HEMOB

Reimagining the Treatment of Coagulation Disorders

August 2026

Disclaimer

Forward-Looking Statements

This presentation contains forward-looking statements that involve substantial risks and uncertainties. All statements, other than statements of historical facts, contained in this presentation, including statements regarding our strategy, future operations, prospects and plans, objectives of management, the anticipated timelines for reporting data from our clinical trials, the anticipated timelines for initiating a Phase 3 clinical trial of sutacimig and first-in-human studies of HMB-003, the clinical potential of sutacimig, HMB-002 and HMB-003, our plans to expand our pipeline, and the sufficiency of our cash resources for the period anticipated, constitute forward-looking statements within the meaning of The Private Securities Litigation Reform Act of 1995. The words "anticipate," "believe," "continue," "could," "estimate," "expect," "intend," "may," "might," "objective," "ongoing," "plan," "predict," "project," "potential," "should," or "would," or the negative of these terms, or other comparable terminology are intended to identify forward-looking statements, although not all forward-looking statements contain these identifying words. We may not actually achieve the plans, intentions or expectations disclosed in these forward-looking statements, and you should not place undue reliance on these forward-looking statements. Actual results or events could differ materially from the plans, intentions and expectations disclosed in these forward-looking statements as a result of various important factors, including: uncertainties inherent in the identification and development of product candidates, including the initiation and completion of preclinical studies and clinical trials; uncertainties as to the availability and timing of results from preclinical studies and clinical trials; the timing of and our ability to initiate and enroll patients in clinical trials; whether results from preclinical studies and earlier clinical trials will be predictive of the results of later clinical trials; whether our cash resources will be sufficient to fund our foreseeable and unforeseeable operating expenses and capital expenditure requirements; as well as the risks and uncertainties identified in our filings with the Securities and Exchange Commission (SEC), including our most recent Form 10-Q and in subsequent filings we may make with the SEC. In addition, the forward-looking statements included in this presentation represent our views as of the date of this presentation. We anticipate that subsequent events and developments will cause our views to change. However, while we may elect to update these forward-looking statements at some point in the future, we specifically disclaims any obligation to do so. These forward-looking statements should not be relied upon as representing our views as of any date subsequent to the date of this presentation.

Market and Industry Data

This presentation includes statistical and other industry and market data that we obtained from independent industry publications and research, surveys and studies conducted by independent third parties as well as our own estimates of the prevalence of certain diseases and conditions. The market data used in this presentation involves a number of assumptions and limitations. Industry publications and third-party research, surveys and studies generally indicate that their information has been obtained from sources believed to be reliable, although they do not guarantee the accuracy or completeness of such information. Our estimates of the patient population with the potential to benefit from treatment with any product candidates we may develop include several key assumptions based on our industry knowledge, industry publications, third-party research and other surveys, which may be based on a small sample size and may fail to accurately reflect the addressable patient population.

Patients Living With Neglected Blood Coagulation Disorders Face Significant Disease Burden

Increased mortality

Fatigue, depression, cognitive impairment commonly reported

Patients report feeling **fear, concern, and a sense of neglect**

0 **prophylactic treatments available**

Existing treatments can be burdensome, including blood transfusions or risky bone marrow transplants

Social stigma and psychological distress from bleeds of the nose and gums, which are highly visible sites

Abnormal bleeding is normalized

Source: Natural history studies - GT360, FVIID360, VWD360.

Bringing Innovation to Patients With Neglected Blood Coagulation Conditions

+20 *Approved Products* — **Hemophilia A**

8 *Approved products* — **Hemophilia B**

HEMAB TODAY

- **Glanzmann Thromb[1].** ~5K *sutacimig*
- **Factor VII Deficiency[1]** ~5K *sutacimig*
- **Von Willebrand Dis[2].** ~120K *HMB-002*
- **Heavy Menstrual Bl.[14]** ~20M *HMB-003*

HEMAB TOMORROW

- **Bernard Soulier Syn.[3]** ~5K
- **Other Platelet Dis.[4]** ~10K
- **Factor X Def.[5]** 1:500K
- **Anti-thrombin Def.[6,7]** 20-50K
- **Factor V Deficiency[8]** ~5K
- **Plasminogen Def.[9]** ~3K
- **Protein C Def.[10,11]** 1:500K

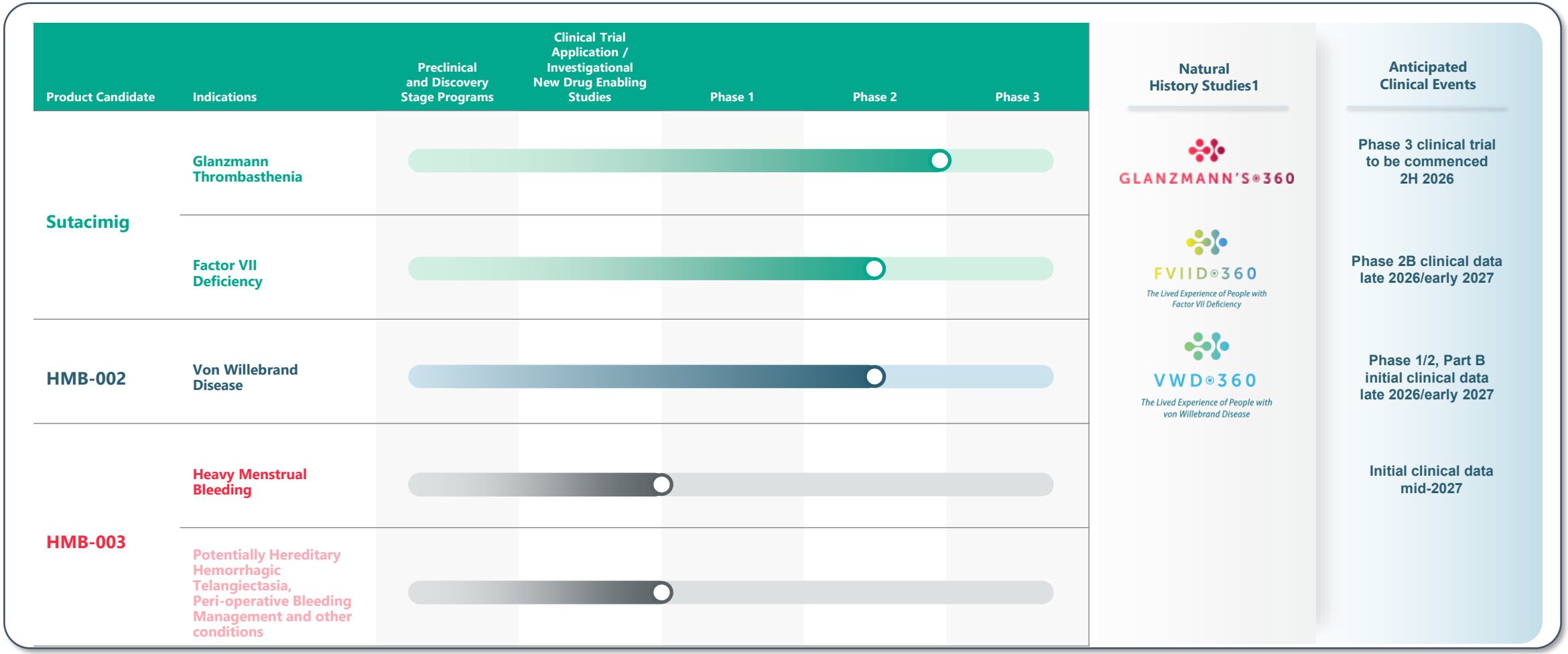
Less common

- **HHT[12,13]** ~1.5M
- **Arterial/Venous Dis.[15,16,17]** ~25-30M
- **Post Thrombotic Syn.[17,18,19,20]** ~600K-1M
- **Coagulopathy PPH[21,22,23]** ~8-14K/yr
- **Factor V Leiden[7, 24, 25]**
- **Trauma & Peri-op.[26, 27, 28, 29]** 250-500K/yr

Larger populations

[1] WFH Annual Global Survey 2023. Available at: wfh.org/research-and-data-collection/annual-global-survey/. [2] cdc.gov/hemophilia-community-counts/php/data-research/htc-population-profile/index.html. [3] Kaya Z. Bernard-Soulier syndrome: a review of epidemiology, molecular pathology, clinical features, laboratory diagnosis, and therapeutic management. Semin Thromb Hemost. 2025;51(2):209–218. PMID: 39191409. [4] Hamostaseologie 2021;41:475–488. [5] Blood (2015) 126 (23): 3511. [6] National Organization for Rare Disorders (NORD). Antithrombin Deficiency. Updated November 2023. Available at: rarediseases.org/rare-diseases/antithrombin-deficiency/. [7] MedlinePlus Genetics (NIH). Hereditary antithrombin deficiency. Estimated prevalence approximately 1 in 2,000–3,000 individuals. Available at:medlineplus.gov/genetics/condition/hereditary-antithrombin-deficiency/. [8] Hematol transfus cell ther. 2024;46(S7):S21–S42. [9] Shapiro AD, Menegatti M, Palla R, et al. An international registry of patients with plasminogen deficiency (History). Haematologica. 2020;105(3):554–561. PMC7049368. [10] National Organization for Rare Disorders (NORD). Protein C Deficiency. Updated March 2025. Available at: rarediseases.org/rare-diseases/protein-c-deficiency/. [11] StatPearls. Protein C Deficiency. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; updated July 2023. Available at: ncbi.nlm.nih.gov/books/NBK542222/. [12] Faughnan ME, Mager JJ, Hetts SW, et al. Second International Guidelines for the Diagnosis and Management of Hereditary Hemorrhagic Telangiectasia. Ann Intern Med. 2020;173(12):989–1001. [13] Ragni MV, et al. Characterizing the healthcare utilization and costs of hereditary hemorrhagic telangiectasia. Am J Hematol. 2025. PMC12417757. [14] Centers for Disease Control and Prevention (CDC). About Heavy Menstrual Bleeding. Updated May 2024. Available at: cdc.gov/female-blood-disorders/about/heavy-menstrual-bleeding.html. [15] CDC Blood Clots – Data & Statistics (2025). [16] Prandoni et al., *Clin Epidemiol* (2010). [17] Heit JA, *Arterioscler Thromb Vasc Biol* (2008). [18] Farrell JJ et al., *Cardiovasc Diagn Ther* (2016). [19] Kahn SR et al., *Circulation* (2014). [20] Prandoni P & Kahn SR, *Br J Haematol* (2009). [21] Wen T et al., *Obstet Gynecol* (2023). [22] Belfort MA, *N Engl J Med* / PMC Review (2023). [23] Callaghan WM et al., *Am J Obstet Gynecol* (2010). [24] Kujovich JL, *GeneReviews* / NCBI (2024). [25] Rosendaal FR et al., *Genetics in Medicine* (2011). [26] Bikdeli B et al., *PubMed* (2018). [27] Geerts WH et al., *PMC* (trauma prophylaxis review). [28] Cripps MW et al., *PMC* (2021). [29] ASH Guidelines, *Blood Advances* (2019).

