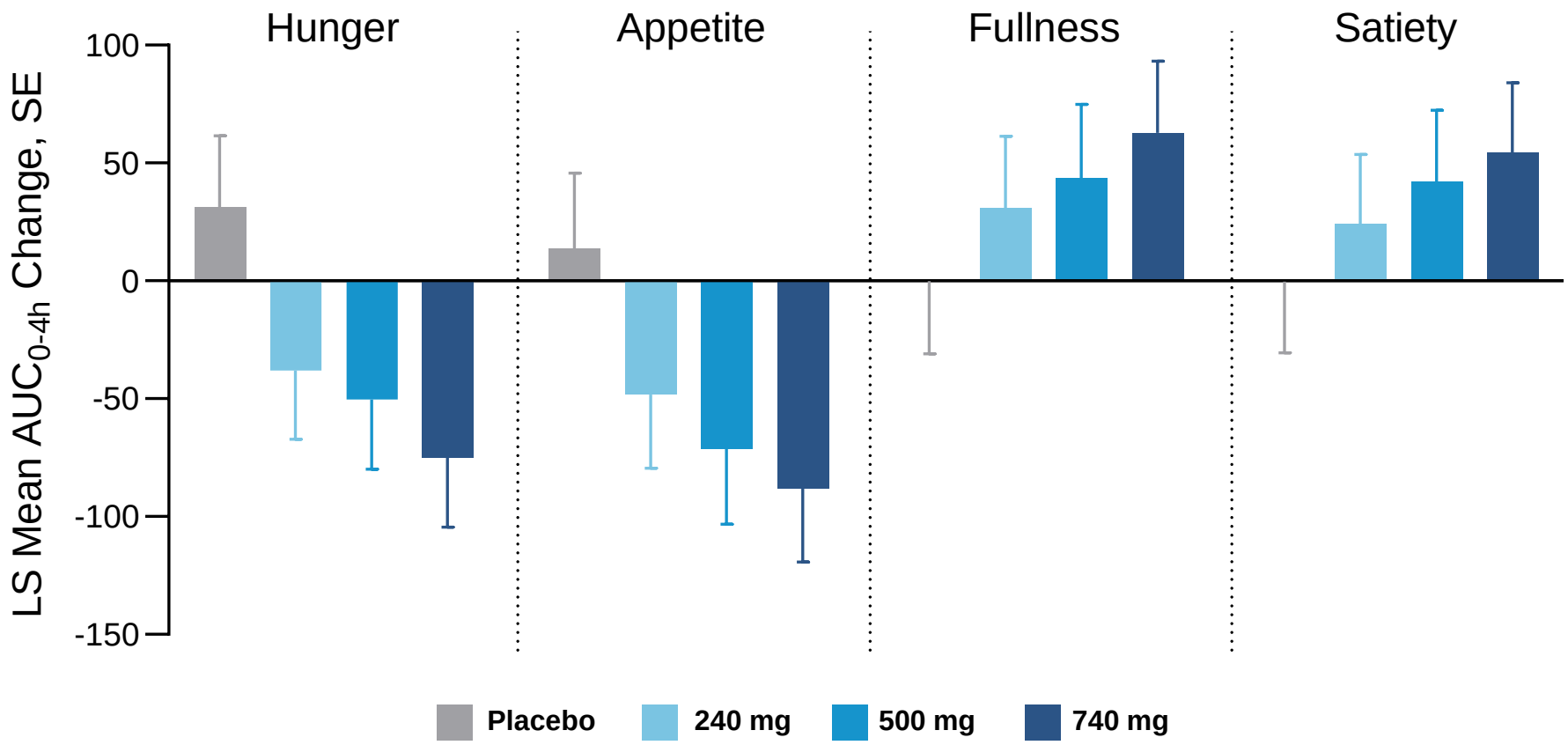
Clear Dose Response With 67% of Participants Losing ≥ 5% Baseline Body Weight at Top Dose



*p-value <0.1; **p-value <0.01, relative to placebo

Meaningful Changes in Hunger/Satiety Scores Seen at All Doses with Clear Dose Relationship

Day 27 Change from Baseline – Participant Appetite Questionnaire



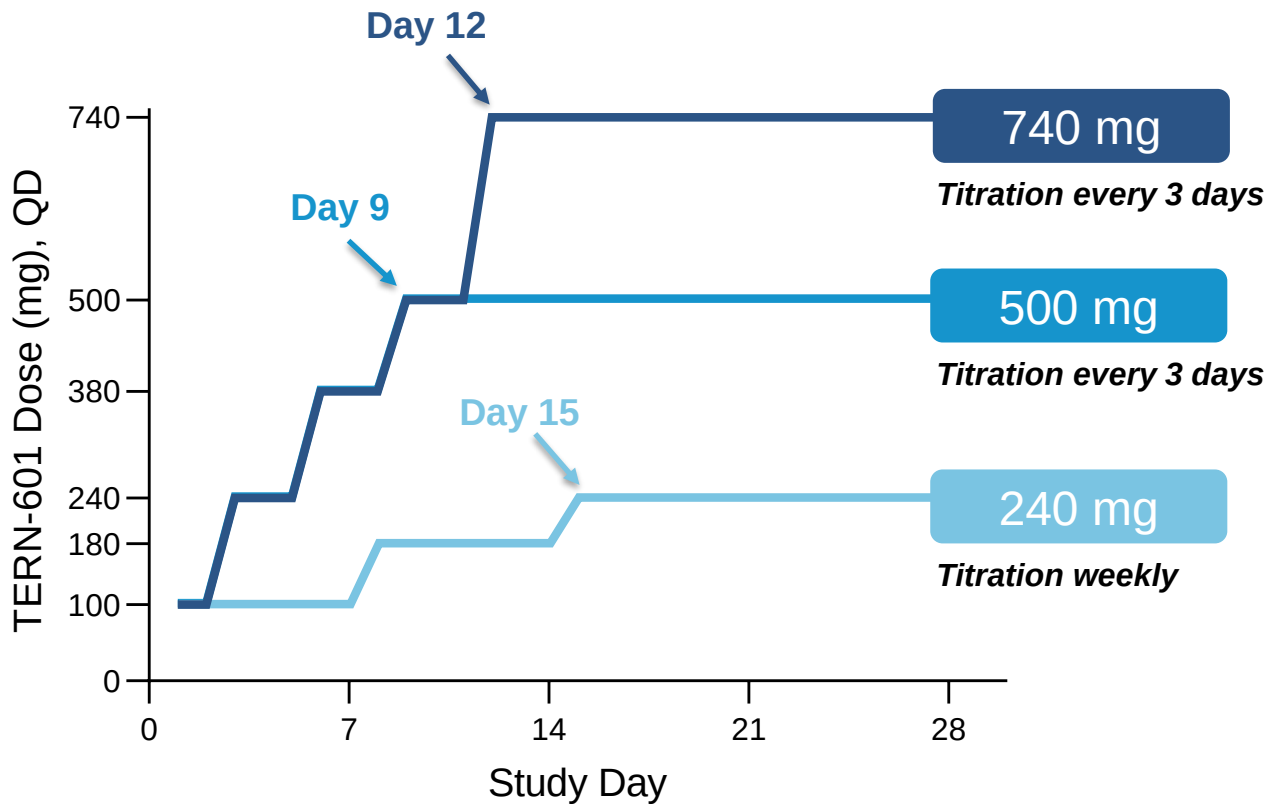
Data based on patient-reported appetite and satiety scores measured using the visual analog scale (0-100 mm)
$AUC_{0\text{-}4hr}$ = area under the curve from timepoint 0 to 4 hr (hr.mm), LS: least squares, SE: standard error

28-Day MAD Design Assessed Tolerability of Fast Titration to Target Doses

Well tolerated despite fast titration suggests potential for improved tolerability in subsequent studies with slower titration

- Safety / tolerability data from completed cohorts guided titration speed and target dose for subsequent cohorts
- Primary measures of tolerability guiding escalation / titration decisions were:
  - Dose interruptions / reductions / discontinuations
  - Severity of GI AEs

All Cohorts Completed Titration Within the First 2 Weeks



GI AEs: gastrointestinal adverse events, MAD: multiple ascending dose, QD: once-daily

TERN-601 Was Well Tolerated With Unremarkable Safety Findings Despite Rapid Titration to Target Doses

- No AE-related discontinuations, interruptions or dose reductions
  - Adverse events were generally mild and evenly distributed across arms, including placebo
  - No drug-related serious adverse events
- Favorable safety profile with no severe or serious AEs
  - >95% of treatment emergent adverse events were mild (Grade 1)
- No clinically meaningful changes in liver enzymes
  - Liver enzymes remained < 1.5X ULN while on treatment at all doses
- Majority of GI-related AEs mild in severity despite fast titration
  - GI AEs consistent with class increased with faster titration to target doses, as expected, and were not dose limiting

Compelling 28-Day Data Amongst Oral GLP-1RA Peers

	TERN-601	danuglipron	GSBR-1290	orforglipron	RGT-075	CT-996
≥3% Placebo-Adjusted Weight Loss	✓	✓	✓	✓	✓	✓
No Dose Interruptions or Reductions Due to AEs	✓	✗	✓	?	✗	✗
No Drug-Related AE Discontinuations	✓	✗	✓	✗	✗	✓
No Severe TEAEs	✓	✗	✓	✓	✓	✓
Rapid Dose Titration (>50% of Days at Highest Dose)	✓	✓	✗	✗	✗	✗

Note: Assessments based on entirety of Phase 1 28-day datasets of peer compounds (any/all doses/cohorts); no head-to-head study has been conducted with TERN-601 against the other drug product candidates. Differences exist in study designs and conditions, and caution should be exercised when comparing data across studies. Data are shown for illustrative purposes only.

