

MAIA Biotechnology, Inc.
(NYSE: MAIA)



Key Statistics

52 Week Range	\$1.40 – \$3.74
Avg. Volume (3 months)	413.64K
Shares Outstanding	32.99M
Market Capitalization	\$52.12M
EV/Revenue	N/A
Cash Balance*	\$10.14M
Analyst Coverage	2

* Cash balance as of June 2025

Revenue (in \$ mm)

Dec - FY	2024A	2025E	2026E
1Q	0.00	0.00	0.00
2Q	0.00	0.00	0.00
3Q	0.00	0.00	0.00
4Q	0.00	0.00	0.00
FY	0.00	0.00	0.00

EPS (in \$)

Dec - FY	2024A	2025E	2026E
1Q	(0.46)	(0.16)	(0.19)
2Q	(0.40)	(0.18)	(0.23)
3Q	(0.11)	(0.22)	(0.16)
4Q	(0.14)	(0.16)	(0.13)
FY	(1.05)	(0.72)	(0.71)

Stock Price Chart (in \$)



Hunter Diamond, CFA

research@diamondequityresearch.com

MAIA Biotechnology Inc. – Advancing Towards Late-Stage Readouts in NSCLC as Ateganosine Secures Fast Track; Phase 2 Data Shows Durable Survival, Global Enrollment Expands, and Multi-Indication Pipeline Progresses

Share Price

\$1.58

Valuation

\$10.27

Investment Highlights

- Strengthens Clinical Momentum in Q2 2025 with Strong Phase 2 NSCLC Survival Data, Fast Track Designation, Global Trial Progress, and Multi-Indication Strategy:** MAIA Biotechnology advanced its lead telomere-targeting candidate, ateganosine (THIO), across clinical, regulatory, and pipeline fronts during the quarter. Updated results from the pivotal Phase 2 THIO-101 study in advanced NSCLC reported a [median overall survival of 17.8 months](#) in the intent-to-treat set of 22 third-line patients, with a 95% confidence interval (CI) lower bound of 12.5 months and a 99% confidence interval lower bound of 10.8 months. Treatment remained generally well tolerated in this heavily pretreated population, and a new partial response, defined as at least a 30% tumor reduction, was confirmed after 20 months of therapy. Operationally, the trial expanded into Asia with first dosing in Taiwan and ongoing screening in Europe and Asia. The company also entered into a master clinical supply agreement with Roche to evaluate ateganosine in sequence with atezolizumab (Tecentriq®) across multiple hard-to-treat cancers and strengthened clinical guidance by appointing two hepatocellular carcinoma specialists to its Scientific Advisory Board. MAIA also published preclinical data from its second-generation ateganosine prodrug platform in Nucleic Acids Research, highlighting dual-pharmacophore constructs designed to enhance therapy and overcome resistance. Furthermore, the FDA granted Fast Track designation for the treatment of non-small cell lung cancer (NSCLC). Beyond NSCLC, hepatocellular carcinoma (HCC), small cell lung cancer (SCLC), colorectal cancer (CRC), the company plans to initiate studies in breast, prostate, gastric, pancreatic, and ovarian cancers, with THIO-103 planned to evaluate first-line NSCLC and SCLC.
- Valuation:** Recent updates further de-risk the THIO program in third-line NSCLC. The Phase 2 THIO-101 dataset now shows a median overall survival of 17.8 months in the intent-to-treat cohort with a generally tolerable safety profile, and a confirmed partial response after 20 months of therapy supports durability. Fast Track designation for ateganosine in NSCLC and a master clinical supply agreement with Roche to evaluate the THIO–atezolizumab sequence enhance the regulatory path and partnering visibility. We continue to expect initiation of the pivotal THIO-104 study in the second half of 2025 with an accelerated approval filing targeted for 2026, contingent on THIO-101 outcomes, while planned multi-tumor studies expand the addressable opportunity. We have revised our valuation model to incorporate the most recent financial results, updated share count, and re-evaluated the comparable company analysis, yielding a valuation of \$10.27 per share, contingent on successful execution by the company.

Company Description

Founded in 2018 and headquartered in Chicago, Illinois, MAIA is a biotechnology company engaged in discovering, developing, and commercializing novel cancer therapies with high unmet medical needs. The company's lead therapeutic candidate is currently being evaluated in a phase 2 clinical trial for the treatment of non-small-cell lung cancer.