Building A Franchise Designed to Address Select Coagulation Disorders With the Potential to Meaningfully Improve Disease Management



[1]*We conduct natural history studies in our patient populations, which capture detailed insights into patients' lived experiences, including bleed rates, treatment patterns, and the impact of bleeding disorders on social life, economic status, psychological wellbeing, and work or education.*

Sutacimig (HMB-001)

Glanzmann thrombasthenia
Factor VII Deficiency
Potentially other platelet disorders

UK

ILAP Designation

EU

Priority Medicines (PRIME) & Orphan Drug Designation

US IND

Breakthrough Therapy, Fast Track & Orphan Drug Designation

Glanzmann Thrombasthenia (GT) and Factor VII Deficiency (FVIID)

Disease Burden

GLANZMANN THROMBASTHENIA

88%	~54%	65%	30%
Experienced ≥1 bleed in the past week	Had ≥3 bleeds per week	Required a hospital visit in the past 6 months	Showed signs for symptomatic depression

FACTOR VII DEFICIENCY

~1.3 bleeds/week

Mean bleed frequency (1.65 women vs. 0.53 men)

24%	50%	61%
used prophylaxis in the last month	Lost time from education	Reported low mood

Market Opportunity

10K

~10,000 patients across GT & FVIID in key addressable markets (mainly US, EU, Japan, GCC countries)

Concentrated provider network

Current treatments are generally reactive, short-acting, IV-based, and high-risk

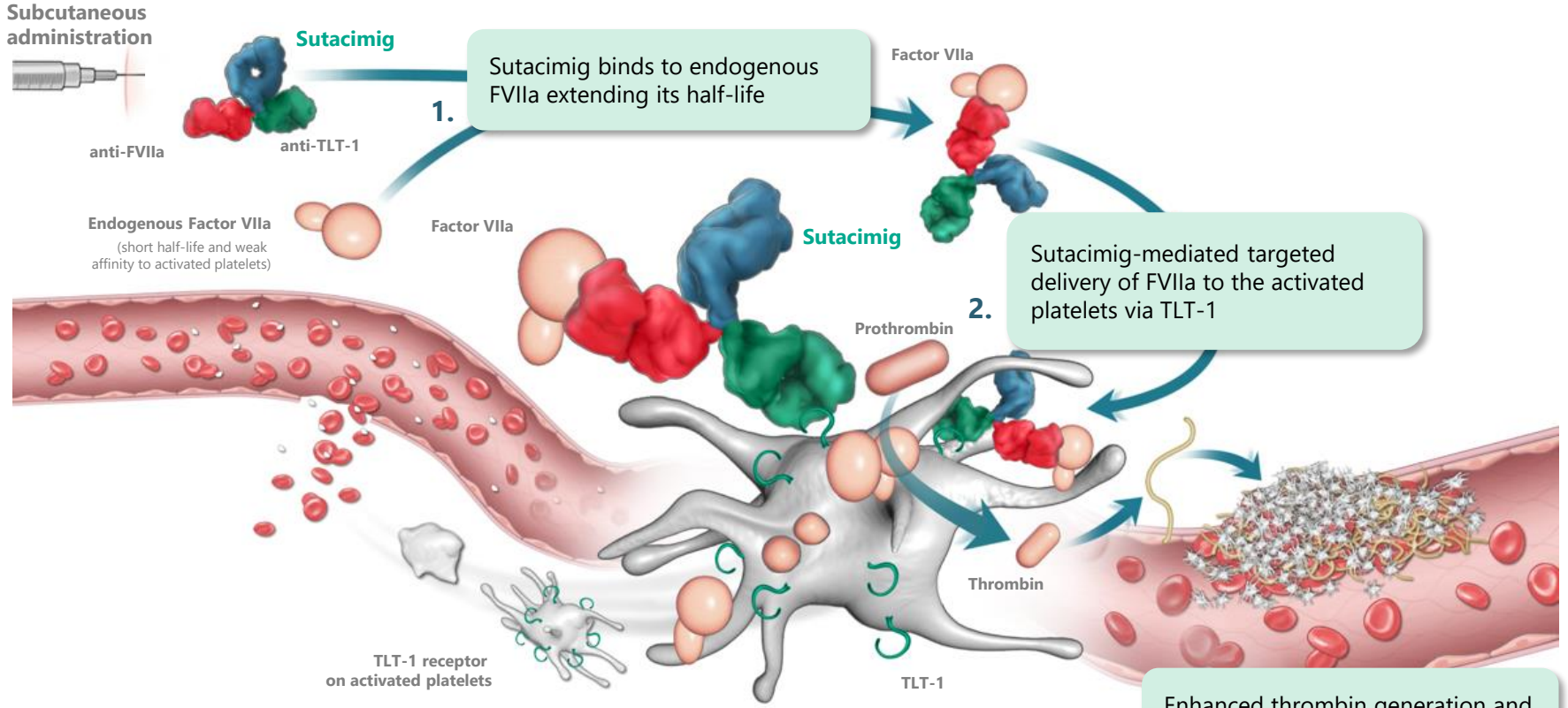
Other options (platelet transfusion, antifibrinolytics, iron/component supplementation) address consequences, not prevention. GT prevalence: ~1:100K (GCC*); ~1:350K–600K in the US. FVIID: ~1:500K globally (moderate/severe). Note: Incl. quantitative survey & qualitative interviews. Source: Res Pract Thromb Haemost. 2024 Oct 9;8(7):102586. N=117 (GT360), N=100 (FVIID360). *GCC=Gulf Cooperation Council.

Sutacimig targets endogenous FVIIa to activated platelet surface, leading to potentiation of FVIIa activity

Bispecific antibody

Low-volume (<1 ml) SQ dose

TLT-1 potentiated FVIIa-mediated thrombin generation

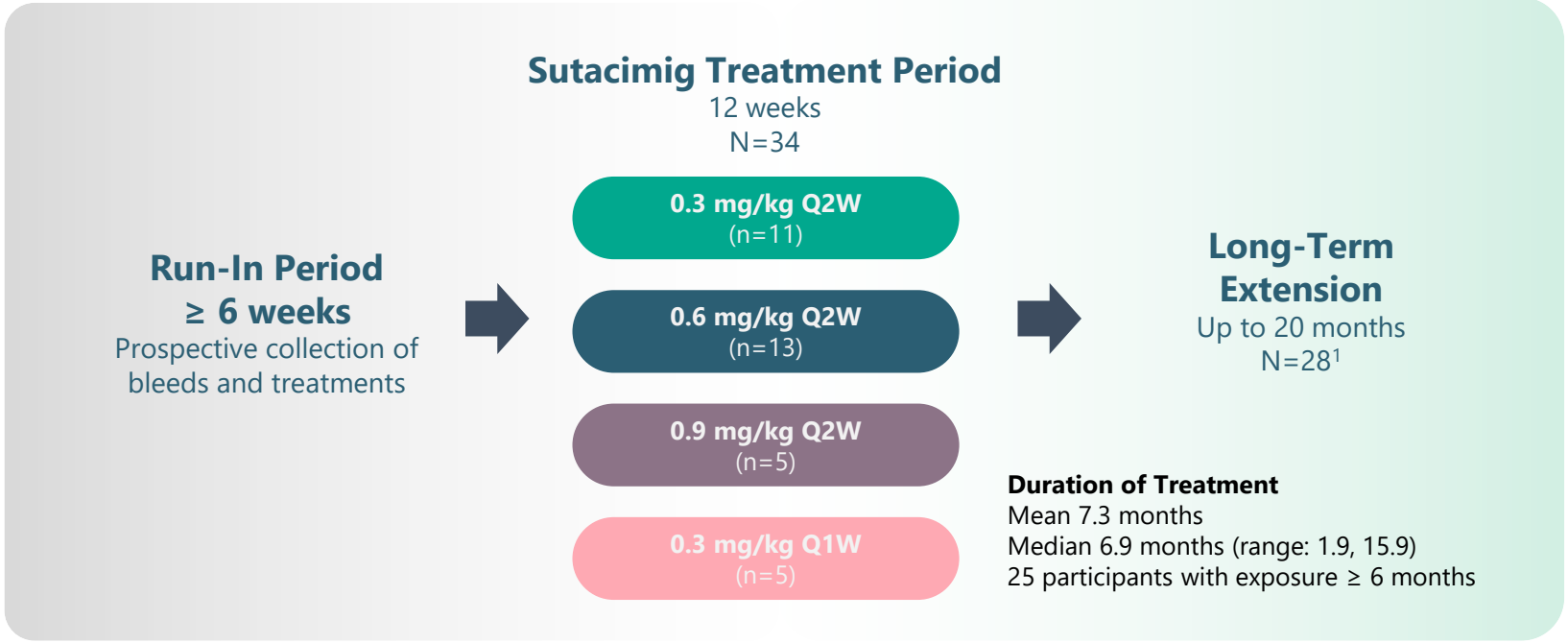


Sutacimig binds endogenous FVIIa and, following vessel lesion, localizes FVIIa to the surface of activated platelets via TLT-1, potentiating activity at the site of bleeding.
TLT-1 is expressed only on activated platelets targeting FVIIa delivery to sites of injury and potentiating its activity.

Abbreviations: FVIIa, activated factor VII; SQ, subcutaneous; TLT-1, TREM-like transcript-1.
References: Gandhi PS, et al. Nat Cardiovasc Res. 2024.

