UNITED STATES
SECURITIES AND EXCHANGE COMMISSION
WASHINGTON, D.C. 20549

FORM 8-K

CURRENT REPORT

Pursuant to Section 13 or 15(d) of the Securities Exchange Act of 1934

Date of Report (Date of earliest event reported): November 04, 2026

CULLINAN THERAPEUTICS, INC.

(Exact name of Registrant as Specified in Its Charter)

Delaware	**001-39856**	**81-3879991**
(State or Other Jurisdiction of Incorporation)	**(Commission File Number)**	**(IRS Employer Identification No.)**
One Main Street Suite 1350 Cambridge, Massachusetts		**02142**
(Address of Principal Executive Offices)		**(Zip Code)**

Registrant's Telephone Number, Including Area Code: 617 410-4650

(Former Name or Former Address, if Changed Since Last Report)

Check the appropriate box below if the Form 8-K filing is intended to simultaneously satisfy the filing obligation of the registrant under any of the following provisions:

☐ Written communications pursuant to Rule 425 under the Securities Act (17 CFR 230.425)

☐ Soliciting material pursuant to Rule 14a-12 under the Exchange Act (17 CFR 240.14a-12)

☐ Pre-commencement communications pursuant to Rule 14d-2(b) under the Exchange Act (17 CFR 240.14d-2(b))

☐ Pre-commencement communications pursuant to Rule 13e-4(c) under the Exchange Act (17 CFR 240.13e-4(c))

Securities registered pursuant to Section 12(b) of the Act:

Title of each class	Trading Symbol(s)	Name of each exchange on which registered
Common Stock, $0.0001 par value per share	CGEM	The Nasdaq Global Select Market

Indicate by check mark whether the registrant is an emerging growth company as defined in Rule 405 of the Securities Act of 1933 (§ 230.405 of this chapter) or Rule 12b-2 of the Securities Exchange Act of 1934 (§ 240.12b-2 of this chapter).

Emerging growth company ☐

If an emerging growth company, indicate by check mark if the registrant has elected not to use the extended transition period for complying with any new or revised financial accounting standards provided pursuant to Section 13(a) of the Exchange Act. ☐

Item 7.01 Regulation FD Disclosure.

On November 4, 2026, Cullinan Therapeutics, Inc. (the "Company") issued a press release announcing that the Company will present updated data from the CLN-049 Phase 1 dose escalation clinical trial in patients with relapsed or refractory acute myeloid leukemia ("AML") or myelodysplastic syndrome ("MDS") at the 68th American Society of Hematology Annual Meeting and Exposition in December. A copy of the press release is furnished as Exhibit 99.1 to this Current Report on Form 8-K and is incorporated herein by reference.

The information in this report furnished pursuant to Item 7.01, including Exhibit 99.1 attached hereto, shall not be deemed "filed" for purposes of Section 18 of the Securities Exchange Act of 1934, as amended (the "Exchange Act") or otherwise subject to the liabilities of that section, nor shall it be deemed incorporated by reference in any filing under the Securities Act of 1933, as amended, or the Exchange Act, except as expressly set forth by specific reference in such filing.

Item 8.01 Other Events.

On November 4, 2026, the Company shared updated data from its Phase 1 dose escalation clinical trial for CLN-049 in patients with relapsed or refractory AML or MDS.

As of the data cutoff, 87 patients (76 AML, 11 MDS) were enrolled across 13 cohorts (target dose range, 1.5-24 µg/kg). The cytokine release syndrome ("CRS") rate was 36% (13/36 patients), which included the only Grade 3 event, in patients receiving one step-up dose. Implementation of a two step-up dosing regimen (1 or 1.5 µg/kg to 6 µg/kg) reduced CRS incidence to 26% (7/27 patients) and severity (Grade 1 and Grade 2 only). ICANS was uncommon (6%, 5/87 patients), with one Grade 3 event, and all events were self-limiting. Grade 3 or greater treatment-emergent adverse events occurring in greater than 10% of patients included febrile neutropenia (18%), transaminase ("TA") elevations (14%), and pneumonia (12%). Utilization of a two step-up dosing regimen with an initial step-up dose of 1 or 1.5 µg/kg mitigated TA elevations (4%, 1/27 patients). Importantly, TA elevations were asymptomatic, reversible, and not associated with other liver lab abnormalities.