- MAIA Biotechnology Reports 40% Reduction in Net Loss for Q2 2025 Despite 35% Rise in Operating Expenses:** For the three months ended June 30, 2025, MAIA Biotechnology reported a net loss of \$5.35 million, a 40% improvement from a net loss of \$8.88 million in Q2 2024. The year-over-year improvement was primarily driven by a sharply lower non-cash loss on the change in fair value of warrant liability (approximately \$0.26 million versus \$5.16 million a year ago), which reduced other expense, net to \$0.18 million from \$5.06 million. Interest income declined 10% to \$79,697, and Australian R&D incentives were nil versus \$18,048 in the prior-year quarter. Total operating expenses rose 35% to \$5.17 million, reflecting a 52% increase in R&D to \$3.11 million due to higher scientific and clinical research outlays, plus modest increases in stock-based compensation and payroll. G&A grew 17% to \$2.06 million, largely on higher professional fees and stock-based compensation, partly offset by lower investor relations spend. Loss from operations was \$5.17 million versus \$3.82 million in the prior-year quarter. Cash and cash equivalents as of June 30, 2025, stood at approximately \$10.14 million, supported primarily by proceeds from private placements and an at-the-market offering.
- MAIA Biotechnology Granted European Patent Covering Next-Generation Ateganosine Analogues and THIO Sequencing Methods in Cancer Therapy:** MAIA Biotechnology announced that the European Patent Office has decided to grant a patent broadly covering a portfolio of ateganosine-based analogues for telomere-targeting anticancer therapy and methods of using ateganosine (THIO) alone or prior to checkpoint inhibitors. The patent, titled “Mercaptopurine Ribonucleoside Analogues for Altering Telomerase Mediated Telomere,” lists Sergei M. Gryaznov, PhD, and Jerry W. Shay, PhD, as inventors, and emphasizes preclinical evidence that mercaptopurine nucleoside analogues disrupt telomere structure and function, reduce immune activity, and induce programmed cancer cell death, with potential for improved cancer-cell specificity and increased anticancer activity. The company’s global IP estate covers telomerase-mediated telomere-altering compounds, treatment of therapy-resistant cancers, and THIO’s sequential combination with checkpoint inhibitors filed worldwide. MAIA now reports 10 issued patents globally, including Europe, validated in 19 countries, and 24 pending patent applications.
- Received FDA Fast Track Designation for Ateganosine in NSCLC; Phase 2 THIO-101 Reported 17.8-Month Median OS and Supports Potential Accelerated Pathway:** MAIA Biotechnology received FDA Fast Track designation for ateganosine (THIO) as a treatment for non-small cell lung cancer, where it is being evaluated in the pivotal Phase 2 THIO-101 trial in sequence with a checkpoint inhibitor. Ateganosine is a first-in-class small molecule that disrupts telomere structure and function via telomerase-mediated action, promoting rapid tumor cell elimination and immune memory and potentially reversing intrinsic or acquired resistance to immune checkpoint inhibitors. As of May 15, 2025, THIO-101 reported a median overall survival of 17.8 months in a heavily pretreated population, with the longest-surviving patient completing 32 cycles and reaching 24.3 months; by comparison, standard-of-care chemotherapy in a similar setting shows overall survival of just 5 to 6 months. Fast Track provides more frequent FDA interactions and rolling review and may confer eligibility for Accelerated Approval and Priority Review, which can reduce decision timelines to six months if criteria are met.
- MAIA Biotechnology Adds Leading Hepatocellular Carcinoma (HCC) Clinician-Scientists to Scientific Advisory Board to Guide Phase 2 Program; HCC Trial Preparation Underway with ODD Support:** MAIA Biotechnology appointed two hepatocellular carcinoma specialists, Claudia Fulgenzi, MD, and David J. Pinato, MD, MRCP (UK), PhD, to its Scientific Advisory Board to advise on the design and protocols of the planned company-sponsored Phase 2 HCC trial and to potentially contribute to future investigator-sponsored studies. Dr. David J. Pinato, a clinician scientist and consultant oncologist at Imperial College London, leads Developmental Cancer Therapeutics and focuses on hepatocellular carcinoma and immunotherapy; his work has been recognized by ASCO and the Society for Immunotherapy of Cancer. Dr. Claudia Fulgenzi, a medical oncologist at Imperial College London, specializes in immuno-oncology and gastrointestinal cancers, has received the ASCO Merit Award and the ILCA Young Investigator Award, and holds clinical roles at Chelsea and Westminster Hospital and the early-phase clinical trials unit at Hammersmith Hospital. The company expects to secure required approvals to begin enrolling patients by year-end. Ateganosine (THIO) holds an FDA Orphan Drug Designation for HCC granted in 2022, which can provide up to seven years of market exclusivity. HCC accounts for roughly 90 percent of liver cancers and ranks fifth in global incidence and third in mortality, emphasizing the clinical need MAIA aims to address with its checkpoint-inhibitor-sequenced strategy.

Company Overview

MAIA Biotechnology Inc. (NYSE: MAIA) is a clinical-stage biotechnology company working in the discovery, development, and commercialization of cancer-targeting therapies. Their major offering is THIO (aka 6-thio-dG, 6-thio-2'-deoxyguanosine), a small molecule drug with the potential first-in-class, being the sole direct telomere targeting agent in cancer cells, and is currently in clinical development. Lung cancer is the company's primary indication which has a global incidence of more than 2,200,000 patients per annum (second to breast cancer) and is also a leading cause of cancer death with a mortality count of more than 1,800,000. MAIA is also in the process of developing second-generation telomere targeting agents potentially with improved anti-cancer activity compared to THIO.

Ateganosine (THIO) Telomere Targeting Agent

Clinical Trial	Indication	Treatment	Status	Preclinical	Phase 1	Phase 2	Phase 3	Rights
THIO-104	NSCLC 3L	Ateganosine → Libtayo®	Initiating Phase 3					Worldwide rights owned by MAIA
THIO-101	NSCLC 2L+	Ateganosine → Libtayo®	Ongoing Phase 2			Clinical supply agreement with REGENERON		
THIO-102-CRC	CRC 3L+	Ateganosine → tislelizumab	Planned Phase 2			Clinical supply agreement with BeiGene		
THIO-102-HCC	HCC 2L+	Ateganosine → tislelizumab	Planned Phase 2			Clinical supply agreement with BeiGene		
THIO-102-SCLC	SCLC 2L+	Ateganosine → tislelizumab	Planned Phase 2			Clinical supply agreement with BeiGene		
THIO-103	NSCLC 1L, SCLC 1L	Ateganosine → tislelizumab	Planned Phase 2/3					

2nd Generation Telomere Targeting Agents

Agent	Indication	Status	Preclinical	Phase 1	Phase 2	Phase 3	Rights
MAIA-2021-020	Multiple Tumor Types	IND Enabling					Developed in-house fully-owned by MAIA
MAIA-2022-012	Multiple Tumor Types	IND Enabling					
MAIA-2021-029	Multiple Tumor Types	IND Enabling					

Exhibit 1: MAIA Product Pipeline. Source: Company Presentation

Telomeres, repetitive d-(TTAGGG) DNA-protein complexes located at the chromosomes' ends and are integral for cancer cell survival. These telomeres are maintained by an enzyme called telomerase in most tumors. Telomerase activity is found in the majority of human cancers, and it significantly contributes to the proliferation and reproductive immortality of cancer cells. THIO targets telomere via telomerase, thus compromising the structure of the telomere and causing the uncapping of chromosome ends which brings about rapid tumor cell death. THIO, along with anti- PD-L1 or anti-PD1 therapy, fully eliminated advanced tumor growth *in vivo* preclinical models and produced cancer cell type-specific memory in the immune system to keep it active against cancer cells for a longer period of time. MAIA's clinical program for THIO in NSCLC is advancing through two key trials. The expanded Phase 2 THIO-101 trial for the second line and later (2L+) NSCLC, is set to progress in 2025 and is evaluating THIO administered sequentially with cemiplimab. Concurrently, the Phase 3 THIO-104 trial, focused on third-line (3L) NSCLC, is planned to assess