Phase 1/2 Study Evaluating Sutacimig in Glanzmann Thrombasthenia

Multiple Ascending Dose (MAD) Evaluation with Long-Term Extension



Baseline ATBR during run-in[2]	Mean[3] (SEM); range Median (Q1, Q3)	**40.2** (11.8); range: 0-324.7 **17.7** (4.0, 47.6)

Objectives

- Examine safety and tolerability of sutacimig
- Estimate effect on annualized treated bleeding rate (ATBR)
- PK/PD

Eligibility criteria

- Age 18–67 years
- Confirmed GT diagnosis
- History of bleeding requiring treatment

Preceded by SAD (Part A)

- 0.2 mg/kg, 0.5 mg/kg and 1.25 mg/kg

Abbreviations: Q2W, every two weeks; Q1W, every one week; SQ, subcutaneous. Data available as of interim data cut-off of October 14, 2025. Additional study details available at ClinicalTrials.gov NCT06211634. [1]Two participants terminated the 12-week treatment period prior to completion, 1 each due to adverse event and physician decision due to participant noncompliance. Four participants did not consent to the long-term extension, 3 for logistical reasons (travel to site, scheduling) and 1 had ongoing medical issues that interfered with trial participation. [2] Efficacy set excludes 3 participants: 2 due to run-in period <6 weeks and 1 due to noncompliance with bleed diary guideline. [3] Weighted by number of evaluable days during the run-in period

Sutacimig

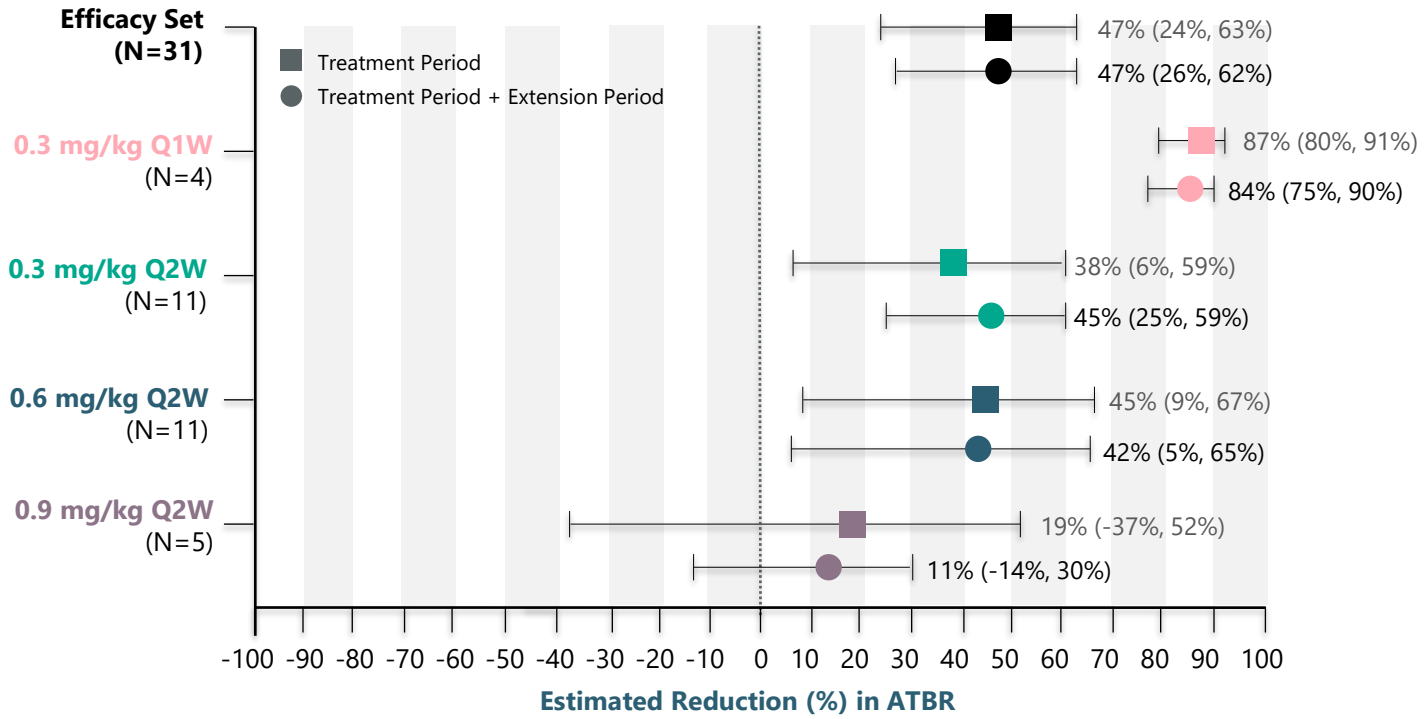
Reduction in Mean ATBR Across Multiple Dose Cohorts

12-Week Treatment Period vs Treatment + Extension Periods (median exposure 6.9 months)

- **Persistent and clinically meaningful ATBR reduction observed**, including over extended treatment
- ATBR reduction across all dose levels
- Enhanced response with weekly dosing

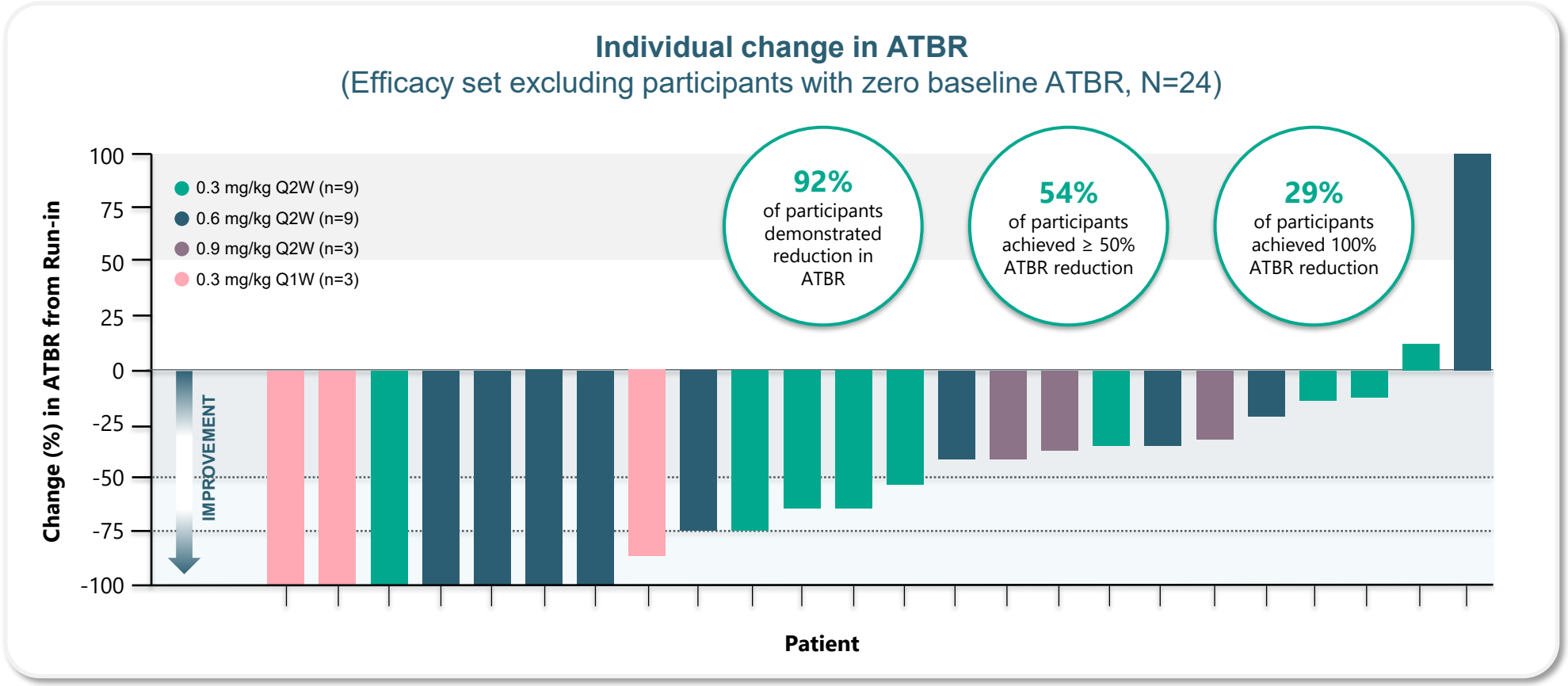
Estimated reduction in ATBR with 95% confidence interval (Efficacy set, N=31)

Treatment Period vs Treatment + Extension Periods



A GEE negative binomial regression model with an offset for duration of study period was fitted to estimate the mean ATBR during each study period and reduction in mean ATBR along with corresponding 95% CIs.
Efficacy analyses included comparison of the run-in and 12-week treatment + extension periods (data cut-off October 14, 2025).

ATBR Reduction Observed Across All Dose Cohorts and in the Majority of Participants

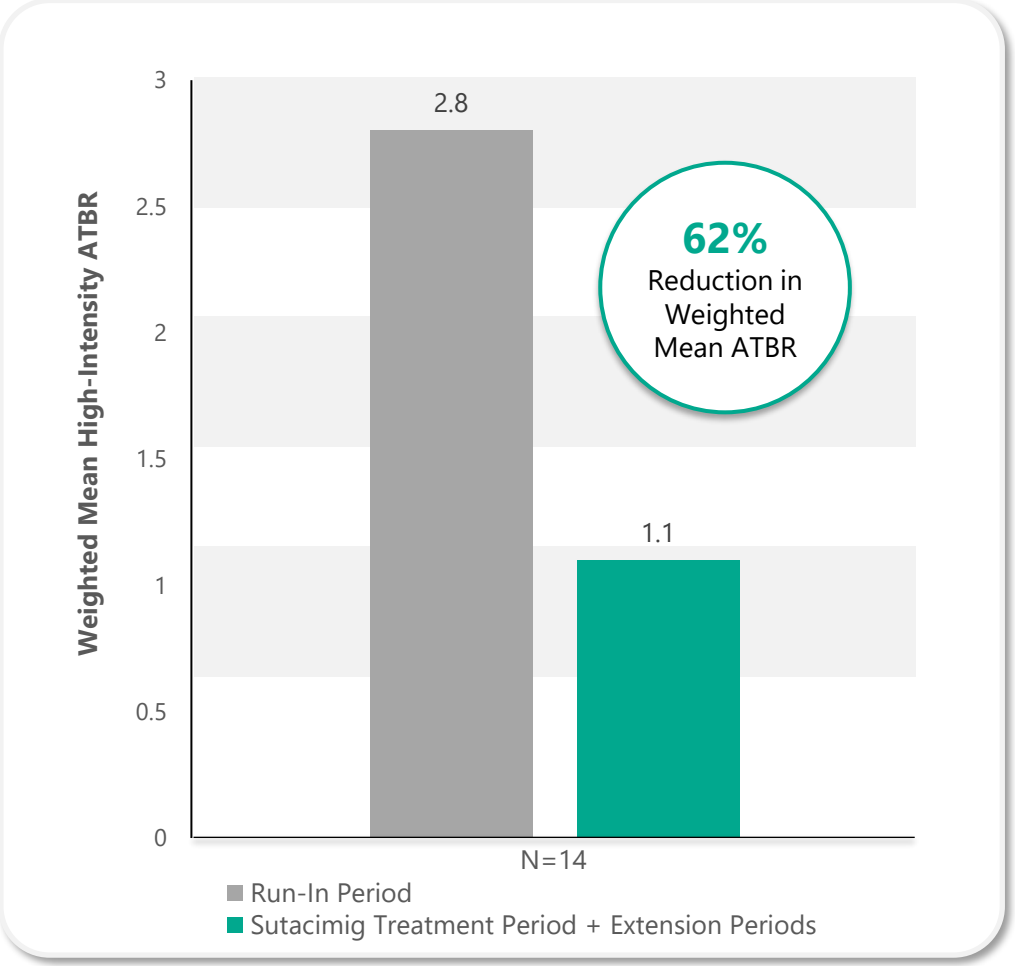


Note: A GEE negative binomial regression model with an offset for duration of study period was fitted to estimate the mean ATBR during each study period and reduction in mean ATBR along with corresponding 95% CIs.
Efficacy analyses included comparison of the run-in and 12-week treatment period for Efficacy Set excluding participants with zero ATBR during run-in period (N=24, data cut-off October 14, 2025).

