



Corporate Presentation

May 2026



Forward-Looking Statements and Risk Factors

This presentation and the accompanying oral commentary contain forward-looking statements that involve risks, uncertainties and assumptions. If the risks or uncertainties ever materialize or the assumptions prove incorrect, our results may differ materially from those expressed or implied by such forward looking statements. All statements other than statements of historical fact could be deemed forward-looking, including, but not limited to, any statements regarding: plans, strategies, and objectives of management for future operations, including our clinical development, regulatory and commercialization plans and timelines; any projections of financial information; historical results that may suggest trends for our business; expectation or belief regarding future events; potential markets, market opportunity or market size; technology developments; our clinical product pipeline, clinical and pre-clinical data or the implications thereof; enforceability of our intellectual property rights, competitive strengths or our position within the industry; anticipated patent exclusivity timelines; anticipated benefits of our collaborations or other strategic transactions; and any statements of assumptions underlying any of the items mentioned.

These statements are based on estimates and information available to us at the time of this presentation and are not guarantees of future performance. Actual results could differ materially from our current expectations as a result of many risks and uncertainties, including but not limited to, risks associated with: the potential impacts of raising additional capital, including dilution to our existing stockholders, restrictions on our operations or requirements that we relinquish rights to our technologies or product candidates; the success, cost, and timing of our product development activities and clinical trials; the timing of our planned regulatory submissions to the FDA for our product candidate bezuclastinib and feedback from the FDA as to our plans; our ability to obtain and maintain regulatory approval for our bezuclastinib product candidate and any other product candidates we may develop, and any related restrictions, limitations, and/or warnings in the label of an approved product candidate; the potential for our identified research priorities to advance toward clinical development; the ability to license additional intellectual property relating to our product candidates from third parties and to comply with our existing license agreements and collaboration agreements; the ability and willingness of our third-party research institution collaborators to continue research and development activities relating to our product candidates; our ability to commercialize our products in light of the intellectual property rights of others; our ability to obtain funding for our operations, including funding necessary to complete further development and commercialization of our product candidates; the scalability and commercial viability of our manufacturing methods and processes; the commercialization of our product candidates, if approved; our plans to research, develop, and commercialize our product candidates; our ability to attract collaborators with development, regulatory, and commercialization expertise; our expectations regarding our ability to obtain and maintain intellectual property protection for our product candidates; business interruptions resulting from public health crises, which could cause a disruption of the development of our product candidates and adversely impact our business; and the fact that interim clinical data may not be indicative of future results, among others. For a further description of the risks and uncertainties that could cause actual results to differ from those expressed in these forward-looking statements, as well as risks relating to our business in general, see our periodic filings filed from time to time with the Securities and Exchange Commission. Unless as required by law, we assume no obligation and do not intend to update these forward-looking statements or to conform these statements to actual results or to changes in our expectations.

All of Cogent Biosciences, Inc.'s ("Cogent") product candidates are investigational product candidates and their safety and efficacy have not yet been established. Cogent has not obtained marketing approval for any product, and there is no certainty that any marketing approvals will be obtained or as to the timelines on which they will be obtained.

Three Positive Pivotal Trials for Bezuclastinib Showcase Potential As Best-in-Class KIT Inhibitor Across All KIT-mutant Driven Indications



Phase 3 trial in 2nd-line Gastrointestinal Stromal Tumors:
sunitinib +/- bezuclastinib 600 mg

Positive Results Announced Nov 2025, BT¹ Granted; NDA Submitted under RTOR² March 2026

\$4 billion+ Global Market Opportunity;

First positive 2nd-line GIST trial in over 20 years, 50% reduction in risk of progression or death, 16.5 month mPFS



Pivotal trial in NonAdvanced Systemic Mastocytosis:
bezuclastinib 100 mg vs. placebo

Positive Results Announced July 2025, NDA Accepted, Assigned Dec 30, 2026 PDUFA

\$3.5 billion+ Global Market Opportunity;

Best-in-class symptomatic improvement and pathobiology data demonstrate potential for complete remission in patients



Pivotal trial in Advanced Systemic Mastocytosis:
bezuclastinib 150 mg

Positive Results Announced Dec 2025

\$500 million+ Global Market Opportunity;

Clearly differentiated safety/tolerability results provide clear path to market leadership

**Total Global Market Opportunity exceeds \$8 billion with limited competition;
IP protection anticipated through 2046 based on strength of COM + PTE and pending patent applications**

¹BTD: Breakthrough Therapy Designation, ²RTOR: Real-time Oncology Review

Cogent Biosciences Poised for Transformative 2026

Three Pivotal Trial Wins Position Bezuclastinib as Best-in-Class KIT inhibitor



PEAK: First ever positive trial in GIST patients using active comparator

SUMMIT: Demonstrated reduction in mast cell burden leads to substantial symptom improvements

APEX: Selective, non-CNS penetrant KIT inhibitor spares toxicities associated with current SOC

Prepare to Launch Bezuclastinib in 2026



- Onboarding highly experienced US commercial team to prepare for anticipated 2H26 launch
- NonAdvSM PDUFA 12/30/26; GIST NDA submitted under RTOR March '26; AdvSM NDA planned 1H26

Strong Balance Sheet & Cash Runway



- \$866 million as of 3/31/26*, runway well into 2028

Creating Next-Gen, Best-in-Class Pipeline



- Focused on two franchises: Oncology & Hematology
- Pan-KRAS and JAK2 V617F inhibitors on track for IND 2026

* Cash balance as of 3/31/2026.

Leadership with Deep Scientific Expertise in Precision Medicine



Andrew Robbins
President &
Chief Executive Officer



Jessica Sachs, MD
Chief Medical Officer



Cole Pinnow
Chief Commercial Officer



John Robinson, PhD
Chief Scientific Officer



Evan Kearns, JD
Chief Legal Officer



John Green
Chief Financial Officer



Erin Schellhammer
Chief People Officer



Brad Barnett
Chief Technology Officer

Building Robust Pipeline Across Two Franchises

	PROGRAM	TARGET	PATIENT POPULATION	PRE-CLINICAL	EARLY CLINICAL	LATE CLINICAL
HEMATOLOGY	Bezuclastinib	KIT D816V	Nonadvanced Systemic Mastocytosis (NonAdvSM)	December 30, 2026 PDUFA		
	Bezuclastinib	KIT D816V	Advanced Systemic Mastocytosis (AdvSM)	NDA submission on track for 1H 2026		
	CGT1145	JAK2 V617F	Myeloproliferative Neoplasms	IND expected 2026		
	Undisclosed Targets					
ONCOLOGY	Bezuclastinib	KIT D816V	Gastrointestinal Stromal Tumors (GIST)	NDA submitted March 2026 under RTOR		
	CGT4255	ErbB2	Breast Cancer, NSCLC	Phase 1 dose escalation ongoing		
	CGT6297	PI3K α	Breast Cancer	Phase 1 dose escalation ongoing		
	CGT1815	pan-KRAS	Solid Tumors	IND expected 2026		
	Undisclosed Targets					

Bezuclastinib: Compelling Long-Term Exclusivity Expected Through 2046



PATENT EXCLUSIVITY Orange Book listable patents

- Compound patent**
Original expiration date: **January 2034**
- Patent term extension**
Expected expiration date: **January 2039**
- Formulation patent** (pending non-provisional application)
Expected expiration date: **December 2043**
- Method of administration patent** (pending provisional application)
Expected expiration date: **December 2046**

REGULATORY EXCLUSIVITY No ANDAs approved by FDA

- New chemical entity (NCE) exclusivity**
5 year: **2026-2031**
- Orphan drug exclusivity (ODE)**
7 year: **2026-2033**



BEZUCLASTINIB in Gastrointestinal Stromal Tumors



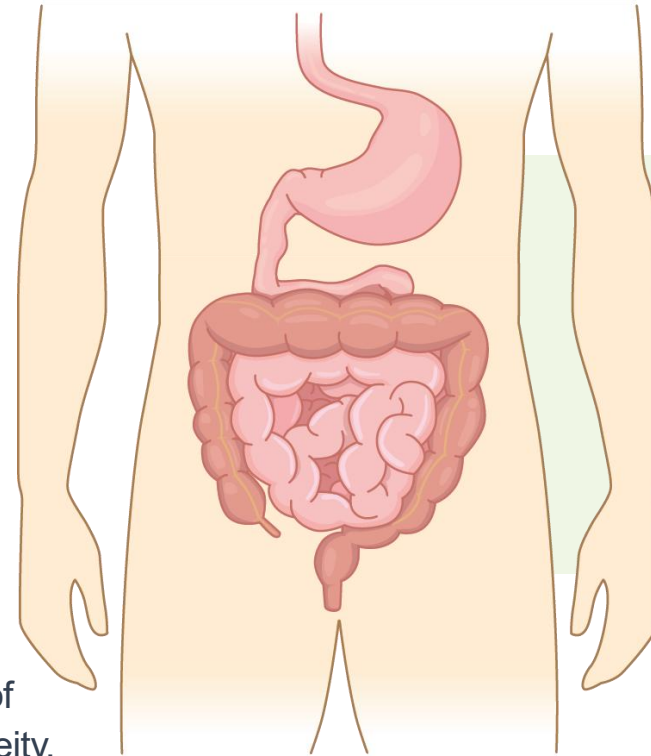
Significant Unmet Need Remains for Patients with Gastrointestinal Stromal Tumors (GIST)

Up to 12,000 GIST cases diagnosed annually in US and EU, over 80% of which express KIT mutations, typically exons 11 and 9.^{1,2}

Tumors can start anywhere in the GI tract, but they occur most often in the **stomach (about 60%)** or the **small intestine (about 35%)**.¹

While **imatinib provides disease control in the majority of patients** in the 1L setting, ~60% of patients with GIST develop resistance within 2 years, primarily due to mutations in exon 13/14 and/or exon 17/18.^{1,2}

Additional FDA-approved sequential lines of therapy include sunitinib, regorafenib, and ripretinib; however, each is only effective against a subset of resistance mutations and disease progression results from clonal heterogeneity.



Symptoms³

Diarrhea, Nausea, Vomiting, Abdominal pain, Bloating, Gastroesophageal reflux disease, GI bleeding, Loss of appetite, Weight loss

¹ Key statistics for gastrointestinal stromal tumors. American Cancer Society.

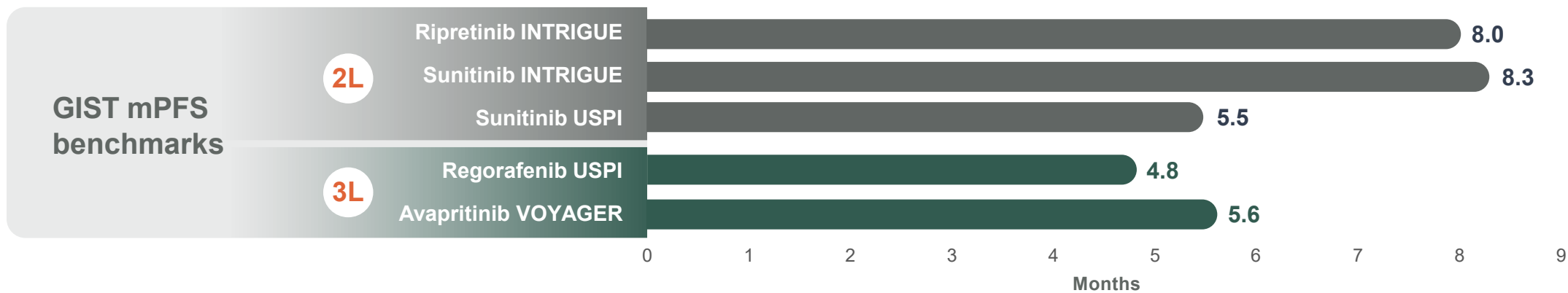
² Gramza AW, Corless CL, Heinrich MC., 2009.

³ Signs and symptoms of gastrointestinal stromal tumors. American Cancer Society. L: Line of Therapy

Unmet Medical Need Remains for Patients with Imatinib-Resistant or Intolerant GIST



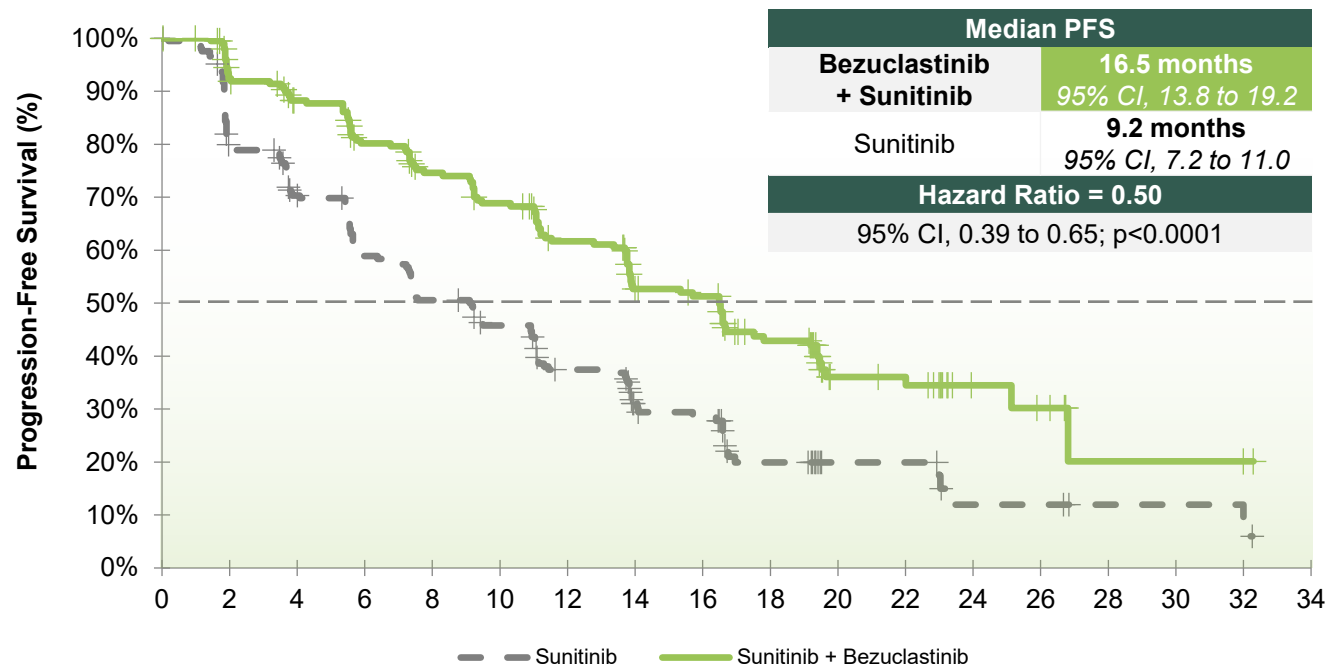
Modest historical performance of novel agents in imatinib-resistant setting emphasizes unmet need



ORR/PFS for all approved agents was obtained from labeled information from those agents

Bezuclastinib Demonstrated Unprecedented Efficacy in 2L GIST

Bezuclastinib Combination Extends PFS with 50% Reduction in Risk of Progression or Death



Patients at Risk

Months

Bezuclastinib + Sunitinib	204	180	166	146	130	119	102	76	72	51	24	23	8	6	2	2	2	0
Sunitinib	209	161	137	113	97	85	64	39	35	19	9	9	4	4	2	2	2	0

Bezuclastinib + Sunitinib Demonstrates Unprecedented 46% Objective Response Rate

ORR per BICR, %, n [95% CI]	
Bezuclastinib + Sunitinib (n=204)	45.6% 93 [38.6, 52.7]
Sunitinib (n=209)	25.8% 54 [20.0, 32.3]
Difference in ORR, % [95% CI]; P Value	
19.8 [10.6, 28.6]; P Value < 0.0001	

BOR per BICR, n (%)	Bezuclastinib + Sunitinib	Sunitinib
Complete Response (CR)	13 (6.4)	4 (1.9)
Partial Response (PR)	80 (39.2)	50 (23.9)
Stable Disease (SD)	91 (44.6)	108 (51.7)
Progressive Disease (PD)	15 (7.4)	41 (19.6)
Not Evaluable (NE)	5 (2.5)	6 (2.9)

Bezuclastinib + Sunitinib is Generally Well Tolerated with a Favorable Safety Profile

	Study Treatment	
	Bezuclastinib + Sunitinib (n=204)	Sunitinib (n=208) ¹
TEAEs, n (%)	204 (100)	207 (99.5)
TRAEs, n (%)	202 (99.0)	204 (98.1)
Gr3+ TRAEs, n (%)	146 (71.6)	109 (52.4)
Bezuclastinib related Gr3+	126 (61.8)	N/A
Sunitinib related Gr3+	141 (69.1)	109 (52.4)
SARs, n (%)	34 (16.7)	24 (11.5)
Bezuclastinib related SAEs	25 (12.3)	N/A
Sunitinib related SAEs	31 (15.2)	24 (11.5)
TRAEs leading to death, n (%)	0	1 (0.5)
Reductions of either drug due to TRAEs, n (%)	114 (55.9)	92 (44.2)
DC of study treatment due to TRAEs, n (%)	15 (7.4)	8 (3.8)

- The incidence of TEAEs and TRAEs was similar between treatment arms
- No TRAEs leading to death in patients on bezuclastinib + sunitinib combination
- Only TRAEs leading to discontinuation of either drug in >1 patient on the combination arm were neutropenia (2.9%), ALT/AST increased (1.5%), and diarrhea (1%)

Randomized Period Data; 1: One patient randomized to sunitinib but never dosed

Data cut-off as of 30Sep2025; SAE: serious adverse event; TEAE: treatment-emergent adverse event; TRAE: treatment-related adverse event; SAR: serious adverse reaction; DC: discontinuation

PEAK Results Dramatically Change 2nd-line GIST Commercial Potential Given Estimated Average Duration of Treatment

Large Population with Unmet Need



US + EU: 12,000 GIST patients diagnosed annually

~85% KIT driven

~60% imatinib resistant within 2 years



~6,000

2nd-line GIST patients annually

Approved KIT Inhibitors Monthly Price Benchmark*

QINLOCK[®]
(ripretinib) 50 mg tablets

\$44,370

AYVAKIT[®]
avapritinib | tablets

\$40,837

PEAK Results Show Dramatic Improvement in Duration of Treatment (DOT)

>19+ months
(est. mean duration treatment)

2nd-line GIST
Total Available Market

\$4 Billion+
Global

* Price listed 30-day supply U.S. WAC pricing

PEAK Results are Transformative and Expected to be Practice Changing

Bezuclastinib combination establishes first new benchmark for 2L GIST in 20 years

50% reduced risk of progression or death compared to current standard of care

16.5 months mPFS compared to 9.2 months for sunitinib alone ($p < 0.0001$)