MAIA Biotechnology is developing potentially safe and effective therapies for multiple forms of difficult-to-treat solid tumors with high unmet medical needs, poor prognosis, and developed therapeutic resistance

the efficacy of THIO in combination with a checkpoint inhibitor compared to the investigator's choice of treatment. This pivotal study includes an interim analysis that could support potential full commercial approval in 2026. The company has plans to evaluate multiple other cancer indications, including small-cell lung cancer, hepatocellular carcinoma, and advanced colorectal cancer.

Understanding Telomere Targeting and The Role of Telomerase Enzymes in Promoting Therapy-resistant Tumors

Therapeutic resistance or drug resistance is a highly common phenomenon in cancer research and treatment, wherein tumor cells become tolerant or resistant to different anticancer agents. Intrinsic resistance (pre-existing) and acquired resistance (induced after therapy) have been responsible for the failure of cancer treatment options, including chemotherapies and targeted therapies. Cancer cells employ various mechanisms contributing to drug resistance. Tumor heterogeneity, tumor microenvironment (TME), target gene alteration, and increased efflux of drugs are a few of the common mechanisms playing an important role in tumorigenesis and drug resistance. Currently, 90% of failures in chemotherapy are during the invasion and metastasis of cancers related to drug resistance.¹ Furthermore, it is found that resistance to anti-PD1 therapy affects up to 60% of the patients treated.²

Drug resistance has raised the need for developing newer treatment modalities for various types of cancers with high unmet medical needs

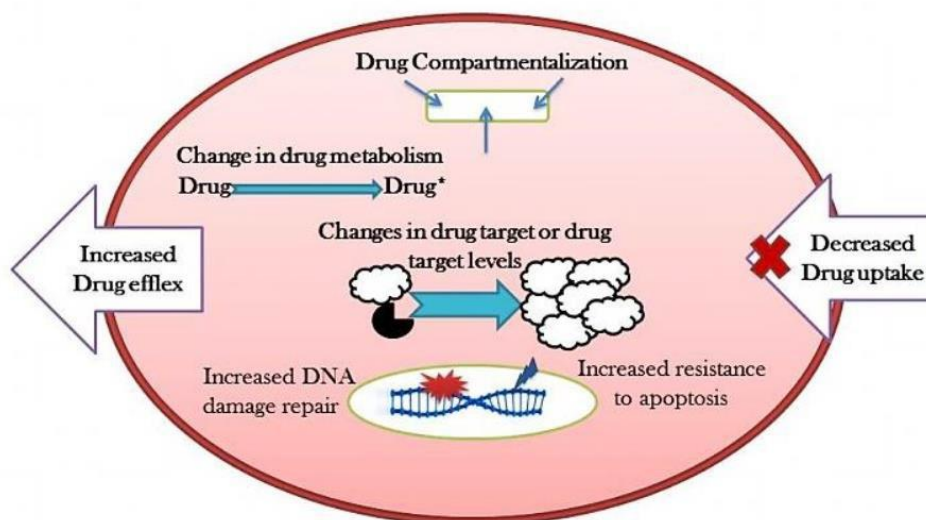


Exhibit 2: Mechanism of Drug Resistance in Cancer Cells. Source: Mansoori et al., 2017

Drug resistance has raised the need for developing newer treatment modalities for various types of cancers with high unmet medical needs. The most frequent form of acquired resistance in NSCLC (the leading cause of cancer death) is secondary mutations in EGFR (e.g., T790M “gatekeeper”) occurring in 60% of patients treated with second-generation TKIs³. Precisely targeting factors that drive cancer growth and proliferation have had remarkable initial success, but as the treatment proceeds, a large majority of patients (30% - 55%)

¹ Mansoori et al., Adv Pharm Bull., 2017

² Jake et al., Cancer Treatment Reviews, 2017

³ Sosa et al., Front Oncol., 2018

with NSCLC develop drug resistance, relapse, and die due to the disease progression.⁴ Combinational and personalized therapies currently represent the optimal treatment option over monotherapies due to their ability to target several cancer traits, inhibit more clones in tumors and make new cancer mutations resistant to a combination therapy much more complex and difficult.⁴

One such treatment modality that has the potential to overcome the drug resistance mechanism is the inhibition of telomerase activity or targeting telomeres functioning in cancer cells. Telomere- Telomerase functioning is highly correlated with cancer cell proliferation and is a hallmark of poor prognosis. Telomeres are a chain of a repetitive sequence of DNA found at both ends of chromosomes. Telomeres protect chromosomes from enzymatic end-degradation and ensure the correct replication of cells in a way that preserves genetic information. Telomeres are analogues to plastic ends of shoelaces that protect them from fraying or unravelling. Each time the cell divides, the protective caps shorten and reach the point of inactivation or replicative senescence. Scientific evidence and research indicate that telomere functioning holds the key to biological aging and cancer proliferation.

