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John D. Vandermosten
312-265-9588 / jvandermosten@zacks.com

scr.zacks.com
101 N. Wacker Drive, Chicago, IL 60606

TuHURA Biosciences, Inc. (HURA: NASDAQ)

HURA: 2025 Financial Results

TuHURA's valuation relies on a DCF model and a 15% discount rate applied to our cash flow estimates. We apply a success probability of 60% to the IFx-2.0 program in MCC and 15% to the anti-VISTA program in AML generating a 50% blended rate. We separately value the ADC program. The adjustments recognize regulatory and commercialization risks. The model includes contributions from the US, EU, Australia & the developed world.

Current Price (4/8/2026)	$1.81
Valuation	**$7.00**

OUTLOOK

TuHURA is a clinical-stage, oncology-focused biotechnology company advancing innate immune agonists, checkpoint inhibitors & antibody-drug conjugates (ADCs). Its IFx platform technology features IFx-2.0 tumoral injection delivery for Merkel cell carcinoma (MCC). IFx encodes a bacterial protein to be expressed in cancer cells, activating the innate immune system & subsequent cascade that may eliminate the tumor. Other assets include an anti-VISTA antibody acquired from Kineta & the ADC Δ-opioid receptor which may be used to treat blood cancers.

IFx-2.0 uses pDNA to encode the production of Emm55 on cancer cells to elicit an immune response. A pivotal trial is underway, with a plan for FDA approval in 24 months using accelerated regulatory pathways & support from Project Frontrunner. Other assets in the portfolio may also advance quickly with supportive early data.

TuHURA merged with Kineta and raised $14 million since the close of the transaction to support its pipeline. Additional capital raises and partnerships with established biopharma companies are expected.

Initial clinical studies target rare and blood cancers. Future opportunities lie in oncology using combinations with other immunotherapies.

SUMMARY DATA

52-Week High	**$4.44**
52-Week Low	**$0.41**
One-Year Return (%)	**-51.7**
Beta	**0.8**
Average Daily Volume (sh)	**1,736,496**
Shares Outstanding (mil)	**63.6**
Market Capitalization ($mil)	**115.2**
Short Interest Ratio (days)	**6.2**
Institutional Ownership (%)	**10.4**
Insider Ownership (%)	**25.7**
Annual Cash Dividend	**$0.00**
Dividend Yield (%)	**0.00**
5-Yr. Historical Growth Rates	
Sales (%)	**N/A**
Earnings Per Share (%)	**N/A**
Dividend (%)	**N/A**
P/E using TTM EPS	**N/A**
P/E using 2025 Estimate	**N/A**
P/E using 2026 Estimate	**N/A**
Zacks Rank	**N/A**

Risk Level	**Above Average**
Type of Stock	**Small-Growth**
Industry	**Med-Biomed/Gene**

ZACKS ESTIMATES

Revenue

(In millions of US$)

	Q1 (Mar)	Q2 (Jun)	Q3 (Sep)	Q4 (Dec)	Year (Dec)
2024	$0.0 A	$0.0 A	$0.0 A	$0.0 A	$0.0 A
2025	$0.0 A	$0.0 A	$0.0 A	$0.0 A	$0.0 A
2026					$0.0 E
2027					$0.0 E

Earnings per Share

	Q1	Q2	Q3	Q4	Year
2024	-$0.34 A	-$0.49 A	-$0.43 A	-$0.16 A	-$1.16 A
2025	-$0.16 A	-$0.21 A	-$0.14 A	-$0.12 A	-$0.63 A
2026					-$0.48 E
2027					-$0.51 E

WHAT'S NEW

Operational and Financial Results

On April 1st, 2026, TuHURA Biosciences, Inc. (NASDAQ: HURA) reported 2025 financial and operational results and filed its Form 10-K with the SEC. The company is conducting Phase III and Phase Ib/IIa trials in Merkel cell carcinoma (MCC) and is expected to soon start a Phase Ib/II trial for TBS-2025 in acute myeloid leukemia (AML). The IFx-2.0 Phase III is expected to read out in 2027. The TBS-2025 anti-VISTA asset is now the subject of FDA meetings centering on the development plan and clinical trials could begin in 2H:26. Other program work includes the identification of a lead antibody drug conjugate (ADC) in AML along with initiation of proof-of-concept studies. Outside of operational efforts, TuHURA added new team members and is presenting at scientific and investor conferences. Below, we summarize the company's 2025 financial results.