46% ORR compared to 26% for sunitinib alone ($p < 0.0001$)

OS immature with event rate of **less than 20%** at time of PFS analysis



Generally well tolerated with **no unique risks observed** when compared to the known safety profile of sunitinib

Estimated **19 months+ mean treatment duration** for bezuclastinib combination patients based on projection for patients remaining on combination therapy

Active **Expanded Access Program** allowing **immediate availability** of the bezuclastinib combination for 2L patients with GIST

NDA submitted under RTOR in March 2026 for bezuclastinib in imatinib-resistant or intolerant GIST

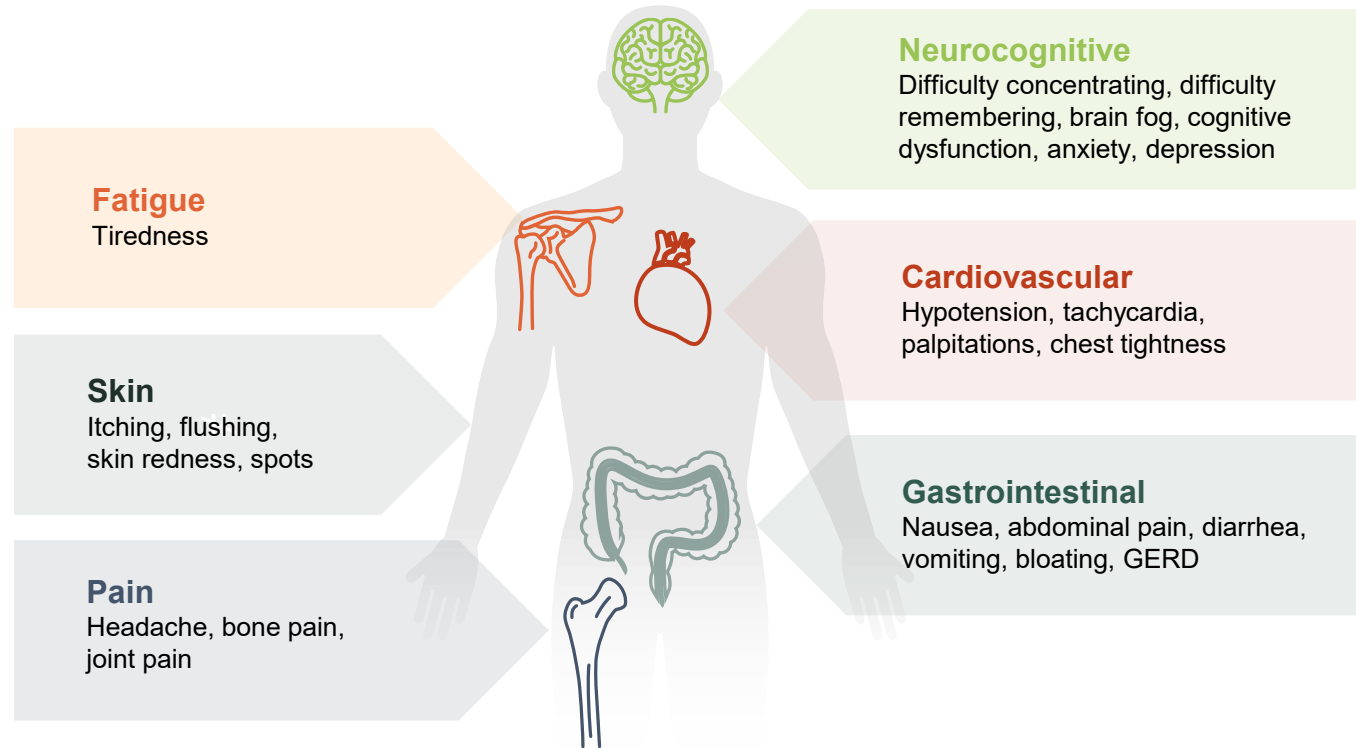
See Additional Data Here →

BEZUCLASTINIB in Systemic Mastocytosis



Bezuclastinib is an oral, potent, and selective type 1 TKI with activity against *KIT* p.D816V, the activating mutation in most patients with systemic mastocytosis¹⁻⁵

Symptoms of NonAdvSM



- Nonadvanced SM (NonAdvSM), including indolent SM, smoldering SM, and bone marrow mastocytosis subtypes, is the most prevalent form of SM^{3,6,7}
- NonAdvSM can be associated with debilitating symptoms, including life-threatening anaphylaxis, which can significantly impair quality of life^{5,8,9}
- Bezuclastinib is highly active against *KIT* p.D816V, has minimal brain penetration and spares closely related kinases, which may minimize off-target toxicities, such as bleeding, cognitive impairment, edema, and pleural effusion^{1,2,10}

GERD, gastroesophageal reflux disease; SM, systemic mastocytosis; TKI, tyrosine kinase inhibitor.

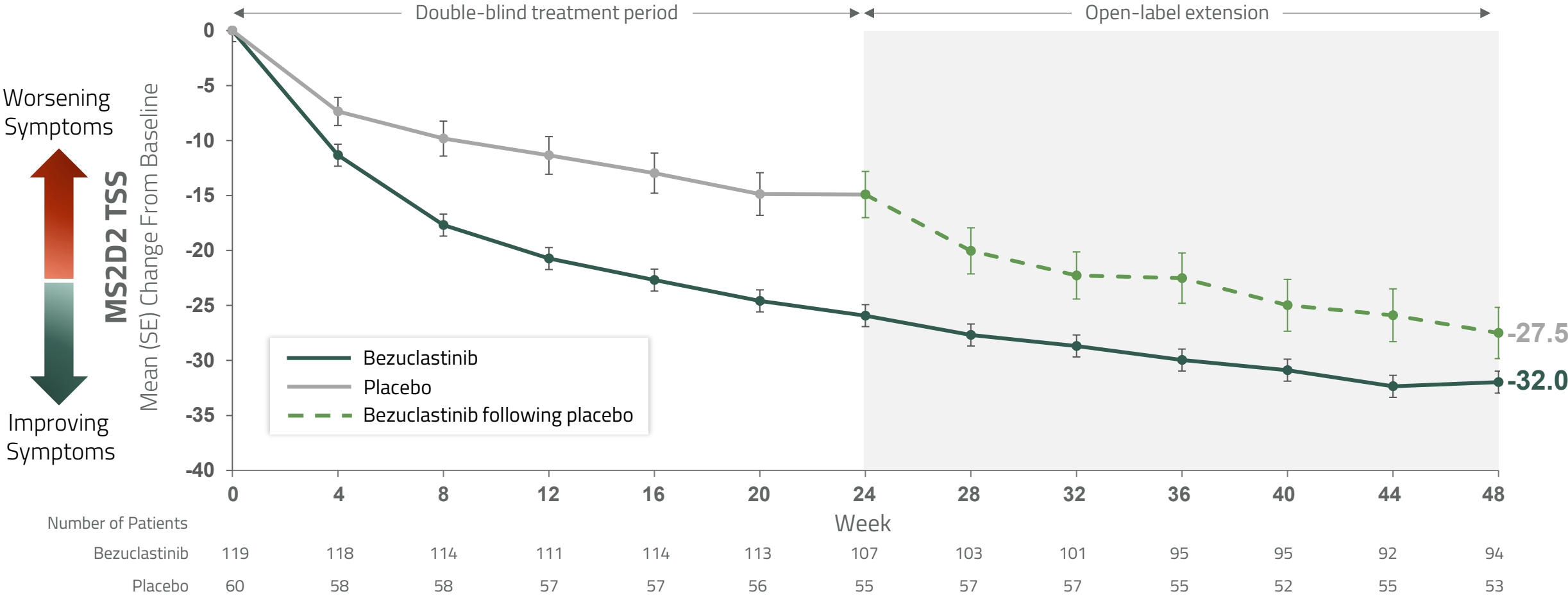
1. DeAngelo DJ, et al. *Hemasphere*. 2022; 6(suppl).
2. Guarnieri A, et al. Poster presented at: AACR Annual Meeting; April 8-13, 2022; Orleans, LA.
3. Ungerstedt J, et al. *Cancers*. 2022;14(16):3942. doi:10.3390/cancers14163942
4. Li JY, et al. *Cancers (Basel)*. 2023. 15(23).
5. Tse KY, et al. *J Allergy Clin Immunol Glob*. 2024.3(4):100316.
6. Scherber RM and Borate U. *Br J Haematol*. 2018;180:11-23.
7. Gilreath JA, et al. *Clin Pharmacol*. 2019;11:77-92.
8. Pardananani A. *Am J Hematol*. 2021;96(4):508-25.
9. Piris-Villaespesa M and Alvarez-Twose I. *Front Pharmacol*. 2020;11(443): doi: 10.3389/fphar.2020.00443.
10. Das A, et al. *Crit Rev Oncol Hematol*. 2021;157:103186.

Minimal Late-Stage Competitive Activity with Clear Path to Best-in-Class Position

		Systemic Mastocytosis		
		avapritinib	elenestinib	bezuclastinib
	D816V potent	✓	✓	✓
	CNS selective	✗	✓	✓
	PDGFR sparing	✗	✗	✓
	FLT3/CSF1R sparing	✗	✗	✓
Active trials	ASM	Approved	Phase 1/2	NDA Submission on track for 1H26
	NonAdvSM	Approved	Phase 2/3	NDA Accepted; 12/30/26 PDUFA

Rapid, durable, and clinically meaningful symptom improvement that continues to deepen to 48 weeks of treatment

Patients crossing over to receive active treatment experience clinically meaningful symptom improvement



Number of Patients

Bezuclastinib	119	118	114	111	114	113	107	103	101	95	95	92	94
Placebo	60	58	58	57	57	56	55	57	57	55	52	55	53

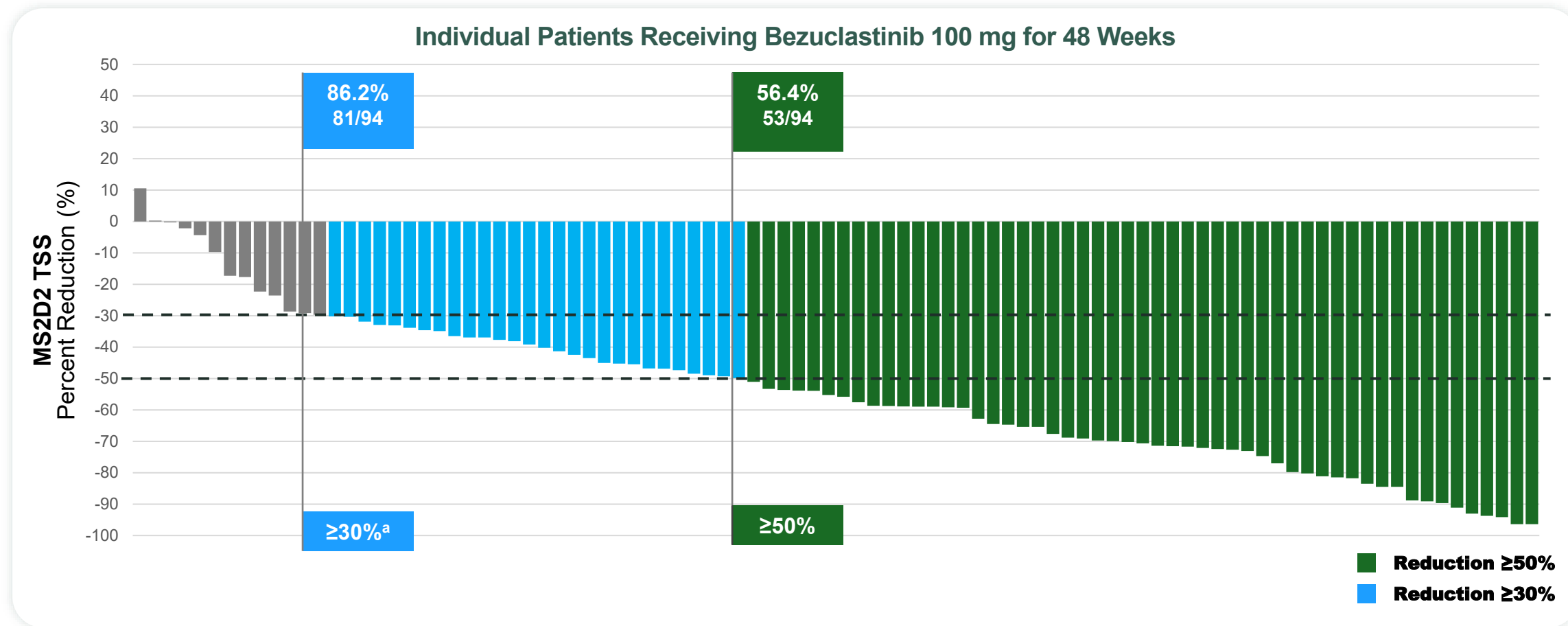
Data cutoff: Nov 7, 2025.

MS2D2, mastocytosis symptom severity daily diary; TSS, total symptom score.



Bezuclastinib achieves robust symptom reduction at 48 weeks

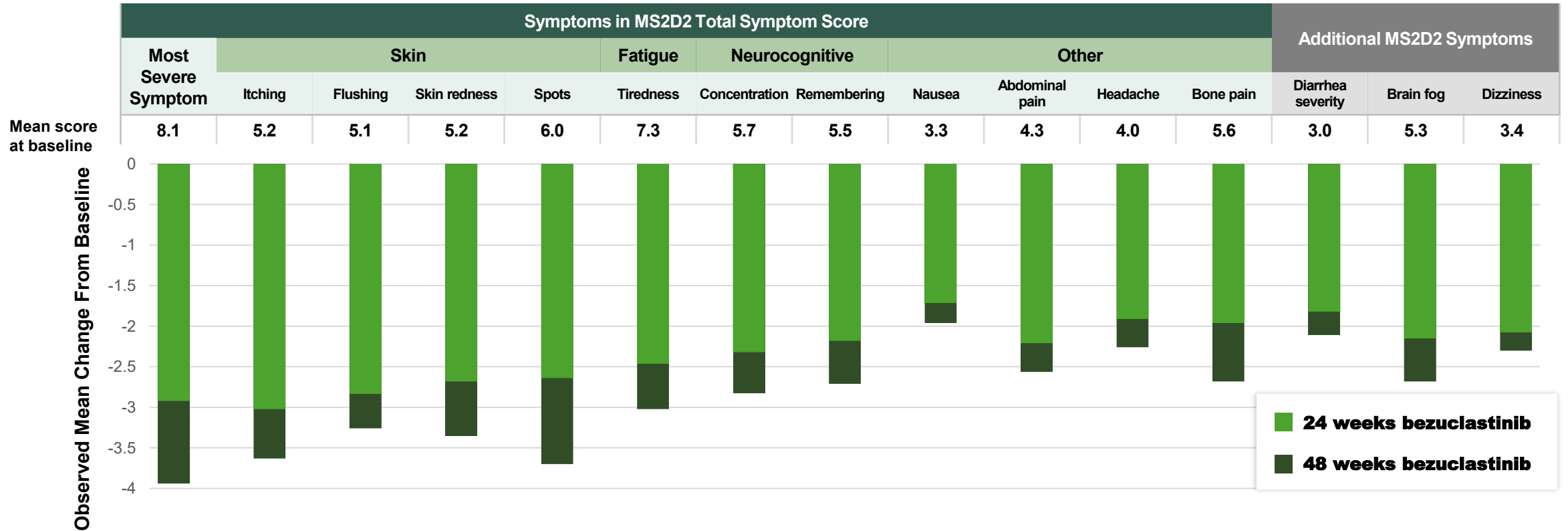
86% of Patients Achieve Clinically Meaningful Symptom Improvement



Data cutoff: Nov 7, 2025.

^aSymptom reductions of ≥30% represent clinically meaningful change as determined by anchor-based analyses.

Bezuclastinib achieves sustained and deepening symptom improvement through 48 weeks in NonAdvSM



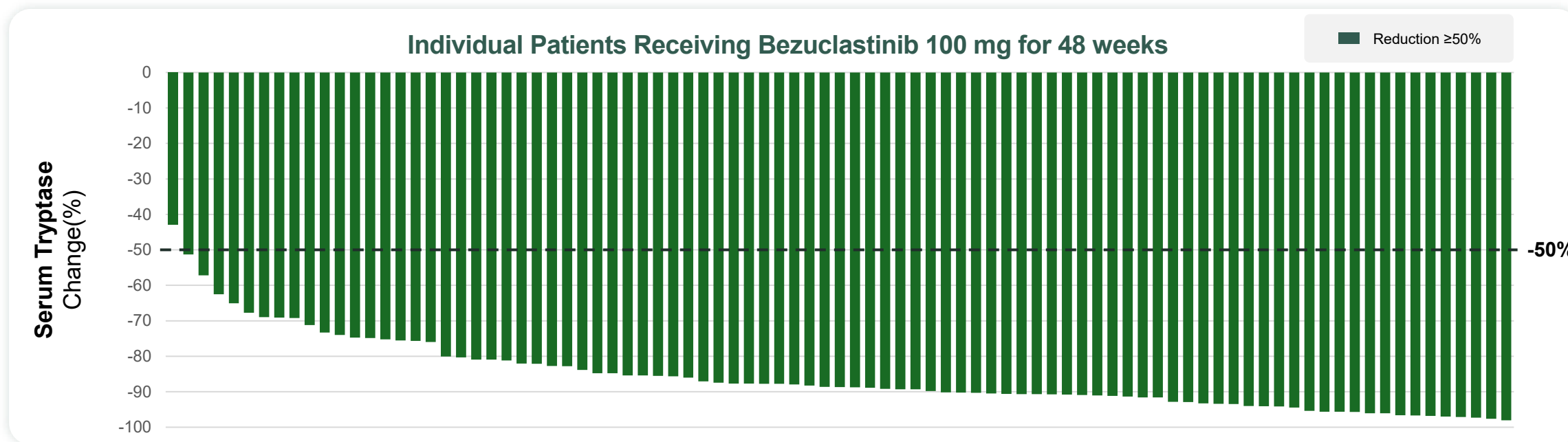
Data cutoff: Nov 7, 2025.

- All symptoms have deepening of improvement to 48 weeks of treatment with bezuclastinib
- At week 48, 50% of those in the bezuclastinib arm had a dose reduction or discontinuation of BSC medication

MS2D2, mastocytosis symptom severity daily diary; TSS, total symptom score.