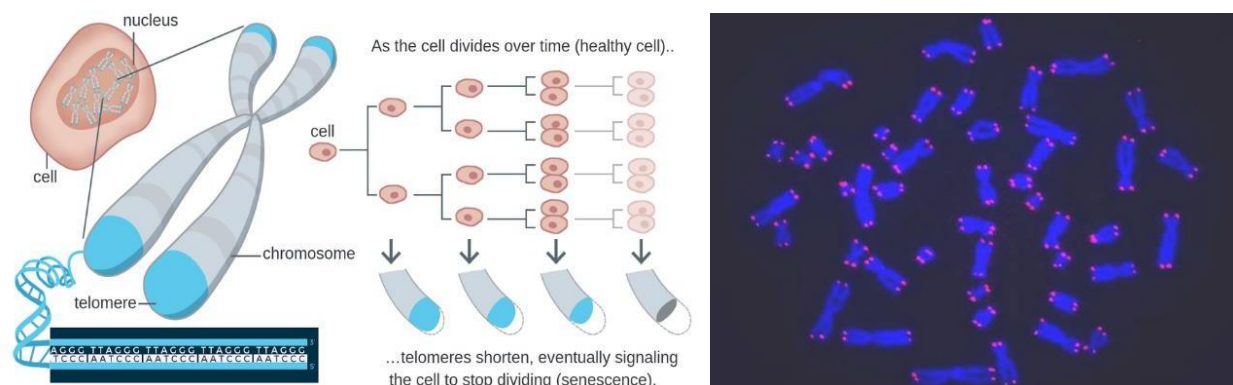


Exhibit 3: Human Cell, Chromosome, Telomere (Left) Visualization of Telomeres Using Digital Fluorescence Microscopy. Source: Labster.com, Sugarman, et al., 2019

Telomerase, a ribonucleoprotein complex, counteracts the telomere-shortening mechanism, which can lead to unlimited cell proliferation and immortality. The protective mechanism of telomerase is absent in human somatic cells, while cancer cells, including over 85% of malignancies, have detectable telomerase enzymic activity.⁵ This represents a major mechanism of cancer cell growth and disease progression.

The telomerase holoenzyme consists of two major components, functional RNA, hTR, and the other is catalytic protein reverse Transcriptase activity (hTERT). Telomeres and the enzyme Telomerase alterations play an important role in drug resistance and sensitivity to current cancer therapies. Inhibiting Telomerase activity, or more importantly, specific modification and alteration of structural integrity of telomeres in cancer cells,

The company's lead therapeutic candidate, 6-thio-dg, or THIO, is a direct telomere targeting agent with the potential to overcome therapeutic resistance and induce an immune response

⁴ Wang et al., Cancer Drug Resist, 2019

⁵ Cong et al., Microbiol Mol Biol Rev., 2002

represents a key pathway to creating successful cancer therapy for multiple types of drug-sensitive and drug-resistant cancers.⁶

THIO - Telomere Targeting Agent with Dual Mechanism of Action

The company's lead therapeutic candidate THIO (6-thio-dG or 6-thio-2'-deoxyguanosine) is a purine nucleoside analog that is incorporated into telomeres positive cells via telomerase and targets telomeres to achieve the desired therapeutic effect. Targeting telomerase directly is ineffective due to high toxicities and the long lag period between initiation and effect of the therapeutic treatment. THIO has been found to decrease the lag period experienced by previous direct telomerase inhibitors and demonstrated independence of telomere length in its ability to affect cancer in a timely manner with a reduced toxicity profile.⁷

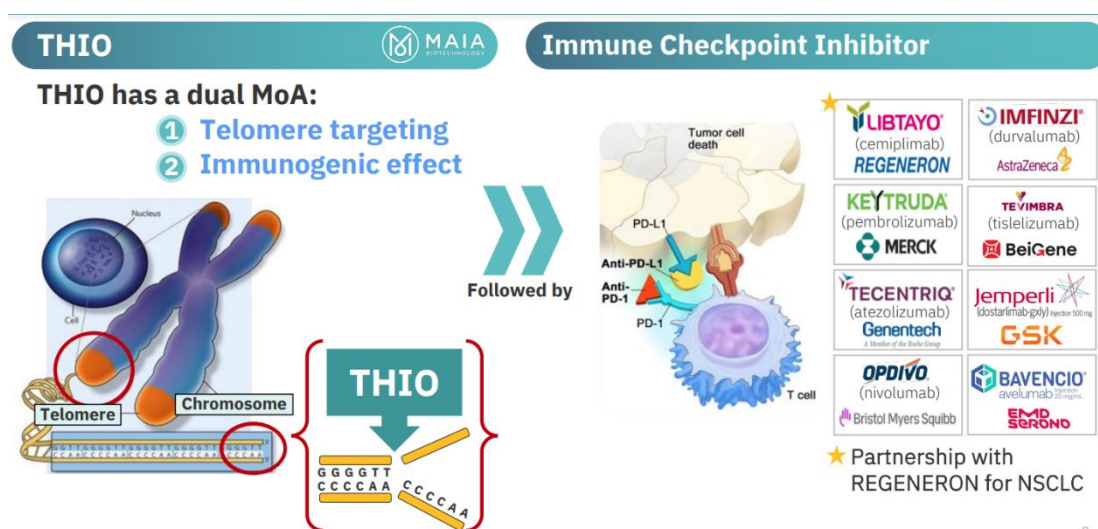


Exhibit 4: THIO Dual Mechanism of Action (MoA). Source: MAIA Corporate Presentation

THIO utilizes a dual mechanism of action that not only directly targets telomere DNA structure and functional integrity, but also yields immunogenic effects converting immunologically cold tumors into hot tumors that are refractory to checkpoint inhibitors. The company is currently evaluating the drug in a phase 2 clinical trial in combination with immune checkpoint inhibitor Libtayo® (cemiplimab) for the potential treatment of non-small cell lung cancer (NSCLC). Telomerase activity is detected in almost all forms of malignancies and thus represents almost a universal oncological target. In line with the underlying findings, THIO has also been evaluated in multiple forms of malignancies in multiple pre-clinical models, with the company planning to further evaluate it in clinical settings in patients with advanced colorectal cancer, hepatocellular carcinoma, and small-cell lung cancer.

THIO has been evaluated in multiple pre-clinical and clinical trials providing insights into its ability to successfully inhibit tumour growth and cause cancer cell death. Data from these trials indicated THIO's robust cancer killing ability and well-established safety profile

⁶ Ryan et al., Molecular Pharmacology, 2005

⁷ Sugarman et al., Mol Carcinog., 2019

THIO Clinical Trials - An Overview of Clinical Research

THIO as a pharmacological agent has been assessed in different forms of cancer in nineteen phases 1 to phase 3 clinical trials enrolling over 600 patients from the 1970s to the early 1980s. Even though the historical clinical trial had significant limitations, including a lack of information regarding the way the statistical significance was set and data points in line with the current ICH Good Clinical Practices, it still provided reasonable insights into the clinical profile of the drug. Given the sample population of over 600 subjects with doses significantly higher than currently being tested, past trials provided important information regarding potential efficacy and, most importantly, the safety profile.