TuHURA generated no revenues in 2025 and expended $31.8 million on operational activities related to advancing IFx-2.0, TBS-2025 and other programs, producing a net loss of $30.1 million or $0.63 per share. For the year ending December 31st, 2025 and versus the same prior year period:

- Research & development expense totaled $20.5 million, increasing 54% from $13.3 million on higher facilities, salary and personnel related costs, greater clinical spending on IFx-2.0 and TBS-2025, and increased allocations to the preclinical IFx-3.0, myeloid derived suppressor cell (MDSC) and REM-001 programs;
- General & administrative expense totaled $11.2 million, which includes acquisition related costs of $3.7 million. The total was up markedly from $4.3 million, due to increases in non-cash stock compensation, merger transaction costs and expenses related to being a public company;
- Other items included $2.2 million that consisted of grant income related to Kintara's REM-001 asset and reimbursements from Health and Human Services and amounts related to share settlement to former Kineta employees, and employee retention tax credit. The most significant item was $1.6 million in income related to the fair value of Kineta merger holdback shares partially offset by a $185,000 loss on Kineta employee separation payments;
- Net interest expense was $489,000;
- Net loss was $30.1 million or $0.63 per share. Removing the nonoperating items from the "Other" line item produces a net loss of $32.3 million or $0.67 per share.

As of December 31st, 2025, TuHURA held $3.6 million in cash on its balance sheet. Cash burn for 2025 was $27.7 million while net cash generated from financing sources was $19.9 million which consisted of proceeds from a bridge note, warrants and common stock issuance partially offset by capital raising costs and transaction and liability payments related to Kintara. In October, TuHURA entered into a $3.0 million loan agreement of which $1.5 million of the loan was advanced upon execution. In November, TuHURA entered into an at-the-market (ATM) facility with HC Wainwright as its sales agent along with the filing of a Form S-3 registration statement making available $50 million in capacity for the ATM. Following the end of the reporting period, the second and third tranches of December's $15.6 million raise were completed. This added gross proceeds of $7 million.

Anti-VISTA (TBS-2025) Program

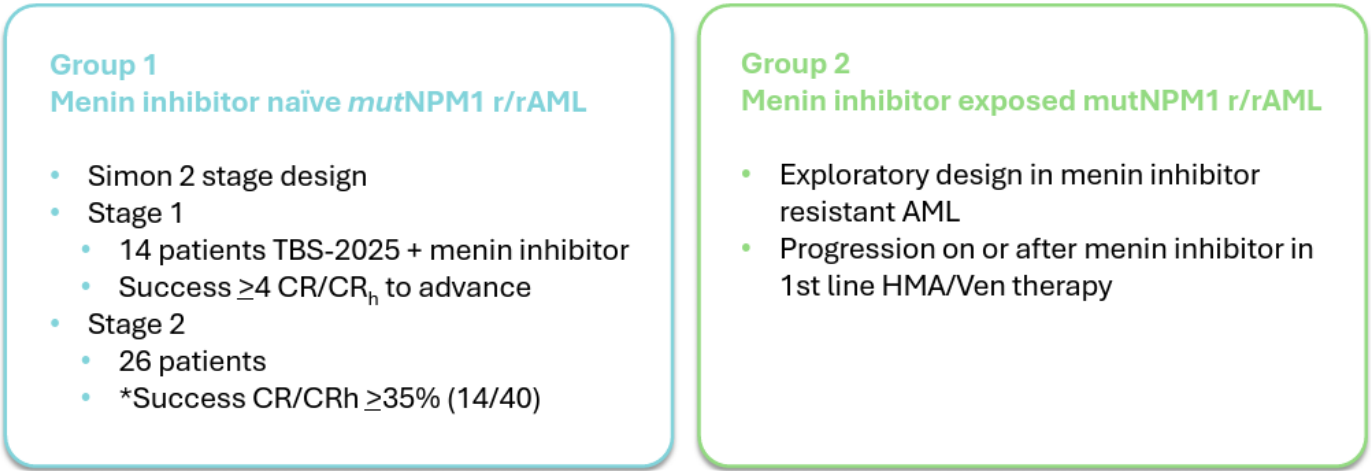
TuHURA closed the Kineta acquisition in June 2025 bringing the latter's anti-VISTA asset into the fold. Now designated TBS-2025, the candidate is a VISTA-blocking immunotherapy developed to reverse immunosuppression in the tumor microenvironment (TME). It is a fully-human engineered IgG1 monoclonal antibody that was designed to bind to VISTA through a unique epitope at physiologic and acidic pH levels. The product is being developed as an intravenous infusion. Under TuHURA's aegis, TBS-2025 is expected to be the subject of a Phase Ib/II trial in patients with relapsed/refractory (r/r) mutated nucleophosmin 1 (mutNPM1) AML.

