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): October 24. 2025

MAIA Biotechnology, Inc.
(Exact name of registrant as specified in its charter)

Delaware	**001-41455**	**83-1495913**
(State or other jurisdiction of incorporation)	**(Commission File Number)**	**(IRS Employer Identification No.)**

444 West Lake Street, Suite 1700	
Chicago, IL	**60606**
(Address of principal executive offices)	**(Zip Code)**

(312) 416-8592
(Registrant's telephone number, including area code)

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	MAIA	NYSE American

Indicate by check mark whether the registrant is an emerging growth company as defined in Rule 405 of the Securities Act of 1933 (17 CFR §230.405) or Rule 12b-2 of the Securities Exchange Act of 1934 (17 CFR §240.12b-2).

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 8.01 Regulation FD Disclosure.

MAIA Biotechnology, Inc. (the "Company") has prepared a poster (the "Poster") entitled "Presentation 1: A Phase 2 Study of Ateganosine (THIO; 6-thio-2'-deoxyguanosine) in Combination with Immune Checkpoint Inhibitor (ICI) in Patients with Advanced Non-Small Cell Lung Cancer (NSCLC) Resistant to Prior ICI and Chemotherapy: THIO-101 Trial." The Poster was presented at the AACR-NCI-EORTC International Conference on Molecular Targets and Cancer Therapeutics in Boston, Massachusetts on October 24, 2025 and posted to the Company's website on such date. A copy the Poster is filed as Exhibit 99.1 to this Current Report on Form 8-K and is hereby incorporated by reference.

The Poster contains forward-looking statements, and as a result, investors should not place undue reliance on these forward-looking statements.

Item 9.01 Financial Statements and Exhibits.

(d) Exhibits.

Exhibit No.	Description
99.1	Poster
104	Cover Page Interactive Data File (embedded within the Inline XBRL document)

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.

Dated: October 24, 2025

MAIA BIOTECHNOLOGY, INC.

By: */s/ Vlad Vitoc*
Name: Vlad Vitoc
Title: Chief Executive Officer

Abstract #12870

A Phase 2 Study of Ateganosine (THIO; 6-thio-2'-deoxyguanosine) in Combination with Immune Checkpoint Inhibitor (ICI) in Patients with Advanced Non-Small Cell Lung Cancer (NSCLC) Resistant to Prior ICI and Chemotherapy: THIO-101 Trial in Progress

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01 Introduction

- Despite recent advances for the first-line treatment of advanced Non-Small Cell Lung Cancer (NSCLC), long-term prognosis remains poor with a 5-year survival rate of 28%[1] and limited options exist in patients' refractory or resistant to immune checkpoint inhibitors (ICI).
- Ateganosine (THIO, 6-thio-2'-deoxyguanosine, 6-thio-dG) is a small molecule, first-in-class direct cancer telomere targeting agent that selectively kills telomerase positive (TERT+) cancer cells:
  - Over 80% of all cancers and approx. 78-83% of all NSCLC types are TERT+.[2,3]
  - Ateganosine is incorporated into de novo synthesized telomeres leading to chromatin uncapping, generation of DNA damage signals, and rapid apoptosis.[4]
- In preclinical models, sequential treatment of Ateganosine and ICIs overcame ICI resistance and showed a potent and durable antitumor activity.[5]
- Preliminary trial results in NSCLC indicates that low doses of Ateganosine induce sensitivity to ICIs when administered prior to an ICI in tumors which otherwise are resistant or do not respond to an ICI.
- Treatment options for immune checkpoint inhibitor (ICI)-resistant patients are limited. Ateganosine, a telomere-targeting agent, modifies telomeres in cancer cells, including circulating tumor cells (CTCs), confirming its mechanism of action and demonstrating efficacy independent of PD-L1 expression.
- Biomarkers assessing telomere damage in cancer cells are becoming increasingly important for accurately determining efficacy following treatment.

02 Methods

- Using a modified 3+3 design, the safety lead-in (Part A) enrolled 10 patients who received Ateganosine 360 mg IV (120 mg QD, D1–3), followed by 350 mg cemiplimab on D5, Q3W. Following completion of Part A, enrollment was opened in the dose-finding portion of the study (Part B).
- Using a Simon 2-stage design, 79 patients were assigned to one of the Ateganosine doses: 360, 180, or 60 mg followed by cemiplimab Q3W for up to 1 year in Part B.
- Disease status is assessed at Cycle 3 Day 1, Cycle 5 Day 1 and every 9 -12 weeks thereafter.
- An expansion cohort started based on data from Part B: up to 48 patients in Part C (one arm with the combination of Ateganosine + cemiplimab, one arm with Ateganosine as monotherapy) and up to 100 patients are planned in Part D.
- CTCs were labeled with TRF1 and γH2AX to detect telomere dysfunction-induced foci (TIFs). TIF fraction in CTCs were measured using flow cytometry.

03 Pharmacodynamics (PD) and Pharmacokinetics (PK)

- Phamacodynamics (Parts A, B, C and D) and Pharmacokinetics (Parts C and D) analysis for THIO-101

Pharmacokinetic parameters:

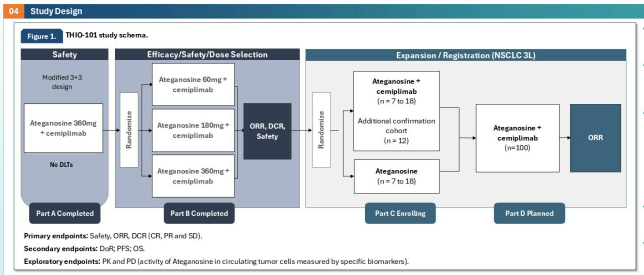
- Ateganosine concentration levels and PK parameters (Full PK collection for Part C and limited PK collection for Parts C and D)
- *Pharmacodynamic parameters and Biomarkers (PD):*
- TIF (Telomere dysfunction-induced foci) formation in CTCs
- CTCs evaluation of PDL1 expression
- Tumor telomerase TERT (+) status (via expression of telomerase mRNA), by ISH-TERT (in situ hybridization for TERT) of paraffin-embedded tumor samples.