99% of patients reached $\geq 50\%$ reduction in serum tryptase at 48 weeks

Serum tryptase normalization persisted to 48 weeks of bezuclastinib treatment



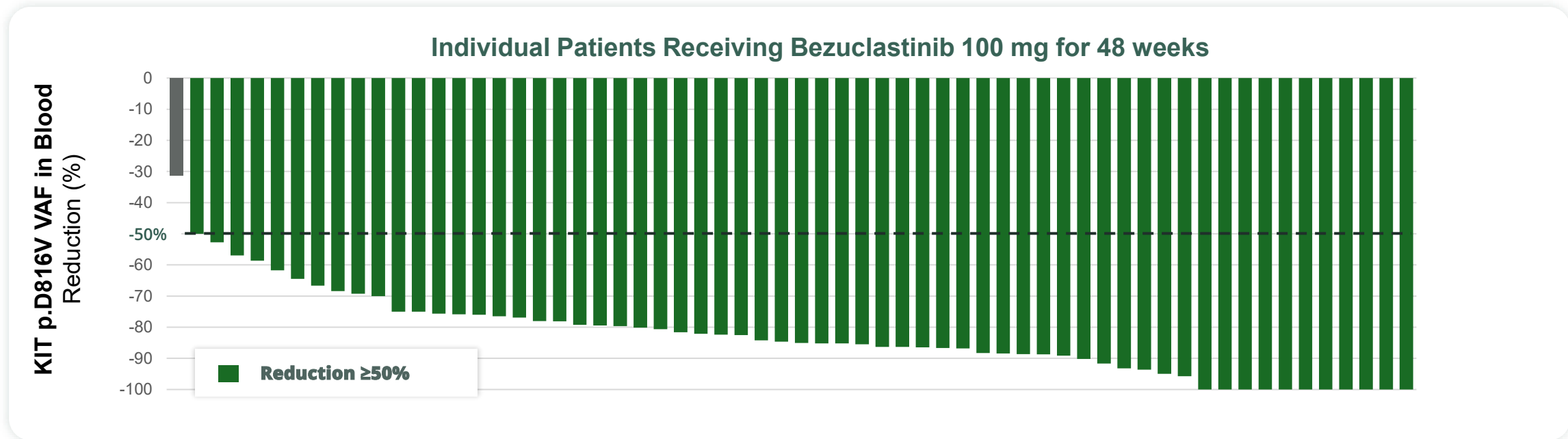
	Week 48	
	Bezuclastinib N=89	Placebo → Bezu N=60
Median serum tryptase, ng/mL (range)	4.7 (1.0-128.0)	5.8 (1.1-143.0)
Patients reaching $\geq 50\%$ reduction in serum tryptase, % (n/N) ^a	98.9% (88/89)	96.1% (49/51)
Patients achieving threshold ^b , % (n/N)		
<20 ng/mL	91.7 (66/72)	90.5 (38/42)
<11.4 ng/mL	83.3 (70/84)	77.6 (38/49)

- At week 48, the majority of placebo crossover patients achieved $\geq 50\%$ serum tryptase reductions and normalization within 24 weeks of bezuclastinib treatment

Data cutoff: Nov 7, 2025.

^aPatients with data at 48 weeks. ^bOf patients with baseline serum tryptase above threshold. ITT, intention to treat.

Bezuclastinib significantly reduced *KIT* p.D816V variant allele frequency, a molecular driver of disease, at 48 weeks



		Week 48	
		Bezuclastinib	Placebo → Bezu
Median <i>KIT</i> p.D816V VAF in blood, % (range)		0.04 (0-10.5)	0.07 (0-12.1)
Patients reaching ≥50% reduction in <i>KIT</i> p.D816V VAF in blood ^b or undetectable mutation, % (n/N)	ITT population^c	98.4 (61/62)	100 (37/37)

Data cutoff: Nov 7, 2025.

- At week 48, placebo crossover patients achieved significant reductions in *KIT* p.D816V within 24 weeks of bezuclastinib treatment

^aPatients with data at 48 weeks. ^bLimit of detection equals 0.03%.
ITT, intention to treat; VAF, variant allele frequency.

Bezuclastinib was well tolerated and no new safety concerns observed with longer-term treatment

TEAEs during OLE	Placebo → Bezuclastinib (n=58)	Bezuclastinib (n=103)
Median (range) duration of bezuclastinib treatment (months) in the OLE	8.8 (2.8 – 14.4)	8.6 (0.46 – 15.0)
TEAEs, n (%)	57 (98.3)	92 (89.3)
Serious TRAEs, n (%)	1 (1.7)	0
Reductions due to TRAEs, n (%)	9 (15.5)	2 (1.9)
DCs due to TRAEs, n (%)	3 (5.2)	1 (1.0)

TEAEs ≥10% either arm, n (%)				
Preferred Term	All Grade	Grade ≥ 3	All Grade	Grade ≥ 3
Hair color changes	37 (63.8)	-	11 (10.7)	-
ALT/AST increased ^a	22 (37.9)	5 (8.6)	10 (9.7)	1 (1.0)
Altered taste ^a	12 (20.7)	-	2 (1.9)	-
Dizziness	9 (15.5)	-	10 (9.7)	-
Nausea	8 (13.8)	-	5 (4.9)	-
Dyspepsia	8 (13.8)	-	5 (4.9)	-
Headache	8 (13.8)	-	9 (8.7)	-
URTI	8 (13.8)	-	7 (6.8)	-
Fatigue	8 (13.8)	2 (3.4)	2 (1.9)	-
Diarrhea	7 (12.1)	-	8 (7.8)	-
ALP increased	7 (12.1)	-	1 (1.0)	-
Arthralgia	6 (10.3)	-	10 (9.7)	1 (1.0)
Insomnia	6 (10.3)	-	4 (3.9)	-

- The majority of TEAEs were low grade and reversible
- Overall median (range) duration of treatment in patients continuing bezuclastinib in the OLE was 13.4 (0.67–20.5) months
- Consistent safety and tolerability profile observed in OLE for patients crossing over to bezuclastinib, similar to the previously reported 24-week experience
- The only hepatic events reported were transient and manageable lab abnormalities, similar to the previously reported 24-week experience
- Discontinuations due to TRAEs remained limited, consistent with the previously reported 24-week experience; all were due to transaminase elevations and all events fully resolved

Data cutoff: Nov 7, 2025.

Only TEAEs that occurred during OLE on bezuclastinib treatment are included. Adverse events per NCI CTCAE v5.0.

^aIncludes pooled terms.

ALT, alanine aminotransferase; AST, aspartate aminotransferase; DC, discontinuation; TEAE, treatment emergent adverse event; TRAE, treatment related adverse event.

Bezuclastinib represents a promising new treatment for patients with NonAdvSM

Disease modification and symptom improvement continue out to 48 weeks of treatment

Rapid, statistically significant, and **clinically meaningful symptom improvement** is durable and continues to deepen to 48 weeks of treatment

- Placebo patients crossing over to receive bezuclastinib experience clinically meaningful symptom improvement

86% of patients achieve clinically meaningful symptom improvement

- All symptoms have deepening of improvement to 48 weeks of treatment with bezuclastinib
- At week 48, 50% in the bezuclastinib arm had a dose reduction or discontinuation of BSC medication



Safety and tolerability profile in OLE, with > 24 weeks and up to 48 weeks of treatment, supports potential for chronic dosing

NDA for broad NonAdvSM population accepted; assigned 12/30/26 PDUFA BTD granted by the FDA for patients with NonAdvSM previously treated with avapritinib and with smoldering SM

Bezuclastinib Systemic Mastocytosis Expanded Access Program (NCT06915766) is currently open to requests from treating physicians in the United States

[See Additional Data Here](#) →



Advanced Systemic Mastocytosis



APEX Part 2: Patients Receiving 150mg Bezuclastinib Achieved Positive Results on Primary (mIWG) and Key Secondary (PPR) Endpoints

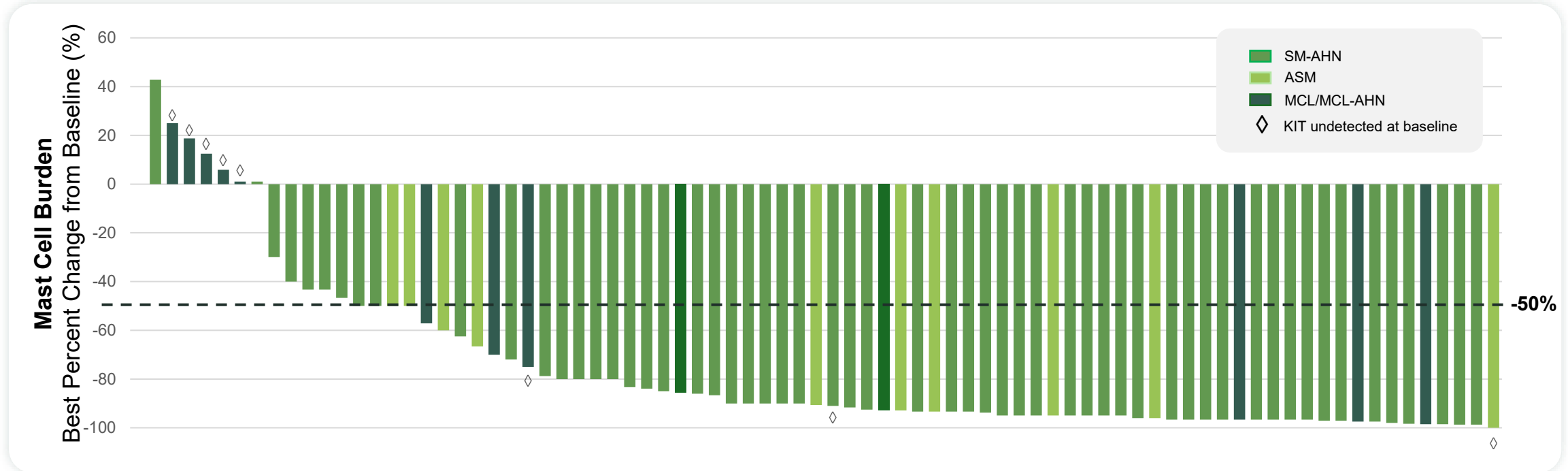
	Best ORR per mIWG, n (%) N=68	Best PPR, n (%) N=81
Overall Response Rate		
CR+CRh+PR+CI	39 (57.4)	-
CR+CRh+PR	33 (48.5)	65 (80.2)
Best Overall Response		
Complete Response (CR+CRh)	9 (13.2)	46 (56.8)
Molecular CR/CRh	-	18 (22.2)
Partial Response (PR)	24 (35.3)	19 (23.5)
Clinical Improvement (CI)	6 (8.8)	-
Stable Disease (SD)	25 (36.8)	15 (18.5)
Progressive Disease (PD)	2 (2.9)	0
Not Evaluable (NE)	2 (2.9)	1 (1.2)

At time of data cut-off, multiple ongoing patients had unconfirmed responses

Data cutoff: May 22, 2025.

Median Duration of Treatment: 9.4 months

APEX Part 2: Bezuclastinib Significantly Reduced Objective Measures of Disease Burden Including Mast Cells, Serum Tryptase, and KIT D816V VAF



	Serum Tryptase N=80 ^a	MC Burden N=80 ^a	KIT D816V VAF N=43 ^b
Best percent change from baseline, mean	-83.4%	-75.5%	-77.3%
Patients reaching $\geq 50\%$ reduction from baseline or defined threshold ^c	89%	89%	91%

Data cutoff: Sept 19, 2025.

^aIncludes patients who had at least one post baseline assessment.

^bIncludes patients who had detectable VAF at baseline and one post baseline assessment.

^cThreshold: for mast cell burden is clearance of MC aggregates and for KIT D816V VAF is reaching undetectable level of KIT (limit of detection is 0.03%).

APEX Part 2: Bezuclastinib was Well Tolerated with a Favorable Safety Profile

150mg QD Bezuclastinib N=81		
TRAEs, n (%)	75 (92.6)	
Drug-related SAEs, n (%)	5 (6.2)	
Reductions due to TRAEs, n (%)	12 (14.8)	
DCs due to TRAEs, n (%)	0	
TRAEs in ≥ 10% of patients; Preferred term, n(%)		
	Any grade	Grade ≥ 3
Hematological events, n (%)		
Neutropenia*	24 (29.6)	19 (23.5)
Thrombocytopenia*	20 (24.7)	11 (13.6)
Anemia	13 (16.0)	8 (9.9)
NON- HEMATOLOGICAL EVENTS, n (%)		
Hair color changes	25 (30.9)	0
Altered taste*	23 (28.4)	0
ALT/AST increased*	17 (21.0)	1 (1.2)
Blood ALP increased	11 (13.6)	4 (4.9)
Diarrhea	12 (14.8)	0
Nausea	12 (14.8)	1 (1.2)
Alopecia	9 (11.1)	0
Peripheral edema	9 (11.1)	0

- Hematological events were reversible and manageable
- Treatment related hepatic events were transient and manageable lab abnormalities
- Dose reductions were primarily due to hematological events; no other adverse event led to dose reduction in more than one patient
- No discontinuations due to treatment-related AEs
- No treatment-related deaths were reported

Data cutoff: Sept 19, 2025.

*Pooled terms

Bezuclastinib Represents a Promising New Treatment Option for ASM Patients

Patients receiving bezuclastinib achieved **high rates of response:**

- 57% ORR (CR+CRh+PR+CI) per mIWG-MRT-ECNM
- 80% ORR (CR+CRh+PR) per PPR

Significant reductions in objective disease markers underscore potent target engagement and impact on KIT-driven disease pathology:

- Serum tryptase: ↓ ≥50% in 89% of patients
- Bone marrow mast cell burden: ↓ ≥50% in 89% of patients
- KIT p.D816V variant allele frequency: ↓ ≥50% in 91% of patients



Bezuclastinib was well-tolerated, with infrequent need for dose reduction and no patients requiring discontinuation for treatment related adverse events

- Only 1 patient experienced Gr 3 AST/ALT, and remains on study following dose reduction
- Encouraging safety profile potentially allows for concomitant treatment in patients who require other cytoreductive therapies for AHN or post-transplant

NDA submission for AdvSM population is expected in 1H 2026

[See Additional Data Here](#) →

Significant Market Opportunity in Systemic Mastocytosis With Bezuclastinib's Clinical Profile

Promising Growth Trends in SM Management

~40%

Growth in TKI Utilization in SM*



In Testing and Diagnostics



Patients Diagnosed

Limited Competition Creates Unmet Market Needs

\$3.5B - NonAdvSM: AYVAKIT has suboptimal therapeutic dosing and is often used off label

\$500M - AdvSM: AYVAKIT has significant safety concerns including risk of intracranial hemorrhage

Large Market Opportunity Across Major Markets

**\$4 Billion+
Global**

Annual Market Opportunity

SUMMIT and APEX results together position bezuclastinib as best-in-class option for SM patients

*Internal Open and Closed Claims Analysis in SM



Best-in-Class Research Pipeline



Cogent Pipeline Highlight: Pan-KRAS Inhibitor

Potency

Best-in-class across prevalent KRAS mutations

Selectivity

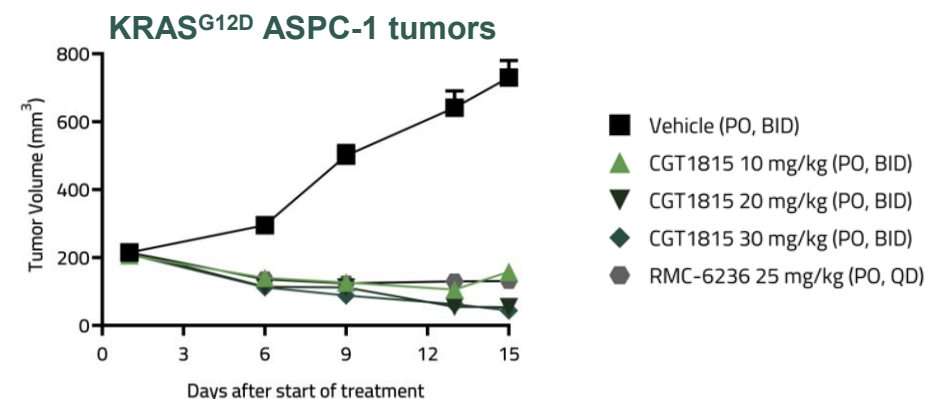
No inhibition of H/NRAS for potential improvement in tolerability vs. multi-RAS inhibitors

Drugability

Pro drug provides QD / low dose option for patients with excellent ability to combine

Cell Line	Tumor Type	KRAS Mutation	CGT1263 Cellular IC ₅₀	RMC-6236 Cellular IC ₅₀
MKN1	Gastric Cancer	Wild Type Amplified	0.65 nM	8.5 nM
NCI-H2009	Non-small cell lung cancer	G12A	0.11 nM	0.89 nM
NCI-H358	Non-small cell lung cancer	G12C	0.22 nM	0.30 nM
AsPC-1	Pancreatic cancer	G12D	0.24 nM	1.28 nM
A549	Non-small cell lung cancer	G12S	0.35 nM	0.65 nM
SW480	Colorectal cancer	G12V	0.37 nM	0.29 nM
HCT116	Colorectal cancer	G13D	1.0 nM	0.96 nM
NCI-H460	Non-small cell lung cancer	Q61H	0.30 nM	0.50 nM

- CGT1263 (the active form of CGT1815) showed pM/nM pERK inhibition across the spectrum of KRAS cell lines shown



Treatment	%TGI (Day 15)	Max %R
CGT1815 10 mg/kg (PO, BIDx14)	78	40 (D13)
CGT1815 20 mg/kg (PO, BIDx14)	93	75 (D13)
CGT1815 30 mg/kg (PO, BIDx14)	94	79 (D15)
RMC-6236 25 mg/kg (PO, QDx14)	82	39 (D15)

- CGT1815 showed superior tumor growth inhibition (>90%) at the 20 and 30 mg/kg BID PO dose levels compared to RMC-6236

Cogent Pipeline Highlight: JAK2 V617F Inhibitor

Selectivity

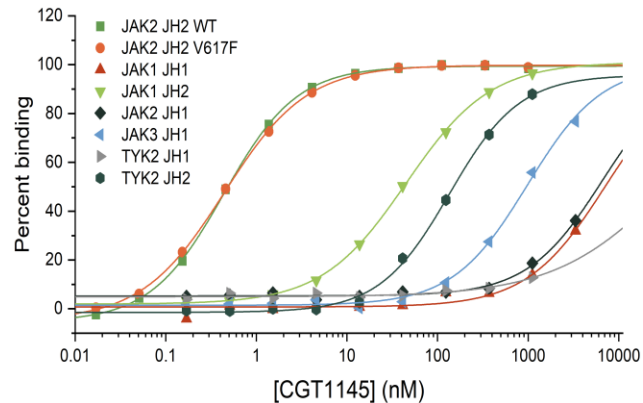
100+ fold selectivity in both binding and cellular assays for JAK2 V617F over JAK2 WT

↑ molecular response vs SOC agents
↓ thrombosis risk, ↓ fibrotic risk, and ↓ inflammation