Based on research that has demonstrated that mutated NPM1 and DNMT3A result in high expression of VISTA on the surface of leukemic blasts.[1] The presence of VISTA on these cells is believed to be the primary mechanism by which leukemic cells escape immune recognition and attack, resulting in a low treatment response rate and a short duration of response in AML.

[1] NPM1 and DNA methyltransferase 3A (DNMT3A) are two of the most common mutations in AML and typically co-mutated in myelodysplasia (MDS).

This February, TuHURA filed an Investigational New Drug (IND) Application with the FDA for TBS-2025. It submitted the document to the Division of Hematologic Malignancies for the treatment of mutNPM1 r/r AML in combination with a menin inhibitor. If cleared, the Phase Ia portion plans to enroll 14 patients and the Phase II, 26 subjects. TuHURA's next steps with the TBS-2025 program are to determine a starting dose in an abbreviated Phase Ib trial. The subsequent Phase II trial will investigate TBS-2025 in combination with a menin inhibitor for patients with mutNPM1 r/r AML who were previously untreated with a menin inhibitor. These studies are slated to begin in 2H:26.

Exhibit I – TBS-2025 Phase Ia/II Trial Design



Source: TuHURA March 2026 Presentation

In the press release announcing the IND, Dr. Bianco pointed out that leukemogenic mutations common in AML may drive the expression of VISTA on the surface of leukemic cells, which in turn shut down the immune response. The anti-VISTA antibody's mechanism raises the shield so the immune system can kill these cells. He continued, noting that complete response rates using menin inhibitors as monotherapy are below 25% and of short duration. Adding TBS-2025 to the treatment paradigm may markedly increase both the magnitude of response and its duration. Success in this endeavor would provide TuHURA the data it needs to seek an accelerated approval route with the FDA.

In March 2026, TuHURA announced that Dr. Craig Tendler would lead the anti-VISTA program in AML. Dr. Tendler's first public association with TuHURA was the company's announcement that he would join TuHURA's board of directors in March 2025. Last month, it was announced that he would take on the responsibilities consistent with those of Chief Medical Officer (CMO) and lead the TBS-2025 program. He will continue his role on the board. A press release provided a biography for the thirty-year industry veteran noting his tenure at Johnson & Johnson. Joining Dr. Tendler is Amanda Garofalo, who was announced as SVP of Clinical Operations on April 7th, 2026. She will assist with the development of TBS-2025 and TuHURA's other clinical programs.

Menin Inhibitor Background

Menin inhibitors are a targeted therapy for NPM1-mutated r/r AML that work by disrupting the menin-dependent transcriptional program that leukemic cells use to maintain HOX/MEIS1 expression, which helps drive survival and differentiation block.[2] In practice, these small molecules can induce differentiation and remissions in this disease, and they are now part of the treatment landscape for this molecular subtype, with activity seen most often in heavily pretreated patients. NPM1-mutated AML is biologically dependent on menin-mediated signaling, so blocking menin can turn off an oncogenic program rather than just broadly killing dividing cells. That makes menin inhibition especially relevant in r/r mutNPM1 AML, where options are limited and targeted therapy is needed.[3,4]