Hematology and Clinical Chemistry:

- Interleukin- 6 (IL-6)
- C-Reactive Protein (CRP)
- Carcinoembryonic Antigen (CEA)
- Lactate dehydrogenase (LDH)
- Neutrophil-lymphocyte ratio (NLR)
- Platelet-lymphocyte ratio (PLR)

04 Study Design

Figure 1. THIO-101 study schema.



Primary endpoints: Safety, ORR, DCR (CR, PR and SD).

Secondary endpoints: DoR; PFS; OS.

Exploratory endpoints: PK and PD (activity of Ateganosine in circulating tumor cells measured by specific biomarkers).

- Patient enrollment for parts A and B of the study was completed in Feb'24 with 79 patients.
- The optimal dose of Ateganosine 180mg was selected in Nov'23 based on evaluations of safety, tolerability, and efficacy.
- At the time of data cut-off (June 30, 2025), the observed Median Overall Survival (OS) was at 17.8 months in third-line NSCLC patients that are resistant to chemotherapy and ICI. In addition, the observed Overall Response Rate (ORR) was at 30% in the intent-to-treat population (3/10).
- Patient enrollment for Part C started in Jul'25, with the first patient dosed in Taiwan.
- The trial is planned to enroll patients in multiple geographies, including Europe, Asia and the U.S.

THIO-101 Part A (Completed enrollment, N=10)

- Modified 3+3 design
- Safety lead-in study of Ateganosine 360 mg per cycle (120 mg on Days 1-3, sequenced with cemiplimab).

THIO-101 Part B (Completed enrollment, N= 69)

- Randomized, dose-finding, Simon's 2-stage design
- 3-arm study (Ateganosine 60 mg, Ateganosine 180 mg, or Ateganosine 360 mg per cycle; all dose levels sequenced with cemiplimab).

THIO-101 Part C (Enrollment initiated, up to N = 48)

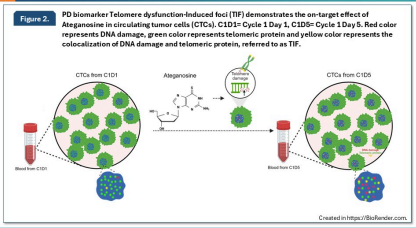
- Randomized, Simon's 2-stage in each arm
- Arm 1: Ateganosine 180 mg/cycle (60 mg IV on D1-3) sequenced with cemiplimab
- Arm 2: Ateganosine 180 mg/cycle (60 mg IV on D1-3)
- Objective: To evaluate the efficacy and safety of Ateganosine 180 mg per cycle sequenced with cemiplimab compared with single-agent Ateganosine 180 mg per cycle as third-line treatment in advanced/metastatic NSCLC patients that are resistant to chemotherapy and ICI.

THIO-101 Part D (Planned, N = 100)

- Single-Arm Efficacy Cohort
- Ateganosine 180 mg/cycle (60 mg IV on D1-3) sequenced with cemiplimab.
- Objective: To evaluate the efficacy and safety of Ateganosine 180 mg per cycle sequenced with cemiplimab as third-line treatment in advanced/metastatic NSCLC patients that are resistant to chemotherapy and ICI.

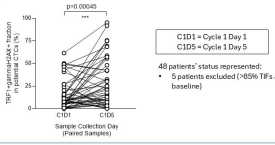
05 Pharmacodynamic biomarker Telomere dysfunction-Induced foci (TIF)

Figure 2. PD biomarker Telomere dysfunction-Induced foci (TIF) demonstrates the on-target effect of Ateganosine in circulating tumor cells (CTCs). C1D1= Cycle 1 Day 1, C1D5= Cycle 1 Day 5. Red color represents DNA damage, green color represents telomeric protein and yellow color represents the colocalization of DNA damage and telomeric protein, referred to as TIF.



06 Biomarker findings from THIO-101 ongoing Phase 2 study

Figure 3. TIFs fraction in circulating tumor cells (CTCs) from THIO-101 patients (60, 180, 360 mg dosed patients).



- Telomere dysfunction-Induced foci (TIF)-positive CTCs were characterized as TRF1+/gammaH2AX+fraction in CD326+/PanKRT+ CTCs.
- TIF analysis demonstrated the intended on-target mechanism of action: modification of telomeres in CTCs by Ateganosine (Figure 3).

07 Conclusions

- **Following the completion of Parts A and B of THIO-101, an expansion of the study was warranted to further validate the safety and efficacy observed with the selected dose of Ateganosine 180mg.**
- **The median observed Overall Survival (OS) in advanced third-line NSCLC patients who are resistant to prior treatment with ICI and chemotherapy was 17.8 months at the data cut-off (June 30, 2025).**
- **In all subjects enrolled in Parts C and D of the study, pharmacokinetic samples will be collected for longitudinal exposure-response assessments. The levels of Ateganosine and 6-TG in human plasma will be determined using a GLP-validated Liquid Chromatography Tandem Mass Spectrometry (LC-MS/MS) method.**
- **Treatment with Ateganosine alone leads to a significant induction in TIF formation in CTCs. This potentially highlights the utility of telomere DNA damage as a biomarker for monitoring the pharmacodynamic (PD) effects of Ateganosine treatment.**
- **Additional biomarker studies from Parts C and D will be conducted as the trial continues.**

References

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Author disclosures

- Veronika Müller has no conflict of interest.

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