Sources: danuglipron: Saxena A, et al. *Nature Medicine*. 2021;27:1079-87; GSBR-1290: Structure Therapeutics Corporate Presentation; GSBR-1290 Phase 1b MAD Results. 2023 September 29; orforglipron: Pratt E, et al. *Diabetes Obes Metab*. 2023;25:2642-49; RGT-075: Priner M. et al. *Diabetes* 2022;71(Supplement_1):94-LB; CT-996: Presented at the 60th European Association for the Study of Diabetes Annual Meeting. Safety, Pharmacokinetics and Pharmacodynamics of CT-996, an Oral Small-Molecule, Signal-Biased GLP-1 Receptor Agonist Over 4 Weeks in Adults with Obesity. 11 September 2024.

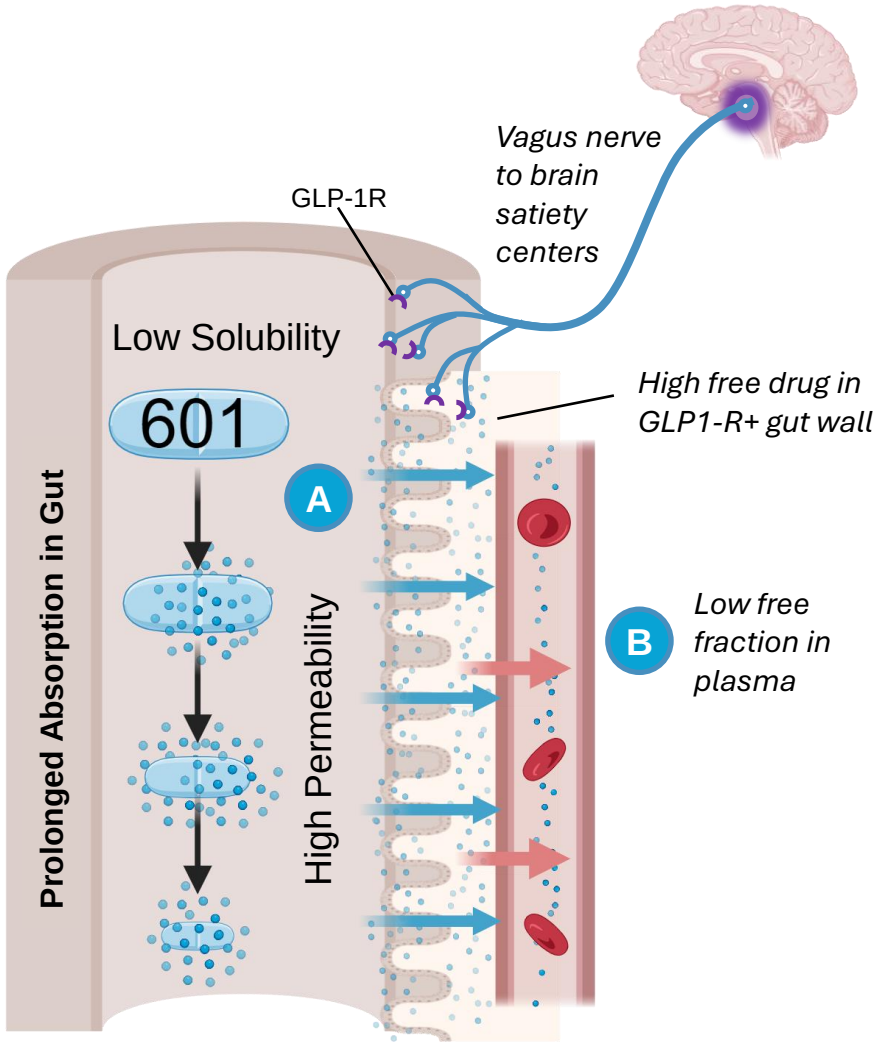
AE: adverse event, GLP-1R agonist: glucagon-like peptide-1 receptor agonist, TEAE: treatment emergent adverse event

Distinct Drug Properties May Confer Advantages For an Orally-Dosed GLP-1R Agonist

	TERN-601 Property	Advantage
Drug Product	Tablet	Convenient once-daily oral dosing
Solubility	Low	Prolonged absorption and flat PK curve
Gut Permeability	High	
Gut wall: Plasma Concentration Ratio	High	High levels of GLP-1R activation in gut
Plasma Protein Binding	High	Allows high doses with good tolerability

GLP-1R: glucagon-like peptide-1 receptor; PK: pharmacokinetic

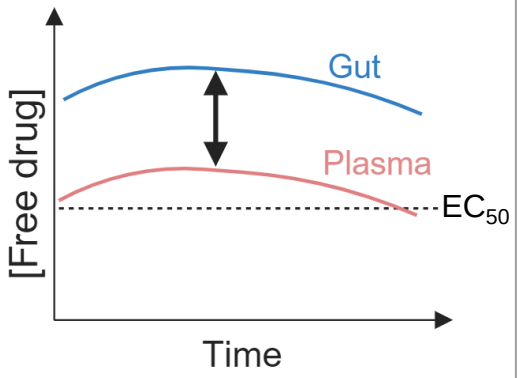
Distinct Properties Enable Tolerable Target Doses that Achieve Robust GLP-1R Activation and Flat PK Curve



A

Low solubility & high permeability results in:

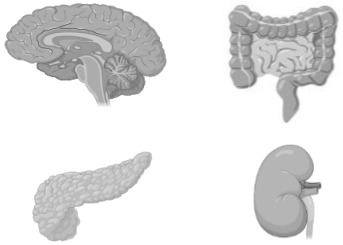
- **Prolonged absorption** and **flat PK curve** allowing **QD dosing**
- **High drug levels in gut wall** that strongly activate GLP-1R in gut triggering satiety centers in brain



B

Low free fraction may allow:

- **Tolerable higher doses** that drive both **gut and systemic GLP-1R** activation



GLP-1R: glucagon-like peptide-1 receptor, PK: pharmacokinetic, QD: once-daily
EC_{50}: concentration at which 50% of maximal activity is observed

TERN-601 Well Positioned for Subsequent Studies: Plan to Initiate Phase 2 in Early 2Q25

Clinical Data To Date:		Potential Impact on Future Development:
✓ Thorough exploration of dose range	→	**No new dose range exploration anticipated**
✓ Well tolerated despite fast titration scheme	→	**Improved tolerability with slower titration**
✓ Flat PK with sustained target coverage	→	**Compelling weight loss over longer durations**
✓ Robust PD effects at all dose levels	→	**Optionality to pursue high/low doses for various patient segments**

PD: pharmacodynamic, PK: pharmacokinetic

Next Steps for TERN-601: Phase 2 Start in Early 2Q25

- ✓ **Positive Phase 1**
- ✓ **Operational and CMC Readiness**
- ✓ **Scientific and Regulatory Feedback**

Phase 2 for Obesity

- Plan to initiate a Phase 2 clinical trial in early 2Q25
- Initial 12-week data expected in 2H25
- Trial will begin with a 12-week portion to optimize dose titration and inform subsequent cohorts

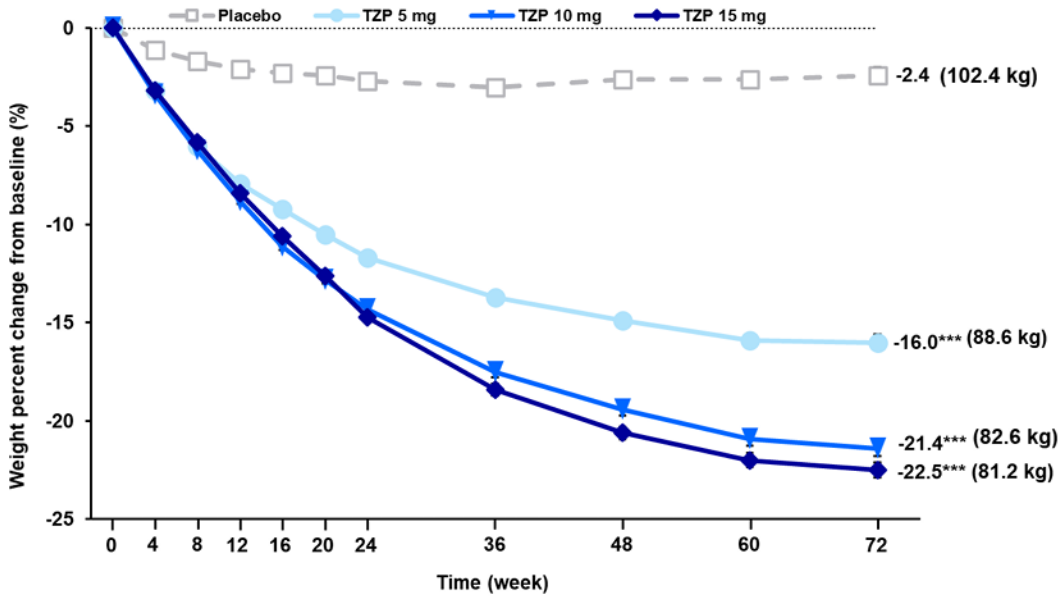
TERN-800 Series

- Prioritizing efforts on nominating a GIPR antagonist development candidate
- Candidate nomination activities ongoing
- Focused on potential class-leading GIPR modulators