Drugability

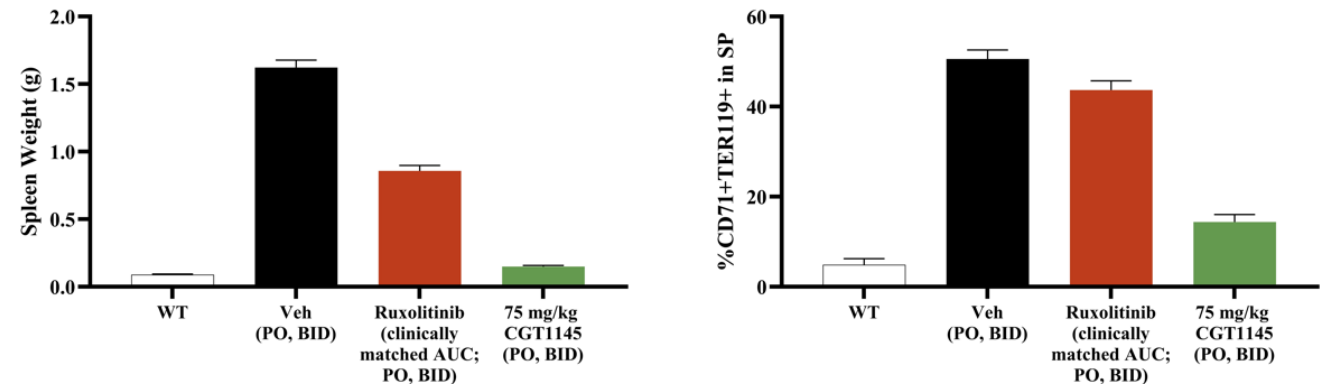
QD dose option for patients with excellent ability to combine

CGT1145 is Selective for the JAK2 JH2 Domain



CGT1145 binds to the JAK2 JH2 domain with sub-nM potency and is over 100x selective vs JAK1 JH2, JAK1/2/3 JH1, and TYK2 enzymes

Spleen Outcomes

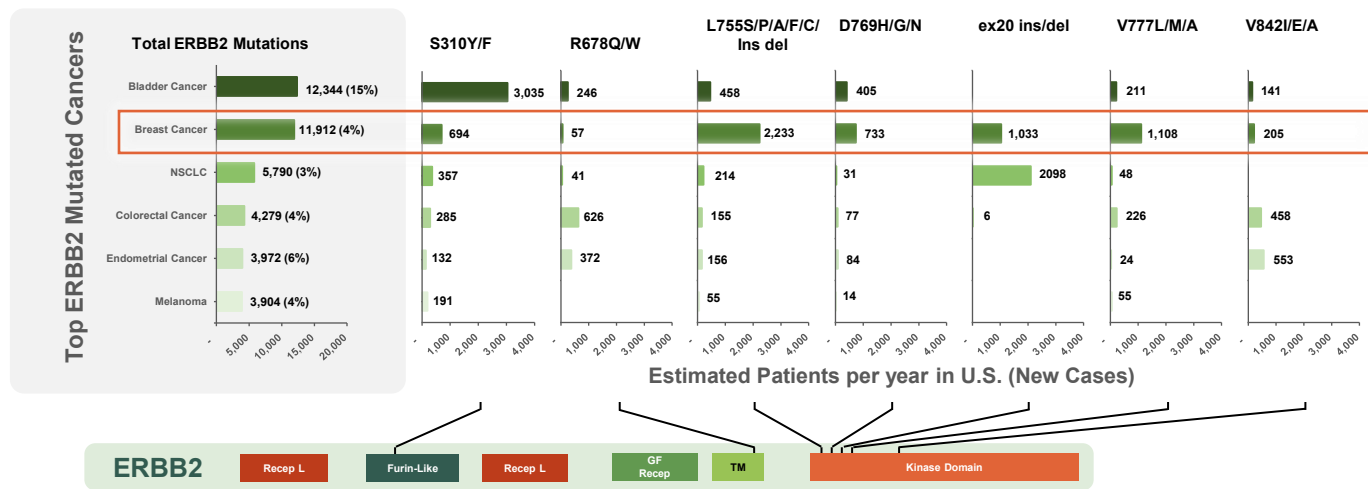


Treatment with CGT1145 led to normalization of spleen weight and decreased CD71+TER119+ erythroid precursor accumulation in the spleen, supporting the potential of CGT1145 as a disease-modifying therapy through restoration of bone marrow function and attenuation of extramedullary hematopoiesis

Creating a Best-in-Class CNS-penetrant, pan-mutant ErbB2 Inhibitor

CGT4255 is a highly potent and selective ErbB2 inhibitor targeting resistance (YVMA), kinase, and extracellular domain mutations, with best-in-class potential performance in multiple underserved patient populations

Prevalence of Oncogenic Mutations of ErbB2,5

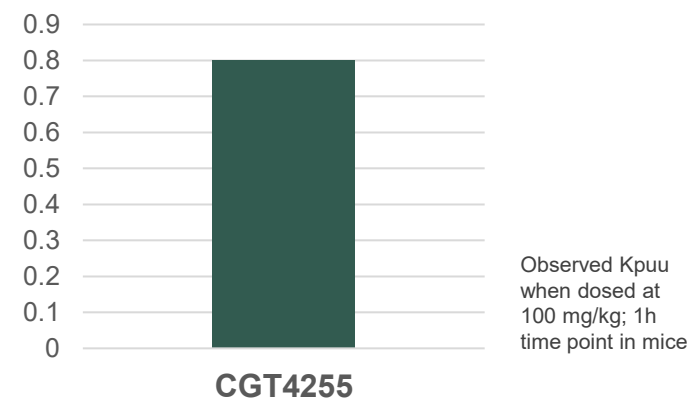


CGT4255

ErbB2	ErbB2 Cellular IC ₅₀ Inhibition of pErbB2				
	WT	L755S	YVMA	S310F	V842I
CGT4255	8 nM	9 nM	3 nM	7 nM	15 nM

Adjusted for FBS-binding

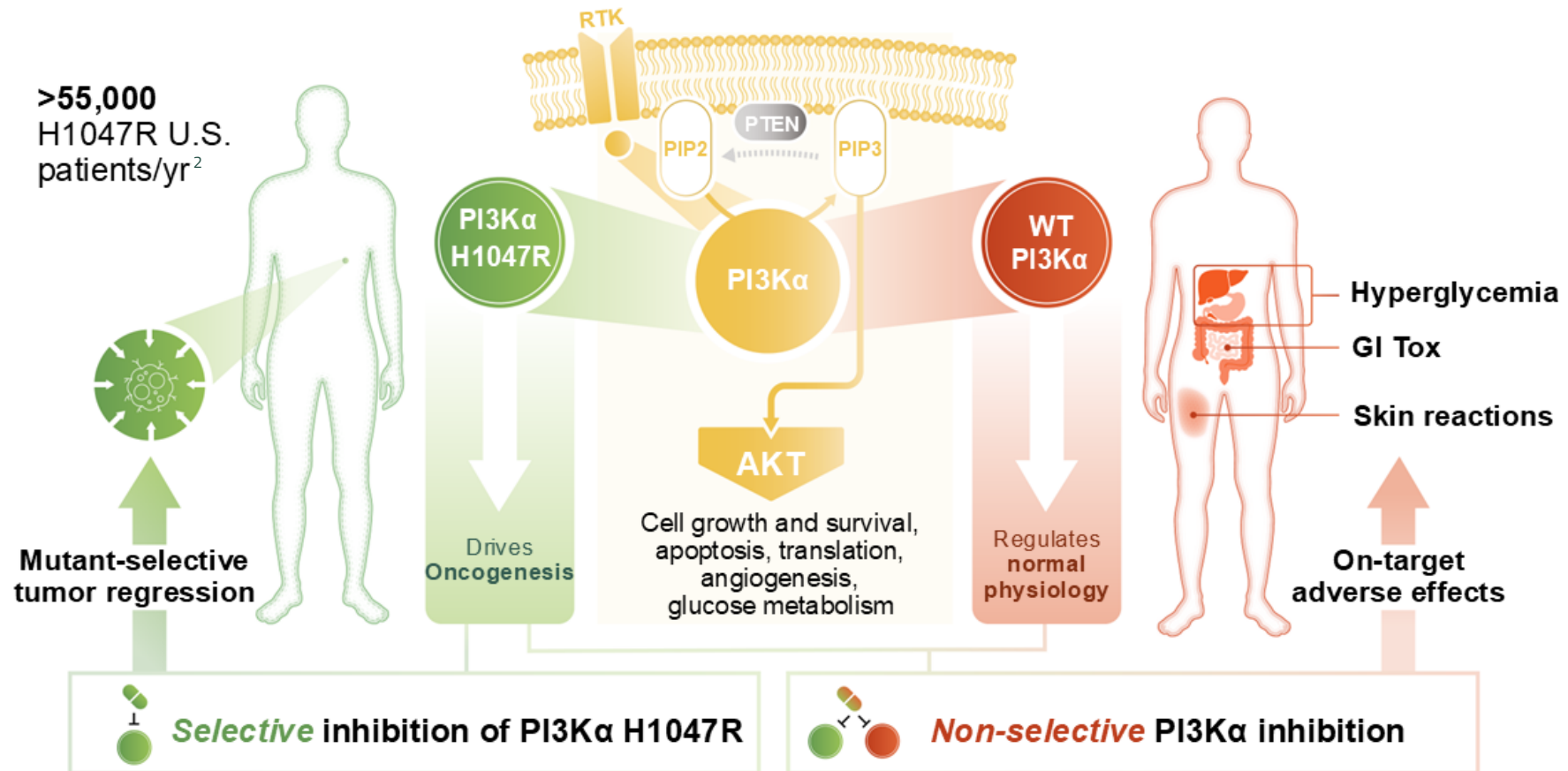
Brain Penetrance (K_{puu})



Outperform:

- Minimal shift across all relevant mutations, YVMA and ErbB2 wt isoforms
- Best in class potential CNS exposure
- Superior whole blood stability across ErbB2-covalent MOA/drug class
- Superior *in vitro* and *in vivo* performance vs. SOC- ex.~Tucatinib
- Ability to combine therapeutically with ADC, other TKIs and mAbs

Selective pan-mutant PI3K α Inhibition Avoids PI3K α Wild Type Toxicity for Improved Efficacy and Tolerability



- On-target inhibition of wild type PI3K α by approved inhibitors leads to tolerability issues including hyperglycemia, hyperinsulinemia, gastrointestinal issues, and skin reactions¹
- Increases in insulin result in activation of PI3K α in tumor cells and diminished efficacy¹

- A mutant selective inhibitor will avoid these toxicities resulting in better tolerability, greater target coverage, and improved efficacy compared to approved agents

1. Hanks, A. B.; Kaklamani, V.; Arteaga, C. L. Challenges for the Clinical Development of PI3K Inhibitors: Strategies to Improve Their Impact in Solid Tumors. *Cancer Discov.* 2019; 9: 482–491

2. The AACR Project GENIE Consortium. AACR Project GENIE: Powering Precision Medicine Through An International Consortium. *Cancer Discov.* 2017; 7: 818–831.

Cogent Biosciences: Major Catalysts Expected in 2026

Bezuclastinib

- Gain approval and successfully launch in NonAdvSM
- ✓ Complete GIST NDA submission (March 2026)
- ✓ Submit NonAdvSM NDA (12/30/26 PDUFA)
- Present detailed GIST data at ASCO, May 30, 2026
- Present detailed AdvSM data at EHA, June 13, 2026

Pipeline

- Submit IND for CGT1815; novel, selective pan-KRAS inhibitor
- Submit IND for CGT1145; novel, selective JAK2 V617F inhibitor
- Share clinical data from Phase 1 study for CGT4859, FGFR 2/3 inhibitor
- ✓ Dose escalation for CGT4255, CNS-penetrant ErbB2 inhibitor
- ✓ Dose escalation for CGT6297, novel, selective PI3K α inhibitor

Corporate

- Build highly capable US commercial team to support bezuclastinib launch
- Identify ex-US commercial partner(s) for bezuclastinib, including European go-to-market strategy

\$866M as of 3/31/26 expected to fund operations through US commercial launch into 2028



**Real Challenges.
Real Solutions.**

[Cogentbio.com](https://cogentbio.com)



APPENDIX



Peak Phase 3 Top-Line Results

Real Challenges. Real Solutions.

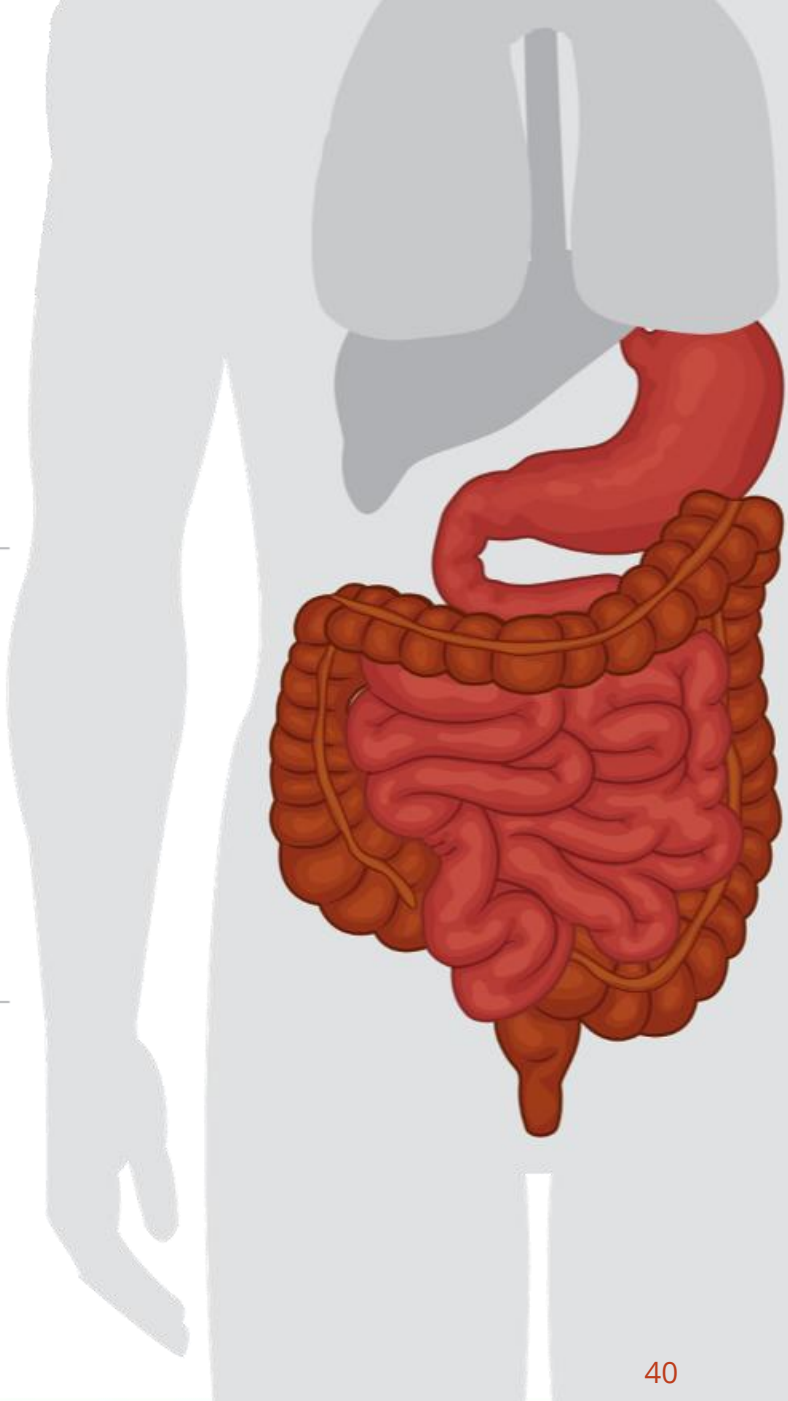
Precision therapies for genetically defined diseases

Significant Unmet Need Remains for Patients with Gastrointestinal Stromal Tumors (GIST)

- Up to 6,000 GIST cases diagnosed annually in US, over 80% of which express KIT mutations, typically exons 11 and 9.^{1,2}
- Tumors can start anywhere in the GI tract, but they occur most often in the stomach (about 60%) or the small intestine (about 35%).¹
- While imatinib provides disease control in the majority of patients in the 1L setting, ~60% of patients with GIST develop resistance within 2 years, primarily due to mutations in exon 13/14 and/or exon 17/18.^{1,2}
- Additional FDA-approved sequential lines of therapy include sunitinib, regorafenib, and ripretinib; however, each is only effective against a subset of resistance mutations and disease progression results from clonal heterogeneity.

Symptoms³

Diarrhea, Nausea, Vomiting,
Abdominal pain, Bloating,
Gastroesophageal reflux
disease, GI bleeding, Loss of
appetite, Weight loss



Combination of Bezuclastinib + Sunitinib Inhibits the Full Spectrum of Primary and Secondary Mutations

- No single TKI inhibits all KIT mutations.¹⁻¹¹
- The combination of bezuclastinib + sunitinib inhibits mutations in KIT exons 9, 11, 13, 14, 17, and 18, targeting the full spectrum of primary and secondary mutations relevant in advanced GIST.

Treatments	Exon 9	Exon 11	Exon 13	Exon 14	Exon 17	D816V	Exon 18
Imatinib	Moderate Inhibition	Strong Inhibition	No Inhibition	No Inhibition	No Inhibition	No Inhibition	No Inhibition
Regorafenib	Strong Inhibition	Strong Inhibition	No Inhibition	Strong Inhibition	Moderate Inhibition	No Inhibition	Strong Inhibition
Velzatinib (IDRX-42)	Strong Inhibition	Strong Inhibition	Moderate Inhibition	No Inhibition	Strong Inhibition	No Inhibition	Strong Inhibition
Ripretinib	Moderate Inhibition	Strong Inhibition	Moderate Inhibition	No Inhibition	Strong Inhibition	Strong Inhibition	Strong Inhibition
Sunitinib	Strong Inhibition	Strong Inhibition	Strong Inhibition	Strong Inhibition	No Inhibition	No Inhibition	No Inhibition
Bezuclastinib + Sunitinib	Strong Inhibition	Strong Inhibition	Strong Inhibition	Strong Inhibition	Strong Inhibition	Strong Inhibition	Strong Inhibition

No Inhibition
 Moderate Inhibition
 Strong Inhibition

¹ Plexikon. Data on file.

² Serrano C et al. Br J Cancer, 2019.

³ Evans EK et al. Sci Transl Med, 2017.

⁴ Trent J et al. CTOS [presentation]. 2020.

⁵ Smith P et al. AACR [poster]. 2018.

⁶ Wagner AJ et al. JAMA Oncol. 2021.

⁷ Serrano C and Fletcher O. Oncotarget, 2019.