Observed adverse events relating to the combination and single-agent historical clinical studies included leukopenia, thrombocytopenia, skin rash, alopecia (reversible), nausea, and vomiting. The past clinical trial results indicated THIO's favorable safety and toxicity profile, which is still a concern in many of the telomerase inhibitor compounds. Further analysis of historical clinical trial results indicate that researchers were unaware of THIO's targeting mechanism, immune system activating ability, and immunosuppressive nature at higher doses.

THIO - Preclinical Trial Design and Results

Multiple preclinical studies were undertaken to evaluate THIO in all major types of malignancies. The *in vitro* cell line assays included telomerase-positive lung, colorectal, liver, prostate, head and neck, melanoma, and brain tumor. THIO has also been evaluated in syngeneic and genetic- deficient mouse models of telomerase-positive lung, colorectal, liver, melanoma, and brain cancers.

In vitro studies, HCT116 (colon cancer), A549 (lung cancer), and a panel of non-small cell lung cancer cell lines (H2882, HCC2429, HCC15, among others) were administered with 0.5 to 10 $\mu\text{mol/L}$ THIO and 6-thioguanine. Similar treatment was administered in BJ human cell lines, colonic epithelial, and cell lines (HCEC1) to understand the effect of THIO-induced telomere dysfunction in normal cells.

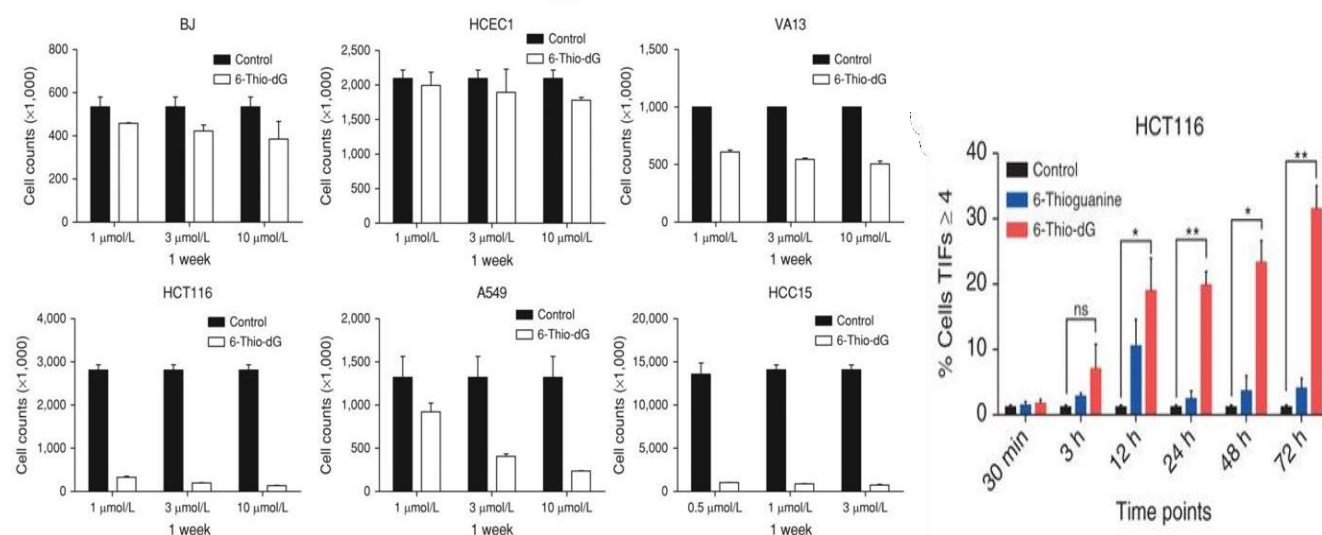


Exhibit 5: Cell Count after One Week of Treatment and TIF Index. Source: Mender et al., 2015

As represented by Exhibit 5, the cancer cells (HCT116, A549, and HCC15) at different dosage levels saw a considerable reduction post one week of administration compared to the control arm. The normal untransformed cells (BJ, HCEC1, and VA13) were comparatively much less affected, providing an indication of THIO's ability to target and induce cancer cell death while not harming or destroying normal healthy cells. Furthermore, telomeric and genomic DNA damage in cancer cells was assessed using TIF Index and genomic DNA damage foci per cell. Telomere dysfunction-Induced Foci (TIFs) is an established biomarker of efficacy indicating telomere-associated DNA damage. TIFs can be identified as the colocalization of telomeres with DNA damage response factors such as γ -H2AX.

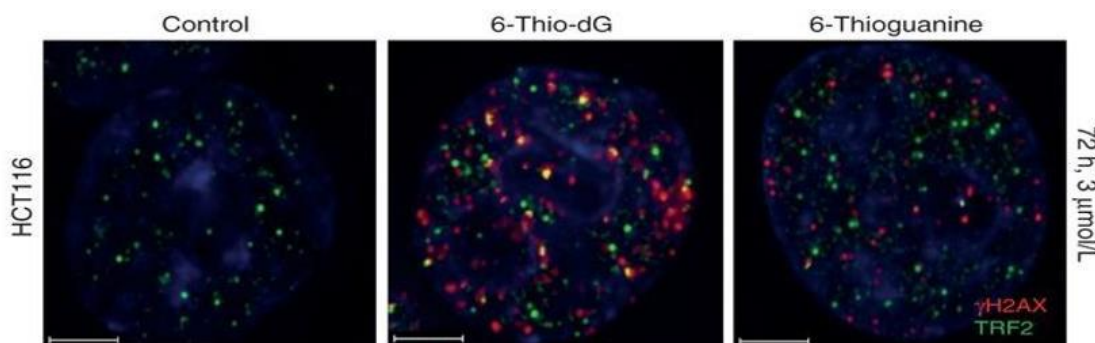


Exhibit 6: Microscopy Image of HCT116 after Treatment with THIO and 6-thioguanine. Source: Mender et al., 2015

The 6-thio-dG treatment induced a 7.8-fold increase in telomeric DNA damage and overall modes increase in genomic damage as compared with 6-thioguanine after 72 hours.⁸ Expected contrasting results were observed in non-cancerous normal cells, which lack telomerase, with no detectable telomeric DNA damage. In Exhibit 6, the red dots show DNA damaging response factor, γ H2AX, the green dots show TRF2, and the yellow dots indicate the presence of TIFs. The increased presence of yellow and red dots in the 6-thio-dG arm indicates the presence of TIFs and γ H2AX as a result of telomere dysfunction cascades.