As of the data cutoff, 80 patients were efficacy evaluable (≥1 response assessment or patients who discontinued without assessment). At the 6 µg/kg target dose level, the CRc rate was 19% (3/16 patients, all CR) and the ORR was 25%. At the 12 µg/kg target dose level, the CRc rate was 21% (6/29 patients, 2 CR) and the ORR was 45%. In patients treated at the 12 µg/kg target dose level who received a starting step-up dose of 1.5 µg/kg or lower, the CRc rate was 26% (5/19 patients, 2 CR) and the ORR was 53%.

Additional clinical observations included:

- Measurable residual disease–negative responses by multiparameter flow cytometry were observed in five patients.
- Two responding patients remained on CLN-049 treatment for longer than 12 months without further consolidation.
- In four additional responding patients, CLN-049 provided a bridge to allogeneic hematopoietic stem cell transplantation.

These results demonstrated that a two step-up dosing regimen on day one (1.5 µg/kg) and day four (6 µg/kg) provided optimal immune priming and favorable clinical safety. The highest CRc rates were observed at target dose levels of 6 or 12 µg/kg. These target dose levels demonstrated anti-leukemic activity with count recovery in a broad population of patients with relapsed or refractory AML or MDS to be further explored in the ongoing Phase 2 clinical trial of CLN-049.

Item 9.01 Financial Statements and Exhibits.

(d) Exhibits

Exhibit No.	Description
99.1	Press release issued by Cullinan Therapeutics, Inc. on November 4, 2026, furnished herewith
104	Cover page from this Current Report on Form 8-K, formatted in Inline XBRL

SIGNATURES

Pursuant to the requirements of the Securities Exchange Act of 1934, the registrant has duly caused this report to be signed on its behalf by the undersigned hereunto duly authorized.

CULLINAN THERAPEUTICS, INC.

Date: November 4, 2026

By: /s/ Mary Kay Fenton
Mary Kay Fenton
Chief Financial Officer

Exhibit 99.1

Cullinan Therapeutics Announces Presentations for CLN-049, a FLT3 T Cell Engager, and Velinotamig, a BCMA T Cell Engager, at the 68th ASH Annual Meeting

CAMBRIDGE, Mass., Nov. 04, 2026 (GLOBE NEWSWIRE) -- **Cullinan Therapeutics, Inc.** (Nasdaq: CGEM), a clinical-stage biopharmaceutical company accelerating potential first- or best-in-class, disease-modifying T cell engagers in autoimmune diseases and cancer, today announced that two of its T cell engager programs, CLN-049, a FLT3xCD3 T cell engager, and velinotamig (CLN-803), a BCMAxCD3 T cell engager, will be featured across four poster presentations at the 68th American Society of Hematology (ASH) Annual Meeting and Exposition, being held December 12-15 in New Orleans, Louisiana.

Cullinan will present updated data from the CLN-049 Phase 1 dose escalation study in patients with relapsed or refractory acute myeloid leukemia (AML) or myelodysplastic syndrome (MDS). The abstract reports the anti-leukemic activity of the selected Phase 2 target doses of 6 µg/kg and 12 µg/kg, and a favorable safety profile for the double step-up dosing regimen. Updated dose escalation results for the selected Phase 2 regimens will be further detailed in the poster presentation.

Additional posters include a Trial in Progress for CLN-049 in combination with azacitidine and venetoclax in newly diagnosed AML and a Trial in Progress for velinotamig in immune thrombocytopenia and warm autoimmune hemolytic anemia, which is also supported by a poster with new preclinical data demonstrating that velinotamig selectively targets BCMA-expressing B-lineage cells that drive autoimmune cytopenias.

