Hemab Therapeutics Presents New Clinical Data from HMB-002 in Von Willebrand Disease and Introduces HMB-003 for Heavy Menstrual Bleeding at the ISTH 2026 Congress

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HMB-002 clinical data demonstrate proof of mechanism, with dose-dependent, greater than or equal to 2.4-fold peak increases in Von Willebrand Factor (VWF) and Factor VIII (FVIII), normalization of peak thrombin generation and activated partial thromboplastin time (APTT), and durability supporting potential monthly subcutaneous dosing

HMB-003 program announced as non-hormonal direct plasmin inhibitor with potent, selective and extended antifibrinolytic activity, designed to reduce bleeding across multiple settings beginning in heavy menstrual bleeding

CAMBRIDGE, Mass. and COPENHAGEN, Denmark, July 12, 2026 (GLOBE NEWSWIRE) -- Hemab Therapeutics (Nasdaq:COAG), a clinical-stage biotechnology company developing therapies that reimagine the treatment of blood coagulation disorders to sustain life and human resilience, today presented new clinical data from HMB-002 in Von Willebrand disease (VWD) and announced the HMB-003 program at the International Society on Thrombosis and Haemostasis (ISTH) 2026 Congress in Paris, France.

"Millions of people living with bleeding disorders including VWD and heavy menstrual bleeding have no reliable way to prevent excessive bleeds," said Benny Sørensen, MD, PhD, CEO of Hemab. "Today, at ISTH 2026 we presented new HMB-002 data demonstrating proof of mechanism in VWD alongside preliminary clinical observations on treated bleeding events. We also unveiled HMB-003, a novel antifibrinolytic built on validated fatty-acid conjugated peptide technology, initially targeting heavy menstrual bleeding – an often-ignored vital sign. It affects one in three women, causing pain, iron-deficiency anemia, fatigue, stigma, and extensive lost days at school and work, with treatment options largely unchanged in decades. HMB-003 is non-hormonal, potent, and selective, engineered to cover the period, then wash out between cycles."

"The new HMB-002 data support a non-replacement approach in VWD: a single subcutaneous dose raising both VWF and FVIII more than 2-fold, with a favorable safety profile across every cohort," said Priyanka Raheja, MD, Consultant Haematologist at Barts Health NHS Trust. "Building on 50 years of clinically validated treatment strategy, HMB-002 shows potential to change the VWD treatment paradigm by addressing the root cause of the disease; a durable increase, and for many patients potential normalization, of both VWF and FVIII."

Program Presentations

HMB-002 is designed to address the underlying biological drivers of VWD through a non-replacement approach—elevating the body's endogenous levels of VWF and FVIII rather than infusing exogenous factor—with early first-in-human data building a compelling case for this paradigm shift.

- **Extended half-life supporting infrequent dosing:** Pharmacokinetic (PK) analysis confirmed dose-dependent increase in Cmax with prolonged duration.
- **Dual VWF and FVIII elevation:** In all dose cohorts tested, VWF and FVIII were elevated in a dose-dependent manner. In cohort A3 (150 mg), ≥2.4-fold elevation in both VWF and FVIII was achieved and accompanied by restoration of thrombin generation, shortening of APTT, and stable multimer distribution. The pharmacokinetic/pharmacodynamic (PK/PD) profile supports potential for monthly dosing.
- **Emerging safety profile:** Most treatment-emergent adverse events (TEAEs) were mild to moderate in severity, no serious TEAEs occurred, no events were considered related to HMB-002, and no participant discontinued the study due to TEAEs. There were no thromboembolic events, no injection site reactions, no thrombocytopenia, and no hypersensitivity reactions.
- **Preliminary clinical observations**: The single ascending dose (SAD) part of the study wasn't designed to measure efficacy, so clinical observations should be considered descriptive in nature. Across the SAD cohorts, 8 of 9 evaluable patients had zero treated bleeds in the 28 days following HMB-002 dosing, with a mean ATBR of 1.6. Among participants treated with HMB-002, the baseline mean ATBR before HMB-002 treatment was 20.1 reflecting significant disease burden in Type 1 VWD, based on bleed data collected up to 5.5 months.

HMB-003 is a novel fatty-acid-conjugated peptide antifibrinolytic, designed to stabilize clots and reduce bleeding across multiple settings, beginning in heavy menstrual bleeding, with preclinical data supporting potent, selective antifibrinolytic activity and extended duration of action.

