MAIA Biotechnology, Inc. (NYSE: MAIA)

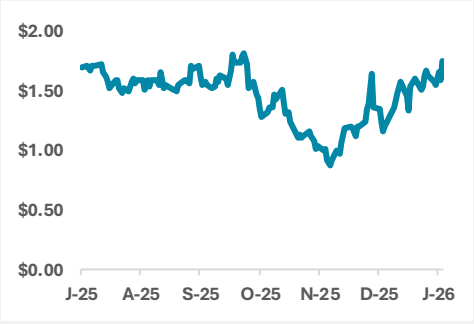
Key Statistics	
52 Week Range	$0.87 – $2.74
Avg. Volume (3 months)	705.54K
Shares Outstanding	37.03M
Market Capitalization	$64.43M
EV/Revenue	N/A
Cash Balance*	$10.89M
Analyst Coverage	2

*** Cash balance as of September 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)	(0.18)
2Q	(0.40)	(0.18)	(0.22)
3Q	(0.11)	(0.27)	(0.16)
4Q	(0.14)	(0.13)	(0.13)
FY	(1.05)	(0.74)	(0.69)

Stock Price Chart (in $)



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MAIA Biotechnology Inc. – Advances to Registrational Phase as First Patient Dosed in Phase 3 THIO-104 Trial; Extends Financial Runway with Insider-Supported Private Placement

Share Price	$1.74	Valuation	$10.27

Investment Highlights

- **MAIA Biotechnology Raises $1.51 Million Through Private Placement with Continued Board Participation, Supporting Financial Runway and Highlighting Insider Conviction Ahead of Phase 3 Trial:** MAIA Biotechnology announced the completion of a $1.51 million private placement through the issuance of 1,233,488 common shares at $1.224 per share, each accompanied by a warrant exercisable at $1.36 with a three-year term. Proceeds from the offering are intended to fund Step 1 of Part C of the Phase II THIO-101 trial and support general working capital as the company advances its clinical programs. Notably, independent directors participated in the financing, purchasing an aggregate 179,737 shares and an equal number of warrants, emphasizing continued insider alignment. Following the transaction, directors and officers collectively hold approximately 5.02 million shares, representing 13.43% ownership. We note that the successful placement, coupled with repeated insider participation, strengthens MAIA's near-term liquidity position and reflects sustained confidence in ateganosine's scientific differentiation and commercial potential, as the company progresses through pivotal Phase 3 development targeting advanced NSCLC.

- **MAIA Initiates Pivotal Phase 3 THIO-104 Trial in Third-Line NSCLC, Marking Transition to Registrational Stage for Ateganosine:** MAIA Biotechnology announced that the first patient has been dosed in its Phase 3 pivotal THIO-104 trial, a key inflection point as the ateganosine (THIO) program advances into a full registrational study in advanced non-small cell lung cancer (NSCLC). The multicenter, open-label trial will enroll up to 300 third-line NSCLC patients who are resistant to prior checkpoint inhibitor and chemotherapy regimens, randomizing patients 1:1 to ateganosine sequenced with a checkpoint inhibitor versus investigator's choice of chemotherapy, with overall survival as the primary endpoint. Regulatory approvals are in place to screen patients across Taiwan, Turkey, select EMA countries, and Georgia, with enrollment now underway. The initiation of THIO-104 builds directly on encouraging Phase 2 THIO-101 data, which demonstrated a median overall survival of 17.8 months, progression-free survival of 5.6 months (more than double standard-of-care chemotherapy), and individual patient survival extending to 30 months. Importantly, the company has disclosed a Bayesian assurance analysis for THIO-104, citing an estimated 96% probability of success at the interim analysis and a 99% probability of success at final analysis, based on the observed Phase 2 survival benefit and the trial's statistical design assumptions. Ateganosine's Fast Track designation from the FDA further supports an expedited regulatory pathway. From our perspective, first patient dosing in THIO-104 de-risks the development timeline, shifts the program into a value-defining phase, and positions MAIA for a potential comparative efficacy readout that could support full regulatory approval if Phase 2 benefits are confirmed at scale.