Presentation Details

CLN-049 Poster Presentations

Title: Updated anti-leukemia activity from a Phase 1 study of CLN-049, a novel anti-FLT3 x anti-CD3 bispecific T cell engager, in relapsed/refractory (R/R) acute myeloid leukemia (AML) and myelodysplastic syndrome (MDS)
Session Name: 616. Acute Myeloid Leukemias: Investigational Drug and Cellular Therapies: Poster II
Session Date: December 13, 2026
Session Time: 6:00 p.m. - 8:00 p.m.
Session Location: MCCNO - Hall F
Poster Number: 3385

Title: Trials in Progress: A multicenter Phase 1b study of the bispecific T-cell engager CLN-049 in combination with venetoclax and azacitidine in patients with newly diagnosed acute myeloid leukemia (AML)
Session Name: 616. Acute Myeloid Leukemias: Investigational Drug and Cellular Therapies: Poster I
Session Date: December 12, 2026
Session Time: 5:30 p.m. - 7:30 p.m.
Session Location: MCCNO - Hall F
Poster Number: 1640

Velinotamig (CLN-803) Poster Presentations

Title: Velinotamig (CLN-803), a BCMA×CD3 Bispecific T-cell Engager, Targets B Cells and Plasma Cells That Drive Autoimmune Cytopenias
Session Name: 703. Cellular Immunotherapies Other Than CAR-T Cells: Basic and Translational: Poster I
Session Date: December 12, 2026
Session Time: 5:30 p.m. - 7:30 p.m.
Session Location: MCCNO - Hall F
Poster Number: 2384

Title: Trials in Progress: A Phase 1b/2a, Open-label Study of a Novel Subcutaneously Administered BCMAxCD3 T-Cell Engager Velinotamig in Relapsed/Refractory Immune Thrombocytopenia and Warm Autoimmune Hemolytic Anemia
Session Name: 311. Disorders of Platelet Number or Function: Clinical and Epidemiological: Poster II
Session Date: December 13, 2026
Session Time: 6:00 p.m. - 8:00 p.m.
Session Location: MCCNO - Hall F
Poster Number: 3001

About CLN-049

CLN-049 is a novel, investigational FLT3xCD3 T cell engager. CLN-049 is designed to target FLT3-expressing leukemia cells, offering a new immunotherapeutic approach for treating acute myeloid leukemia (AML) and myelodysplastic syndrome (MDS). CLN-049 binds to both mutated and non-mutated FLT3, allowing targeted action regardless of FLT3 mutational status, making the investigational treatment widely applicable to a broad population.

CLN-049 is being studied in a Phase 1/2, open-label, multicenter, multiple ascending dose study evaluating safety, tolerability, pharmacokinetics (PK), pharmacodynamics (PD), and preliminary efficacy of intravenously (IV) administered CLN-049 in patients with relapsed/refractory AML or MDS (NCT05143996).

CLN-049 is also being studied in a Phase 1/2, open-label, multicenter, dose escalation and dose expansion study to evaluate the safety, tolerability, PK, and PD of IV-administered CLN-049 in combination with azacitidine and venetoclax in patients with newly diagnosed AML (NCT07722767).

CLN-049 has received Orphan Drug designation and Fast Track designation from the U.S. FDA for the treatment of relapsed/refractory AML.

About Acute Myeloid Leukemia (AML)

Acute myeloid leukemia (AML) is a cancer of the blood and bone marrow, and the most common form of acute leukemia in adults.[1] It is characterized by the rapid growth of abnormal white blood cells that crowd out healthy cells, leading to infections, fatigue, and bleeding.[2] Each year in the U.S., approximately 23,000 people are diagnosed with AML, and about half as many lives are lost to the disease.[3] Globally, AML affects an estimated 145,000 people annually, with approximately 130,000 deaths.[4]

Despite recent advances, outcomes for patients with AML remain poor, particularly for those with relapsed or refractory disease, where five-year survival is 10% or less.[5] Patients with high-risk genetic features, such as complex karyotype or TP53 mutations, face especially limited options.[6,7] Intensive treatments like chemotherapy and stem cell transplantation may be inaccessible for many older patients due to severe side effects.[7] Currently, there are no approved immunotherapies for AML, underscoring the urgent need for novel therapeutic approaches that can improve outcomes for patients and their families facing this life-threatening disease.[8]

About Velinotamig (CLN-803, GR1803)

Velinotamig is a BCMAxCD3 T cell engager designed to redirect cytotoxic T cells to target BCMA-expressing cells including pathogenic, autoantibody-producing plasma cells. Velinotamig is engineered with high-affinity binding to BCMA and lower-affinity binding to CD3, with the goal of enhancing target cell depletion while minimizing non-specific T cell activation and associated cytokine-related toxicity. By targeting BCMA-expressing cells, velinotamig has the potential to address autoimmune diseases driven by pathogenic autoantibodies.