- **Potent and selective plasmin inhibition:** HMB-003 directly inhibits plasmin at its active site and inhibits fibrinolysis across both tPA- and uPA-driven pathways, while showing no effect on thrombin generation, platelet function, or coagulation in nonclinical studies.

- **Extended antifibrinolytic activity:** Following a single subcutaneous dose in minipigs, HMB-003 achieved peak plasma levels within hours and sustained antifibrinolytic activity for approximately one week, supporting the potential for cycle-matched dosing in heavy menstrual bleeding.

About Von Willebrand Disease
Von Willebrand Disease (VWD) is the most common inherited bleeding disorder, characterized by quantitative or qualitative defects in Von Willebrand Factor (VWF), often resulting in frequent mucocutaneous bleeding events and heavy menstrual bleeding in women. The severity of bleeding ranges from low-volume events to potentially life-threatening hemorrhages. Chronic blood loss frequently leads to iron deficiency anemia, exacerbating the disease burden and reducing quality of life, particularly for those with clinically understated subtypes. Despite its prevalence, current treatment options for VWD primarily focus on managing symptoms rather than addressing the underlying biology of the disease.

About HMB-002
HMB-002 is a monovalent human antibody being developed as the first-in-class subcutaneous prophylactic treatment for Von Willebrand Disease targeting the underlying cause of the disease, a condition driven by a deficiency or defect in Von Willebrand Factor (VWF), a key regulator of hemostasis. By specifically targeting the C-terminal CK domain of VWF, which is distinct from regions critical to its essential interactions, HMB-002 shields the protein from degradation, boosting endogenous levels without compromising its function. Clinical and nonclinical data suggest strong potential for meaningful therapeutic benefit. For more information, please visit clinicaltrials.gov (NCT06610201 and NCT06754852).

HMB-003
HMB-003 is a subcutaneously administered peptide-based plasmin inhibitor with a durable half-life — a proven therapeutic target in coagulation medicine — being developed as a novel antifibrinolytic designed to reduce bleeding across multiple settings. Engineered to directly inhibit plasmin at its active site, HMB-003 blocks fibrinolysis independently of the plasminogen activation pathway. HMB-003 is optimized to provide sustained bleed protection across multiple high-unmet-need conditions, ranging from heavy menstrual bleeding and hereditary hemorrhagic telangiectasia to peri-operative bleeding management.

About Hemab Therapeutics
Hemab Therapeutics Holdings, Inc. is a clinical-stage biotechnology company developing therapies that reimagine the treatment of blood coagulation disorders to sustain life and human resilience. Hemab's mission is to discover, develop, and commercialize innovative therapies for the millions of patients worldwide suffering from serious bleeding and thrombotic diseases. Hemab is building a franchise of innovative therapeutics designed to address critical gaps in the treatment of coagulation disorders, including sutacimig (HMB-001), a bispecific antibody in clinical development for the prophylactic treatment of Glanzmann thrombasthenia and Factor VII deficiency, HMB-002, a monovalent antibody in clinical development for the prophylactic treatment of Von Willebrand Disease, and HMB-003, an anti-fibrinolytic targeting plasmin inhibition in preclinical development for multiple high-unmet-need conditions, ranging from heavy menstrual bleeding, hereditary hemorrhagic telangiectasia to peri-operative bleeding management.

Learn more at hemab.com. Follow us on LinkedIn, Facebook, Instagram, and X.

Forward-Looking Statements
This press release contains forward-looking statements that involve substantial risks and uncertainties. All statements, other than statements of historical facts, contained in this press release, including statements regarding Hemab's strategy, future operations, prospects and plans, objectives of management, the anticipated timelines for reporting data from Hemab's 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, Hemab's plans to expand its pipeline, and the sufficiency of Hemab's 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. Hemab 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 Hemab's 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 Hemab's cash resources will be sufficient to fund the Company's foreseeable and unforeseeable operating expenses and capital expenditure requirements; as well as the risks and uncertainties identified in Hemab's filings with the Securities and Exchange Commission (SEC), including the Company's most recent Form 10-Q and in subsequent filings Hemab may make with the SEC. In addition, the forward-looking statements included in this press release represent Hemab's views as of the date of this press release. Hemab anticipates that subsequent events and developments will cause its views to change. However, while Hemab may elect to update these forward-looking statements at some point in the future, it specifically disclaims any obligation to do so. These forward-looking statements should not be relied upon as representing Hemab's views as of any date subsequent to the date of this press release.

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