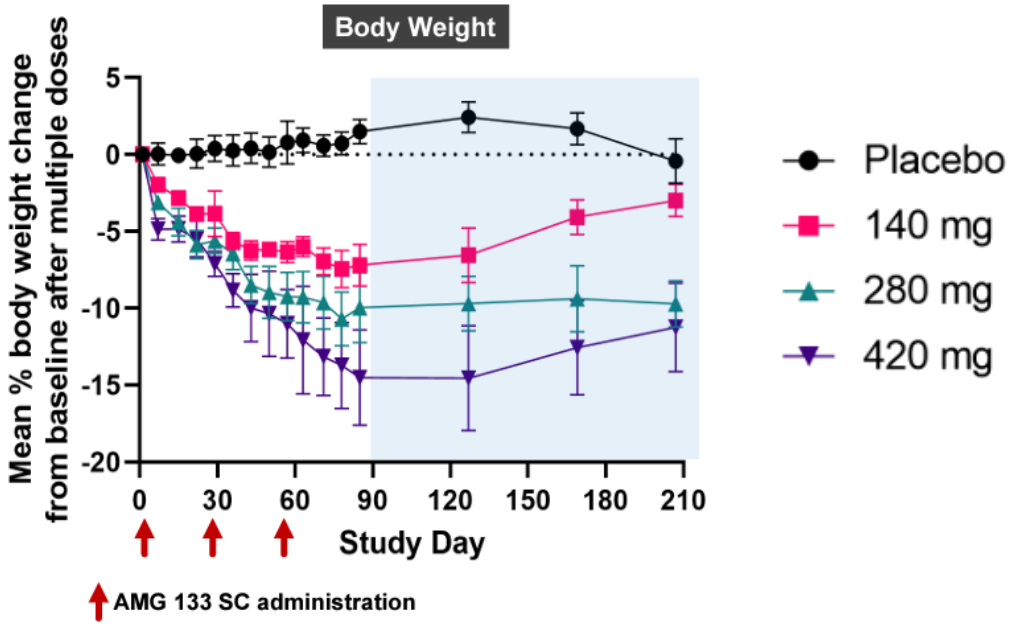
GIPR Modulators Have Shown High Potential in Weight Loss (~15% - ~20%)

Terns' GIPR discovery efforts are ongoing; prioritizing GIPR antagonist for candidate nomination

tirzepatide, a GLP-1 / GIPR *agonist*, showed ~20% mean weight loss over 72 weeks:



AMG-133, a GLP-1 agonist / GIPR *antagonist*, also showed significant weight loss up to 150 days:



Source: Campbell Mol Metab. 2021 Apr; 46: 101139; AMGN data disclosure; LLY data disclosure

TERN-800 Series is Underway: Prioritizing Efforts Towards Nominating a GIPR Antagonist Candidate

GIPR Antagonist in Lead Optimization

- Prioritizing efforts on nominating a GIPR antagonist development candidate based on in house discoveries and growing scientific rationale supporting GLP-1 agonist & GIPR antagonist combos for obesity

GIPR Modulator Discovery Efforts Ongoing

- Combining chemistry expertise with leading synthesis to develop initial set of ‘800 series
- Focused on modulators that can be combined with GLP-1s

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TERN-501

Highly-Selective THR-β Agonist

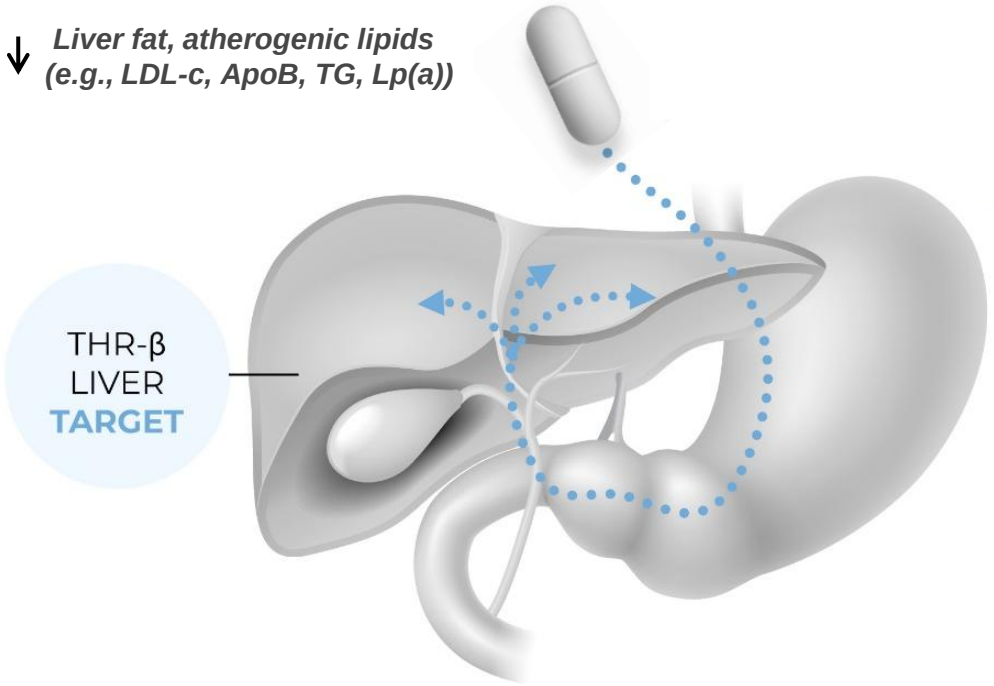
- Potential best-in-class THR-β agonist on efficacy and tolerability based on Phase 2 clinical data
- Emerging superior profile for combinations with GLP-1s to enhance weight loss and metabolic health
- Evaluating opportunities to further develop TERN-501 as a combo therapy for cardiometabolic disease

TERN-501: A Differentiated THR-β Agonist

THR-β regulates key aspects of energy metabolism (e.g., fatty acid & lipid synthesis, liver fat removal through fatty acid oxidation)

↑ *Sex hormone binding globulin*

↓ *Liver fat, atherogenic lipids (e.g., LDL-c, ApoB, TG, Lp(a))*



Other THR-β agonists face limitations with off-target effects, unpredictable PK, or need for CYP metabolism

- TERN-501 was screened for **a differentiated, potentially best-in-class profile**
  - High β/α selectivity → low dose, broad therapeutic window, low CV side effects and improved efficacy
  - Better gastrointestinal profile vs peer molecules → improved tolerability
  - Predictable PK, once-daily dosing with low drug-drug interaction potential → attractive partner for combinations
- **Positive top-line DUET results** announced August 2023: compelling profile of **efficacy, tolerability & combinability** vs peers