Cullinan holds a global (ex-Greater China), all indication, exclusive license to velinotamig from Genrix Bio. Genrix Bio is evaluating velinotamig in a Phase 1b/2a clinical trial in patients with refractory systemic lupus erythematosus (SLE) in China. Cullinan Therapeutics is evaluating subcutaneously administered velinotamig in a global Phase 1b/2a study in patients with relapsing and refractory immune thrombocytopenia and warm autoimmune hemolytic anemia (NCT07738822).

About Immune Thrombocytopenia (ITP)

Immune thrombocytopenia (ITP) is a heterogeneous autoimmune disease characterized by increased platelet destruction and impaired platelet production.[9] Platelets play a critical role in blood clotting, and reduced platelet levels can lead to bruising, bleeding, debilitating fatigue, and impaired health-related quality of life.[9,10] In severe cases, bleeding may be life-threatening.[9] ITP may present as an acute, self-limited condition or follow a chronic and relapsing course, particularly in adults.[9] In the U.S., the prevalence of chronic primary ITP among adults has been estimated at 23.6 per 100,000 people, or approximately 62,000 adults.[11] Although currently available therapies can help manage platelet counts and reduce bleeding risk, many patients continue to experience relapses and the burden of ongoing disease management.[9,10]

About Warm Autoimmune Hemolytic Anemia (wAIHA)

Warm autoimmune hemolytic anemia (wAIHA) is a rare autoimmune disease in which warm-reactive autoantibodies cause the premature destruction of red blood cells, resulting in anemia.[12] People living with wAIHA may experience debilitating fatigue, shortness of breath, jaundice, and other complications of anemia. The disease can be serious and potentially life-threatening.[12] The prevalence of wAIHA among adults in the U.S. is estimated at approximately 33,300 adults.[13,14] Achieving sustained disease control can remain challenging, particularly for patients with relapsing or refractory wAIHA, underscoring the need for additional treatment options.[15]

About Cullinan Therapeutics

Cullinan Therapeutics, Inc. (Nasdaq: CGEM) is a biopharmaceutical company developing potential first- or best-in-class, disease-modifying T cell engagers for autoimmune diseases and cancer. Cullinan pursues promising therapeutic targets while leveraging core expertise in T cell engagers, which are established in oncology and are now advancing into autoimmune diseases. With a clinical-stage pipeline built on a rigorous scientific approach and purposeful innovation, Cullinan is advancing its mission to deliver new standards of care for patients. Learn more about Cullinan at **https://cullinantherapeutics.com/**, and follow Cullinan on **LinkedIn** and **X**.

Forward-Looking Statements

This press release contains forward-looking statements within the meaning of The Private Securities Litigation Reform Act of 1995. These forward-looking statements include, but are not limited to, express or implied statements regarding the company's beliefs and expectations regarding: the clinical and therapeutic potential of CLN-049, our plans regarding future data presentations and other statements that are not historical facts. The words "believe," "continue," "could," "estimate," "expect," "intends," "may," "plan," "potential," "project," "pursue," "will," and similar expressions are intended to identify forward-looking statements, although not all forward-looking statements contain these identifying words.