TBS-2025 may pair well with a menin inhibitor because the combination targets two different resistance layers in AML. Anti-VISTA antibodies may reverse immune suppression in the tumor microenvironment while menin inhibitors suppress the leukemic oncogenic transcription program in mutNPM1 AML. In murine mutNPM1 AML, loss of VSIR (the gene encoding VISTA) was associated with an immune response and improved survival. This suggests VISTA blockade could help the immune system clear leukemic cells that remain after menin inhibition.[5]

[2] Differentiation block refers to a state where leukemia cells are held in an immature, rapidly dividing stage and are unable to mature into functional white blood cells.
[3] Isidori, A., Marconi, G. The role of menin inhibitors in acute myeloid leukemia. Current Opinion in Oncology. November 2025.
[4] Fiskus, W., *et al*. Effective Menin inhibitor-based combinations against AML with MLL rearrangement or NPM1 mutation (NPM1c). Blood Cancer Journal. January 2022.
[5] TuHURA Biosciences Pipeline. Accessed April 2026.

ASH Presentation

TuHURA addressed the American Society of Hematology (ASH) Annual Meeting and Exposition with a presentation and poster on Sunday, December 7th in Orlando, Florida. Updated results from the delta opioid receptor (DOR) program were provided. The content highlighted DOR expression on myeloid-derived suppressor cells (MDSCs) and the effects of DOR inhibition on tumor-associated macrophages (TAMs).

The oral presentation was entitled *Delta Opioid Receptor (DOR) Expression on Myeloid-Derived Suppressor Cells (MDSCs) Represents a Novel Target to Overcome Resistance to Immune Checkpoint Inhibitors (ICIs)*. The newly appointed Vice President Immunology at TuHURA, Dr. Michael Turner, presented the data validating DOR expression on MDSCs. The data further showed that the pharmacological antagonism of the DOR reduced the suppressive activity of MDSCs. The study showed that antagonism of the DOR with a specific inhibitor modulated a variety of direct and indirect MDSC-mediated immunosuppressive factors and reversed T cell suppression. These features suggest that the DOR may be a novel target to reprogram MDSC-induced immunosuppression in the tumor microenvironment (TME).

The poster presentation was entitled *Delta Opioid Receptor (DOR): A Novel Target for Reprogramming Tumor-Associated Macrophage (TAM) Immunosuppressive Phenotype to Overcome Acquired Resistance and Enhance the Effectiveness of Cancer Immunotherapies.* It was presented by Dr. Krit Ritthipichai, TuHURA's Director of Immunology. The poster presented data showing that DOR is highly expressed on tumor-infiltrating myeloid cells, particularly TAMs. The poster concluded that the TME induces DOR upregulation relative to peripheral macrophages and that targeting the DOR may be a strategy to address several vulnerabilities. These include reprogramming suppressive TAMs and MDSCs, alleviating T-cell dysfunction, and potentially overcoming resistance to checkpoint blockade and other immunotherapies.

Data from this work show that the DOR is expressed on Tregs and further controls the expression of FOXP3, a critical immunosuppressive gene. In parallel with work conducted by Nobel Prize winners Mary E. Brunkow, Fred Ramsdell, and Shimon Sakaguchi and their groundbreaking discoveries on peripheral immune tolerance, TuHURA's work is important because it shows that the tumor microenvironment can be tuned to be either more or less immunosuppressive by agonizing or antagonizing the DOR. This is especially important for TuHURA's anticipated ADC that will combine a DOR inhibitor with anti-VISTA, the combination of which is expected to increase the activity of T cells immune action against cancer cells.

After reviewing laboratory work identifying the expression of the DOR on various cells, the poster offered several conclusions:

- DOR is highly expressed in tumor-infiltrating myeloid cells, particularly TAMs, indicating that the tumor microenvironment induces DOR upregulation relative to peripheral macrophages;
- M1 macrophages consistently exhibited the highest DOR expression, whereas M2 macrophages, which are key drivers of immunosuppression showed the lowest, revealing a polarization-dependent regulation of DOR;
- Macrophage polarization states were validated by their characteristic immunophenotypes, functional activities, and cytokine secretion profiles, supporting the biological relevance of DOR expression patterns;
- Pharmacological inhibition of DOR effectively reversed M2 macrophage–mediated suppression of T-cell proliferation, demonstrating a functional role for DOR in driving immunosuppressive signaling;
- Targeting DOR provides a promising strategy to reprogram suppressive TAMs and MDSCs, alleviate T-cell dysfunction, and potentially overcome resistance to checkpoint blockade and other immunotherapies.