Source: Sinha et al. Thyroid (2019); TERN-501 EASL 2020 Poster

TERN-501 Has Best-in-Class Potential

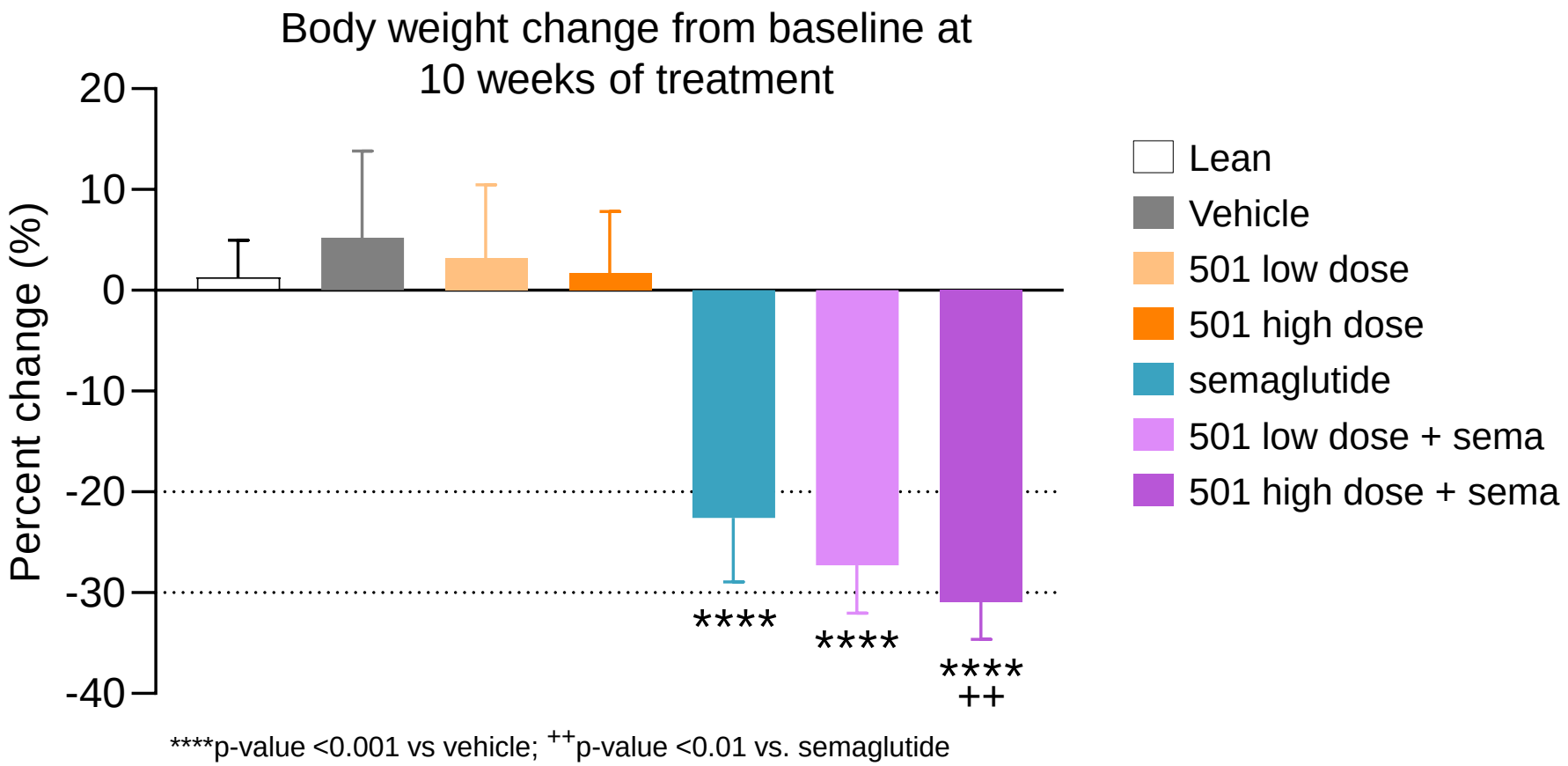
Comparison of THR-βs	TERN-501	Resmetirom	VK2089	ALG-055009	ASC41
Class Leading Liver Fat Reductions	✓	-	✓-	?	-
Once-Daily Dosing	✓	✓	?	✓	✓
Safe/Efficacious @ Low Dose	✓	-	?	-	-
High THR-β / α Selectivity	✓	✓	-	✓	-
Combinability (Linear, Non-variable PK)	✓	-	-	✓	-
Not Metabolized by CyP	✓	-	-	✓	-
Lack of Cardiovascular AEs	✓	✓	-	✓	✓
Lack of Central Thyroid Effects	✓	✓	-	-	-
Lack of GI Adverse Events	✓	-	✓	-	✓
Total Score	**9**	4	2	5	3

Scoring based on publicly available data; comparisons were not done on a head-to-head basis and includes cross-trial and/or cross-phase comparisons; AEs refer to treatment-related AEs; references available upon request.

Non-clinical Data Suggests TERN-501 May Augment Weight Loss Effects of GLP-1R Agonist

Preliminary data in diet-induced obese (DIO) NASH mice[1]

- Semaglutide induces significant body weight loss after 10-weeks of treatment
- TERN-501 **significantly enhances** body weight loss effects of semaglutide



****p-value <0.001 vs vehicle; $^{++}$p-value <0.01 vs. semaglutide

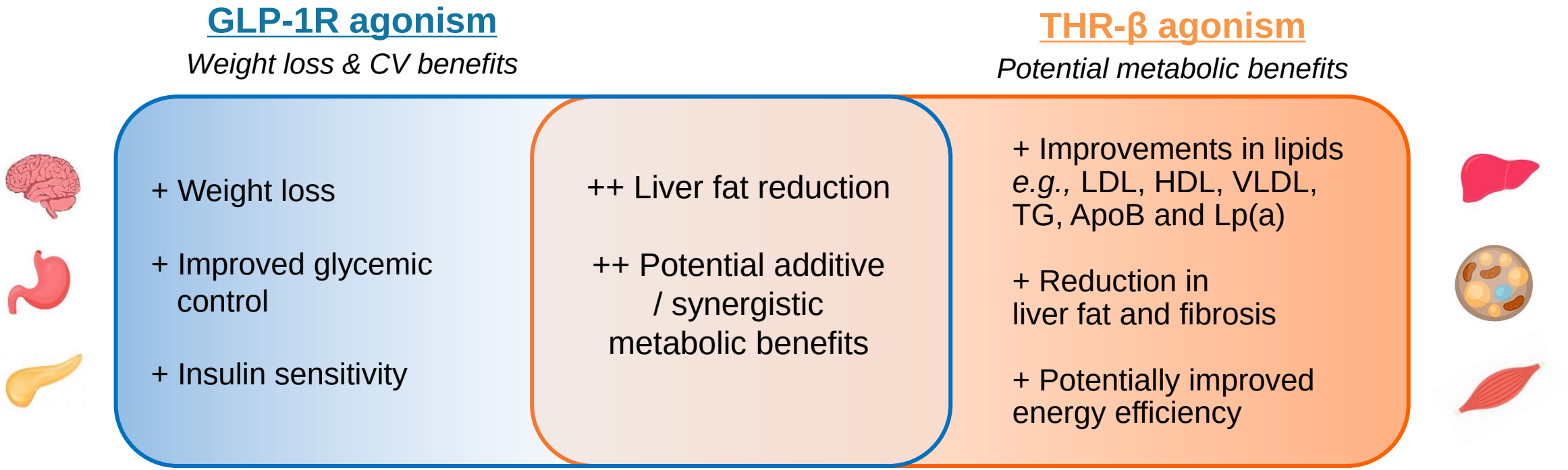
1. Body weight change after 10-weeks of treatment; mice on Gubra amylin high fat, cholesterol, and fructose diet for >35-weeks prior to study start

Note: TERN-501 dosed orally, once-daily; semaglutide dosed subcutaneously, once-daily. The same doses of TERN-501 and semaglutide monotherapy arms were used in combination arms

Combination of GLP-1 and THR-β Has the Potential to Improve Multiple Metabolic Disorders

Potential beneficial effects of simultaneously targeting multiple pathways involved in weight control and metabolism

- Terns is uniquely positioned to develop an oral GLP-1 + THR-β combination



Conclusions

- Strong Balance Sheet
- Multiple upcoming milestones

Strong Financial Position Supports Upcoming Milestones

* As of September 30, 2024; shares include common stock and prefunded warrants

Key Completed and Upcoming Milestones

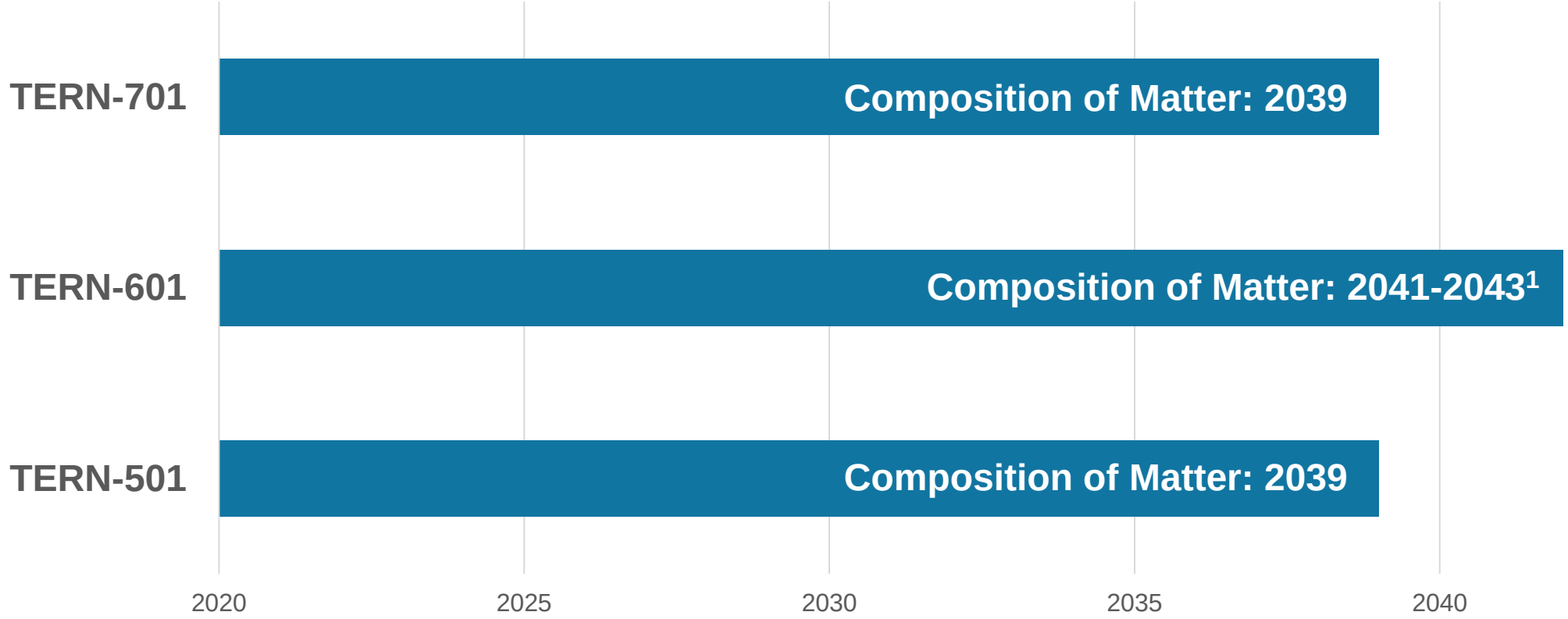
Multiple clinical milestones expected across Terns' pipeline

	2023	2024	2025	
TERN-701 (Allosteric BCR-ABL)	✓ U.S. Ph 1 Trial initiated (Dec 2023)	✓ **Interim data from initial Ph 1 cohorts** (Dec 24)	CARDINAL Dose expansion initiation (1H25)	**Additional Phase 1 data** (4Q25)
TERN-601 (GLP-1 Agonist)	✓ Phase 1 trial initiated (Nov 2023)	✓ **Phase 1 top-line data** (Sept 24)	Phase 2 initiation (early 2Q25)	**Phase 2 initial 12-week data** (2H25)
TERN-501 (THR-β Agonist)	✓ DUET MASH Phase 2a combo trial top-line data (Aug 2023)			

Note: Check mark (✓) denotes completed milestones, all other milestones are anticipated future milestones. Relative position of completed or expected milestones on illustration does not denote or imply chronological order

Terns: Robust Intellectual Property

- Patent exclusivity could be extended for a period of up to 5 years through patent term extension
- Issued patents and pending applications cover polymorphs, methods of treatment/dosing, and combination treatment approaches



All figures above denote US timelines only, similar coverage periods assumed for other territories.