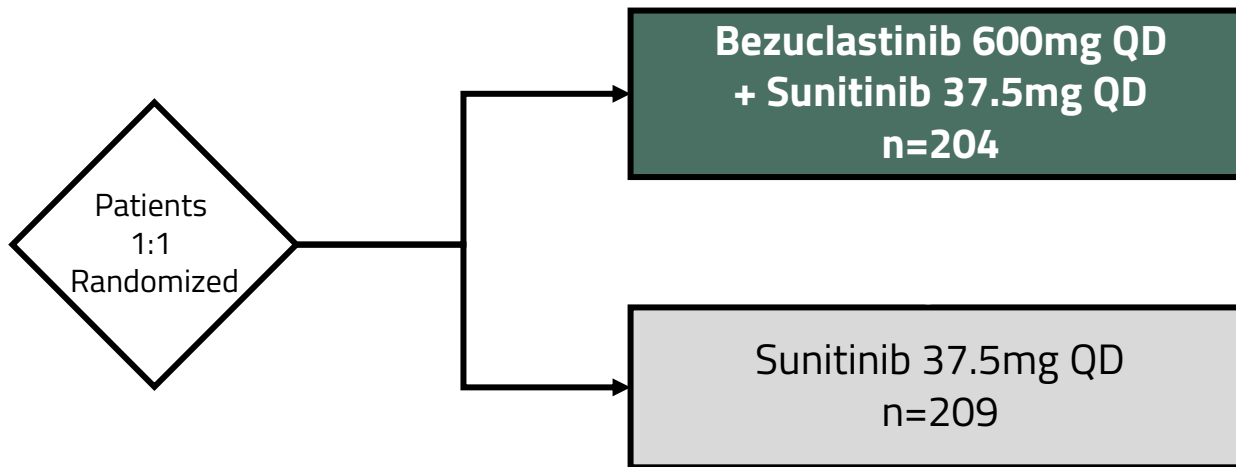
⁸ Muhlenberg T et al. J Clin Oncol, 2024.

⁹ Heinrich MC, et al. Nature Medicine, 2024.

¹⁰ Blum SM, et al. JMedChem, 2023.

¹¹ Wagner AJ et al. CTOS 2022.

Peak: Randomized Clinical Study Evaluating Bezuclastinib in Combination with Sunitinib in Patients with GIST



Crossover allowed following BICR confirmed PD

Patient Eligibility

- Age \geq 18 years
- Histologically confirmed GIST with at least 1 measurable lesion per mRECIST v1.1
- Locally advanced, unresectable or metastatic GIST
- Documented disease progression on or intolerance to imatinib

Primary Endpoint

- Progression Free Survival per BICR

Key Secondary Endpoints

- Objective Response Rate per BICR
- Overall Survival

Secondary Endpoints

- Progression Free Survival per Investigator
- Disease Control Rate
- Time to Response
- Duration of Response

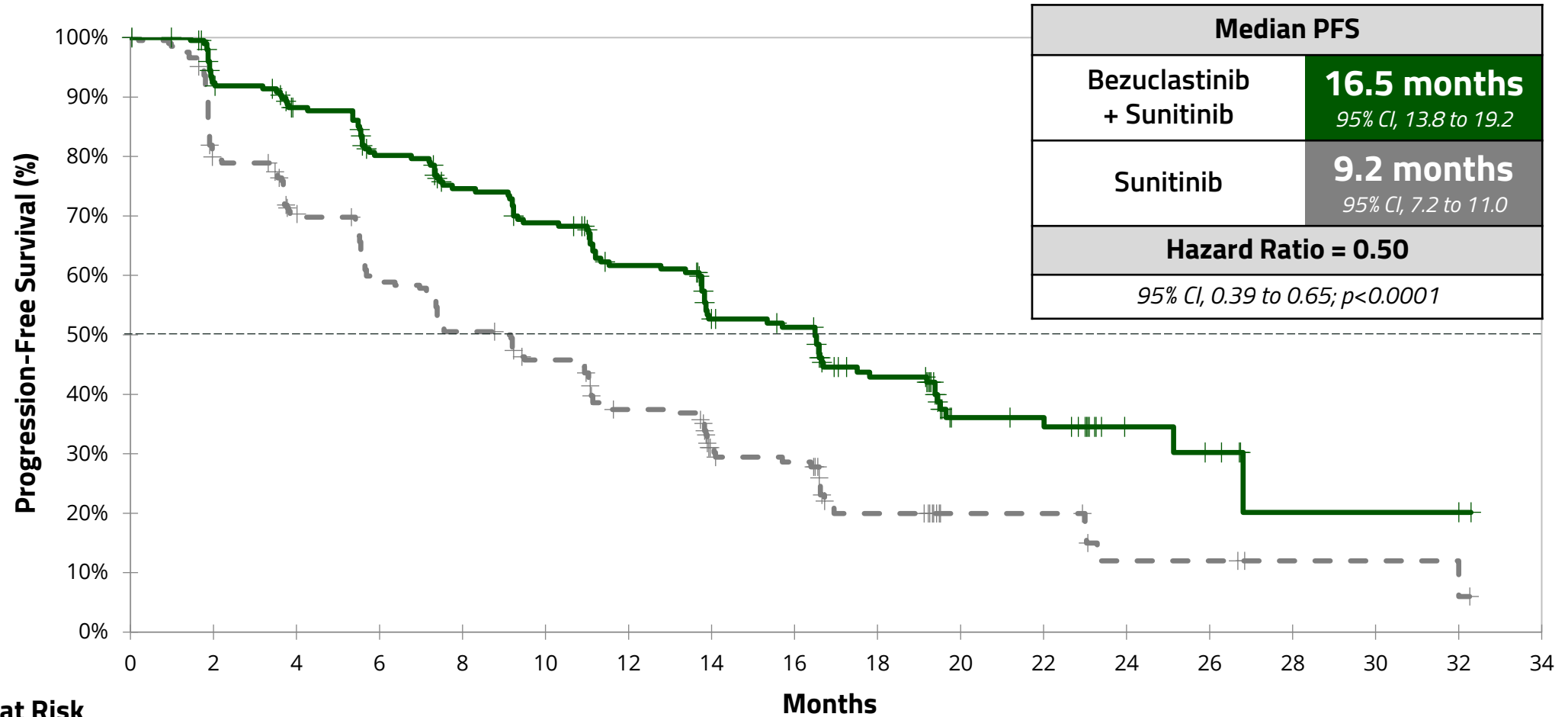
Peak Part 2 Population is Representative of Second-Line Patients with GIST

Patient Demographics	Bezuclastinib + Sunitinib	Sunitinib	Overall
# Patients	204	209	413
Male, n (%)	131 (64.2)	133 (63.6)	264 (63.9)
Median Age in years, (range)	63 (32 - 83)	64 (30 - 88)	63 (30 - 88)
ECOG PS at baseline, n (%)			
0	140 (68.6)	132 (63.2)	272 (65.9)
1	61 (29.9)	74 (35.4)	135 (32.7)
2	3 (1.5)	3 (1.4)	6 (1.5)

Region	Bezuclastinib + Sunitinib	Sunitinib	Overall
North America, n (%)	76 (37.3)	85 (40.7)	161 (39.0)
Europe, n (%)	94 (46.1)	94 (45.0)	188 (45.5)
Latin America, n (%)	20 (9.8)	11 (5.3)	31 (7.5)
Asia-Pacific, n (%)	14 (6.9)	19 (9.1)	33 (8.0)

Baseline Characteristics	Bezuclastinib + Sunitinib	Sunitinib	Overall
KIT Mutations per molecular pathology report, n (%)			
Mutation Detected			
Any Exon 9	31 (15.2)	34 (16.3)	65 (15.7)
Exon 11 only	120 (58.8)	126 (60.3)	246 (59.6)
Neither Exon 9 nor 11	10 (4.9)	11 (5.3)	21 (5.1)
Other	30 (14.7)	34 (16.3)	64 (15.5)
No KIT Mutation Detected	13 (6.4)	4 (1.9)	17 (4.1)
Treatment History			
Imatinib intolerance	6 (2.9)	8 (3.8)	14 (3.4)
Prior Radiotherapy	14 (6.9)	8 (3.8)	22 (5.3)
Prior Anti-Cancer Surgery	156 (76.5)	167 (79.9)	323 (78.2)

Bezuclastinib Combination Extends PFS with 50% Reduction in Risk of Progression or Death



Patients at Risk	Months																	
	0	2	4	6	8	10	12	14	16	18	20	22	24	26	28	30	32	34
Bezuclastinib + Sunitinib	204	180	166	146	130	119	102	76	72	51	24	23	8	6	2	2	2	0
Sunitinib	209	161	137	113	97	85	64	39	35	19	9	9	4	4	2	2	2	0



PFS: Progression-Free Survival; mRECIST v1.1: modified Response Evaluation Criteria in Solid Tumors version 1.1; BICR: Blinded Independent Central Review

Data cut-off as of 30Sep2025

Bezuclastinib + Sunitinib Demonstrates Unprecedented 46% Objective Response Rate

ORR per BICR , %, n [95% CI]	
Bezuclastinib + Sunitinib (n=204)	45.6% 93 [38.6, 52.7]
Sunitinib (n=209)	25.8% 54 [20.0, 32.3]
Difference in ORR, % [95% CI]; P Value	
19.8 [10.6, 28.6]; P Value <0.0001	

BOR per BICR, n (%)	Bezuclastinib + Sunitinib	Sunitinib
Complete Response (CR)	13 (6.4)	4 (1.9)
Partial Response (PR)	80 (39.2)	50 (23.9)
Stable Disease (SD)	91 (44.6)	108 (51.7)
Progressive Disease (PD)	15 (7.4)	41 (19.6)
Not Evaluable (NE)	5 (2.5)	6 (2.9)

Bezuclastinib + Sunitinib is Generally Well Tolerated with a Favorable Safety Profile

	Study Treatment	
	Bezuclastinib + Sunitinib (n=204)	Sunitinib (n=208) ¹
TEAEs, n (%)	204 (100)	207 (99.5)
TRAEs, n (%)	202 (99.0)	204 (98.1)
Gr3+ TRAEs, n (%)	146 (71.6)	109 (52.4)
<i>Bezuclastinib related Gr3+</i>	126 (61.8)	N/A
<i>Sunitinib related Gr3+</i>	141 (69.1)	109 (52.4)
SARs, n (%)	34 (16.7)	24 (11.5)
<i>Bezuclastinib related SAEs</i>	25 (12.3)	N/A
<i>Sunitinib related SAEs</i>	31 (15.2)	24 (11.5)
TRAEs leading to death, n (%)	0	1 (0.5)
Reductions of either drug due to TRAEs, n (%)	114 (55.9)	92 (44.2)
DC of study treatment due to TRAEs, n (%)	15 (7.4)	8 (3.8)

Randomized Period Data; 1: One patient randomized to sunitinib but never dosed

- The incidence of TEAEs and TRAEs was similar between treatment arms
- No TRAEs leading to death in patients on bezuclastinib + sunitinib combination
- Only TRAEs leading to discontinuation of either drug in >1 patient on the combination arm were neutropenia (2.9%), ALT/AST increased (1.5%), and diarrhea (1%)

All Grade TEAEs ≥ 20% Demonstrate Balance Between Arms

Preferred term, n (%)	Bezuclastinib + Sunitinib (n=204)		Sunitinib (n=208) ¹	
	All Grade	Grade 3+	All Grade	Grade 3+
Diarrhea	159 (77.9)	16 (7.8)	138 (66.3)	15 (7.2)
ALT/AST increased*	115 (56.4)	22 (10.8)	35 (16.8)	3 (1.4)
Hypertension	106 (52.0)	60 (29.4)	108 (51.9)	57 (27.4)
Taste disorder*	97 (47.5)	0	52 (25.0)	0
Nausea	81 (39.7)	1 (0.5)	56 (26.9)	2 (1.0)
Hair color changes	79 (38.7)	0	37 (17.8)	0
Fatigue	72 (35.3)	9 (4.4)	70 (33.7)	5 (2.4)
Neutropenia*	71 (34.8)	31 (15.2)	70 (33.7)	32 (15.4)
PPE	59 (28.9)	6 (2.9)	95 (45.7)	5 (2.4)
Vomiting	56 (27.5)	2 (1.0)	45 (21.6)	4 (1.9)
Decreased appetite	55 (27.0)	6 (2.9)	46 (22.1)	0
Anemia	54 (26.5)	19 (9.3)	42 (20.2)	10 (4.8)
Abdominal pain	51 (25.0)	6 (2.9)	52 (25.0)	4 (1.9)
Stomatitis	46 (22.5)	6 (2.9)	68 (32.7)	10 (4.8)
GERD	45 (22.1)	0	30 (14.4)	0
Dyspepsia	43 (21.1)	5 (2.5)	29 (13.9)	0
Thrombocytopenia*	39 (19.1)	2 (1.0)	55 (26.4)	9 (4.3)

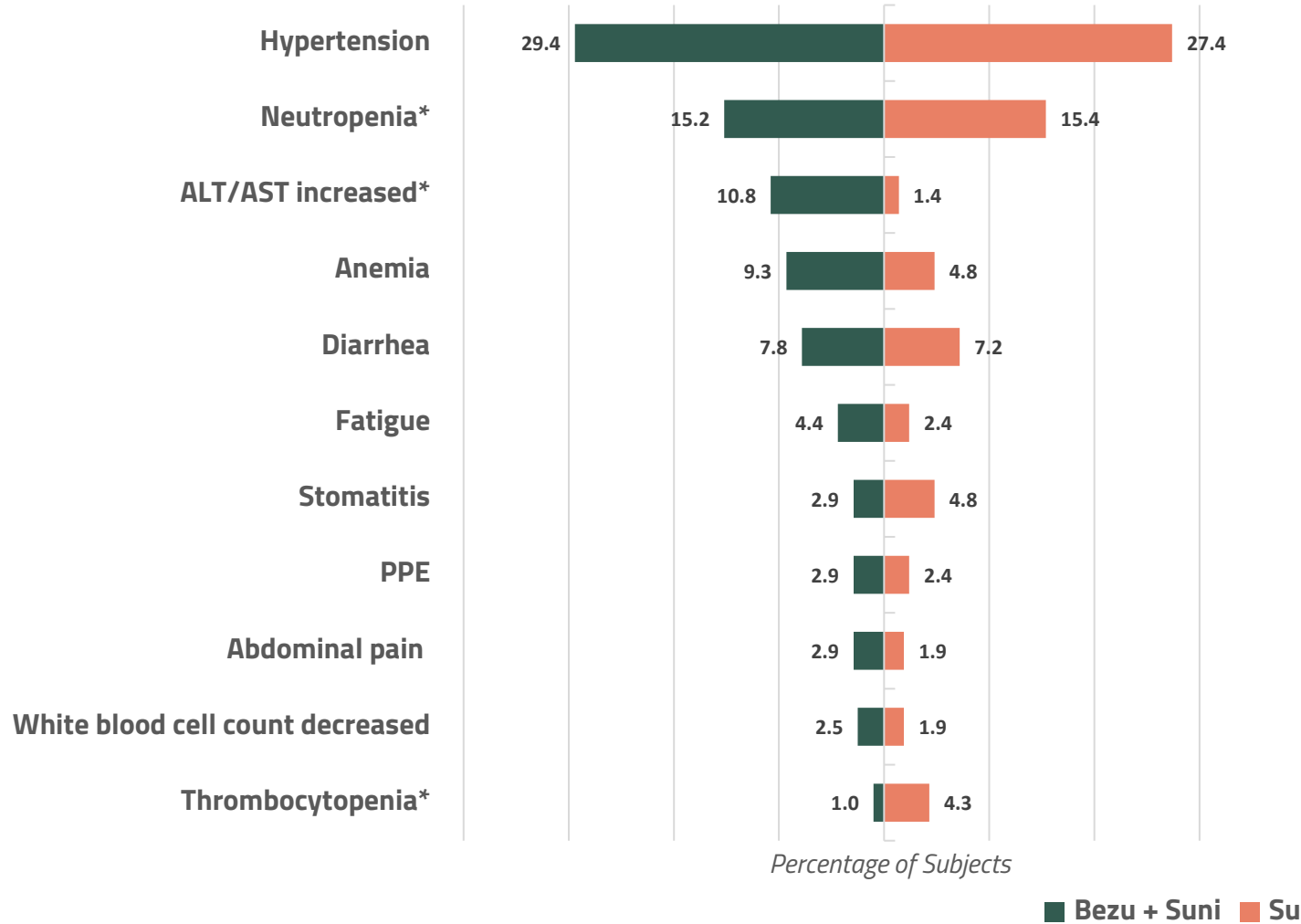
- TEAEs reported at a higher frequency (>15%) in combination arm: ALT/AST increased, taste disorder, and hair color changes
- TEAEs reported less frequently in combination arm: PPE, stomatitis and thrombocytopenia
- The safety profile of bezuclastinib combination is generally consistent with the known safety profile of sunitinib alone and no new risks were identified with the combination

Randomized Period Data; 1: One patient randomized to sunitinib but never dosed



Data cut-off as of 30Sep2025; *Pooled terms; TEAE, treatment-emergent adverse event; PPE, Palmar-Plantar Erythrodysesthesia; GERD Gastroesophageal Reflux Disease