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.

- **Ateganosine Positioned as a Potential First-in-Class Telomere-Targeting Therapy Addressing a Critical Treatment Gap in Advanced NSCLC:** MAIA Biotechnology outlined its view that the advanced non-small cell lung cancer (NSCLC) treatment landscape is approaching a strategic inflection point, particularly for patients without actionable mutations who have become refractory to checkpoint inhibitors (CPIs) and chemotherapy. While CPIs continue to dominate NSCLC treatment, accounting for an estimated $50 billion in global sales in 2024, their limitations in later-line settings leave a substantial unmet need. MAIA believes ateganosine represents a novel therapeutic class targeting telomerase activity, a near-universal feature of cancer cells, rather than mutation-specific or PD-1/PD-L1–dependent pathways. Supported by Fast Track designation from the FDA and the initiation of the Phase 3 THIO-104 trial, ateganosine is positioned to address this refractory patient population with a dual mechanism designed to induce direct cancer cell death while activating immune responses. From a commercial perspective, the NSCLC market is projected to grow from approximately $34 billion to $66 billion by 2032, and ateganosine's opportunity extends beyond lung cancer, with FDA Orphan Drug Designations already granted in glioblastoma, hepatocellular carcinoma, and small cell lung cancer. In our view, the convergence of clinical progress, regulatory momentum, and a differentiated mechanism places MAIA at a potentially pivotal juncture, where successful execution could establish telomere targeting as a new foundational approach in oncology.

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.