Telomeres-telomerase activity has also been found to be a potentially attractive therapeutic target in different deadly brain tumors. An analysis of 18,430 samples across 31 cancer types identified TERT promoter mutations in 89% of glioblastoma (GBM) and 45% of low-grade glioma (LGG).⁹ Multiple pre-clinical trials have been conducted evaluating THIO's anti-tumor activity in brain tumor cell lines.

Multiple preclinical studies were undertaken to evaluate THIO in all major types of malignancies. The cancer cells (HCT116, A549, and HCC15) at different dosage levels saw a considerable reduction post one week of administration compared to the control arm

⁸ Mender et al., Cancer Discover, 2015

⁹ Yu et al., Clin Cancer Res., 2021

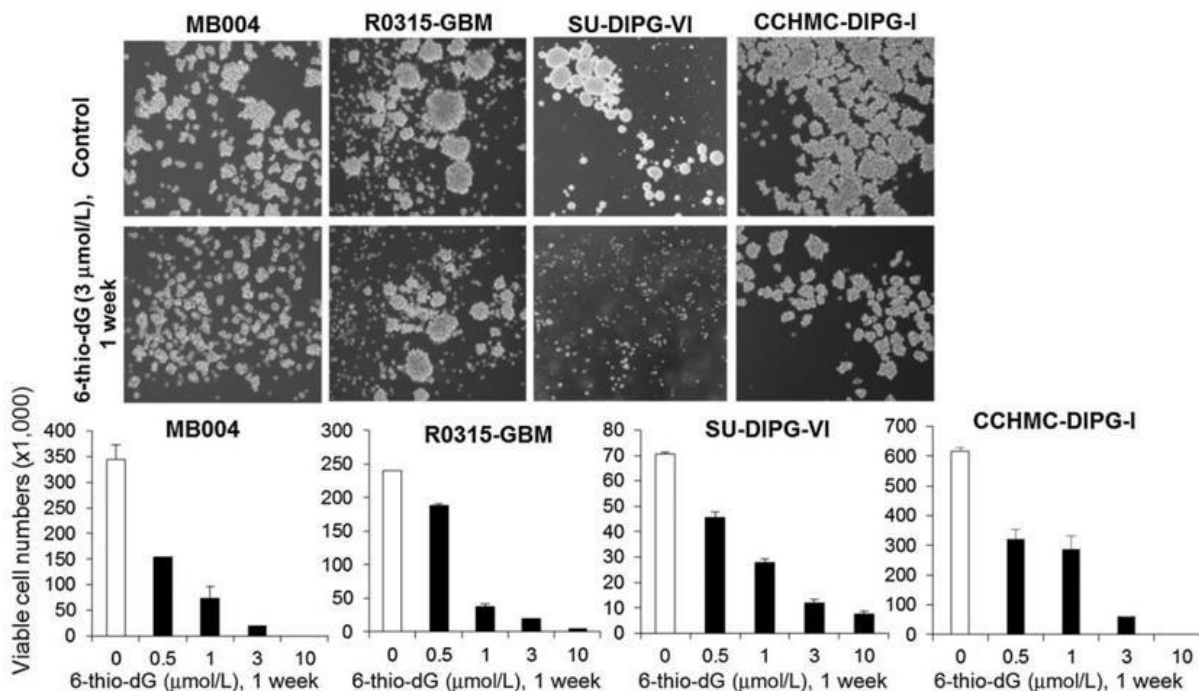


Exhibit 7: TER2+ Pediatric Brain Tumor Cells. Source: Sengupta, S. et al., 2018

As observed previously in NSCLC and colon cancer models, treatment with THIO was able to inhibit growth in TERT(+) medulloblastoma, GBM, and DIPG cancer cells. THIO caused an acute increase in the number of cells with TIFs (~25%) in telomerase-positive cells after 2 days, with the effect being amplified, reaching the TIF-positive cell count of approximately 34% at day 5.¹⁰ Another *in vitro* study of 3 different mouse glioma cell lines and 17 human glioma cell lines exhibited similar results, inhibiting cancer cell proliferation, and inducing senescence and apoptosis. THIO treatment *in vitro* brain tumor studies were able to impair cancer cell viability and cell division by inducing telomeric DNA damage.

Further *in vivo* studies were not only directed to confirm the results observed in multiple cell line models but also to understand THIO's safety profile and interaction between cancer cells and the innate adaptive immune system. These underlying studies also provided crucial insights into THIO's effectiveness in a combinational therapeutic setting as compared to monotherapy.

To determine general toxicity levels, 129S2 wild-type female mice were administered daily for 25 days with 1.67 mg/kg and 5mg/kg dose levels of both THIO and 6-thioguanine. 5 mg/kg treatment of THIO did not result in any deaths, with the weight of the mouse remaining stable over the course of the treatment period. In contrast, treatment with 5mg/kg of 6-thioguanine resulted in three deaths, and dosage levels above 3 mg/kg were found to be toxic in mice. Further analysis indicated THIO, when compared to control, did not cause any toxic effects when evaluating the histopathology of the liver, kidney spleen, and colon.