Marked Reduction in Bleeding Events Treated with High-Intensity Treatments

Subpopulation with Prior High-Intensity Treatment Use – Run-In vs Treatment + Extension Periods

- **Mean reduction of high-intensity ATBR of 62%**
- High-intensity treatment definition:
  - Systemic hemostatic intervention (rFVIIa, platelet transfusion, plasma, cryoprecipitate)
  - Medical, surgical, or radiologic procedures for hemostasis



Abbreviations: rFVIIa: recombinant factor VIIa

A subset of participants were identified as experiencing bleeding events treated with high-intensity treatments within 12-month period prior to first dose of sutacimig. Efficacy analyses included comparison of the run-in and 12-week treatment + extension periods (data cut-off October 14, 2025).

Sutacimig Use During Procedures

Successful hemostatic outcomes with minimal intervention

Gingival Plaque Removal

0.6 mg/kg Q2W

rFVIIa **5 mcg/kg IV** + topical rFVIIa

✓ **Successful hemostasis**

Moh's Surgery, Basal Cell Carcinoma, Scalp

0.9 mg/kg Q2W

Oral tranexamic acid (peri-procedural)

✓ **Successful hemostasis**

Partial Mastectomy & Lymph Node Biopsy

0.3 mg/kg Q1W

IV TXA 1 g + rFVIIa **7 mcg/kg** (pre-procedure)
Oral TXA 1300 mg TID × 10 days (discharge)

✓ **Successful hemostasis**

Individualized bleed management plans were followed during sutacimig treatment, including administration of lower than standard dose of rFVIIa, if needed

Abbreviations: rFVIIa: recombinant factor VIIa, IV TXA: intravenous tranexamic acid
Data available as of interim data cut-off of October 14, 2025.

Sutacimig Generally Well Tolerated in Combination of Part B + C

Median Exposure: 6.9 months (range: 1.9, 15.9) – Data Cut as of 14-Oct-25

Majority of observed TEAEs were mild to moderate in severity, and without specific pattern

- Headache (23.5%), Fibrin D-dimer increased (23.5%) nasopharyngitis (20.6%), back pain (17.6%) most common

No related TEAE ≥ Grade 3[1]

Grade 2 VTEs[1]

- N=3 patients, Assessed as related
- All treated at higher dose levels and/or
- ≥ 3 concurrent VTE risk factors present
- Drug discontinued , received anticoagulation
- All were resolved (SSPE, IDDVT) or resolving (DVT) at last visit

6/34 participants developed ADA titers

- 2/6 received single dose in Part A prior to Part B
- 3/6 resolved/resolving at datacut
- No associated safety events
- FVIIa did not drop below LLN

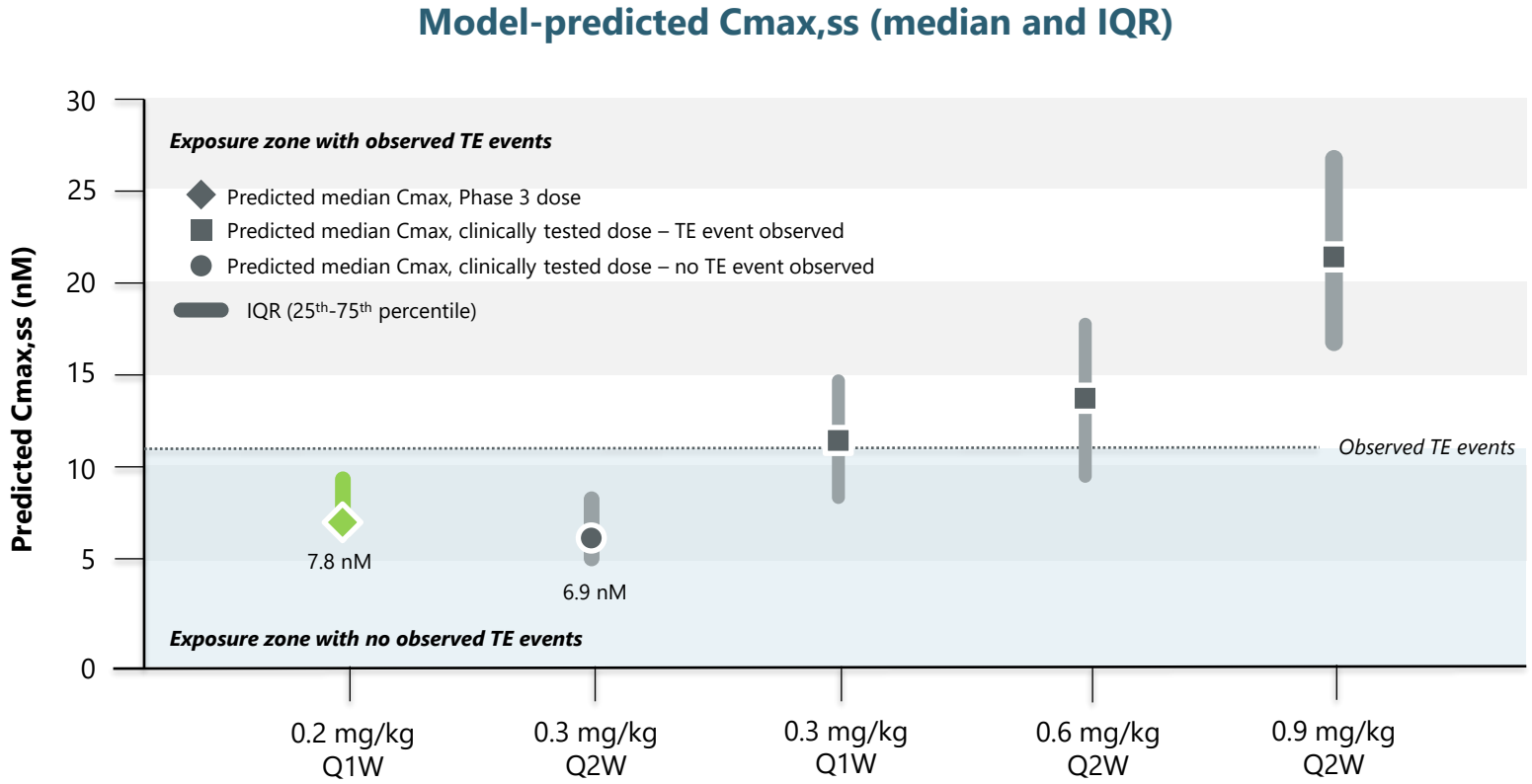
	Treatment + Extension Period Part B + Part C (N=34)
All TEAE	32 (94.1)
Related TEAEs[a]	15 (44.1)
Grade 3+ TEAEs by Maximum CTCAE Grade[2]	7 (20.6)
Serious Adverse Events	5 (14.7)
Deaths	0
Related Serious Adverse Events	2 (5.9)
TEAEs leading to Study Discontinuation	4 (11.8)

Pt	TEAE by PT	AE Description	SAE	Grade	Related	Drug discontinued due to AE	Assigned Dose	Last Dose Prior to Event	Potential contributing risk factors
1	DVT	Proximal DVT lower extremity	Yes	2	Yes	Yes	0.9 mg/kg Q2W	0.9 mg/kg Q2W	Hypertension; Obesity; Nicotine – vaping
2	DVT	Isolated distal DVT lower extremity	No	2	Yes	Yes	0.6 mg/kg Q2W	0.6 mg/kg Q2W	Age >40 yrs; Obesity; Nicotine – smoking
3	Pulmonary embolism	Subsegmental PE	Yes	2	Yes	Yes	0.6 mg/kg Q2W	0.3 mg/kg Q1W	Age >40 yrs; Active GI bleed; Hospitalization; Platelet transfusion

[1]7 pts experienced Grade 3 AE : bleeding events due to underlying GT (3) , back pain (1), neutropenia (1), invasive ductal carcinoma(1) and 1 pt with dental carries & post procedural hemorrhage. Grade 3 GT-related bleeding events consist of GI hemorrhage (1), intra-abdominal hemorrhage(1- same pt as SSPE), epistaxis (1). [2]Venous thrombotic events, [3] If a patient experienced more than one adverse event, the participant was counted once at the most severe or most related event . [4].Total SAE(5): 2 VTE above and the 3 GT bleeding events above. SSPE = subsegmental pulmonary embolism ; IDDVT: Isolated distal deep vein thrombosis; DVT: Deep vein thrombosis; LLN: Lower limit of normal, Pt = participant, PT = Preferred Term, ,SAE = Serious Adverse Event, TEAE = Treatment-emergent adverse event, CTCAE = common terminology criteria for adverse events.. 5. AE leading to study drug discontinuation: 3 VTE above and invasive ductal carcinima (1).