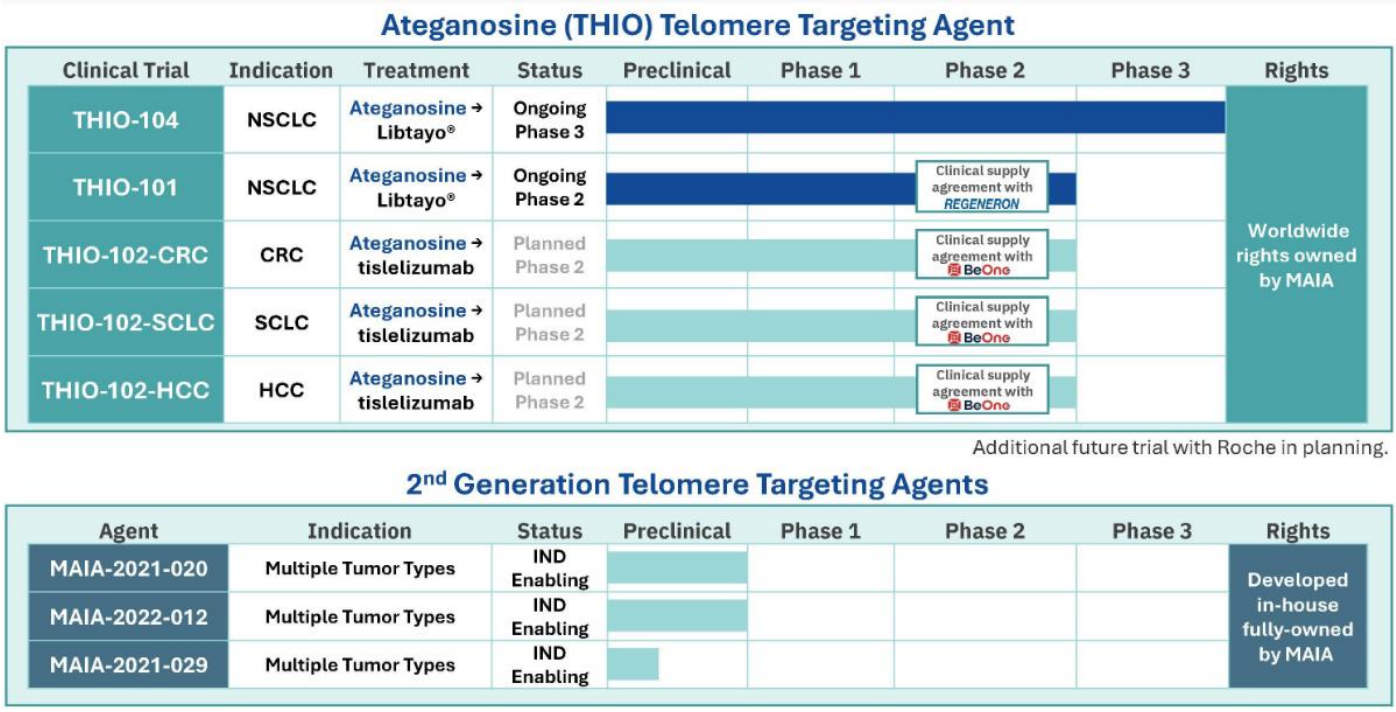

Exhibit 1: MAIA Product Pipeline. Source: Company Presentation

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

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 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

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

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]

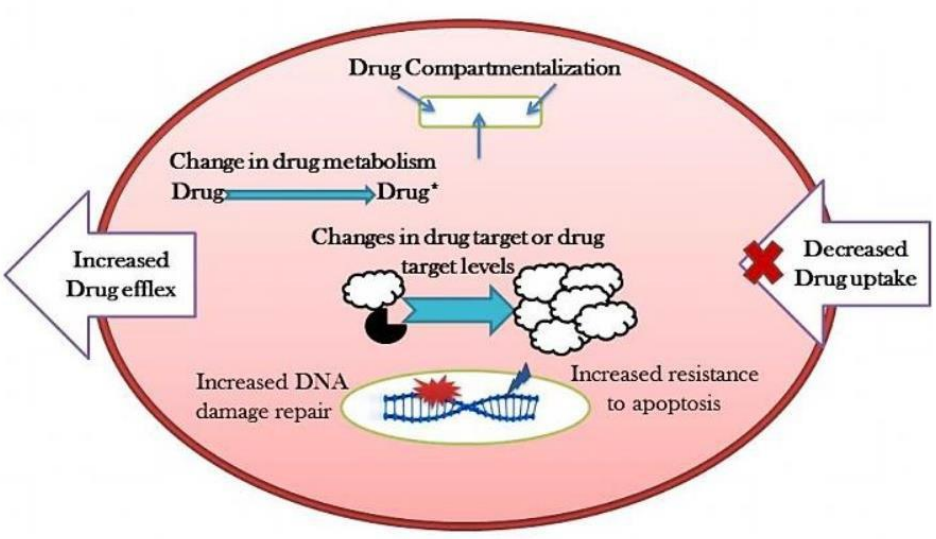

Exhibit 2: Mechanism of Drug Resistance in Cancer Cells. Source: Mansoori et al., 2017

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 TKIs [3]. Precisely targeting factors that drive cancer growth and proliferation

[1] Mansoori et al., Adv Pharm Bull., 2017
[2] Jake et al., Cancer Treatment Reviews, 2017
[3] Sosa et al., Front Oncol., 2018

have had remarkable initial success, but as the treatment proceeds, a large majority of patients (30% - 55%) 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.