Any forward-looking statements in this press release are based on management's current expectations and beliefs of future events and are subject to known and unknown risks and uncertainties that may cause our actual results, performance or achievements to be materially different from any future results, performance or achievements expressed or implied by the forward-looking statements. These risks include, but are not limited to, the following: uncertainty regarding the timing and results of regulatory submissions; the risk that any INDs, NDAs or other global regulatory submissions we may file with the United States Food and Drug Administration or other global regulatory agencies are not cleared or approved on our expected timelines, or at all; the success of our clinical trials and preclinical studies; the risks related to our ability to protect and maintain our intellectual property position; the risks related to manufacturing, supply, and distribution of our product candidates; the risk that any one or more of our product candidates, including those that are co-developed, will not be successfully developed and commercialized; the risk that the results of preclinical studies or clinical studies will not be predictive of future results in connection with future studies; the effect of changes in global economic conditions, including uncertainties related to international trade policies, tariffs and supply chain dynamics on our business and operations; and the success of any collaboration, partnership, license or similar agreements. These and other important risks and uncertainties discussed in our filings with the Securities and Exchange Commission, including under the caption “Risk Factors” in our most recent Annual Report on Form 10-K and subsequent filings with the SEC, could cause actual results to differ materially from those indicated by the forward-looking statements made in this press release. While we may elect to update such forward-looking statements at some point in the future, we disclaim any obligation to do so, even if subsequent events cause our views to change, except to the extent required by law. These forward-looking statements should not be relied upon as representing our views as of any date subsequent to the date of this press release. Moreover, except as required by law, neither the company nor any other person assumes responsibility for the accuracy and completeness of the forward-looking statements included in this press release. Any forward-looking statement included in this press release speaks only as of the date on which it was made.

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1. American Association for Cancer Research. (2025). Acute Myeloid Leukemia. https://www.aacr.org/patients-caregivers/cancer/acute-myeloid-leukemia/
2. National Cancer Institute. (2025). Acute Myeloid Leukemia Treatment (PDQ®)–Patient Version. https://www.cancer.gov/types/leukemia/patient/adult-aml-treatment-pdq
3. Leptidis J., et al. (2014). Fatal cardiac tamponade as the first manifestation of acute myeloid leukemia. Am J Emerg Med 32(10). https://doi.org/10.1016/j.ajem.2014.02.045
4. National Cancer Institute. (2025). Cancer Stat Facts: Leukemia — Acute Myeloid Leukemia (AML). https://seer.cancer.gov/statfacts/html/amyl.html
5. Zhou, Y., et al. (2024). Global, regional, and national burden of acute myeloid leukemia, 1990–2021: A systematic analysis for the Global Burden of Disease Study 2021. Biomarker Research, 12(101). https://doi.org/10.1186/s40364-024-00649-y
6. Moore, CG., et al. (2025). Treatment of Relapsed/Refractory AML—Novel Treatment Options Including Immunotherapy. Am J Hematol. (100)2. https://doi.org/10.1002/ajh.27584
7. Shahzad, M., et al. (2024) What have we learned about *TP53*-mutated acute myeloid leukemia?. Blood Cancer J. 14(202). https://doi.org/10.1038/s41408-024-01186-5
8. Kantarjian H., et al. (2021). Acute myeloid leukemia: current progress and future directions. Blood Cancer J. 11(2). https://doi.org/10.1038/s41408-021-00425-3
9. Cooper, N. et al. (2019). Immune Thrombocytopenia. *New England Journal of Medicine*; 381:945-955. https://doi.org/10.1056/NEJMcp1810479
10. Cooper, N., et al. (2025). Patient survey in immune thrombocytopenia (ITP): Identifying unmet needs related to treatment and disease control in patients living in the United States. *British Journal of Haematology*, 207(3), 1038-1046. https://doi.org/10.1111/bjh.20257
11. Feudjo-Tepie, M. A., et al. (2008). Prevalence of diagnosed chronic immune thrombocytopenic purpura in the US: analysis of a large US claim database: a rebuttal. *Journal of Thrombosis and Haemostasis*; 6:711-712. https://doi.org/10.1111/j.1538-7836.2008.02911.x
12. Brodsky, R. A. (2019). Warm Autoimmune Hemolytic Anemia. *New England Journal of Medicine*, 381, 647-654. https://doi.org/10.1056/NEJMcp1900554
13. Krumme, A. et al. (2025). Prevalence and demographics of autoimmune hemolytic anemia in the United States. *Blood*, 146(Supplement 1):6239. https://doi.org/10.1182/blood-2025-6239

14. Theodosia A. (2016) Warm antibody autoimmune hemolytic anemia. *Hematology Am Soc Hematol Educ Program* 2016 (1): 690–697. https://doi.org/10.1182/asheducation-2016.1.690

15. Barcellini, W., & Fattizzo, B. (2024). Autoimmune Hemolytic Anemias: Challenges in Diagnosis and Therapy. *Transfusion Medicine and Hemotherapy*, 51, 321-331. https://doi.org/10.1159/000540475