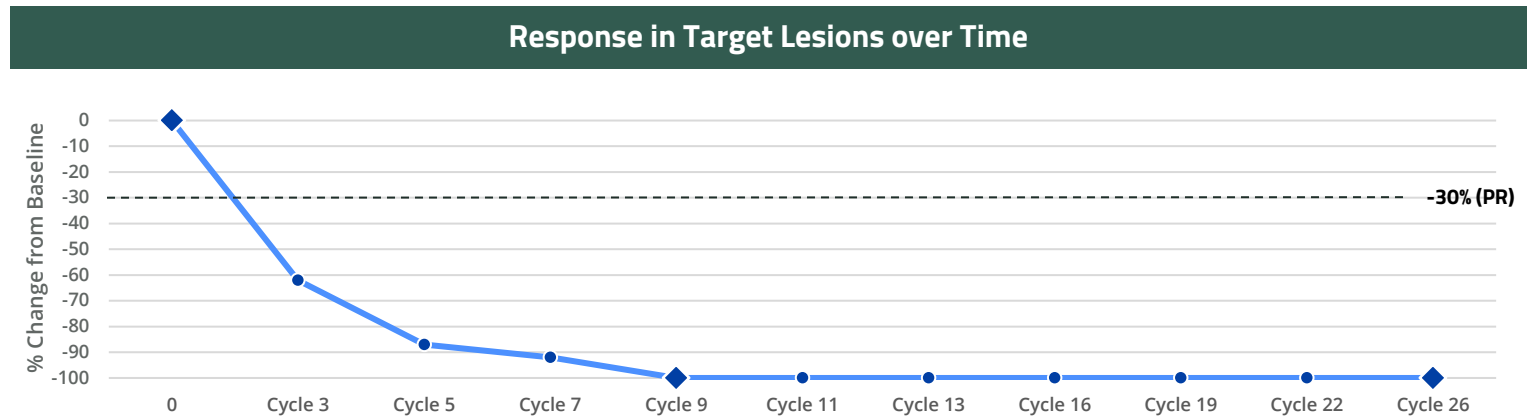
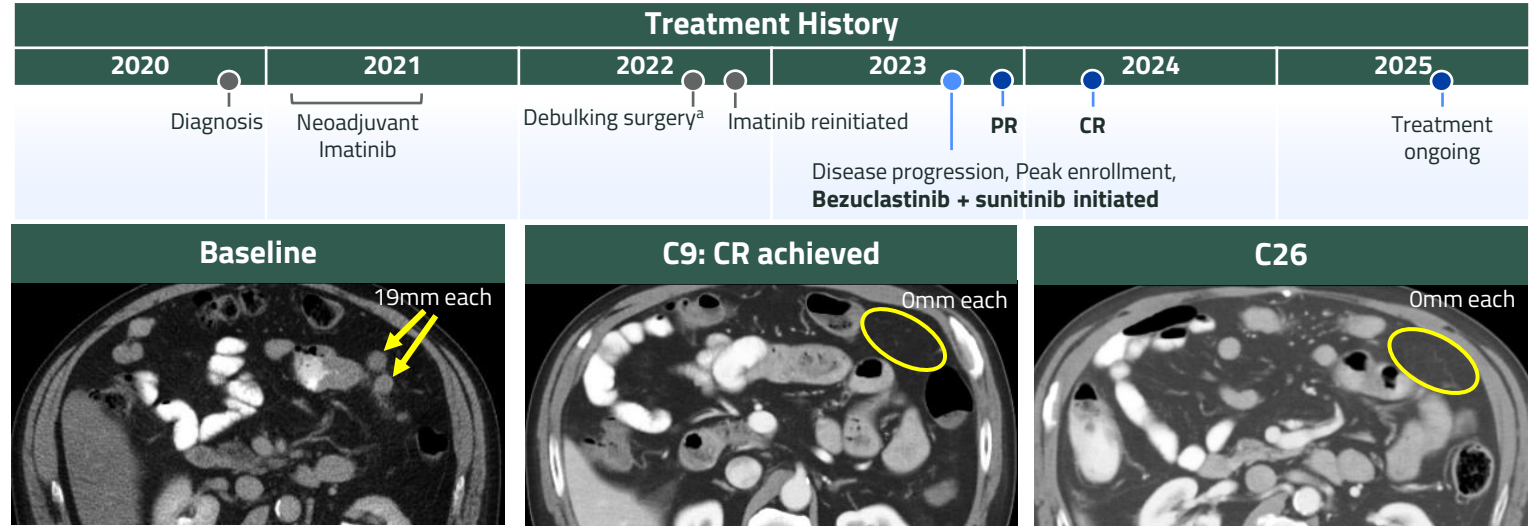
Incidence of Grade 3+ TEAEs ($\geq 2\%$) Balanced Across Arms



- Majority of the Gr 3+ TEAEs were reported at a similar rate between combination and monotherapy arms; ALT/AST increase and anemia reported at higher incidence in the combination arm
- No increase in frequency of severe events observed in combination arm for some key risks seen with sunitinib (hypertension, neutropenia and diarrhea)
- ALT/AST elevations led to bezuclastinib dose reductions in 12.7% of patients and only 1.5% of patients discontinued. All Grade 3 ALT/AST events resolved, and no Grade 4 elevations were reported across the study

66 yo Man with Metastatic GIST who Experienced an Early (PR at C3, CR at C9) and Durable Response to bezuclastinib + sunitinib (ongoing at C26)

Relevant Medical History	
Site of tumor diagnosis: Jejunum/Ileum	
Sites of disease:	
<ul style="list-style-type: none"> Target lesions: Peritoneum, mesentery, small intestine Baseline Sum of Diameters: 84 mm 	
Relevant comorbidities: Obesity; hypertension; anemia; elevated ALT and creatinine; abdominal distention; leg swelling; back pain; GERD	
Peak Treatment and Dose Modifications	
Bezuclastinib 600 mg QD + sunitinib 37.5 mg QD	
<ul style="list-style-type: none"> Sunitinib reduced to 25 mg for diarrhea 	
TRAEs (maximum Gr reported)	
Gr 1	<ul style="list-style-type: none"> Acneiform dermatitis Hair color changes Nausea
Gr 2	<ul style="list-style-type: none"> Localized edema
Gr 3	<ul style="list-style-type: none"> Diarrhea (resolved) Neutropenia (resolved)



Data cut-off as of 30Sep2025; C: Cycle; 1 cycle = 28 days; ^aExcision of abdominal tumors, intestinal resection; AE: adverse event; ALT: alanine transaminase; Gr: grade; GERD: gastroesophageal reflux disease; TRAE: treatment-related AE

69 yo Man with Metastatic GIST Responded to bezuclastinib + sunitinib Treatment at Cycle 5 (PR) and is Continuing to Benefit at Cycle 38

Relevant Medical History

Site of tumor diagnosis: Small intestine/bowel

Sites of disease:

- Target lesions: Peritoneum
- Baseline Sum of Diameters: 65 mm

Relevant comorbidities: Hemorrhoids; Gr 1 hypertension, Gr 1 anemia, anxiety, sleep apnea, hyperlipidemia, irregular heartbeat

Peak Treatment and Dose Modifications

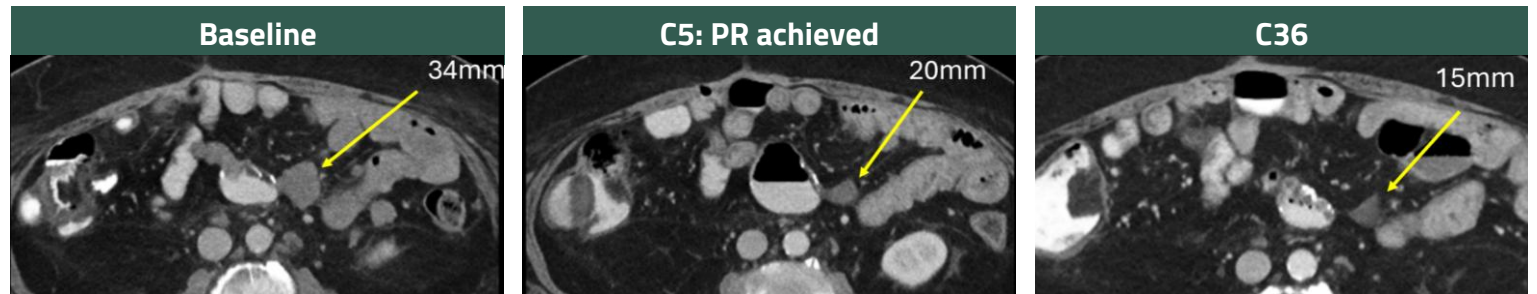
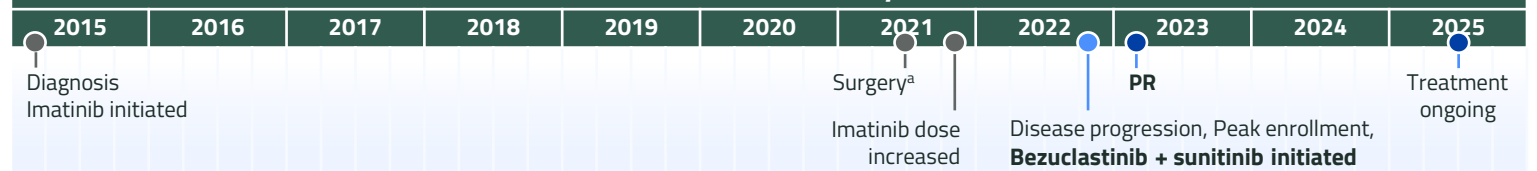
Bezuclastinib 600 mg QD + sunitinib 37.5 mg

- Following Gr 3 anemia/neutropenia:
 - Bezuclastinib interrupted → resumed at 600mg
 - Sunitinib interrupted → reduced to 25 mg

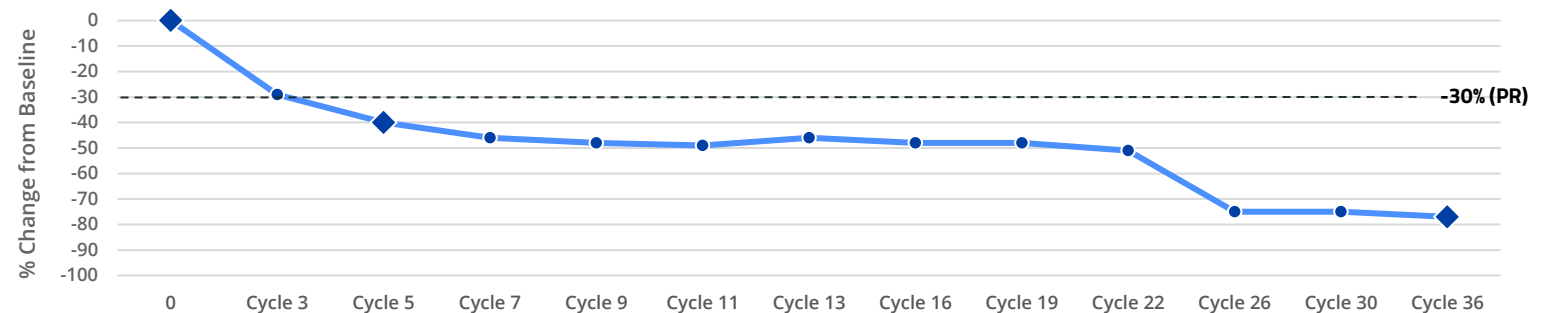
TRAEs (maximum Gr reported)

Gr 1	<ul style="list-style-type: none"> • Diarrhea • Hair color changes
Gr 2	<ul style="list-style-type: none"> • Hypertension • Hypothyroidism
Gr 3	<ul style="list-style-type: none"> • Neutropenia (resolved) • Anemia (related to Sunitinib only, resolved)

Treatment History



Response in Target Lesions over Time



We Believe Peak Results are Transformative and Practice Changing

- Bezuclastinib combination establishes first new benchmark for 2L GIST in 20 years
 - **50% reduced risk of progression or death** compared to current standard of care
 - **16.5 months mPFS** compared to 9.2 months for sunitinib alone ($p < 0.0001$)
 - **46% ORR** compared to 26% for sunitinib alone ($p < 0.0001$)
 - OS immature with event rate of less than 20% at time of PFS analysis
- Generally well tolerated with **no unique risks observed** when compared to the known safety profile of sunitinib
- Estimated **19 months+ mean treatment duration** for bezuclastinib combination patients based on projection for patients remaining on combination therapy
- Active **Expanded Access Program allowing immediate availability** of the bezuclastinib combination for 2L patients with GIST
- **NDA submission** for bezuclastinib in imatinib-resistant or intolerant GIST planned 1H 2026 based on results of the Peak trial

Expanded Results From the Phase 2 Summit Trial: Bezuclastinib in Adults With Non-Advanced Systemic Mastocytosis

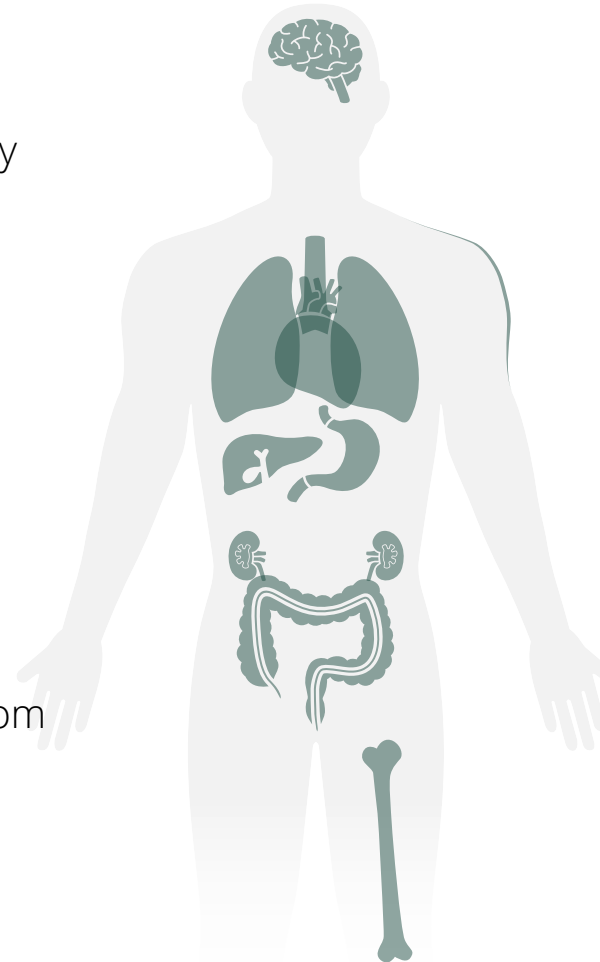
Nathan A Boggs¹, Lindsay Rein², Prithviraj Bose³, Brian D Modena⁴, Vito Sabato⁵, Karin Hartmann⁶, Cem Akin⁷, Tracy I George⁸, Cecilia Arana Yi⁹, Hanneke Oude Elberink¹⁰, Deepti H Radia¹¹, Andreas Reiter¹², Miguel Piris-Villaespesa¹³, Ingunn Dybedal¹⁴, Jens Panse^{15,16}, Stephen T Oh¹⁷, Pankit Vachhani¹⁸, Anthony M Hunter¹⁹, Mariana Castells²⁰, Cristina Livideanu²¹, Paul van Daele²², Arnold Kirshenbaum²³, Iván Alvarez-Twose²⁴, Jennifer E Vaughn²⁵, Minakshi Taparia²⁶, Sonia Cerquozzi²⁷, Andrzej Mital²⁸, Marek Hus²⁹, Alessandra Romano³⁰, John M Fahrenholz³¹, Frederick Lansigan³², Cristina Papayannidis³³, Helena Pomares-Marin³⁴, Michela Rondoni³⁵, Celalettin Ustun³⁶, Richard Herrscher³⁷, Amanda Pilla³⁸, Jenna Zhang³⁸, Lei Sun³⁸, Nisha Shah³⁸, Hina Jolin³⁸, Rachael Easton³⁸, Jessica Sachs³⁸, Frank Siebenhaar^{39,40}, Daniel J DeAngelo⁴¹

¹Department of Medicine, Uniformed Services University & Walter Reed National Military Medical Center, Bethesda, MD, USA; ²Duke Cancer Institute, Durham, NC, USA; ³MD Anderson Cancer Center, Houston, TX, USA; ⁴Modena Allergy and Asthma, La Jolla, CA, USA; ⁵Antwerp University Hospital (UZA), Edegem, Belgium; ⁶University Hospital Basel and University of Basel, Basel, Switzerland; ⁷University of Michigan, Ann Arbor, MI, USA; ⁸Huntsman Cancer Institute, University of Utah, ARUP Laboratories, Salt Lake City, UT, USA; ⁹Mayo Clinic Arizona, Phoenix, AZ, USA; ¹⁰University Medical Center Groningen, Groningen Research Institute of Asthma and COPD, University of Groningen, Groningen, The Netherlands; ¹¹Guy's & St Thomas' Hospitals NHS Foundation Trust, London, UK; ¹²Universitätsmedizin Mannheim, Mannheim, Germany; ¹³Ramón y Cajal Hospital, Madrid, Spain; ¹⁴Oslo University Hospital, Oslo, Norway; ¹⁵Center for Integrated Oncology Aachen Bonn Cologne Düsseldorf (CIO ABCD), Aachen, Germany; ¹⁶Department of Hematology, Oncology, Hemostaseology and Stem Cell Transplantation, University Hospital RWTH Aachen, Aachen, Germany; ¹⁷Washington University School of Medicine, St. Louis, MO, USA; ¹⁸University of Alabama at Birmingham, Birmingham, AL, USA; ¹⁹Emory University School of Medicine, Atlanta, GA, USA; ²⁰Brigham and Women's Hospital, Boston, MA, USA; ²¹CHU de Toulouse, Hopital Larrey, Toulouse, France; ²²Erasmus Medical Center, Rotterdam, The Netherlands; ²³Allervie, Glenn Dale, MD, USA; ²⁴Instituto de Estudios de Mastocitosis de Castilla-La Mancha, Hospital Virgen del Valle, Toledo, Spain; ²⁵The Ohio State University, Columbus, OH, USA; ²⁶University of Alberta, Edmonton, Alberta, Canada; ²⁷University of Calgary, Calgary, AB, Canada; ²⁸Uniwersyteckie Centrum Kliniczne, Klinika Hematologii i Transplantologii, Gdnask, Poland; ²⁹Department of Hematooncology and Bone Marrow Transplantation, Medical University of Lublin, Lublin, Poland; ³⁰Azienda Ospedaliero Universitaria Policlinico Rodolico San Marco, Catania, Italy; ³¹Vanderbilt University Medical Center, Nashville, TN, USA; ³²Dartmouth Cancer Center, Lebanon, NH, USA; ³³IRCCS Azienda Ospedaliero-Universitaria di Bologna, Policlinico di Sant'Orsola, Bologna, Italy; ³⁴Institut Catala d'Oncologia L'Hospitalet, Barcelona, Spain; ³⁵Azienda Unità Sanitaria Locale della Romagna, Ospedale S.Maria delle Croci, Ravenna, Italy; ³⁶Rush University Medical Center, Chicago, IL, USA; ³⁷AirCare, Plano, TX, USA; ³⁸Cogent Biosciences Inc., Waltham, MA, USA; ³⁹Institute of Allergology, Charité-Universitaetsmedizin Berlin, Berlin, Germany; ⁴⁰Fraunhofer Institute for Translational Medicine and Pharmacology ITMP, Immunology and Allergology IA, Berlin, Germany; ⁴¹Department of Medical Oncology, Dana-Farber Cancer Institute, Boston, MA, USA






Presented at the American Academy of Allergy, Asthma & Immunology (AAAAI) 2026 Annual Meeting | February 28, 2026 | Philadelphia, PA, USA

Bezuclastinib (CGT9486) is an oral, potent, and selective type 1 TKI that reduces symptoms of nonadvanced systemic mastocytosis (NonAdvSM)¹⁻⁵

- NonAdvSM is the most prevalent form of SM and is associated with debilitating symptoms that significantly impair quality of life^{3,5-10}
- Bezuclastinib is highly active against KIT D816V, has minimal brain penetration, and spares closely related kinases, which minimizes off-target toxicities, such as bleeding, cognitive impairment, edema, and pleural effusion^{1,2,11}
- The MS2D2^a is a comprehensive PRO measure of symptom severity in patients across the spectrum of NonAdvSM
 - Severity of each symptom within the Total Symptom Score (TSS) is assessed daily from 0 (none) to 10 (worst possible)
 - TSS is analyzed as a 14-day average



Eleven symptoms within 4 domains are included in MS2D2 TSS

Domain	Symptom
 Neurocognitive	<ul style="list-style-type: none"> • Difficulty concentrating • Difficulty remembering
 Fatigue	<ul style="list-style-type: none"> • Tiredness
 Skin	<ul style="list-style-type: none"> • Itching • Flushing • Skin redness • Spots
  Other	Gastrointestinal <ul style="list-style-type: none"> • Nausea • Abdominal pain Pain <ul style="list-style-type: none"> • Headache • Bone pain

^aMS2D2 developed according to FDA Guidance for Industry PRO measures and regulatory agency feedback and reached alignment with FDA for use of MS2D2 TSS in Part 2 of the registration-directed Summit trial.