1. We own multiple composition of matter patent application families directed to our GLP-1R agonist compounds, including TERN-601, for which claims have not yet been granted. Any patents that may issue from applications in these families are generally projected to expire in 2041-2043, not including any patent term adjustments and/or patent term extensions that may be available.

Mission. Vision. Core Values.

MISSION

To advance transformative medicines that address serious diseases

VISION

To pioneer significant innovations across the lifecycle of drug development

TRUST
EVOLVE
RESPECT
NURTURE
SOAR

Trust: empowered and accountable to do the right thing

Evolve: learning and growing from our successes, failures and changes in the environment

Respect: celebrating the diversity of our backgrounds, opinions and experiences

Nurture: fostering internal and external relationships

Soar: aiming high and being your best

Appendix

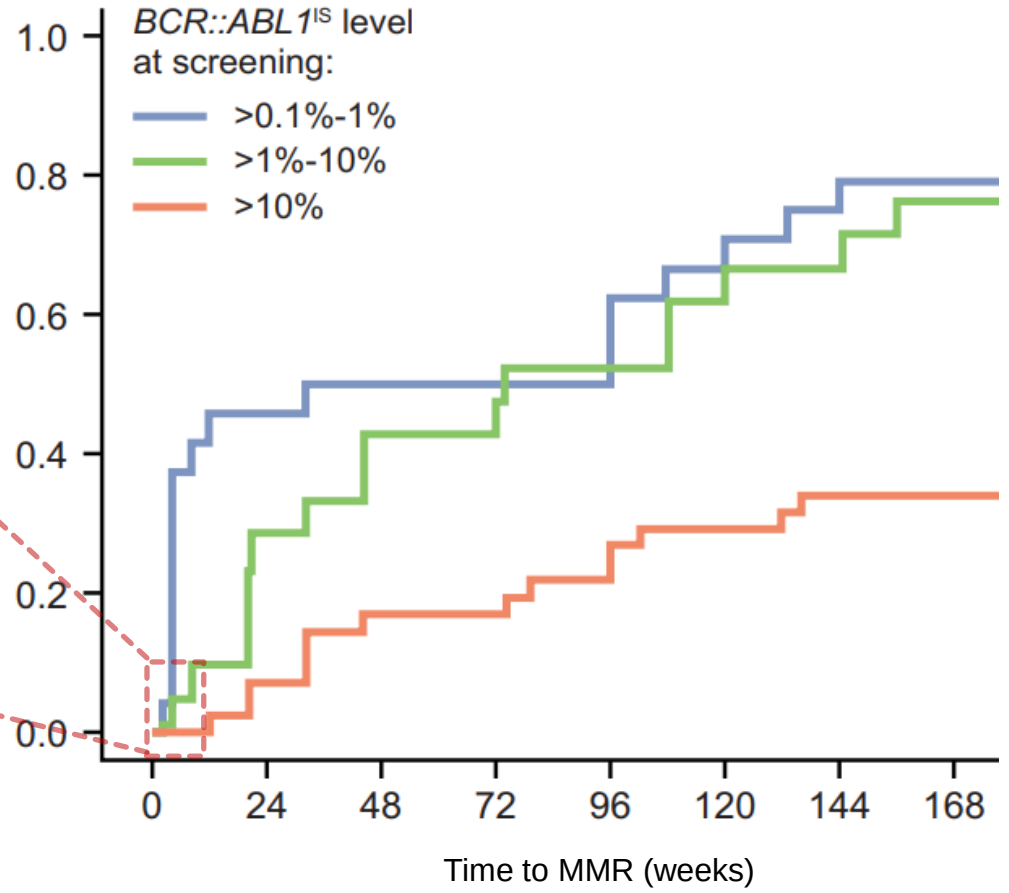
Early Molecular Response Data are Trending Favorably

Encouraging 3-month MMR in non-T315Im CML in a more refractory patient population than asciminib Ph1

- At 3 months, TERN-701 shows:
  - 1/4* with BCR::ABL1 >10% achieves DMR; 4/4 have decrease in transcript with treatment ongoing
  - 1/2 with BCR::ABL1 >1-10% achieves MR2 post-asciminib; 2/2 have decrease in transcript with treatment ongoing
- Asciminib showed <5% and <10% MMR at 3 months in patients without T315Im with BCR::ABL1 >10% and >1-10%, respectively

Baseline BCR::ABL1	Asciminib MMR at 3 months
>10% (N=41)	<5%
>1-10% (N=21)	<10%

Incidence of MMR in non-T315I mutant CP CML in asciminib Phase 1



* 4 response-evaluable patients without T315I mutation and baseline transcript >10%
Note: No head-to-head study has been conducted with TERN-701 against asciminib or any other drug or product candidate. Differences exist in study designs and conditions, and caution should be exercised when comparing data across studies. Data and comparisons are shown for illustrative purposes only; CP: chronic phase; DMR: deep molecular response; MMR: major molecular response; MR2: at least a 2-log reduction (i.e., BCR::ABL1^{IS} ≤ 1%)
Mauro MJ, et al. Leukemia. 2023 May;37(5):1048-1059. Supplemental Material.

No Concerning Safety Signals for Hematologic AEs

Majority of treatment-emergent hematologic adverse events are low grade

No hematologic DLTs or treatment related AEs > Grade 2

Hematologic Treatment-Emergent Adverse Events

Parameter SOC/PT n (%)	160 mg QD (N=7)		320 mg QD (N=5)		400 mg QD (N=3)		All patients (N=15)	
	All grade	≥ Grade 3	All grade	≥ Grade 3	All grade	≥ Grade 3	All grade	≥ Grade 3
Thrombocytopenia	2 (29%)	0	2 (40%)	0	0	0	4 (27%)	0
Anemia	1 (14%)	0	2 (40%)	1 (20%)*	0	0	3 (20%)	1 (7%)
Neutropenia	1 (14%)	0	3 (60%)	1 (20%)*	0	0	4 (27%)	1 (7%)
Thrombocytosis	0	0	2 (40%)	0	0	0	2 (13%)	0

* Neither grade ≥ 3 event was considered related to TERN-701

AEs: adverse events; DLT: dose limiting toxicities; G: grade; SOC: system organ class; PT: preferred term; QD: once-daily

No Concerning Safety Signals for Non-Hematologic AEs

Majority of non-hematologic treatment-emergent adverse events are low grade

No non-hematologic DLTs or treatment related AEs > Grade 2

Non-Hematologic Treatment-Emergent Adverse Events in > 1 Patient

Parameter SOC/PT n (%)	160 mg QD (N=7)		320 mg QD (N=5)		400 mg QD (N=3)		All patients (N=15)	
	All Grade	≥ Grade 3	All Grade	≥ Grade 3	All Grade	≥ Grade 3	All Grade	≥ Grade 3
Nausea	1 (14%)	0	1 (20%)	0	1 (33%)	0	3 (20%)	0
Headache	3 (43%)	0	0	0	0	0	3 (20%)	0
Dizziness	1 (14%)	0	1 (20%)	0	0	0	2 (13%)	0
Fatigue	1 (14%)	0	1 (20%)	0	0	0	2 (13%)	0
Oedema peripheral	1 (14%)	0	1 (20%)	0	0	0	2 (13%)	0

AEs: adverse events; DLTs: dose limiting toxicities; PT: preferred term; QD: once-daily; SOC: system organ class

Incidence of Dose Limiting Toxicities (DLTs) for TERN-701 Trending Lower than Asciminib Phase 1

Both Phase 1 studies assessed DLTs during first 28 days of treatment

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)