Phase 3 Dose: Potential to Optimize for Safety and Efficacy

- 0.2 mg/kg Q1W Cmax comparable to 0.3 mg/kg Q2W

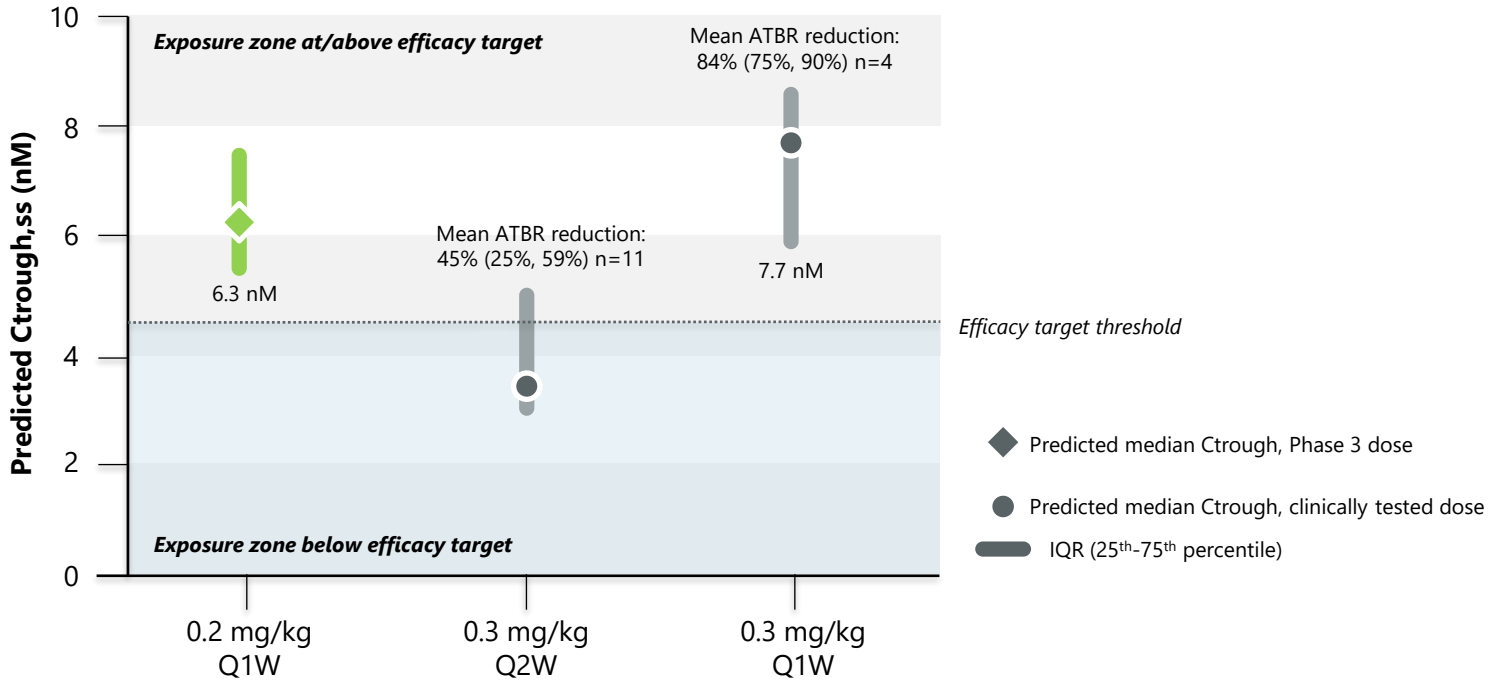


Cmax,ss values are model-predicted medians with IQR band from exposure-response modeling and simulation (N=1500 simulated patients).
Abbreviations: IQR, interquartile range; ss, steady state

Phase 3 Dose: Potential to Optimize for Safety and Efficacy

- Substantial elevation of trough with weekly dosing
- 0.2 mg/kg Q1W predicted to achieve exposure levels at or above efficacy target threshold

Model-predicted Ctrough,ss (median and IQR)



Patients with predicted Ctrough,ss > 4.5nM (N=23) showed a 2-fold greater mean ATBR reduction than those below this threshold

Ctrough, ss values are model-predicted medians with IQR band from exposure-response modeling and simulation (N=1500 simulated patients).
Abbreviations: IQR, interquartile range; ss, steady state

Planned Phase 3 Study in Glanzmann Thrombasthenia

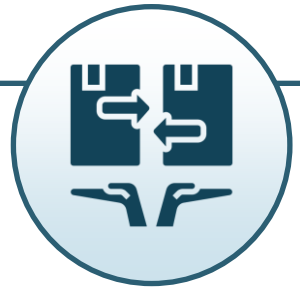
1

Multinational Multicenter Trial

2

Open Label

3



FDA endorsement

Phase 3 dose of 0.2 mg/kg Q1W

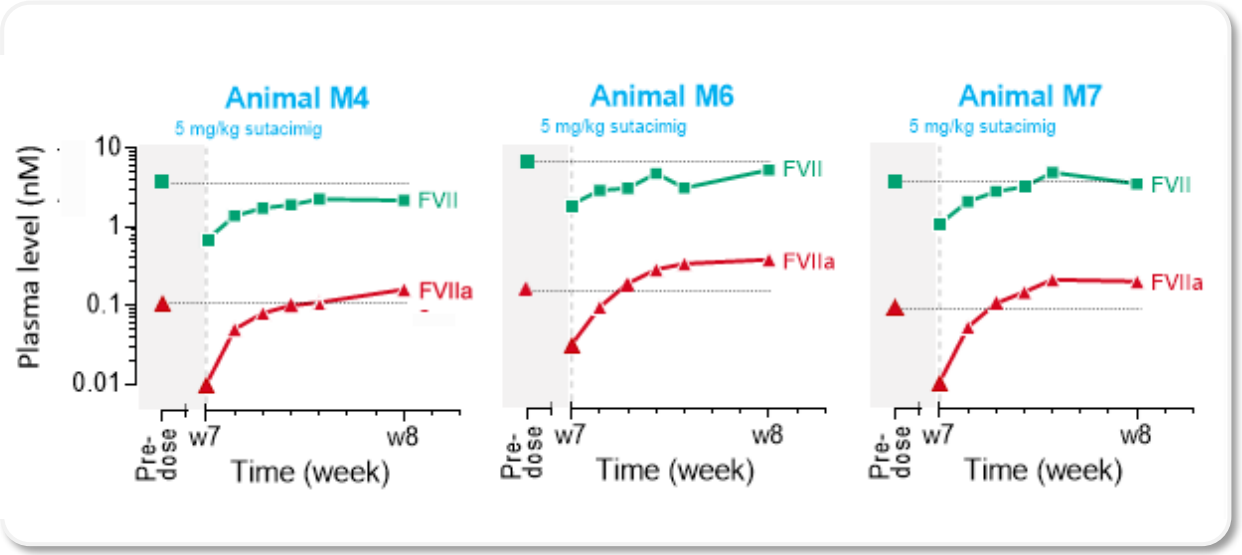
Planned Phase 3 study expected to initiate in 2H 2026

Sutacimig in Factor VII Deficiency

Preclinical POC of Sutacimig for Factor VII Deficiency

Accumulation of FVII/FVIIa to within normal range in NHP FVIID model

- NHP model[1] with 11-13% residual activity
- FVIID rose to within normal range following SC sutacimig (5 mg/kg)



Ongoing Phase 2B

- Open-label
- Single SC dose
- Severe congenital FVIID FVII:C < 10%

Objectives: Safety/tolerability, PK PD through Day 57

Interim analyses for safety & PD expected late 2026 / early 2027

Abbreviations: POC: proof of concept, NHP: non-human primate, SC: subcutaneous, PK: Pharmacokinetics, PD: Pharmacodynamics

[1] NHP Model: Sustained FVII knockdown induced using siRNA (XL10-LNP)-mediated knock-down of endogenous FVII in cynomolgus monkeys

HMB-002

- Von Willebrand Disease Type 1,2,3
- Bleeding related to low levels of VWF

HMB-002

Von Willebrand Disease (VWD)

Disease Burden

Type 1 ~70-80%, **Type 2** ~20% , **Type 3** <1%

1.33 bleeds/week

Type 1: 1.2/wk Type 2: 1.4/wk Type 3: 1.9/wk
Mean per patient (range 0–10)

Similar bleeding patterns across all VWD types despite different severity classifications

IMPACT & OVERALL BURDEN

93%
of female participants reported heavy menstrual bleeding

57%
of females reported a bleed-related hospital admission (past year)

35–40%
have iron deficiency and anemia

Market Opportunity

120K

~120,000 symptomatic VWD patients in key addressable markets (US, EU, Japan, GCC)

140,000 diagnosed in the US (all severities), more than 50k require treatment, 1 in 5,000–10,000 global moderate-to-severe disease prevalence

Current VWD treatment is generally reactive, short-acting, IV-based, and burdensome

1. Leebeek FWG et al. N Engl J Med. 2016;375:2067-2080. 2. Connell NT et al. ASH ISTH NHF WFH 2021 Guidelines. Blood Adv. 2021;5(1):301-325. 3. Bowman M et al. J Thromb Haemost. Volume 8, Issue 1, 213–216. 4. Komodo claims analysis presented at ASH 2024. N=611 (ISTH 2025, Washington, USA – Poster). Type 1 (217), Type 2A (97), Type 2B (49), Type 2M (55), Type 2N (40), Type 3 (45).

HMB-002: Monoclonal Antibody to VWF Designed to Increase VWF and FVIII

HMB-002 Designed to:

Accumulate VWF to restore hemostasis in VWD

- Rescues VWF from degradation through engagement of the FcRn recycling pathway[1]
- *Primary Hemostasis*: Elevated VWF enhances platelet recruitment
- *Secondary Hemostasis*: Elevated VWF levels drive accumulation of FVIII and support thrombin generation & clot formation