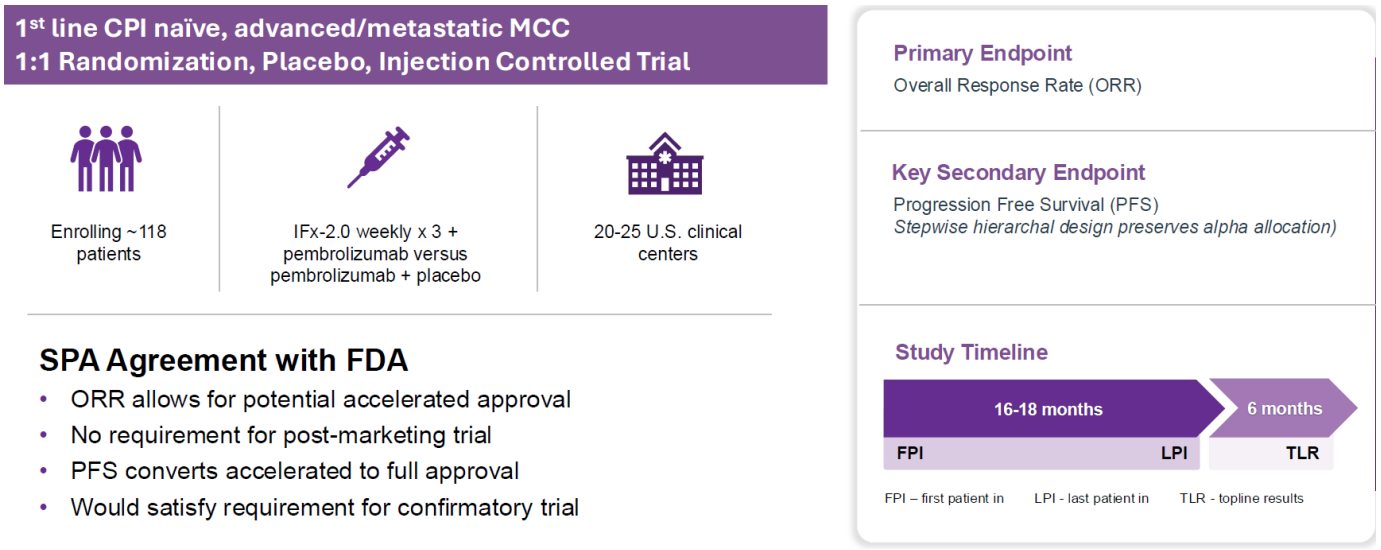
Financing and Corporate Update

TuHURA announced a registered direct offering on December 9th that raised $15.6 million through the issuance of 9,462,423 shares of common stock and warrants. The purchase price for the equity shares was set at $1.65 and the exercise price for the warrants was $1.95. Warrants can be exercised six months after issue. The closing of the transaction was scheduled to occur in three tranches, with all completed by the end of February 2026.

Phase III IFx-2.0 Trial in MCC

TuHURA launched its pivotal Phase III study for its IFx-2.0 candidate in Merkel cell carcinoma (MCC) in June 2025. In the latest earnings report, TuHURA provided its latest set of milestones for the program. They include obtaining Orphan Drug Designation for IFx-2.0 in MCC in 1H:26, reporting preliminary data from the Phase Ib/IIa study of IFx-2.0 in 2H:26 and a topline readout of the Phase III study in 2H:27.