Additional *in vivo* studies included the evaluation of 3 mg/kg THIO in immunocompetent mice inoculated with TERT+ murine colon cancer cells (MC38). The results were similar to as observed in *in vitro* models with treatment-inducing tumor growth inhibition and decreased tumor volume (exhibit 8, left). To understand the

¹⁰ Sengupta., Mol Cancer Therapeutics, 2018

stimulatory role of the immune system, similar studies were conducted in Rag-1 knockout immunodeficient mice lacking T- and B- cells. Notably, THIO treatment was not able to yield any therapeutic effect when compared to the control. The tumor volume increased as the treatment progressed, indicating an important role in the immune response to inhibit tumor growth when treated with THIO. Upregulation of CD8+ T cell proliferation was observed (in immunocompetent mice), as indicated by elevated Ki67 expression confirming the dependence of THIO's therapeutic effect on T cells.

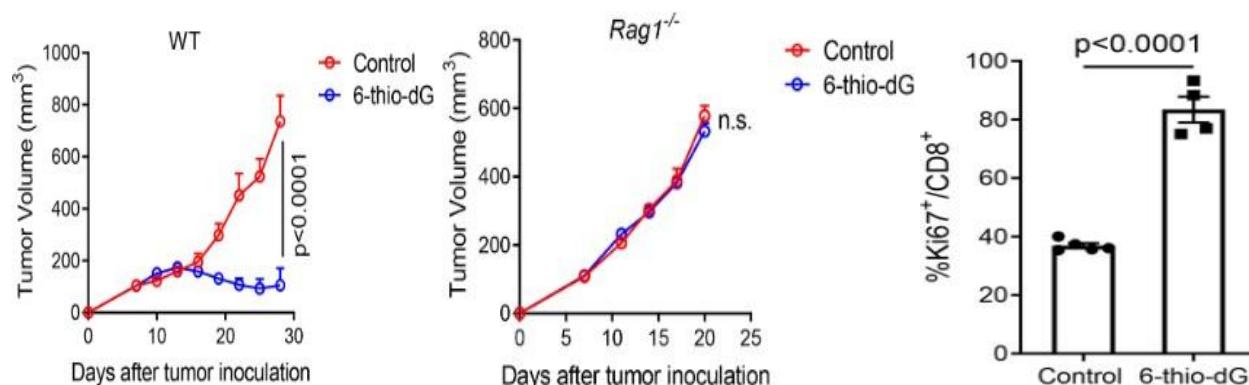


Exhibit 8: Tumor Volume after Treatment with THIO in Immunocompetent Mice (Left) and Immunodeficient Mice (Middle). Frequency of Ki67+CD8+ T Cells (Left). Source: Mender et al., 2020

In addition to T-cell proliferation, THIO treatment also induced the upregulation of PD-1 expression. PD-1 is an immune checkpoint receptor found on T cells that inhibits immune cells' effector function and blocks T cell activity during the immune response. The elevated PD-1 expression might eventually inhibit the cytotoxic CD8+ T cell function after 6-thio-dG treatment.¹¹ Based on this reasoning, THIO has been evaluated in a combinational setting with anti-PD-1/PD-L1 agents, which is expected to show a sustained anti-tumor response while overcoming the PD-L1 blockade resistance mechanism.

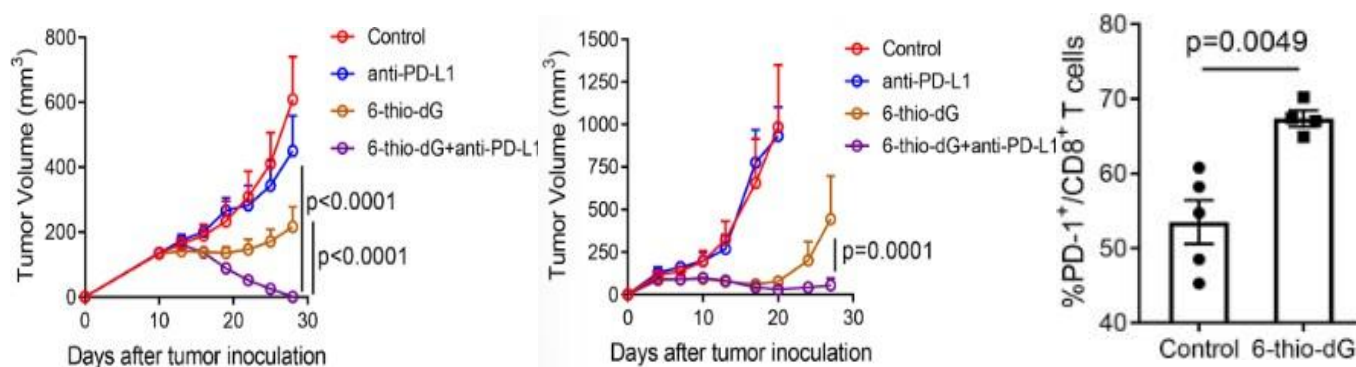


Exhibit 9: Tumor Volume in Mice Bearing TERT+ MC38 tumor (Left) and TERT+ LLC (Middle). Frequency of PD-1+CD8+ T cells (Right). Source: Mender et al., 2020

In C57BL/6, mice inoculated with MC38 tumor cells were administered, twice, with THIO and anti-PD-L1 alone as a monotherapy. In another arm, both the therapies, THIO, and anti-PD-L1 agent were sequentially administered. Tumors in only the combination treatment group were completely inhibited achieving a 100% survival rate. Anti-PD-L1 or THIO as monotherapies were not able to induce sustained tumor growth inhibition

¹¹ Mender et al., Cancer Cell., 2020

(exhibit 9, left). This combination therapy was further tested in a comparatively less immunogenic Lewis lung carcinoma (LLC) tumor model. The results were consistent with the MC38 tumor model, with combination therapy achieving essentially complete tumor inhibition, while anti-PD-L1 monotherapy exhibited no therapeutic effect (exhibit 9, middle). Notably, 40% of mice eventually completed rejected tumors in a combinational setting.¹¹ Treatment with 6-thio-dG also led to the development of a tumor-specific immune memory which stimulates an anti-tumor response when the animals with the observed complete responses to the treatment were reinjected with LLC cells. This tumor-protecting effect was also observed when the agent's combination-treated mice with MC38 tumors were followed for 5 weeks and re-challenged with MC38 tumor cells. The results indicated the development of *antigen-specific immune memory* that rejected the re-challenged MC38 tumor cells.