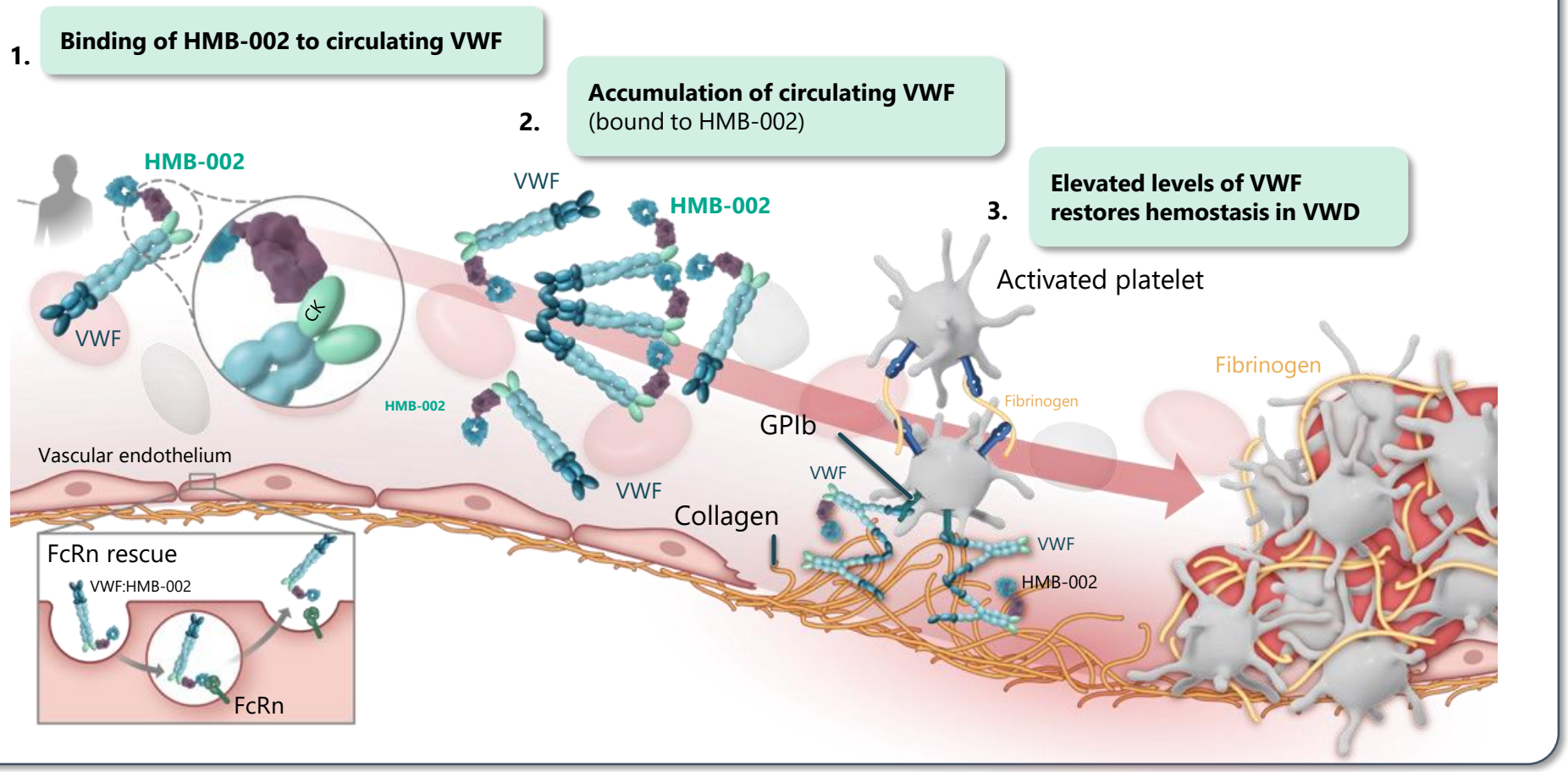
Preserve Regulation

- Maintains ADAMTS13-mediated VWF processing

Clinically validated therapeutic hypothesis

- Increasing endogenous VWF is clinically validated by 50 years of desmopressin use

Mechanism of Action



References: 1. Häger M, et al. ISTH 2025. Oral OC 08.4. 2. Häger, Mattias, et al. "HMB-002: A Monovalent Antibody that Elevates Circulating VWF and FVIII Levels for Treatment of Von Willebrand Disease." Blood Advances (2026).
Abbreviations: VWD, Von Willebrand Disease; VWF, von Willebrand factor.
Desmopressin is a synthetic hormone that lowers urine production and helps blood clot

VELORA Program Overview

Enrolling

VELORA Discover

PROSPECTIVE, NATURAL HISTORY STUDY

(N=100+)

Large prospective natural history study capturing real-world bleeding burden and quality of life in patients with VWD.

Patient progression

VELORA Pioneer

PHASE 1/2 CLINICAL STUDY OF HMB-002

- **VELORA Pioneer** is a three-part protocol
  - **Part A:** evaluate PK, PD, and safety of HMB-002 following a single fixed SC dose — Enrolling
  - **Part B:** evaluate PK, PD, and exploratory efficacy following multiple SC doses — Enrolling
  - **Part C:** evaluate single dose HMB-002 with concomitant IV factor concentrate — Enrolling

KEY INCLUSION CRITERIA:

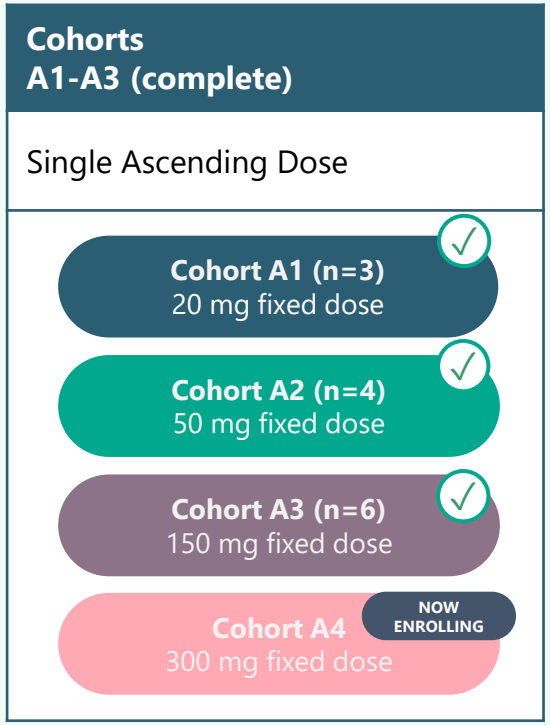
- Type 1 VWD (including Type 1C), Type 2A, and Type 3 (Part C only)
- VWF activity ≤50% at baseline | FVIII activity ≤70% at baseline
- Males and females | Age 18 to 65 years
- Minimum 3 treated bleeds per year
- Oral contraception containing estrogen allowed

Abbreviations: IV, intravenous; PD, pharmacodynamics; PK, pharmacokinetics; SC, subcutaneous; VWD, Von Willebrand disease. Full eligibility criteria available at ClinicalTrials.gov (NCT06754852). Interim data cutoff as of 07 May 2026.

HMB-002

VELORA Pioneer: Phase 1/2 Study of HMB-002 in Individuals with VWD

Baseline Demographics and Disease Characteristics



	Cohort A1 20 mg (n=3)	Cohort A2 50 mg (n=4)	Cohort A3 150 mg (n = 6)	Total (n=13)
Demographics				
Age (yr), mean (range)	39 (27 – 62)	30 (21 – 53)	37 (20 – 54)	35 (20 – 62)
Sex, n (%)				
Female	1 (33)	3 (75)	3 (50)	7 (54)
Male	2 (67)	1 (25)	3 (50)	6 (46)
Weight (kg), mean (range)	74 (62 – 88)	73 (70 – 78)	69 (54 – 80)	72 (54 – 88)
VWD Subtypes	Type 1: 3 (100)	Type 1: 4 (100)	Type 1: 4 (67) Type 1C: 2 (33)*	Type 1: 11 (85) Type 1C: 2 (15)*
Baseline Laboratory Values, mean (range)				
VWF:Ac (%)	24.2 (21.1 – 26.0)	19.2 (9.5 – 36.0)	18.4 (7.3 – 29.4)	19.9 (7.3 – 36.0)
VWF:Ag (%)	23.6 (23.0 – 23.9)	22.4 (12.8 – 39.8)	16.9 (8.4 – 30.2)	20.2 (8.4 – 39.8)
FVIII:C (%)	43.7 (42.1 – 46.8)	38.9 (17.2 – 60.1)	29.8 (4.8 – 63.1)	35.8 (4.8 – 63.1)

*A total of 3 participants have Type 1C VWD; one participant was reclassified as Type 1C in the clinical database following the data cut.
Abbreviations: FVIII:C, factor VIII coagulant activity; VWD, Von Willebrand Disease; VWF:Ac, Von Willebrand Factor activity; VWF:Ag, Von Willebrand Factor antigen
Full eligibility criteria available at ClinicalTrials.gov (NCT06754852). Interim data cutoff as of 07 May 2026.

HMB-002

Interim Safety Summary

The emerging safety profile of HMB-002 remained favorable across all cohorts studied [a]

- One participant experienced a Grade 1 increase in fibrin D-dimer, considered secondary to strenuous physical activity and unrelated to HMB-002

Key Findings

- **Most TEAEs were mild to moderate in severity and resolved**
- **No serious TEAEs**
- **No TEAEs considered related to HMB-002**
- **No discontinuations due to TEAE**
- **No thromboembolic events, injection site reactions, thrombocytopenia, or hypersensitivity**
- **No trends in inflammatory markers or vital signs**
- **Transient ADA increase in one Cohort A3 participant; no safety or PK/PD impact**

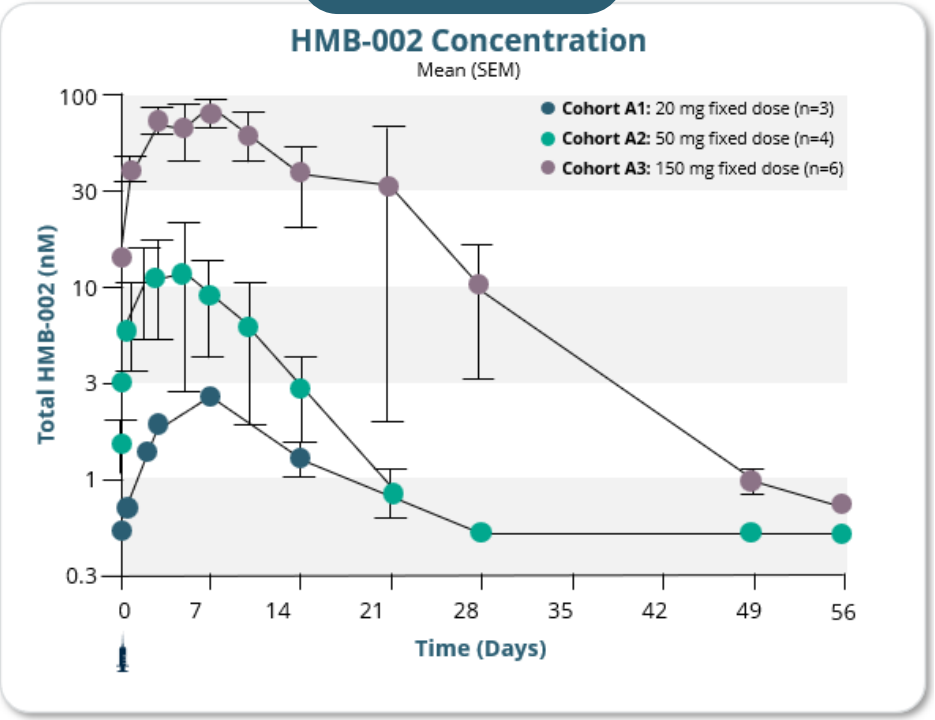
Category	N=13 [b] n (%)
All TEAEs	7 (53.8%) [c]
≥ Grade 3 TEAEs	1 (7.7%)
Serious TEAEs	0 (0%)
TEAEs leading to discontinuation	0 (0%)

Cohort	#TEAEs
A1	0
A2	7
A3	5

[a] Cohort A1 and Cohort A2 completed (56-days follow-up) and Cohort A3 on study (28-72 days follow-up); [b] N = number of participants and (%) = number of participants/N*100; [c] If a participant experienced more than on TEAE, the participant is counted once at the most severe or most related event; Abbreviations: ADA, anti-drug antibody; PD, pharmacodynamics; PK, pharmacokinetics; SAE, serious adverse event; TEAE, treatment-emergent adverse event. Interim data cutoff of 07 May 2026.