Exhibit II – IFx-2.0 Phase III Trial Design

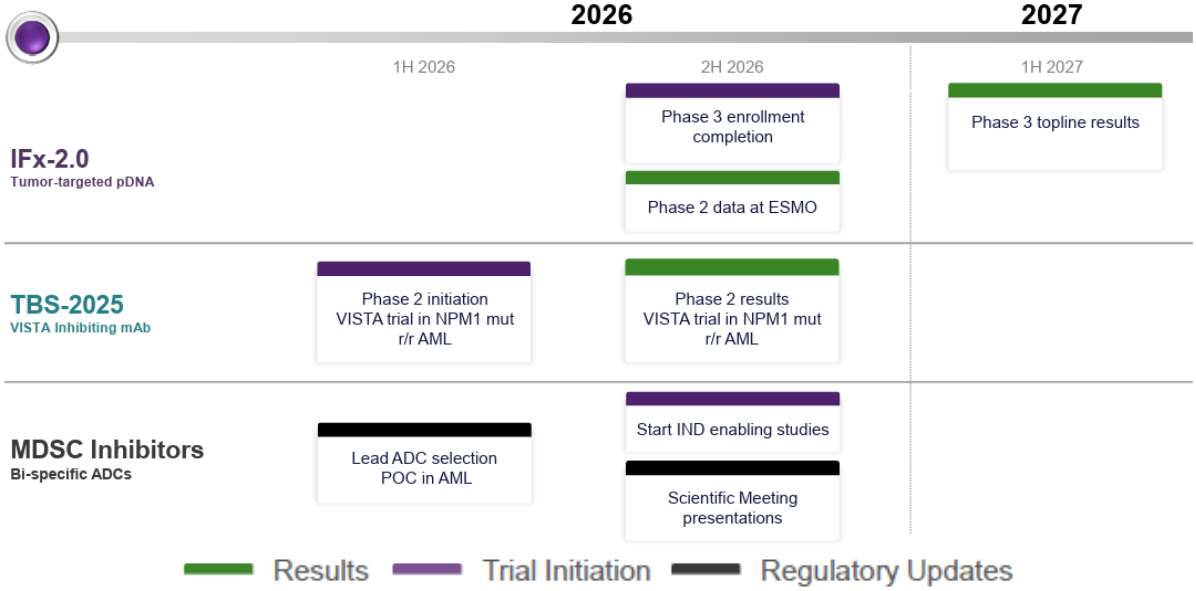


Source: TuHURA September 2025 Presentation

IFx-2.0 will prepare for a new drug application using the FDA's accelerated approval program under a special protocol assessment (SPA). The trial was designed with the input of the FDA's Oncology Center of Excellence (OCE). Accelerated approval allows the sponsor to use surrogate endpoints that predict clinical benefit. In most cases, an accelerated approval will require post-market confirmatory trials to verify the clinical benefit. However, in this case, the FDA has indicated that secondary endpoints that demonstrate clinical benefit may be used. If successfully achieved, the trial may satisfy the requirements for full approval.

Upcoming Milestones

Exhibit III – TuHURA Milestones



Source: TuHURA March 2026 Corporate Presentation

Milestones

- IFx-2.0 MCC trial launch – June 2025
- IFx-2.0 MCC of unknown origin basket trial launch – May 2025
- Close of Kineta acquisition – June 2025
- Appointment of Dr. Michael Turner as VP of Immunology – November 2025
- Delta Opioid Receptor presentation at ASH – December 7th, 2025
- FDA grants Orphan Drug Designation for IFx-2.0 in melanoma – February 2026
- IND filed for TBS-2025 in AML – February 2026
- Presentation at Oppenheimer Healthcare Life Sciences Conference – February 2026
- Compliance regained with NASDAQ minimum bid requirements – February 2026
- Presentations at Citizens Life Science & Leerink Global Healthcare conferences – March 2026
- Dr. Tendler to lead TuHURA's anti-VISTA program – March 2026
- Selection of lead ADC/DOR inhibitor – 1Q:26
- Appointment of Amanda Garofalo as SVP of Clinical Operations – April 2026
- IFx-2.0 Phase Ib/IIa preliminary results – 2Q:26
- FDA meeting for TBS-2025 – June 2026
- Initiate ADC *in vivo* proof of concept studies – 2H:26
- TBS-2025 Phase II initiation – 2H:26
- First proof of concept with *in-vivo* results for lead ADC – 2H:26
- Scientific meeting presentations for lead ADC selection – 2H:26
- IFx-2.0 Phase III topline results – 2H:27