In multiple in-vivo and in-vitro preclinical trials, 6-thio-dg has shown to successfully induce telomere dysfunction via telomerase that successfully led to DNA damage and cancer cell apoptosis

In multiple in vivo and in vitro preclinical studies (Mender et al., 2015, Sengupta. et al., 2018, Mender et al., 2018, Mender et al., 2020, Yu et al., 2021) 6-thio-dG has shown to successfully induce telomere dysfunction via telomerase activity that not only successfully leads to DNA damage and cancer cell apoptosis, but also accomplishes it with the shorter lag period between treatment and expected effect that results in prolonged treatment and hematological toxicities. Further, the combination sequential treatment of THIO and anti-PD-L1 demonstrated a robust anti-tumor response overcoming immunological resistances in advanced tumor models in vivo. The ability of 6-thio-dG treatment to develop tumor-specific immune memory protection further accentuates the positive preclinical efficacy data. The combination sequential treatment with THIO and immune check point inhibitors is believed to yield optimal safety and efficacy data, particularly in therapy-resistant tumors, which is currently being evaluated in human clinical trials.

Appendix

Income Statement	FY2023 A	FY2024 A	FY2025 E	FY2026 E	FY2027 E	FY2028 E
Net sales	-	-	-	-	66,267,110.2	396,551,993.5
Cost of sales	-	-	-	-	(16,566,777.6)	(99,137,998.4)
Gross profit	-	-	-	-	49,700,332.7	297,413,995.1
Operating expenses						
General and Administrative Expenses	(9,070,124.0)	(6,947,981.0)	(8,337,577.2)	(10,005,092.6)	(33,133,555.1)	(178,448,397.1)
Marketing Expense	-	-	-	-	(9,940,066.5)	(39,655,199.4)
Ratchet share expense	-	-	-	-	-	-
Research and Development	(11,112,257.0)	(10,009,229.0)	(15,013,843.5)	(24,022,149.6)	(33,133,555.1)	(47,586,239.2)
EBITDA	(20,182,381.0)	(16,957,210.0)	(23,351,420.7)	(34,027,242.2)	(26,506,844.1)	31,724,159.5
Depreciation and amortization expenses	-	-	-	-	(250,000.0)	(599,400.7)
Other income/ (expense)	-	-	-	-	-	-
License Agreement Payments	-	-	-	-	-	-
Other expenses/income	176,719.0	79,954.0	119,931.0	720,664.5	662,671.1	475,862.4
EBIT	(20,005,662.0)	(16,877,256.0)	(23,231,489.7)	(33,306,577.8)	(26,094,173.0)	31,600,621.2
Interest Income	34,490.0	318,367.0	192,026.0	24,287.2	51,802.1	45,732.1
Interest Expense	(6,863.0)	(57.0)	-	-	-	-
Profit before exceptional items, extraordinary items and tax	(19,978,035.0)	(16,558,946.0)	(23,039,463.7)	(33,282,290.5)	(26,042,370.9)	31,646,353.3
Paycheck protection program loan forgiveness	-	-	-	-	-	-
Change in fair value of warrant liability	205,130.0	(6,682,758.0)	565,387.0	-	-	-
Loss on fair value of warrants over proceeds	-	(12,952.0)	-	-	-	-
Other exceptional items	-	-	-	-	-	-
Profit before tax from continuing operations	(19,772,905.0)	(23,254,656.0)	(22,474,076.7)	(33,282,290.5)	(26,042,370.9)	31,646,353.3
Income tax (expense) benefit	-	-	-	-	-	(6,645,734.2)
Net earnings including noncontrolling interests	(19,772,905.0)	(23,254,656.0)	(22,474,076.7)	(33,282,290.5)	(26,042,370.9)	25,000,619.1

Exhibit 10: Income Statement Snapshot. Source: Diamond Equity Research

Risks

- **Clinical Development Risks:** The success of the company heavily relies on the success of the THIO clinical trial. They may face risks with the emergence of pandemics, epidemics, or outbreaks. It is also important to note that clinical trials are expensive, time-consuming, and difficult to plan and implement, all with the risk of an uncertain outcome. MAIA products are based on novel technologies, which make it difficult to predict the cost, timing, and results of product candidates. Other parts of the trial process, such as patient retention, are also complicated and could be disrupted by negative externalities.
- **Financial/Dilution Risks:** MAIA has a limited operating history, is not profitable yet, and might never achieve or sustain profitability. Even if THIO is successful, it will need further financing to develop new products. This runs the risk of dilution. There is also the risk of concentrating scarce resources on a product candidate that fails to yield returns and fails to capitalize on a profitable drug.
- **Regulatory Risks:** Any disruptions in the FDA or other authorities, domestic or foreign, could impact development and commercialization. FDA and other regulatory processes are lengthy, costly, uncertain, and time-consuming. Serious side effects or other adverse findings might emerge after final approval leading to discontinuation of the product, losing approval on all products, or if discovered after marketing approval, it could lead to the loss of marketing authorizations on their other product candidates. Besides regulatory approvals for product candidates, there are the regulatory requirements required for continued marketing.
- **Commercialization Risks:** MAIA has never commercialized a product, so it is difficult to determine the viability of a new product. The market opportunity for THIO might also be smaller than anticipated. The company faces major competition from other biotechnology and pharma companies. They may also face early generic drug competition for THIO or other products.
- **Counterparty Risks:** Counterparties such as employees and independent contractors such as clinical trial sites, principal investigators, contract research organizations (CROs), consultants, contract manufacturing organizations (CMOs), and other third parties could engage in malpractices, renege on the terms of their contract, etc. and lead to a major operational loss and hindrances to development. Furthermore, the number and nature of collaborations could harm potential partnerships, and any loss of relationships would significantly damage the business.

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