PRO, patient-reported outcome; SM, systemic mastocytosis; TKI, tyrosine kinase inhibitor.

1. DeAngelo DJ, et al. *Hemasphere*. 2022; 6(suppl). 2. Guarnieri A, et al. Poster presented at: AACR Annual Meeting; April 8-13, 2022; New Orleans, LA. 3. Ungerstedt J, et al. *Cancers*. 2022;14(16):3942. doi:10.3390/cancers14163942 4. Li JY, et al. *Cancers (Basel)*. 2023.15(23). 5. Tse KY, et al. *J Allergy Clin Immunol Glob*. 2024.3(4):100316. 6. Scherber RM and Borate U. *Br J Haematol*. 2018;180:11-23. 7. Gilreath JA, et al. *Clin Pharmacol*. 2019;11:77-92. 8. Pardananani A. *Am J Hematol*. 2021;96(4):508-25. 9. Piris-Villaespesa M and Alvarez-Twose I. *Front Pharmacol*. 2020;11(443): doi:10.3389/fphar.2020.00443. 10. Farmer I and Radia DH. *Curr Hematol Malig Rep*. 2024;19:197-207. 11. Das A, et al. *Crit Rev Oncol Hematol*. 2021;157:103186.

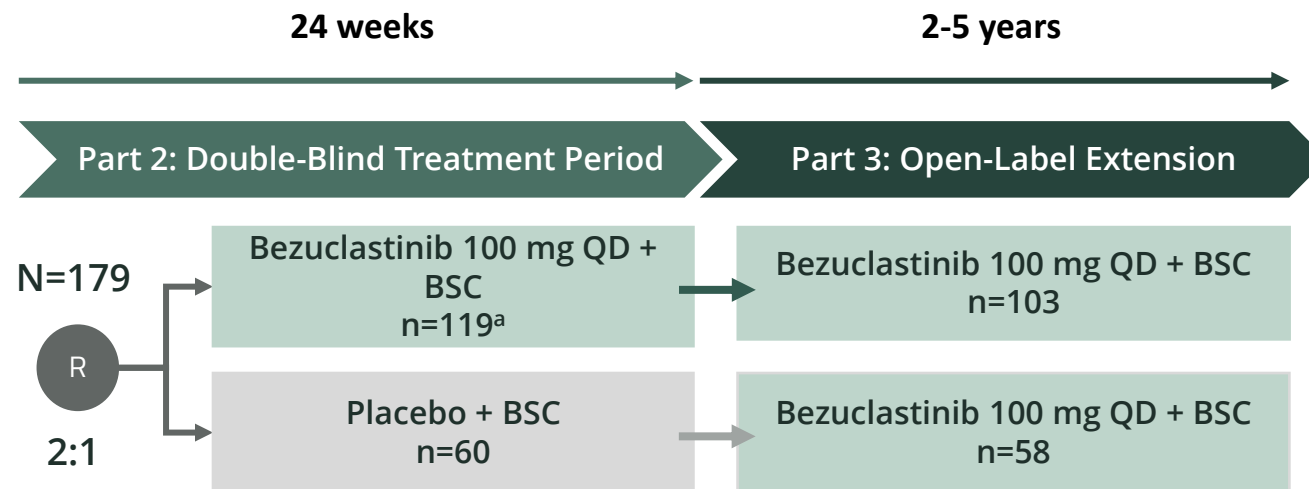
Summit (NCT05186753): Pivotal phase 2 multicenter, randomized, double-blind, placebo-controlled study evaluating bezuclastinib in NonAdvSM

Eligibility

ISM, BMM, or SSM based on WHO 2022 classification

Inadequate symptom control (moderate to severe symptoms) despite ≥ 2 anti-mediator therapies

BSC medications include H1RA, H2RA, cromolyn, LTRAs, corticosteroids, omalizumab, and PPIs



Part 3 OLE Objectives

- Characterize long-term safety and tolerability of bezuclastinib
- Durability of effect at the selected dose
- Change of the most severe symptom and most severe subdomain of MS2D2
- Evaluate additional efficacy parameters in patients with NonAdvSM
- Determine the effects of bezuclastinib on serum tryptase
- Explore changes in concomitant medication use for the treatment of NonAdvSM

^aOne patient withdrew consent and did not receive treatment.

BM, bone marrow; BMM, bone marrow mastocytosis; BSC, best supportive care; H1RA, histamine receptor type 1 antagonist; HR2A, histamine receptor type 2 antagonist; ISM, indolent SM; LTRA, leukotriene receptor antagonists; MC, mast cell; MS2D2, mastocytosis symptom severity daily diary; NonAdvSM, non-advanced systemic mastocytosis; PPI, proton pump inhibitors; QD, once daily; R, randomized; SSM, smoldering SM; TSS, total symptom score; VAF, variant allele frequency; WHO, World Health Organization.

Patient demographics and characteristics were representative of a broad NonAdvSM population

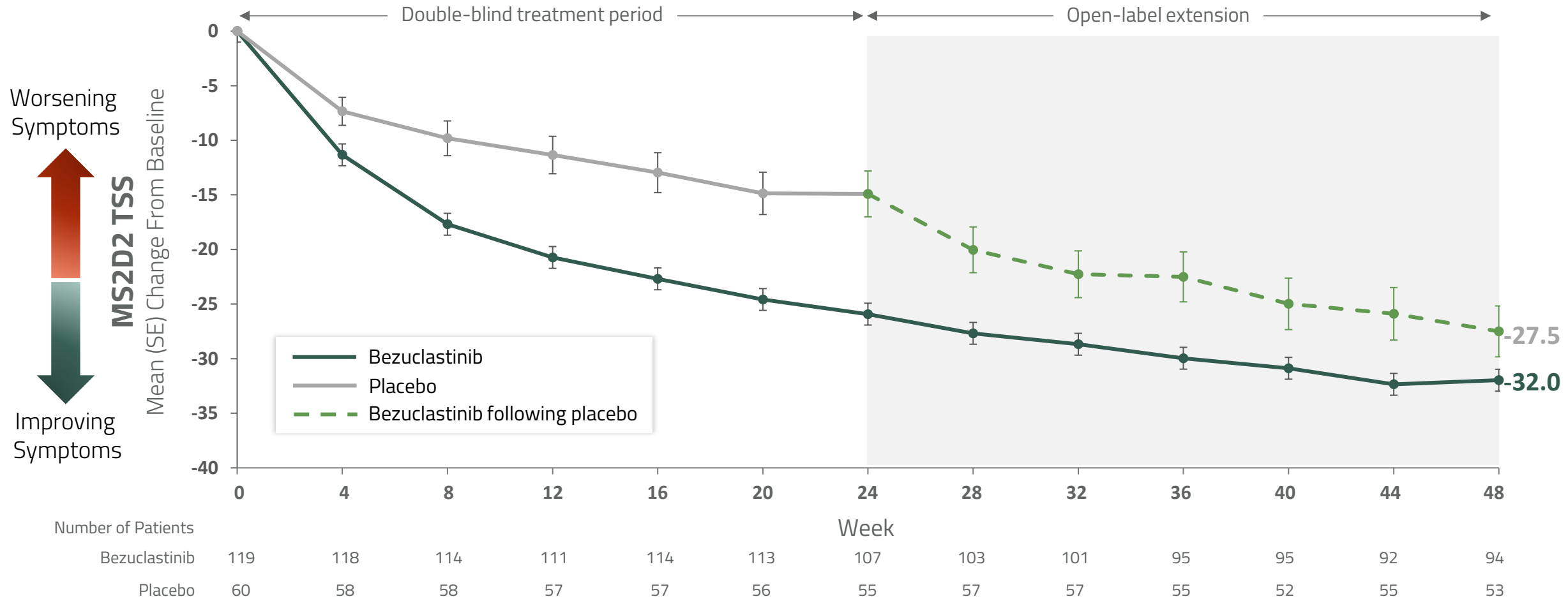
Baseline Characteristics	Bezuclastinib 100 mg QD (N=119)	Placebo (N=60)
Female, n (%)	74 (62.2)	44 (73.3)
Median age in years, (range)	51 (24–73)	52 (23–78)
NonAdv subtype, n (%)		
Indolent SM	97 (81.5)	50 (83.3)
Smoldering SM	8 (6.7)	4 (6.7)
BM mastocytosis	14 (11.8)	6 (10.0)
Region, n (%)		
North America	53 (44.5)	28 (46.7)
Europe	64 (53.8)	30 (50.0)
Asia-Pacific	2 (1.7)	2 (3.3)
SM therapy		
Prior KIT inhibitor ^a , n (%)	17 (14.3)	5 (8.3)
Best supportive care medications, median (range)	3 (0–6)	3 (1–7)

Disease Severity	Bezuclastinib 100 mg QD (N=119)	Placebo (N=60)
Mean MS2D2 TSS, (range)	57.1 (18–105.2)	52.6 (12.8–91.3)
<i>KIT</i> p.D816V ^b in whole blood, detected, n (%)	91 (76.5)	48 (80.0)
Median <i>KIT</i> p.D816V VAF ^b , % (range)	0.22 (0 ^c –32.3)	0.30 (0 ^c –33.6)
Median bone marrow MC burden, % (range)	10 (1–75)	10 (1–75)
Median serum tryptase, ng/mL (range)	39.9 (6.3–448.0)	41.2 (7.1–692.0)
Serum tryptase <20 ng/mL, n (%)	22 (18.2)	10 (16.7)
Most severe MS2D2 TSS symptoms at baseline in >10% of patients, n (%)		
Tiredness	54 (45.4)	24 (40.0)
Spots	39 (32.8)	21 (35.0)
Bone pain	16 (13.4)	9 (15.0)

^aKIT inhibitors included avapritinib, imatinib, midostaurin, dasatinib, and masitinib. ^bLimit of detection equals 0.03%. ^cUndetected. BM, bone marrow; MC, mast cell; MS2D2, mastocytosis symptom severity daily diary; NonAdvSM, nonadvanced SM; QD, once daily; SM, systemic mastocytosis; TSS, total symptom score; VAF, variant allele frequency.

Rapid, durable, and clinically meaningful symptom improvement that continues to deepen to 48 weeks of treatment

Patients crossing over to receive active treatment experience clinically meaningful symptom improvement

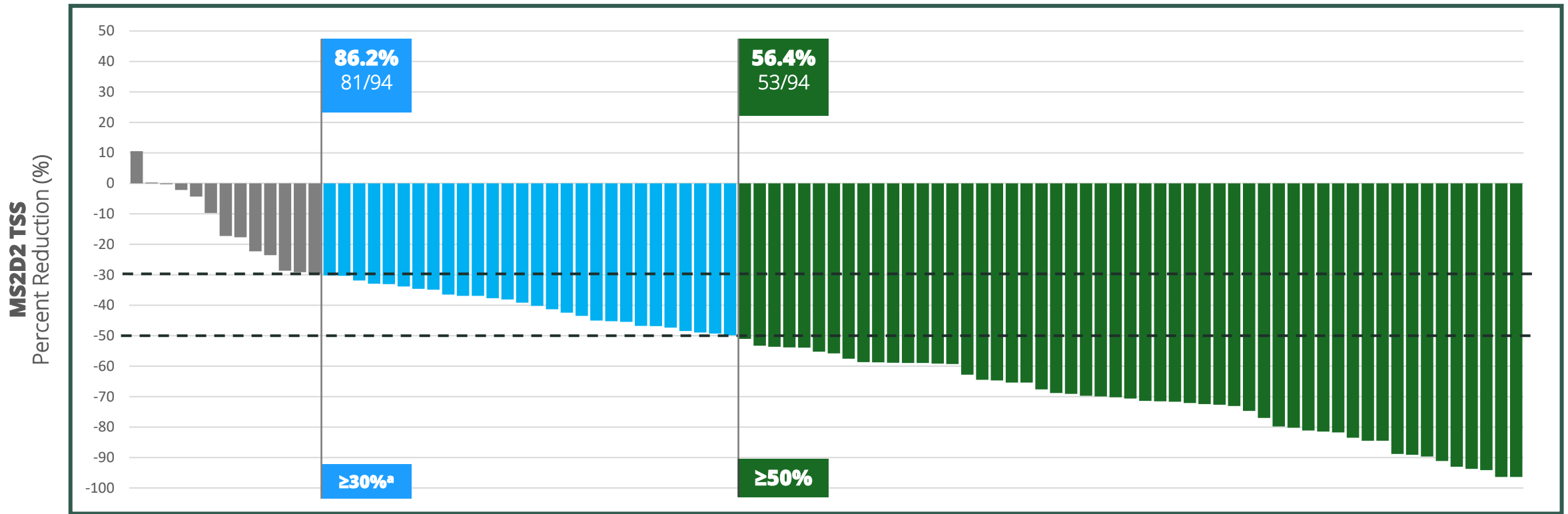


MS2D2, mastocytosis symptom severity daily diary; SE, standard error; TSS, total symptom score.

Bezuclastinib achieves robust symptom reduction at 48 weeks

86% of Patients Achieve Clinically Meaningful Symptom Improvement

Individual Patients Receiving Bezuclastinib 100 mg for 48 Weeks

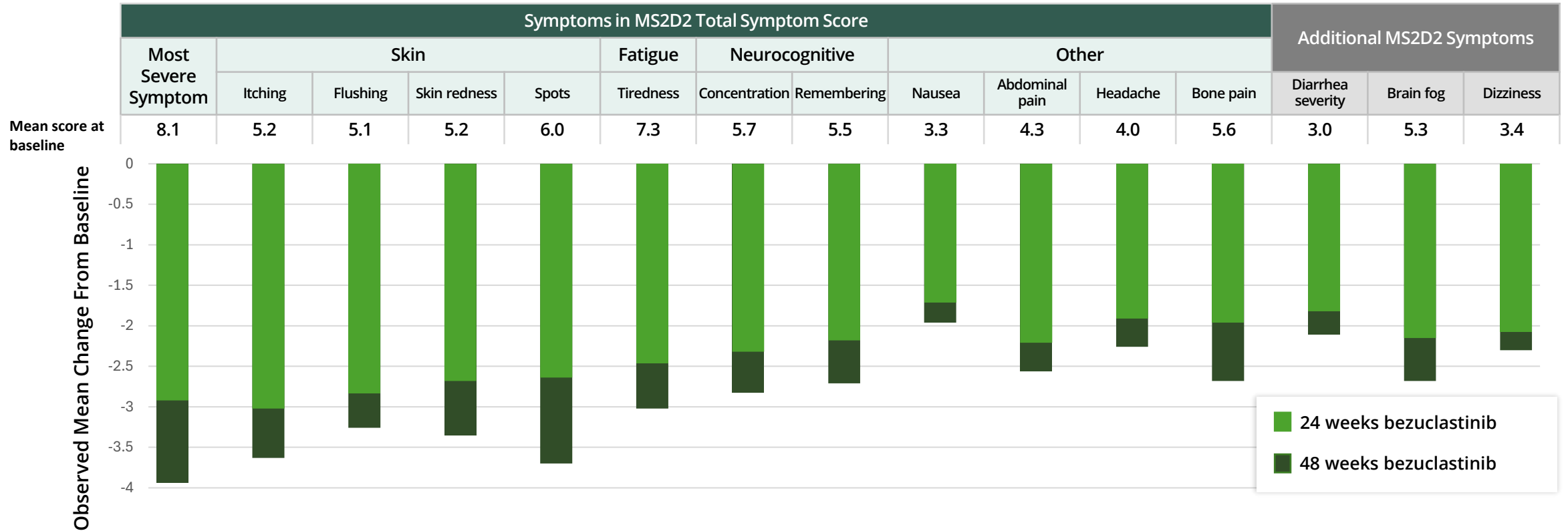


^aSymptom reductions of $\geq 30\%$ represent clinically meaningful change as determined by anchor-based analyses.