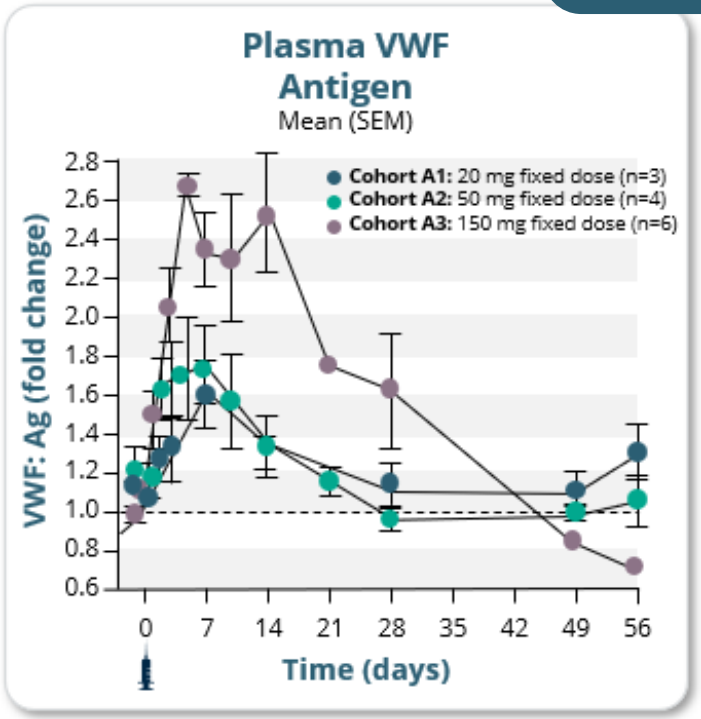
Dose-dependent Increase in PK Achieved >2-Fold Peak Elevation in PD Markers

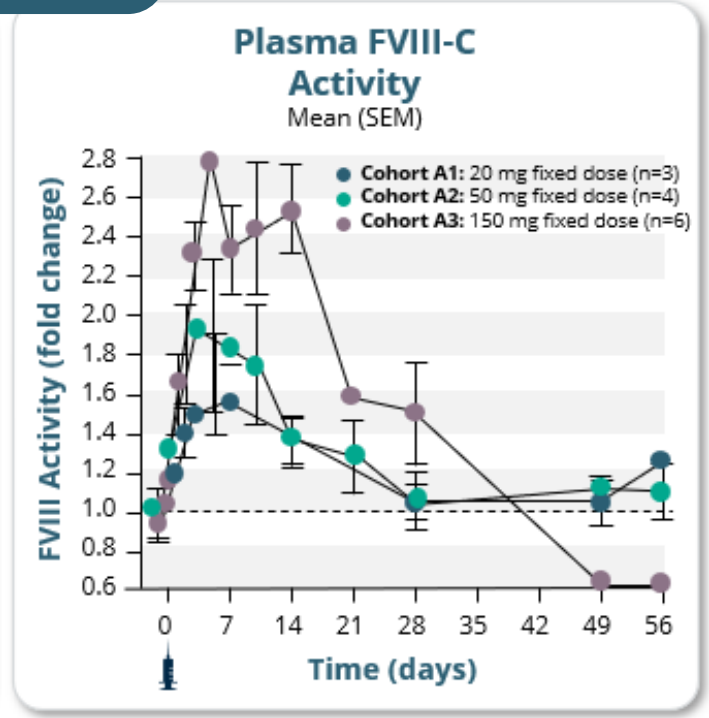
PK



- Observed a dose-dependent increase in C_{max}
- T_{max} was observed between Day 6-9
- Cohort A3 (150 mg) maintained detectable levels out to Day 56, suggesting extended duration potential

PD





- Dose-related increases in VWF antigen and FVIII activity were observed following HMB-002 administration
- Higher dose levels were associated with greater and more sustained PD responses
- ≥2.4-fold VWF antigen and FVIII activity at 150 mg dose
- At all doses, VWF and FVIII activity remained within protocol defined upper limit of normal (<200 IU/dL)
- Multimer distribution remained stable through Day 29 across all dose levels

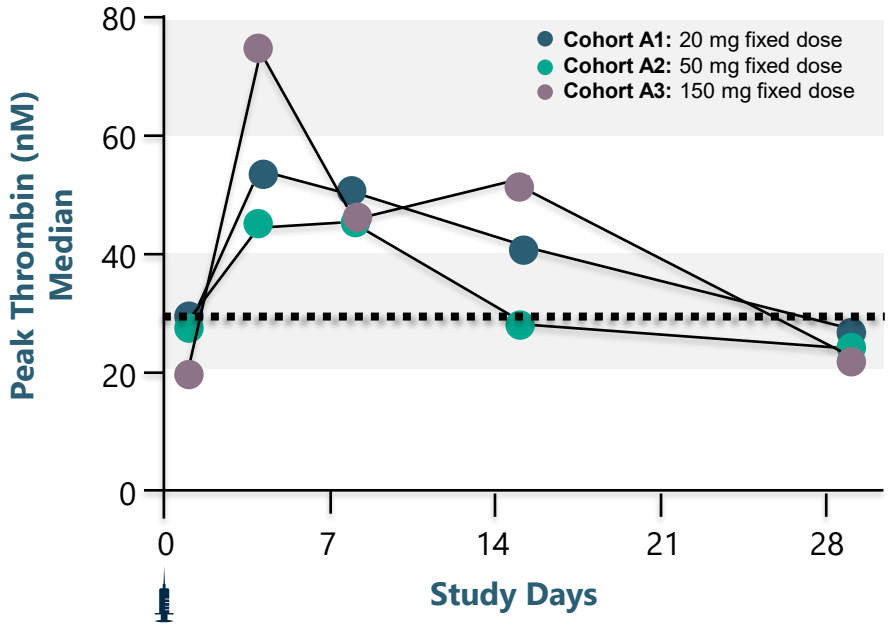
Abbreviations: FVIII:C, factor VIII coagulant activity; VWD, Von Willebrand Disease; VWF, Von Willebrand factor; VWF:Ag; PD, pharmacodynamics; SEM, standard error of the mean

HMB-002

Parallel Normalization of TG and APTT Correction Consistent with FVIII Accumulation

Thrombin Generation

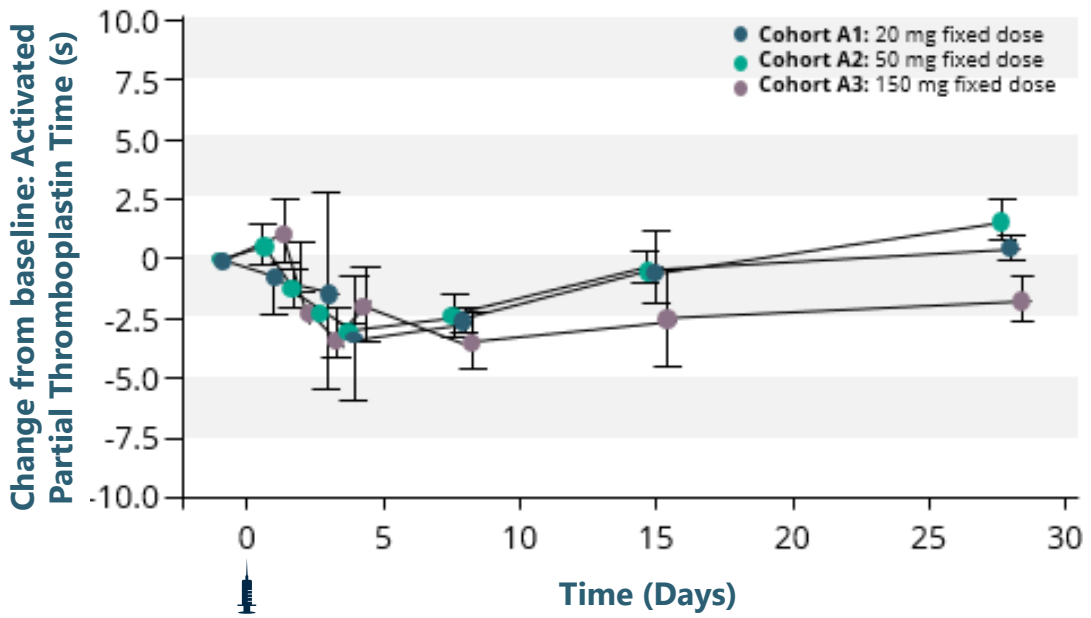
Median Peak Thrombin Levels (nM)



Thrombin generation was elevated to within normal range in all cohorts (reference range: 29.0 – 172.7 nM)

APTT Improvement

Median Peak Thrombin Levels (nM)



Improvement of APTT as measured by standard assays observed across all cohorts

Abbreviations: FVIII, factor VIII; APTT, activated partial thromboplastin time; SEM, standard error of the mean; TG, thrombin generation.
Methods: Calibrated Automated Thrombography, 0.5 pM tissue factor, platelet-poor plasma.
Note: Exploratory endpoint in Phase 1 (n=2, 4 and 5 evaluable in Cohorts A1, A2, and A3, respectively; two participants excluded due to baseline interference, reference interval in healthy donors: 29.0 – 172.7 nM thrombin).

Preliminary Clinical Observations

	Velora Discover Baseline ATBR 10 of 13 with baseline diary data[a]	Velora Pioneer Post HMB-002 ATBR 9 of 13 with post-dose diary data [b]
Mean	20.1	1.6
Median	5.8	0.0
Range	0.0 – 101.5	0.0 – 14.6 [c]

Velora Discover: number of evaluable days, mean (range): 97.3 (7 – 166)
Velora Pioneer: number of evaluable days, mean (range): 22.7 (22 – 25)

- **Prospectively collected bleed data before HMB-002**
  - 2 out of 10 of participants had zero treated bleeding events on Velora Discover during the up to ~5.5 months observation period
- **Prospectively collected bleed data post HMB-002**
  - 8 out of 9 of participants experienced zero treated bleeding events on Velora Pioneer in the 28-day observation period d following a single dose of HMB-002

[a] Bleed diary data unavailable for 3 participants; Velora Discover participation is not required for enrollment on Pioneer Part A. [b] Bleed diary data unavailable for 4 participants; diary completion was optional in Cohorts A1 and A2. [c] One treated bleed was reported in 1 participant in Cohort A2 on Day 4. [d] Velora Pioneer bleed analysis includes bleeds reported between Day 4-28 to reflect the pharmacological profile of HMB-002. ATBR, annualized treated bleed rate is derived as the total number of treated bleeds/evaluable days (year) during Velora Discover or Pioneer study. Evaluable days: days with a valid date entered in the bleed diary, regardless of the reported number of bleeds. Analyses based on data cutoff of 04 May 2026; data are preliminary and subject to change following further review and cleaning of the ongoing clinical study database

HMB-003

Heavy Menstrual Bleeding
Potentially Hereditary Hemorrhagic Telangiectasia,
Peri-operative Bleeding Management,
and other conditions

HMB-003

Heavy Menstrual Bleeding (HMB)

