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TuHURA Biosciences, Inc. (HURA: NASDAQ)

HURA: Initiating an Immune Response

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 (2/3/2025)	$4.85
Valuation	**$9.25**

INITIATION

TuHURA is a clinical-stage, oncology-focused biotechnology company advancing innate immune agonists, checkpoint inhibitors & antibody-drug conjugates (ADCs). It offers the IFx platform technology featuring the IFx-2.0 tumoral injection delivery approach for Merkel cell carcinoma (MCC) & the IFx-3.0 intravenous delivery approach for lymphoma. IFx encodes a bacterial protein to be expressed in cancer cells, activating the innate immune system and subsequent cascade that may eliminate the tumor. Other assets include the anti-VISTA KVA12, recently acquired from Kineta & the ADC Δ-opioid receptor which may be appropriate for 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 planned for 1H:25, 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 recently reverse merged into a shell company and raised $31 million to support its pipeline of products. Additional capital raises and partnerships with established biopharma companies are expected.

While initial clinical studies target rare and blood cancers, we see broader applicability in oncology using combinations with other immunotherapies.

SUMMARY DATA

52-Week High	**7.93**
52-Week Low	**2.84**
One-Year Return (%)	**10.9**
Beta	**0.9**
Average Daily Volume (sh)	**336,332**
Shares Outstanding (mil)	**43.6**
Market Capitalization ($mil)	**211.5**
Short Interest Ratio (days)	**0.3**
Institutional Ownership (%)	**13**
Insider Ownership (%)	**50**
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 USD)

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

Earnings per Share

	Q1	Q2	Q3	Q4	Year
2024	-$1.79 A	-$2.60 A	-$2.42 A	-$0.09 E	-$1.60 E
2025	-$0.15 E	-$0.15 E	-$0.17 E	-$0.17 E	-$0.73 E
2026					-$0.65 E
2027					-$0.60 E

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INITIATION

We are initiating coverage of TuHURA Biosciences, Inc. (NASDAQ: HURA) with a valuation of $9.25 per share. This value is based on our estimates for successful development and commercialization of IFx-2.0 in patients with Merkel cell carcinoma (MCC) and KVA12 which will be advanced in acute myeloid leukemia (AML). IFx-2.0 is scheduled to begin its Phase III study in 1H:25 under a Special Protocol Assessment (SPA) agreement with the FDA. The trial will benefit from the FDA's Project FrontRunner initiative which will allow IFx-2.0 to be evaluated in an earlier clinical setting with only one Phase III required for accelerated approval. The recently acquired anti-V-domain Immunoglobulin suppressor of T-cell activation (VISTA) asset is completing a Phase I dose-finding study in advanced solid tumors. This class of biologic may work particularly well in blood cancers with mutated nucleophosmin 1 (NPM1). The antibody may be combined with a delta opioid receptor (DOR) inhibitor and/or a menin inhibitor in future studies if supported by clinical work. TuHURA will pursue an indication in AML, where there is evidence of a signal. A Phase IIa/b trial for the candidate in mutated NPM1 (mNPM1) AML is slated to begin later this year following the close of the merger with Kineta. TuHURA's antibody-drug conjugate (ADC) is expected to traverse a novel route in this space, binding to the DOR and carrying an immunotherapy, possibly TuHURA's very own anti-VISTA agent. We see the ADC asset as a desirable partnering asset with established biopharma companies.

IFx-2.0 is an intratumorally injected plasma DNA (pDNA) that infects cancer cells to produce bacterial proteins on their surface. These proteins stimulate an innate immune response which destroys cancer cells and subsequently activates an adaptive immune response to overcome resistance. TuHURA has demonstrated safety and proof of concept in several small early-stage trials. To date, the company has completed four Phase I studies. They have shown an overall response rate (ORR) for MCC in many subjects. The dose escalation portion of the study established that IFx-2.0 was well-tolerated at all dose levels. Further study evaluated MCC patients who had previously received checkpoint therapy. Results showed that IFx-2.0 may overcome primary resistance to checkpoint inhibitor therapy and may provide support for administering the agent before checkpoint inhibitor therapy to increase response rates.

Recent research has shown a correlation between high NPM1 mutations and VISTA expression, particularly in AML. As mutated NPM1 (mNPM1) drives leukemogenesis through menin and the efficacy of menin inhibitors in producing treatment responses in mNPM1-associated AML, combination therapy using a VISTA-inhibiting antibody is a promising approach to improve response rates in AML. In 2H:25, TuHURA expects to evaluate this further with a proof-of-concept study. The anti-VISTA asset may be considered as a monotherapy or in combination with the ADC or IFx programs.