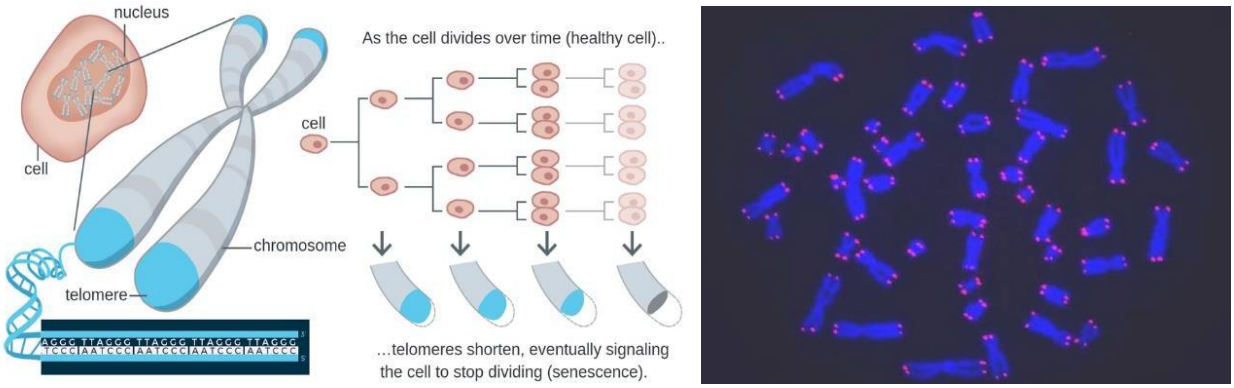

Exhibit 3: Human Cell, Chromosome, Telomere (Left) Visualization of Telomeres Using Digital Fluorescence Microscopy. Source: Labster.com, Sugarman, et al., 2019

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 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

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,

[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]

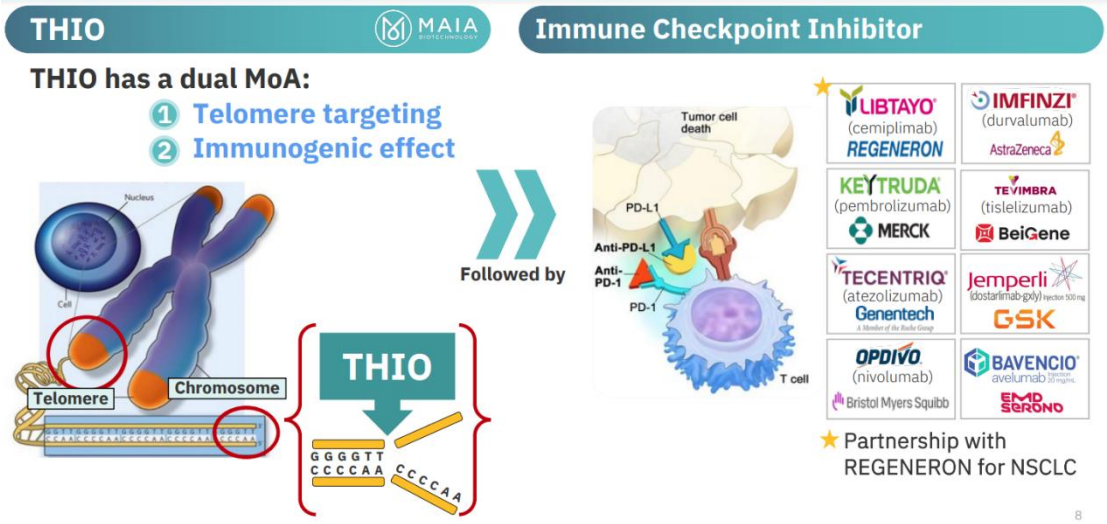

Exhibit 4: THIO Dual Mechanism of Action (MoA). Source: MAIA Corporate Presentation

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 µmol/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.