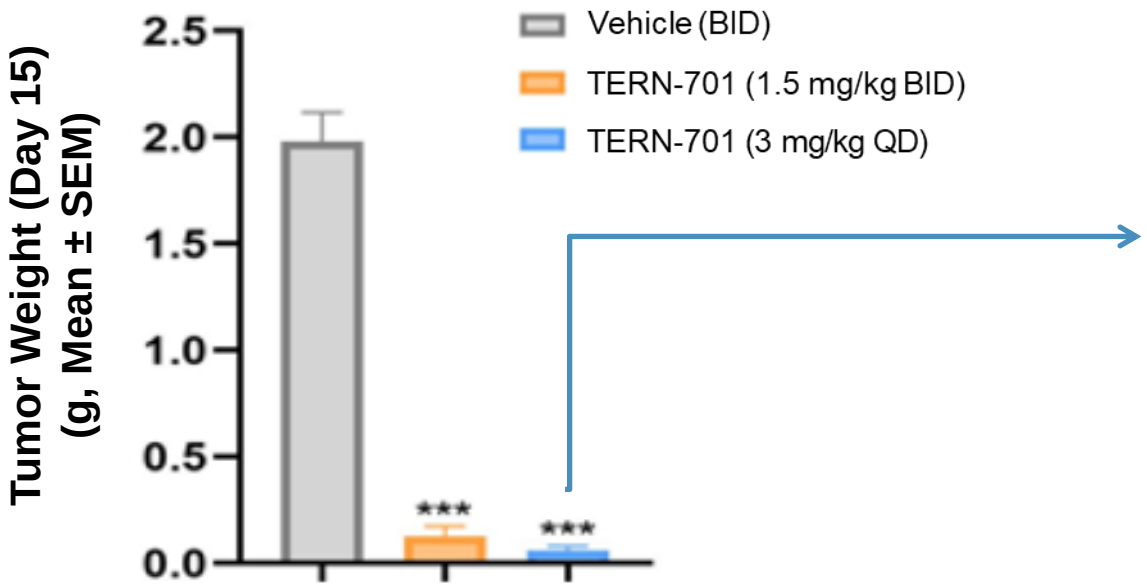
TERN-701 Dose	Dose Limiting Toxicities
160 mg QD	No DLTs
320 mg QD	No DLTs
400 mg QD	No DLTs
500 mg QD	Undergoing evaluation

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.

TERN-701 Showed Robust Tumor Growth Inhibition with High Tumor Drug Levels in CML Mouse Models

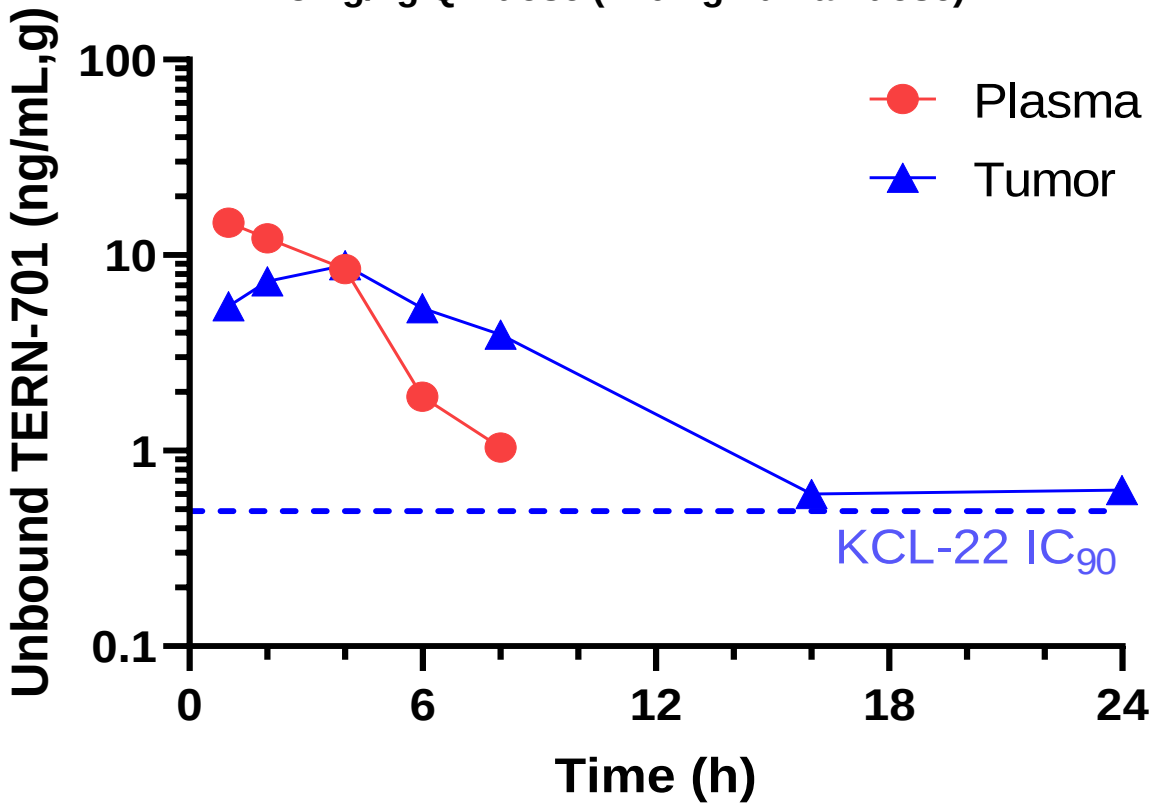
TERN-701 showed robust tumor growth inhibition in KCL-22 mouse xenograft at low doses

In vivo tumor growth inhibition in KCL-22 mouse xenograft



TERN-701 achieved robust and prolonged target coverage in leukemic cells in mouse model

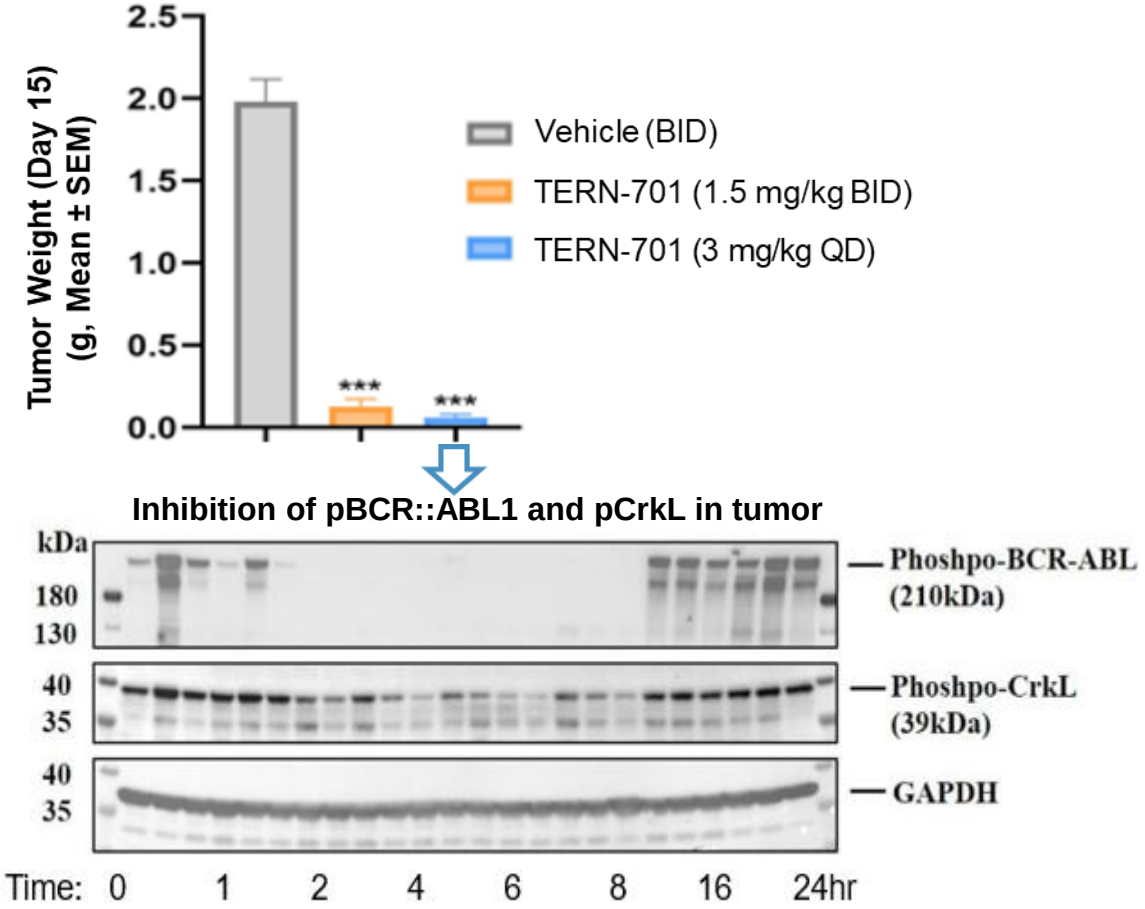
TERN-701 mouse plasma and tumor concentrations at 3mg/kg QD dose (~40mg human dose)



All error bars represent the SEM ***p<0.001.
BID: twice (two times) a day; PD: pharmacodynamic; QD; once-daily