Disease Burden

1 in 3

reproductive-age women affected by heavy menstrual bleeding[1,2]

#1

Most common cause of iron deficiency and iron-deficiency anemia in reproductive-age women[3]

2x

Rate of depression in adolescents with HMB[4]

>$94B

annual cost to the US economy; **3.6 working weeks lost per year**[5]

~50%

switch hormonal treatment within 1 year[5]

Market Opportunity

23M

Over 23M women affected by heavy menstrual bleeding annually in the US alone

One of the most common conditions women report to their healthcare provider

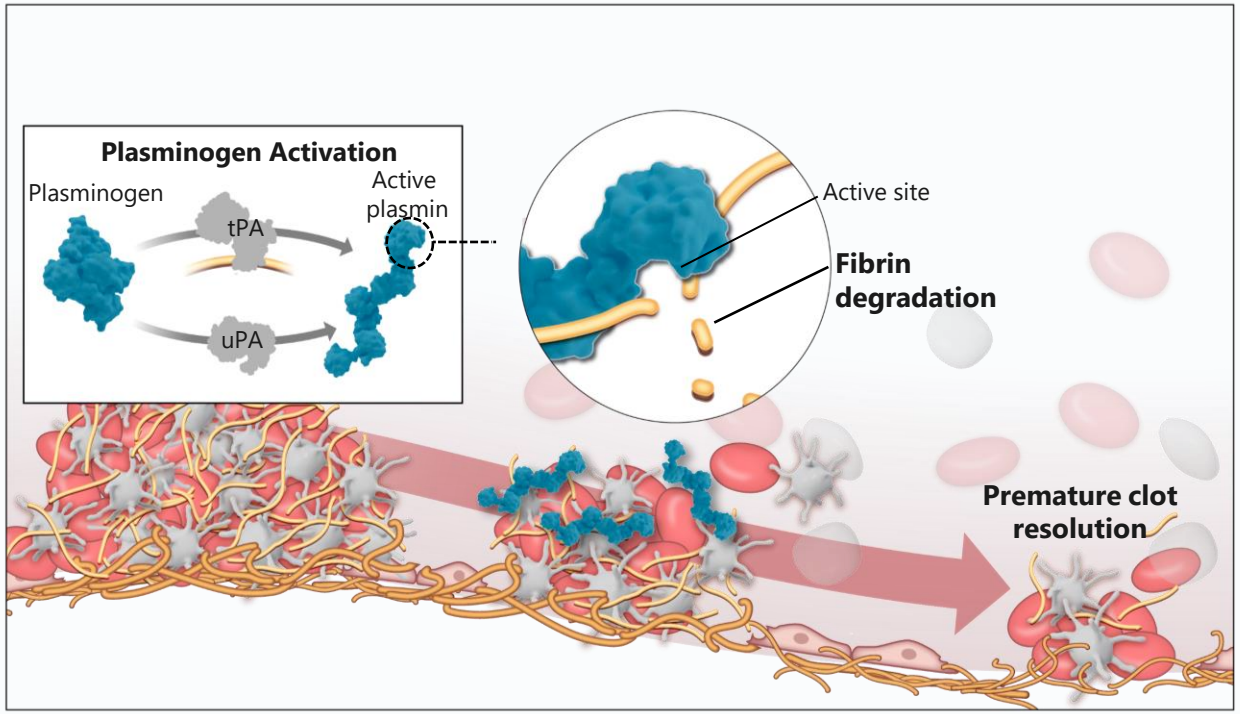
Only approved non-hormonal treatment requires multiple daily doses throughout menses

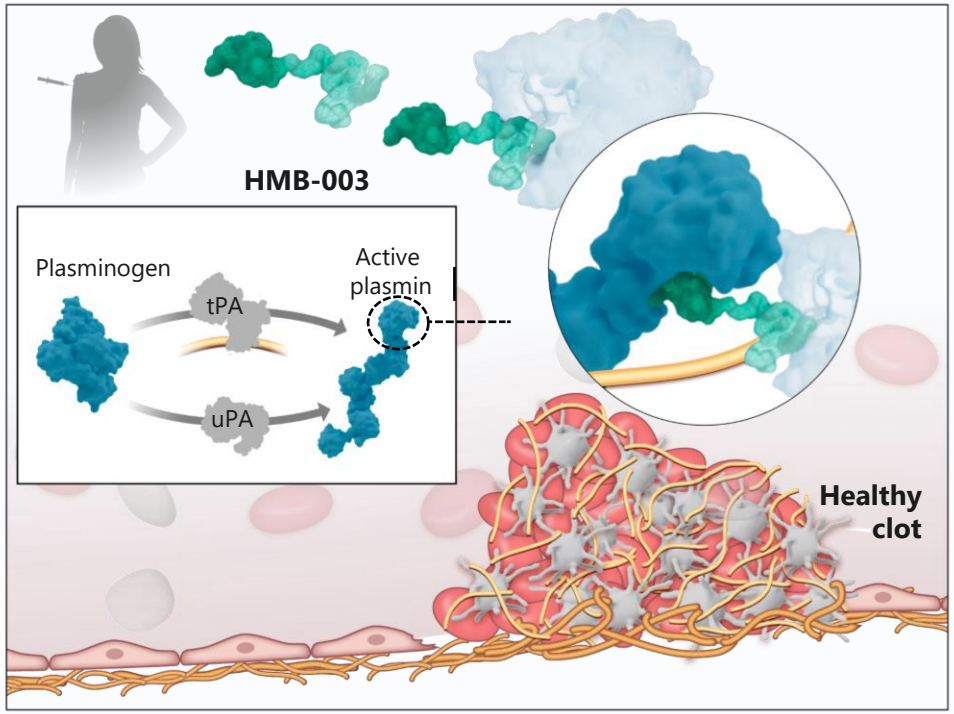
[1] In clinical terms, the NHS defines HMB as the loss of 80ml or more in each period, having periods that last longer than 7 days, or both. On average women lose between 30-40ml of blood during a period. [2] Fraser, I.S., Mansour, D., Breymann, C., Hoffman, C., Mezzacasa, A. and Petraglia, F. (2015), Prevalence of heavy menstrual bleeding and experiences of affected women in a European patient survey. International Journal of Gynecology & Obstetrics, 128: 196-200. https://doi.org/10.1016/j.ijgo.2014.09.027. Other sources: CDC, About Heavy Menstrual Bleeding, cdc.gov, rev. May 2024; Wellcome Leap, The Missed Vital Sign, wellcomeleap.org, accessed Jul 2026 [3]Munro MG, Int J Gynecol Obstet 2023;162(S2):7-13. [4]Weyand et al., J Pediatr 2022;240:171-176. [5]Wellcome Leap, 2025.

HMB-003 Mechanism of Action Inhibits Plasmin to Prevent Premature Fibrinolysis

HMB-003 is designed to block excess plasmin to prevent premature clot breakdown

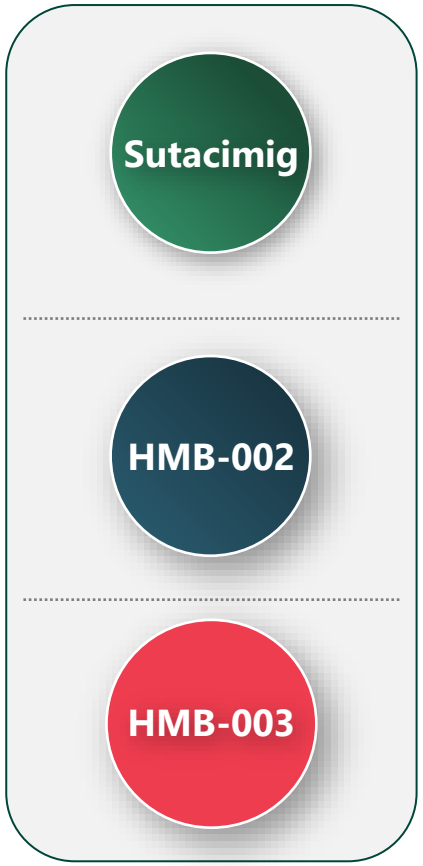
- *Without inhibition:* tPA/uPA convert plasminogen to active plasmin, degrading fibrin and driving premature clot resolution
- *With HMB-003:* Plasmin activation is blocked, preserving fibrin so the clot stabilizes normally. Targets the root cause of excess menstrual blood loss, not just symptoms
- *Non-Hormonal, Targeted Approach:* Acts locally on the fibrinolytic pathway without altering hormonal signaling or systemic clotting





Abbreviations: tPA: Tissue Plasminogen Activator , uPA: Urokinase-type Plasminogen Activator
Source: Rolf Urbanus, et al. ISTH 2026. Oral OC 05.1

Anticipated Clinical Events



2026		2027
H2		H1

Glanzmann thrombasthenia:
Commencement of Phase 3 clinical trial

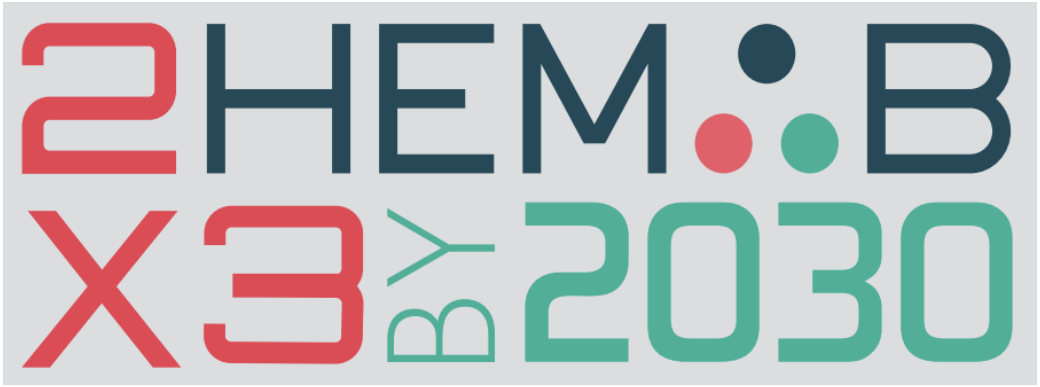
Factor VII Deficiency:
Phase 2B clinical data (late 2026 / early 2027)

Von Willebrand Disease:
Phase 1/2, Part B initial clinical data (late 2026 / early 2027)

Heavy Menstrual Bleeding:
Expected to enter clinical development

Heavy Menstrual Bleeding:
Initial clinical data
(mid-2027)

Executing an Ambitious Strategic Roadmap to Deliver Therapeutics with Meaningful Clinical Benefit



2 Commercial Medicines

2 New Late-Stage Clinical Programs

2 New Early-Stage Clinical Programs

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HEMOB

Reimagining the Treatment of Coagulation Disorders