Company Pipeline

Exhibit IV – TuHURA Pipeline

PROGRAM	DRUG CANDIDATE	INDICATION	PRECLINICAL / PHASE 1 / PHASE 2 / PHASE 3	Upcoming Milestone Targets
Innate Immune Agonists	IFx-2.0 Tumor-targeted pDNA	1st Line Merkel Cell Cancer Keytruda® + IFx-2.0 or placebo[1]		**2H 2027:** Phase 3 Expected Topline Results
		Primary Checkpoint Inhibitor Resistant Metastatic Cancer "Basket" Trial		**2H 2026:** Phase 1a/2b "Basket" trial results ESMO*
TME Modulators Negative Immune Regulators	TBS-2025 VISTA inhibiting mAb[1]	*mut*NPM1 Acute Myeloid Leukemia		**2H 2026:** Expected Phase 1b/2 Trial Initiation
TME Modulators MDSC Inhibitors	Bi-specific ADCs	Myelodysplasia Acute Myeloid Leukemia		**2H 2026:** Expected to initiate ADC *in vivo* POC studies

*European Society of Medical Oncology

Source: TuHURA 2025 Form 10-K

Valuation

We updated our valuation to reflect revised shares outstanding and anticipated capital raises over the next 18 months. Due to the decline in TuHURA share price, the number of shares expected to be issued has increased. While our enterprise value has not changed, the increase in the denominator reduces our valuation to $7.00 per share.

Summary

TuHURA reports 2025 financial results with two active clinical trials advancing IFx-2.0 in MCC and one for TBS-2025 planned for 2H:26. Additional work is planned to advance the ADC towards proof-of-concept studies. Beyond program work, TuHURA board member Dr. Tendler assumed the duties of Chief Medical Officer and will be assisted by recent hire Amanda Garofalo, the SVP of Clinical Operations. Other efforts include participation in scientific and investor conferences. TuHURA laid out several milestones for its programs which provide a number of anticipated newsworthy events as 2026 progresses. We adjust our valuation for actual and anticipated equity shares to generate a valuation of $7.00 per share.

PROJECTED FINANCIALS

TuHURA Biosciences, Inc. - Income Statement

TuHURA Biosciences, Inc.	2024 A	Q1 A	Q2 A	Q3 A	Q4 A	2025 A	2026 E	2027 E
Total Revenues ($USD)	**$0**	**$0**	**$0**	**$0**	**$0**	**$0**	**$0**	**$0**
Research & Development	$13,335	$4,582	$4,927	$4,969	$6,055	$20,533	$24,000	$30,000
General & Administrative	$4,314	$2,435	$4,949	$1,761	$2,118	$11,263	$12,000	$13,500
Other Operational Items								
Income from Operations	($17,649)	($7,017)	($9,876)	($6,730)	($8,173)	($31,796)	($36,000)	($43,500)
Other Items	($256)	$253	$323	($362)	$2,020	$2,233	$0	$0
Net Interest Expense	($3,777)	$100	$29	($10)	($608)	($489)	$0	$0
Pre-Tax Income	($21,682)	($6,664)	($9,524)	($7,102)	($6,761)	($30,052)	($36,000)	($43,500)
Provision for Income Tax	$0	$0	$0	$0	$0	$0	$0	$0
Tax Rate								
Net Income	**($21,682)**	**($6,664)**	**($9,524)**	**($7,102)**	**($6,761)**	**($30,052)**	**($36,000)**	**($43,500)**
Net Margin								
Reported EPS	**($1.16)**	**($0.16)**	**($0.21)**	**($0.14)**	**($0.12)**	**($0.63)**	**($0.48)**	**($0.51)**
YOY Growth								
Basic Shares Outstanding	18,663	42,250	44,555	50,666	56,212	47,927	75,000	85,000

Source: Company Filing // Zacks Investment Research, Inc. Estimates

HISTORICAL STOCK PRICE

TuHURA Biosciences, Inc. – Share Price Chart[6]

[6] Source: Barchart. Note that the price chart measures share price since the October 18th, 2024 date of the reverse merger with Kintara.

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