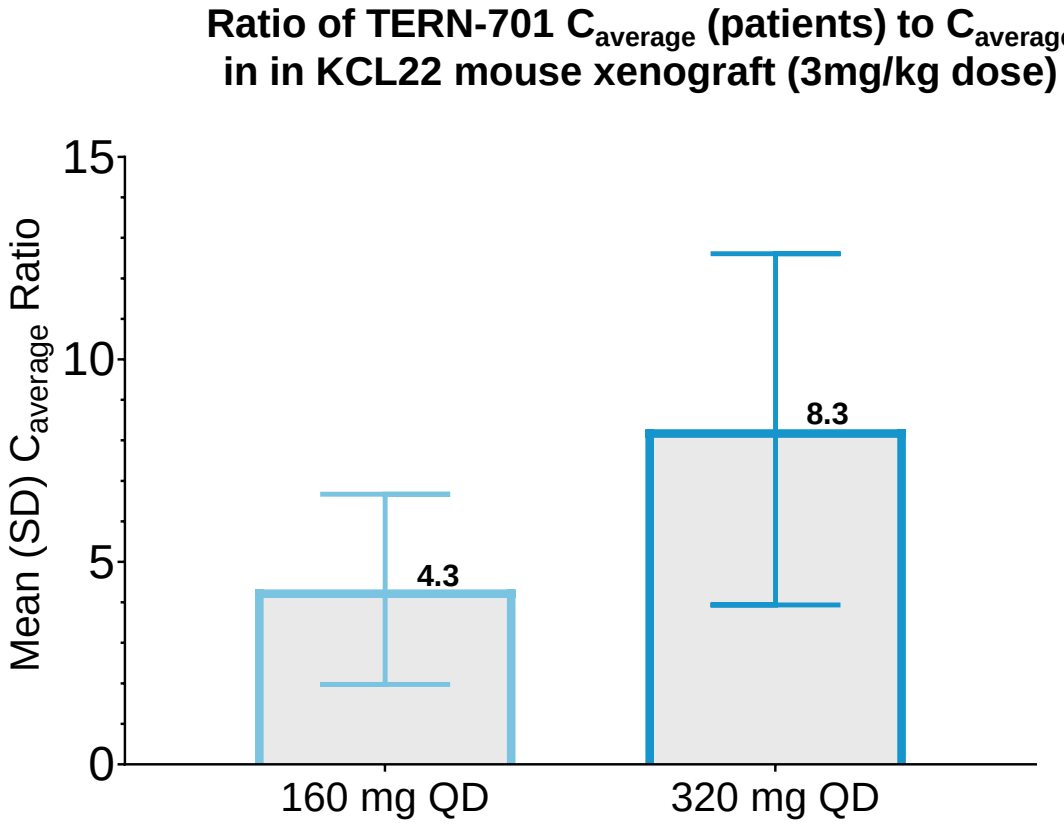
TERN-701 Doses in CARDINAL Study are Associated with Potent Pharmacodynamic Inhibition of BCR::ABL1 Signaling

TERN-701 3mg/kg dose potently inhibits BCR::ABL1 signaling pathway in KCL-22 mouse xenograft



All error bars represent the SEM ***p<0.001.
BID: twice (two times) a day; PD: pharmacodynamic; QD; once-daily

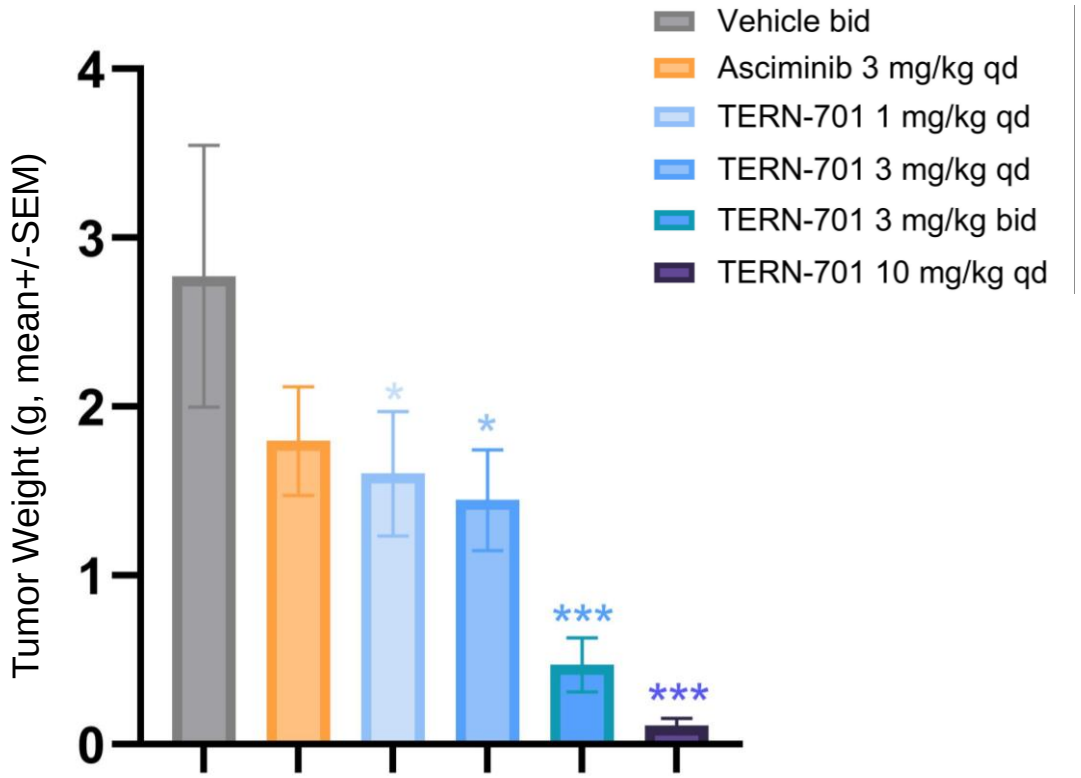
Clinical doses in CARDINAL achieve exposures with robust target coverage relative to 3mg/kg dose in mouse model



TERN-701 Showed a Greater Anti-Tumor Effect vs. asciminib in Additional Mouse Models of CML

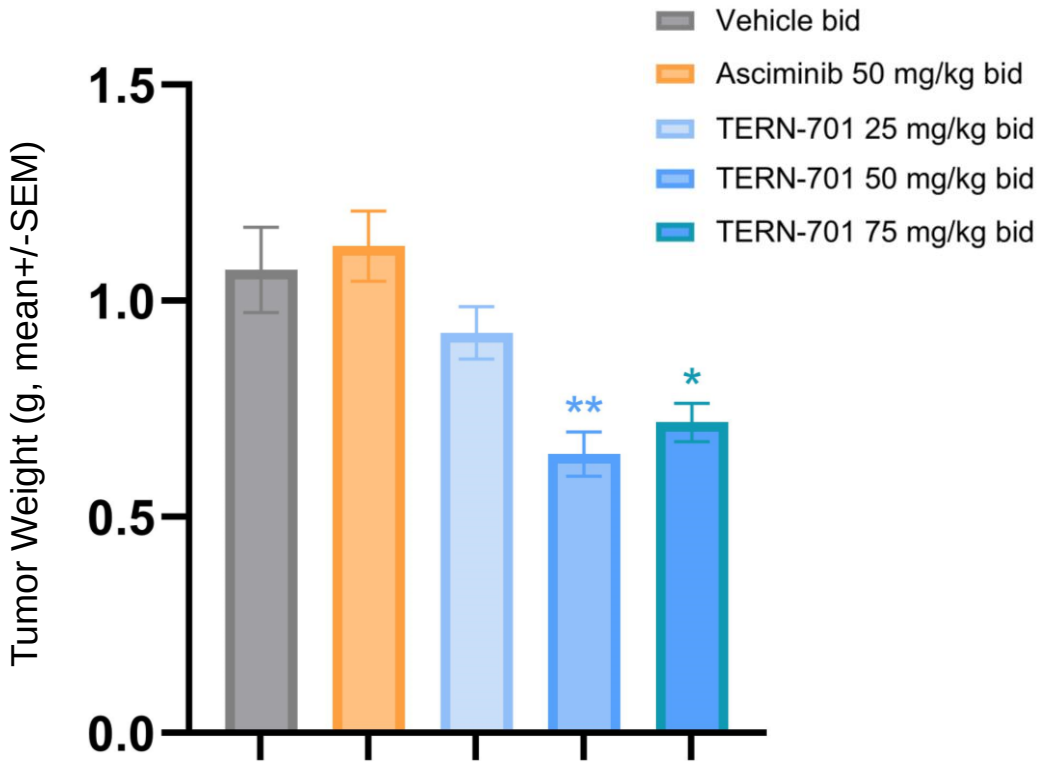
K562 Xenograft

(Day 14)



Ba/F3 BCR-ABL1-T315I Allograft

(Day 15)