■ Reduction $\geq 50\%$
■ Reduction $\geq 30\%$

Data cutoff: November 7, 2025

Bezuclastinib achieves sustained and deepening symptom improvement through 48 weeks in NonAdvSM

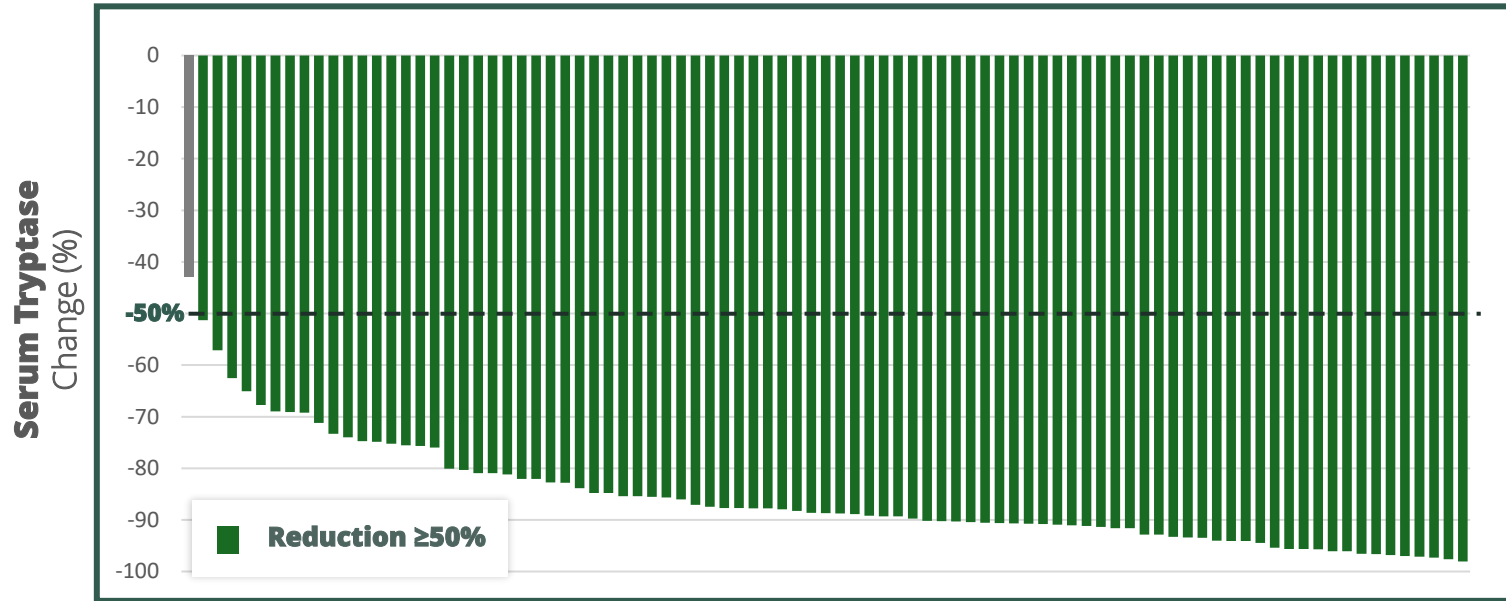


- All symptoms have deepening of improvement to 48 weeks of treatment with bezuclastinib
- At week 48, 50% of those in the bezuclastinib arm had a dose reduction or discontinuation of BSC medication

99% of patients reached $\geq 50\%$ reduction in serum tryptase at 48 weeks

Serum tryptase normalization persisted to 48 weeks of bezuclastinib treatment

Individual Patients Receiving Bezuclastinib 100 mg for 48 weeks



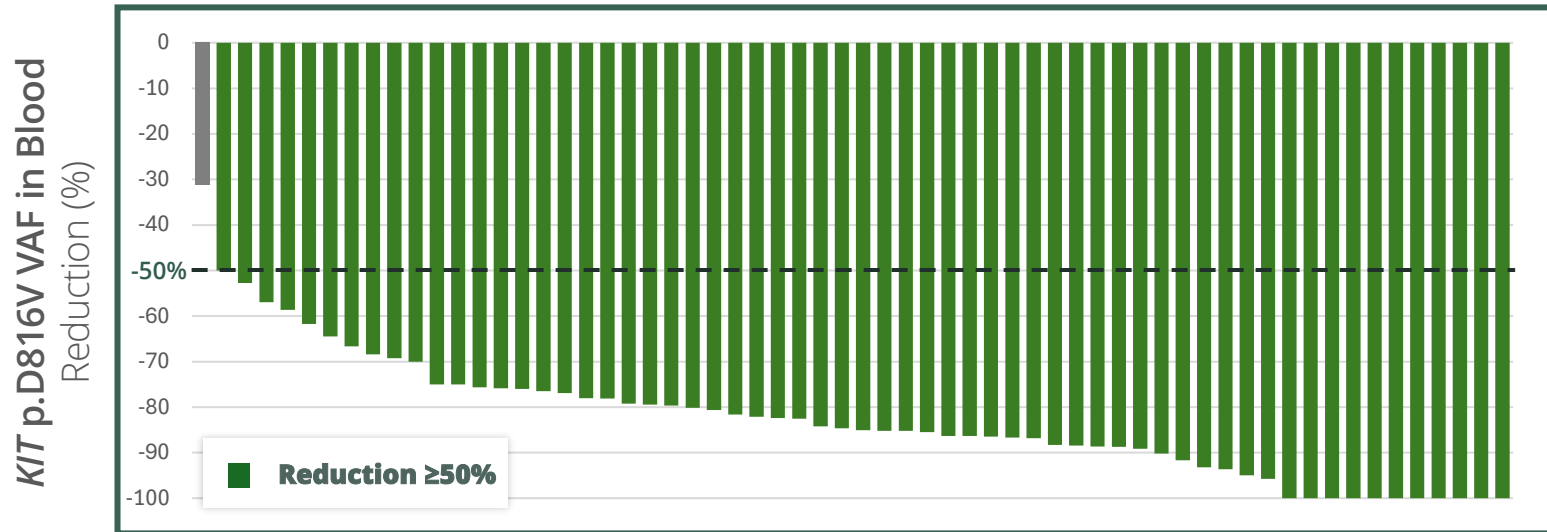
		Week 48	
		Bezuclastinib N=89	Placebo → Bezu N=60
Median serum tryptase, ng/mL (range)		4.7 (1.0-128.0)	5.8 (1.1-143.0)
Patients reaching $\geq 50\%$ reduction in serum tryptase, % (n/N) ^a		98.9% (88/89)	96.1% (49/51)
Patients achieving threshold ^b , % (n/N)	<20 ng/mL	91.7 (66/72)	90.5 (38/42)
	<11.4 ng/mL	83.3 (70/84)	77.6 (38/49)

- At week 48, the majority of placebo crossover patients achieved $\geq 50\%$ serum tryptase reductions and normalization within 24 weeks of bezuclastinib treatment

^aPatients with data at 48 weeks. ^bOf patients with baseline serum tryptase above threshold. ITT, intention to treat.

Bezuclastinib significantly reduced *KIT* p.D816V variant allele frequency, a molecular driver of disease, at 48 weeks

Individual Patients Receiving Bezuclastinib 100 mg for 48 weeks



		Week 48	
		Bezuclastinib	Placebo → Bezu
Median <i>KIT</i> p.D816V VAF in blood, % (range)		0.04 (0-10.5)	0.07 (0-12.1)
Patients reaching ≥50% reduction in <i>KIT</i> p.D816V VAF ^a in blood ^b or undetectable mutation, % (n/N)	ITT population	98.4 (61/62)	100 (37/37)

- At week 48, placebo crossover patients achieved significant reductions in *KIT* p.D816V within 24 weeks of bezuclastinib treatment

^aPatients with data at 48 weeks. ^bLimit of detection equals 0.03%.
ITT, intention to treat; VAF, variant allele frequency.

Bezuclastinib was well tolerated and no new safety concerns observed with longer-term treatment

TEAEs during OLE		Placebo → Bezuclastinib (n=58)		Bezuclastinib (n=103)	
Median (range) duration of bezuclastinib treatment (months) in the OLE		8.8 (2.8 – 14.4)		8.6 (0.46 – 15.0)	
TEAEs, n (%)		57 (98.3)		92 (89.3)	
Serious TRAEs, n (%)		1 (1.7)		0	
Reductions due to TRAEs, n (%)		9 (15.5)		2 (1.9)	
DCs due to TRAEs, n (%)		3 (5.2)		1 (1.0)	
TEAEs ≥10% either arm, n (%)					
Preferred Term	All Grade	Grade ≥ 3	All Grade	Grade ≥ 3	
Hair color changes	37 (63.8)	-	11 (10.7)	-	
ALT/AST increased ^a	22 (37.9)	5 (8.6)	10 (9.7)	1 (1.0)	
Altered taste ^a	12 (20.7)	-	2 (1.9)	-	
Dizziness	9 (15.5)	-	10 (9.7)	-	
Nausea	8 (13.8)	-	5 (4.9)	-	
Dyspepsia	8 (13.8)	-	5 (4.9)	-	
Headache	8 (13.8)	-	9 (8.7)	-	
URTI	8 (13.8)	-	7 (6.8)	-	
Fatigue	8 (13.8)	2 (3.4)	2 (1.9)	-	
Diarrhea	7 (12.1)	-	8 (7.8)	-	
ALP increased	7 (12.1)	-	1 (1.0)	-	
Arthralgia	6 (10.3)	-	10 (9.7)	1 (1.0)	
Insomnia	6 (10.3)	-	4 (3.9)	-	

- The majority of TEAEs were low grade and reversible
- Overall median (range) duration of treatment in patients continuing bezuclastinib in the OLE was 13.4 (0.67–20.5) months
- Consistent safety and tolerability profile observed in OLE for patients crossing over to bezuclastinib, similar to the previously reported 24-week experience
- The only hepatic events reported were transient and manageable lab abnormalities, similar to the previously reported 24-week experience
- Discontinuations due to TRAEs remained limited, consistent with the previously reported 24-week experience; all were due to transaminase elevations and all events fully resolved

Only TEAEs that occurred during OLE on bezuclastinib treatment are included. Adverse events per NCI CTCAE v5.0.


^aIncludes pooled terms.

ALT, alanine aminotransferase; AST, aspartate aminotransferase; DC, discontinuation; TEAE, treatment emergent adverse event; TRAE, treatment related adverse event.

Bezuclastinib represents a promising new treatment for patients with NonAdvSM

Disease modification and symptom improvement continue out to 48 weeks of treatment

- Rapid, statistically significant, and clinically meaningful symptom improvement is durable and continues to deepen to 48 weeks of treatment
 - Placebo patients crossing over to receive bezuclastinib experience clinically meaningful symptom improvement
- 86% of patients achieve clinically meaningful symptom improvement
 - All symptoms have deepening of improvement to 48 weeks of treatment with bezuclastinib
- At week 48, 50% in the bezuclastinib arm had a dose reduction or discontinuation of BSC medication
- Safety and tolerability profile in OLE, with > 24 weeks and up to 48 weeks of treatment, supports potential for chronic dosing
- NDA for a broad NonAdvSM population was submitted in December 2025; Breakthrough Therapy Designation granted by the FDA for patients with NonAdvSM previously treated with avapritinib and with smoldering SM
- Bezuclastinib Systemic Mastocytosis Expanded Access Program (NCT06915766) is currently open to requests from treating physicians in the United States

A silhouette of a person standing on a rocky peak, with their arms raised in a gesture of triumph or achievement. The background consists of a range of mountains under a dark, overcast sky.

APEX Trial Bezuclastinib in AdvSM Top-Line Results

Real Challenges. Real Solutions.

Precision therapies for genetically defined diseases

APEX Part 2: Phase 2 Registration-Directed, Open-Label Clinical Study of Bezuclastinib in Patients with Advanced Systemic Mastocytosis

PART 1: DOSE OPTIMIZATION ORIGINAL FORMULATION

50 mg BID
100 mg BID
200 mg BID
400 mg QD

Stage 1 Confirmation

150mg QD
n=10

Selected
Dose:
150mg QD

300 mg QD
n=15

PART 2: PIVOTAL TRIAL

Stage 2 Expansion

ASM, SM-AHN and MCL patients with mIWG measurable C-findings; 150 mg QD
n=58

Stage 2 Additional Cohorts

ASM, SM-AHN and MCL patients without mIWG measurable C-findings; 150 mg QD
n=13

~20 high-risk SM-AHN patients w/concurrent azacitidine or hydroxyurea; 150 mg QD (ongoing enrollment)

Apex Part 2 Results:

- 81 patients evaluable for safety and PPR
- 68 patients evaluable for mIWG ORR

APEX Part 2: Patients Demographics and Baseline Characteristics

Primary Endpoint: ORR (CR/CRh + PR + CI) per modified IWG-MRT-ECNM response criteria as assessed by CRRC
Key Secondary Endpoint: ORR (CR/CRh + PR) per PPR

Patient Demographics	Part 2 150mg QD
# Patients	81
Female, n (%)	23 (28.4)
Median Age in years, (range)	70 (43, 87)
ECOG PS at screening, n (%)	
0	24 (29.6)
1	37 (45.7)
≥2	20 (24.7)
Clinical Characteristics	
AdvSM Subtype per Central Eligibility Review, n (%)	
ASM	11 (13.6)
SM-AHN	57 (70.4)
MCL and MCL-AHN	13 (16.0)
SRSF2/ASXL1/RUNX1 Mutation in PB	48 (59.3)

SM Therapy	Part 2 150mg QD
Prior AdvSM or AHN Therapy	36 (44.4)
Prior TKI Inhibitor, n (%)	28 (34.6)
Prior Avapritinib, n (%)	6 (7.4)
Prior Midostaurin, n (%)	24 (29.6)
Baseline Mast Cell Burden	
Median KIT D816V in Whole Blood, % (range) BLD, n (%)	6.7 (0.0 - 91.4) 8 (9.9)
Median BM MC Burden, % (range)	35 (3, 95)
Median Serum Tryptase at baseline, ng/mL (range) % Tryptase < 20	191 (23, 1987) 0
mIWG-MRT-ECNM Evaluable Population	
Patients evaluable per mIWG-MRT-ECNM by EC, n (%)	68 (84.0)



AdvSM, advanced systemic mastocytosis; ASM, aggressive SM; CRRC, Central Response Review Committee; SM-AHN, SM with associated hematologic neoplasm; MCL, mast cell leukemia; MCL-AHN, mast cell leukemia with associated hematologic neoplasm; MC, mast cell; PB, Peripheral Blood; PPR, Pure Pathologic Response Criteria; VAF, variant allele frequency

Data cut-off as of 19Sep25

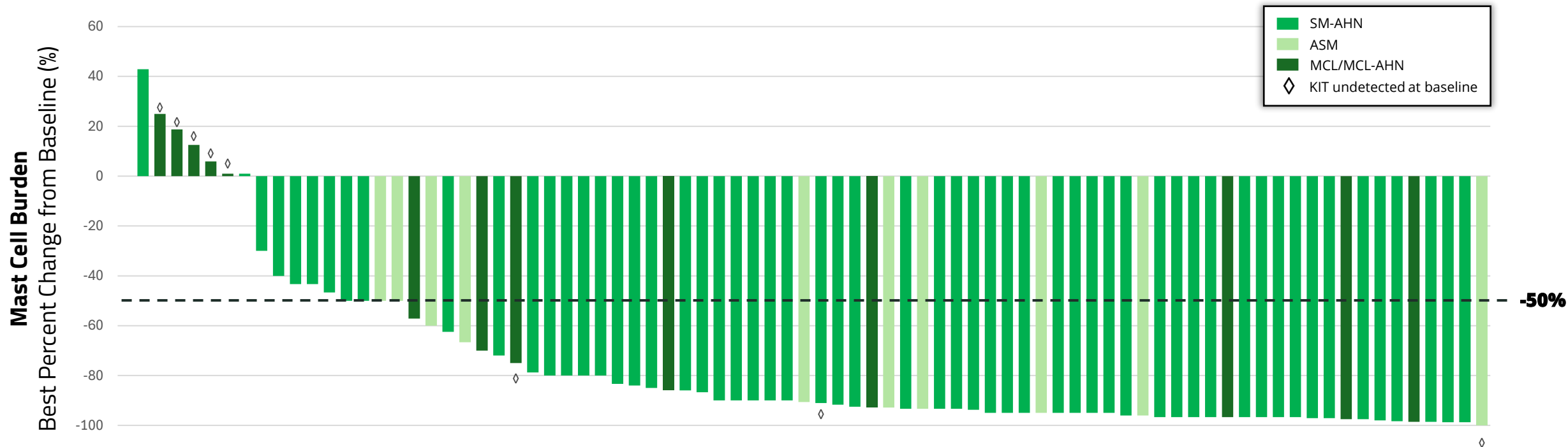
APEX Part 2: Patients Receiving 150mg Bezuclastinib Achieved Positive Results on Primary (mIWG) and Key Secondary (PPR) Endpoints

	Best ORR per mIWG, n (%) N=68	Best PPR, n (%) N=81
Overall Response Rate		
CR+CRh+PR+CI	39 (57.4)	-
CR+CRh+PR	33 (48.5)	65 (80.2)
Best Overall Response		
Complete Response (CR+CRh)	9 (13.2)	46 (56.8)
Molecular CR/CRh	-	18 (22.2)
Partial Response (PR)	24 (35.3)	19 (23.5)
Clinical Improvement (CI)	6 (8.8)	-
Stable Disease (SD)	25 (36.8)	15 (18.5)
Progressive Disease (PD)	2 (2.9)	0
Not Evaluable (NE)	2 (2.9)	1 (1.2)

Median Duration of Treatment: 9.4 months

At time of data cut-off, multiple ongoing patients had unconfirmed responses

APEX Part 2: Bezuclastinib Significantly Reduced Objective Measures of Disease Burden Including Mast Cells, Serum Tryptase, and KIT D816V VAF



	Serum Tryptase N=80 ^a	MC Burden N=80 ^a	KIT D816V VAF N=43 ^b
Best percent change from baseline, mean	-83.4%	-75.5%	-77.3%
Patients reaching $\geq 50\%$ reduction from baseline or defined threshold^c	89%	89%	91%



^aIncludes patients who had at least one post baseline assessment.

^bIncludes patients who had detectable VAF at baseline and one post baseline assessment.

^cThreshold: for mast cell burden is clearance of MC aggregates and for KIT D816V VAF is reaching undetectable level of KIT (limit of detection is 0.03%).

Data cut-off as of 19Sep25

APEX Part 2: Bezuclastinib was Well Tolerated with a Favorable Safety Profile

150mg QD Bezuclastinib N=81		
TRAEs, n (%)	75 (92.6)	
Drug-related SAEs, n (%)	5 (6.2)	
Reductions due to TRAEs, n (%)	12 (14.8)	
DCs due to TRAEs, n (%)	0	
TRAEs in ≥ 10% of patients; Preferred term, n(%)		
	Any grade	Grade ≥ 3
Hematological events, n (%)		
Neutropenia*	24 (29.6)	19 (23.5)
Thrombocytopenia*	20 (24.7)	11 (13.6)
Anemia	13 (16.0)	8 (9.9)
Non- Hematological events, n (%)		
Hair color changes	25 (30.9)	0
Altered taste*	23 (28.4)	0
ALT/AST increased*	17 (21.0)	1 (1.2)
Blood ALP increased	11 (13.6)	4 (4.9)
Diarrhea	12 (14.8)	0
Nausea	12 (14.8)	1 (1.2)
Alopecia	9 (11.1)	0
Peripheral edema	9 (11.1)	0

- Hematological events were reversible and manageable
- Treatment related hepatic events were transient and manageable lab abnormalities
- Dose reductions were primarily due to hematological events; no other adverse event led to dose reduction in more than one patient
- No discontinuations due to treatment-related AEs
- No treatment-related deaths were reported

Bezuclastinib Represents a Promising New Treatment Option for ASM Patients

- Patients receiving bezuclastinib achieved high rates of response:
 - 57% ORR (CR+CRh+PR+CI) per mIWG-MRT-ECNM
 - 80% ORR (CR+CRh+PR) per PPR
- Significant reductions in objective disease markers underscore potent target engagement and impact on KIT-driven disease pathology:
 - Serum tryptase: ↓ ≥50% in 89% of patients
 - Bone marrow mast cell burden: ↓ ≥50% in 89%
 - *KIT* p.D816V variant allele frequency: ↓ ≥50% in 91%
- Bezuclastinib was well-tolerated, with infrequent need for dose reduction and no patients requiring discontinuation for treated related adverse events
 - Only 1 patient experienced Gr 3 AST/ALT, and remains on study following dose reduction
 - Encouraging safety profile potentially allows for concomitant treatment in patients who require other cytoreductive therapies for AHN or post-transplant
- NDA submission for AdvSM population is expected in 1H 2026



**Real Challenges.
Real Solutions.**

[Cogentbio.com](https://www.cogentbio.com)