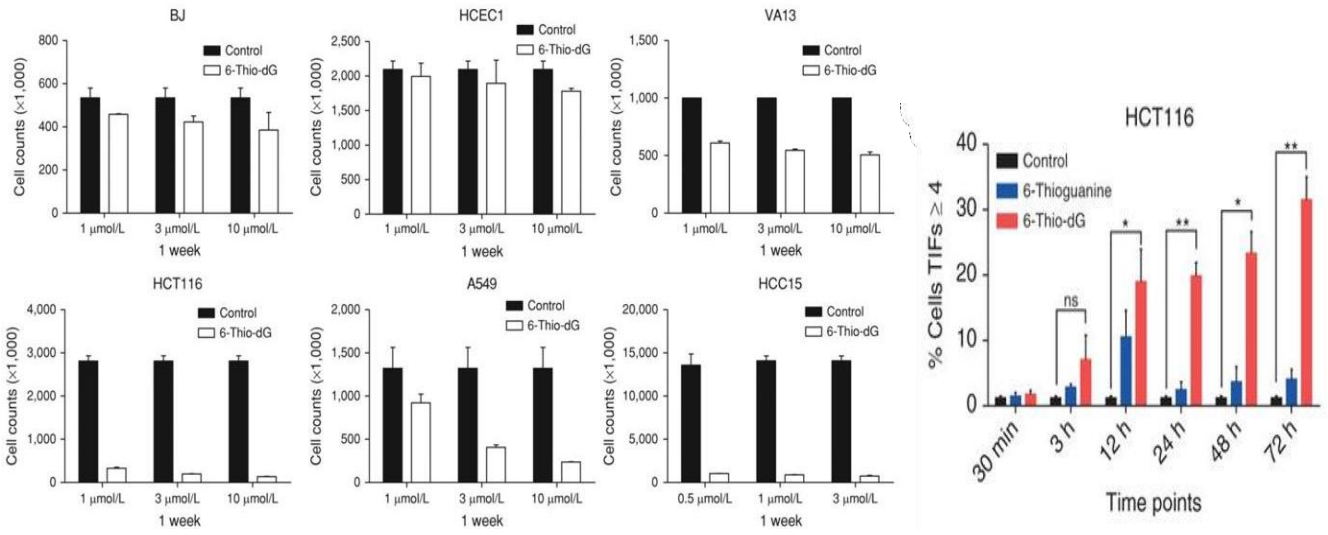

Exhibit 5: Cell Count after One Week of Treatment and TIF Index. Source: Mender et al., 2015

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.

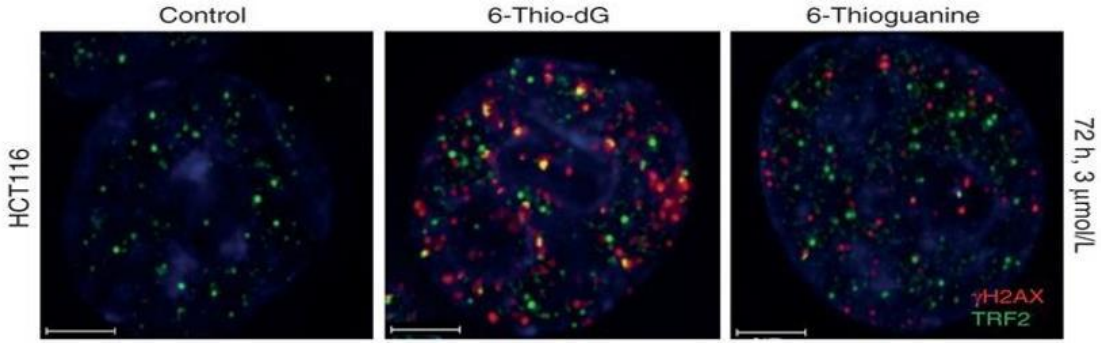

Exhibit 6: Microscopy Image of HCT116 after Treatment with THIO and 6-thioguanine. Source: Mender et al., 2015

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, yH2AX, 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 yH2AX as a result of telomere dysfunction cascades.

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

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.