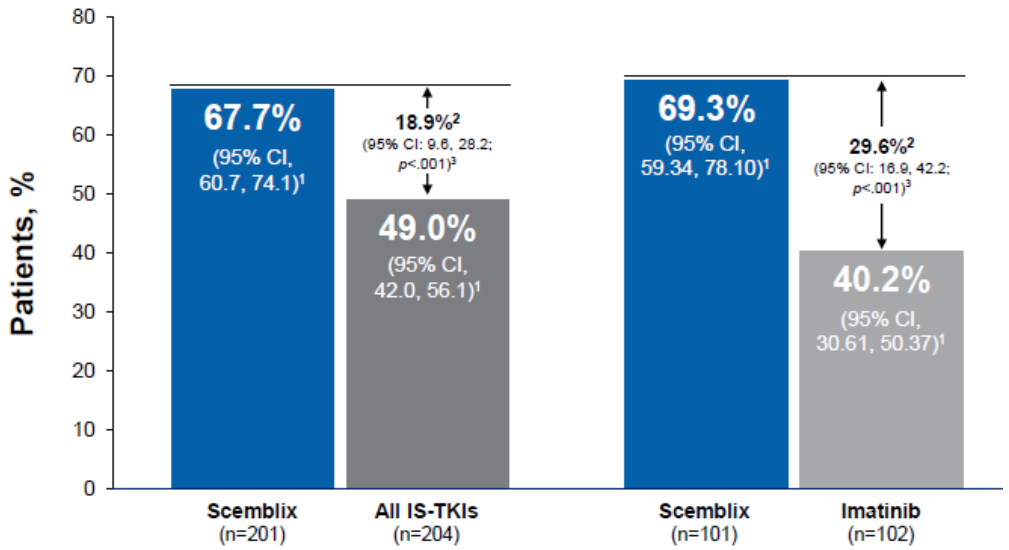
Source: ASPET TERN-701 poster

Note: NOD-SCID (K562) and BALB/c nude mice (Ba/F3T315I) were implanted with CML cells, randomized, and administered the indicated TKIs once tumor volumes reached a mean size of 110 mm. Mean tumor weights for each of the treatment groups at the conclusion of the study. All error bars represent the SEM. *p<0.05, **p<0.01, ***p<0.001.
asciminib was utilized as the free base, TERN-701 was formulated as an optimized salt form

The Only Approved Allosteric TKI for CML has Shown a Benefit Over 2nd Gen Active-site TKIs, Leading to Blockbuster Expectations

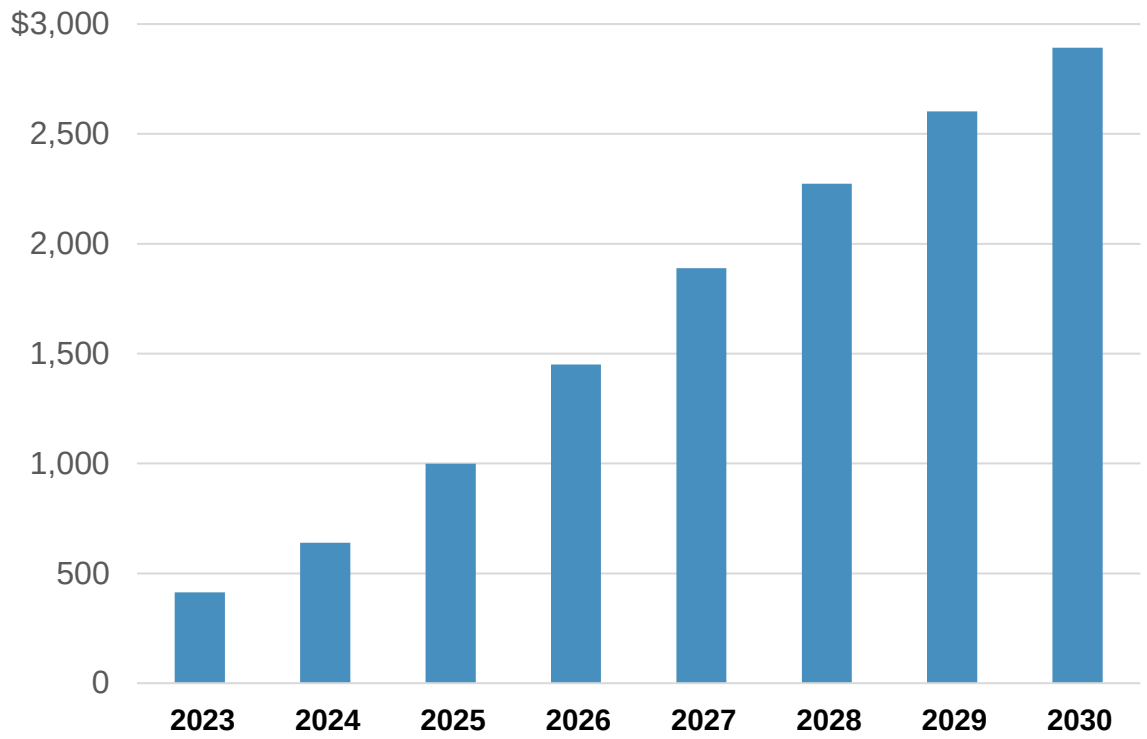
- Asciminib has demonstrated **superior benefit-risk profile** vs standard-of-care TKIs in 1L setting[1], with:
  - Better efficacy with fewer AEs and treatment discontinuations
  - Numerically higher MMR rate vs 2G TKIs[2]
  - Half the discontinuation rate of imatinib or 2G TKIs[2]

ASC4FIRST: MMR rate at week 48 vs IS-TKI and vs imatinib



- Analysts expect asciminib to rapidly approach **blockbuster sales**

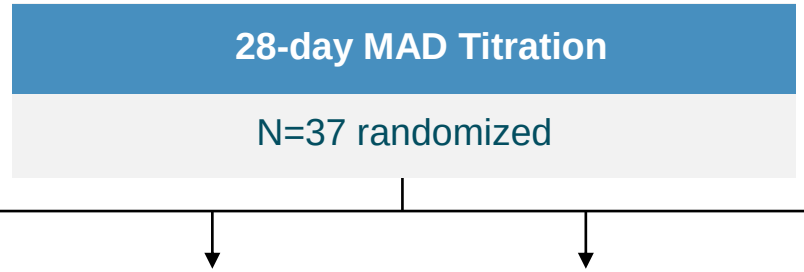
Consensus Sales Estimates ($mm)[3]



Note: 3L: 3rd line; BID: twice-daily; MMR: major molecular response; Scemblix has 3L+ U.S. market share of NBRx 43%, TRx 22% as of 4Q23 (NVS 4Q23 Earnings)

1. Novartis ASCO Investor Event June 2, 2024; 2. Investigator selected 2G TKIs – nilotinib, dasatinib, bosutinib; 3. Estimates from EvaluatePharma; may include sales beyond 3L setting

No Drug-Related Discontinuations, Interruptions or Dose Reductions



	Placebo pooled (N=9)	240 mg (N=10)	500 mg (N=9)	740 mg (N=9)
Completed Treatment	9 (100%)	9 (90%)*	9 (100%)	9 (100%)
Discontinued Study Drug Due to Related-AE	0	0	0	0
Dose Interruption Due to AE	0	0	0	0
Dose Reduction Due to AE	0	0	0	0

* 1 participant discontinued study early due to unrelated Grade 1 AE (menstrual bleeding determined to be unrelated to study drug); participant was replaced
AE: adverse event, MAD: multiple ascending dose, N: number of participants in analysis set

Favorable Safety Profile with No Severe or Serious AEs

>95% of treatment emergent adverse events were mild (Grade 1)

Treatment Emergent AEs by Maximum Severity

Event, N (%)	Placebo pooled (N=9)	240 mg (N=10)	500 mg (N=9)	740 mg (N=9)
Grade 1 (Mild)	5 (55.6%)	5 (50%)	9 (100%)	3 (33.3%)
Grade 2 (Moderate)	0	1 (10%)	0	6 (66.7%)
Grade ≥3 (Severe)	0	0	0	0
Serious Adverse Events	0	0	0	0

- Majority of AEs were consistent with known effects of GLP-1R agonist class (e.g. gastrointestinal)
- No clinically meaningful changes in ECGs, heart rate or blood pressure

AE: adverse event, ECG: electrocardiogram, N: number of participants in analysis set, GLP-1R agonist: glucagon-like peptide-1 receptor agonist

No Clinically Meaningful Changes in Liver Enzymes

Liver enzymes remained ≤ 1.5X ULN while on treatment at all doses

Mean (SD) Change from Baseline to Day 29	Placebo pooled (N=9)	240 mg (N=10)	500 mg (N=9)	740 mg (N=9)
ALT (U/L)	-3.4 (7.6)	-4.0 (6.4)	-9.0 (6.4)	-9.0 (9.7)
AST (U/L)	-2.4 (4.6)	-1.3 (3.3)	-7.0 (4.6)	-5.1 (8.7)
Bilirubin (mg/dL)	0.01 (0.11)	0.15 (0.14)	0.09 (0.35)	0.18 (0.47)

ULN: upper limit of normal; ALT: alanine transaminase; AST: aspartate transaminase; N: number of participants in analysis set; SD: standard deviation

Majority of GI-Related AEs Mild in Severity Despite Fast Titration

GI AEs consistent with class increased with faster titration to target doses, as expected, and were not dose limiting

Treatment Emergent GI AEs by Maximum Severity

Event, N (%)	Placebo pooled (N=9)	240 mg (N=10)	500 mg (N=9)	740 mg (N=9)
Nausea				
Grade 1 (Mild)	2 (22.2%)	0	7 (77.8%)	2 (22.2%)
Grade 2 (Moderate)	0	0	0	6 (66.7%)
Vomiting				
Grade 1 (Mild)	0	0	4 (44.4%)	6 (66.7%)
Grade 2 (Moderate)	0	0	0	1 (11.1%)
Diarrhea				
Grade 1 (Mild)	0	0	2 (22.2%)	2 (22.2%)
Grade 2 (Moderate)	0	0	0	0
Constipation				
Grade 1 (Mild)	0	1 (10.0%)	0	5 (55.6%)
Grade 2 (Moderate)	0	1 (10.0%)	0	0

AE: adverse event, GI: gastrointestinal, N: number of participants in analysis set, GLP-1R agonist: glucagon-like peptide-1 receptor agonist