[8] Mender et al., Cancer Discover, 2015
[9] Yu et al., Clin Cancer Res., 2021

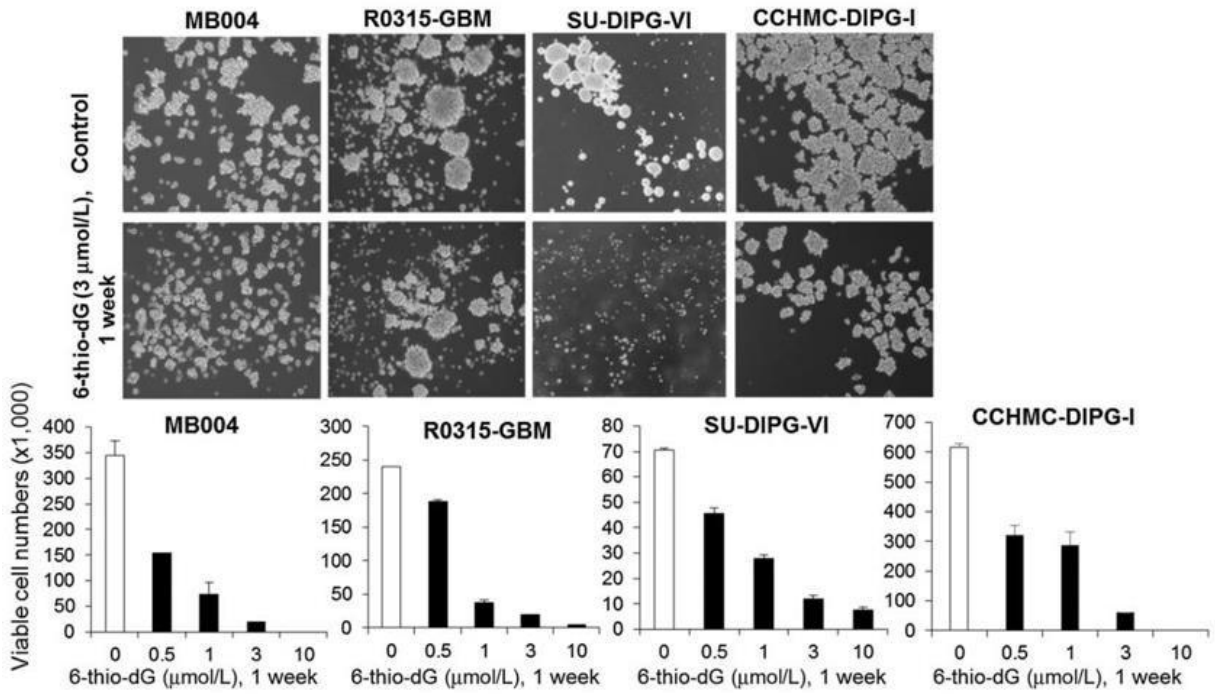

Exhibit 7: TER2+ Pediatric Brain Tumor Cells. Source: Sengupta, S. et al., 2018

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.

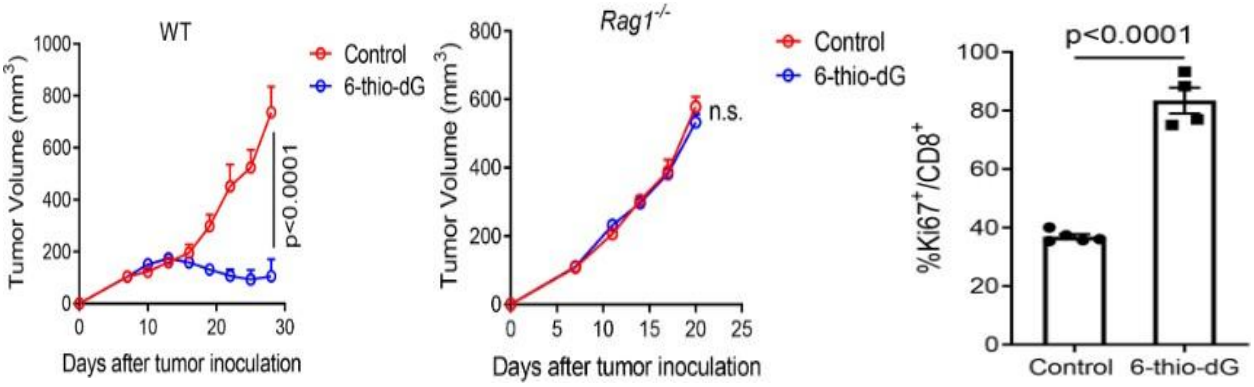

Exhibit 8: Tumor Volume after Treatment with THIO in Immunocompetent Mice (Left) and Immunodeficient Mice (Middle). Frequency of Ki67+CD8+ T Cells (Left). Source: Mender et al., 2020

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' effecter 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.

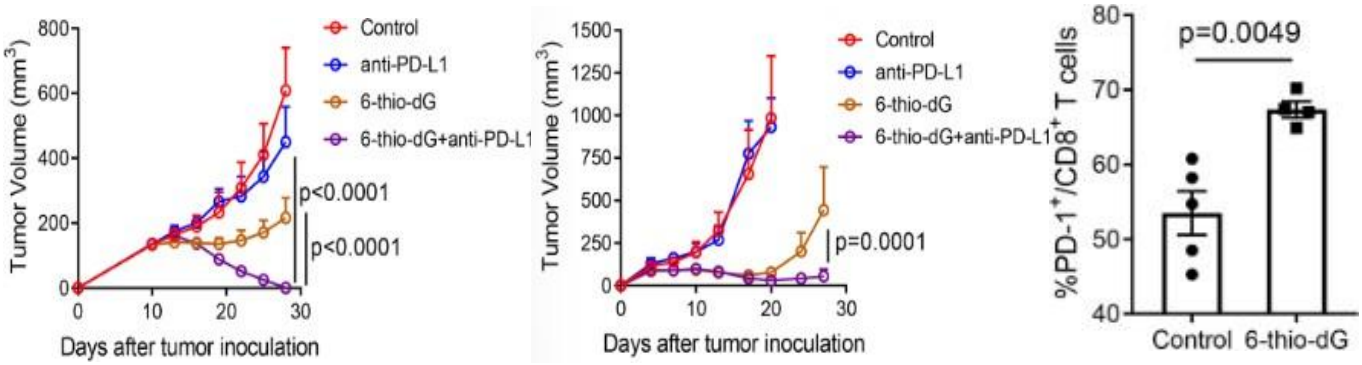

Exhibit 9: Tumor Volume in Mice Bearing TERT+ MC38 tumor (Left) and TERT+ LLC (Middle). Frequency of PD- 1+CD8+T cells (Right) Source: Mender et al., 2020

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)	(9,032,375.3)	(9,935,612.8)	(33,133,555.1)	(158,620,797.4)
Marketing Expense	-	-	-	-	(9,940,066.5)	(39,655,199.4)
Ratchet share expense	-	-	-	-	-	-
Research and Development	(11,112,257.0)	(10,009,229.0)	(16,014,766.4)	(24,022,149.6)	(33,133,555.1)	(47,586,239.2)
EBITDA	**(20,182,381.0)**	**(16,957,210.0)**	**(25,047,141.7)**	**(33,957,762.4)**	**(26,506,844.1)**	**51,551,759.2**
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	127,926.4	720,664.5	662,671.1	475,862.4
EBIT	**(20,005,662.0)**	**(16,877,256.0)**	**(24,919,215.3)**	**(33,237,097.9)**	**(26,094,173.0)**	**51,428,220.9**
Interest Income	34,490.0	318,367.0	316,842.8	137,974.8	286,646.4	215,079.4
Interest Expense	(6,863.0)	(57.0)	-	-	-	-
Profit before exceptional items, extraordinary items and tax	**(19,978,035.0)**	**(16,558,946.0)**	**(24,602,372.5)**	**(33,099,123.2)**	**(25,807,526.6)**	**51,643,300.3**
Paycheck protection program loan forgiveness	-	-	-	-	-	-
Change in fair value of warrant liability	205,130.0	(6,682,758.0)	944,698.0	-	-	-
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)**	**(23,657,674.5)**	**(33,099,123.2)**	**(25,807,526.6)**	**51,643,300.3**
Income tax (expense) benefit	-	-	-	-	-	(10,845,093.1)
Net earnings including noncontrolling interests	**(19,772,905.0)**	**(23,254,656.0)**	**(23,657,674.5)**	**(33,099,123.2)**	**(25,807,526.6)**	**40,798,207.2**

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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