Merkel cell carcinoma (MCC) is a type of skin cancer originating from Merkel cells and can grow rapidly if not treated. While a relatively high proportion of MCC patients respond to checkpoint inhibitors, there remains an unmet need in patients that do not respond or those that relapse. The annual incidence of MCC is about 3,300 persons in the United States and 4,500 in Europe and Australia.

Acute myeloid leukemia (AML) has a substantially higher incidence than MCC with over 20,000 cases per year and global incidence of about 1.5 per 100,000. AML is a cancer of the blood and bone marrow where immature blood cells proliferate and interfere with normal blood cell production. It primarily affects white blood cells called myeloid cells and progresses quickly without treatment. About a third of AML patients carry the NPM1 mutation, which drives overexpression on AML blasts, most of which, coincidentally, have high VISTA expression.

In addition to the lead programs for IFx-2.0 and VISTA, TuHURA has a portfolio of other programs in line for further development or divestiture. The IFx-3.0 program seeks to achieve intravenous delivery of the Emm55 mRNA targeting CD22, which is overexpressed on cancerous B-cells. The antibody-drug conjugate (ADC) program features a delta-receptor-selecting-small-molecule designed to target myeloid derived suppressor cells (MDSCs) and counteract their immunosuppressive effect. As part of its merger with Kintara, TuHURA inherited a photodynamic therapy for unresectable cutaneous metastatic breast cancer (CMBC) designated REM-001. The asset is in a small proof of concept trial that, if successful, will qualify Kintara shareholders for a contingent value right (CVR) to be paid in shares. Another party will advance REM-001 beyond this point.

Based on the strong data generated, TuHURA recently raised $31 million as part of the reverse merger into Kintara last October. $15 million was spent to acquire Kineta in cash and stock. In addition to the cash on hand, TuHURA may raise additional funds via the private investment in public equity (PIPE) route. Company management has stated that it has sufficient capital to fund operations into late 2025. We expect another capital raise to provide further financial support to the pipeline programs.

TuHURA has outlined a plan for 2025 to initiate the IFx-2.0 Phase III trial, begin the KVA12 Phase II trial in mutated NPM1 AML and conduct further preclinical research in its IFx-3.0 and targeted ADC programs. The company expects that it will be able to fully enroll the IFx-2.0 trial within twelve months of dosing the first patient. Six months later, topline results can be available for regulatory submission. Accelerated approval will allow for an expedited review of the application, a timeline that, if everything goes as planned, could support marketing IFx-2.0 about two years after starting the trial. KVA12's pathway could be similarly streamlined after a short proof-of-concept trial slated for 2H:25.

We expect further updates to provide specific timing for the pipeline programs throughout 2025 and should see clinical trial updates at society events including AACR, ASCO, ESMO, SITC and ASH.

Key reasons to own TuHURA Biosciences shares:

- **IFx-2.0 offers the potential to overcome resistance to immunotherapies**
  - **Employs an pDNA that produces a bacterial protein on the tumor cell**
  - **Overcomes tumor resistance mechanisms**
  - **Provides synergies with checkpoint inhibitors**
  - **Activates both the innate and adaptive immune systems**
  - **Credible 24-month study timeline to approval**
- **Recently acquired VISTA asset**
  - **Addresses mechanism of cancer immune resistance**
  - **Limited competition from other VISTA assets**
  - **Integrates innate and adaptive immune responses**
  - **Demonstrates synergies with checkpoint inhibitors (anti-PD-1/PD-L1/CTLA-4)**
  - **Favorable safety profile with no evidence of cytokine release syndrome (CRS)**
  - **Can be used as monotherapy or in combination with other TuHURA assets**
- **Offers preclinical programs that address shortcomings in other therapies**
  - **IFx-3.0 tumor-targeted mRNA**
  - **Bi-specific antibody and peptide drug conjugates (ADCs & PDCs)**
- **Access to expedited treatment from the FDA**
  - **Single Phase III accelerated approval for IFx-2.0**
  - **Special Protocol Assessment agreement with FDA**
  - **Visibility to accelerated approval pathway for VISTA in AML**
- **Robust intellectual property**
  - **Over 40 IFx platform patents internally owned**
  - **DOR & ADC patents jointly owned with West Virginia University & Moffitt Cancer Center**
  - **Acquired Kineta patents for anti-VISTA antibodies**

In the following sections, we introduce immuno-oncology, paying particular attention to innate immune agonists and checkpoint inhibitors. We examine the innate and adaptive immune systems work together to generate abscopal effects throughout the body. We follow with a description of TuHURA's therapeutic candidates including IFx-2.0, KVA12, ADCs and the next generation IFx-3.0 infused therapy. The following chapter reviews the company's clinical programs including IFx-2.0 Phase I and anticipated Phase III study and KVA12 which has completed a Phase I and is preparing for a Phase II. We also include a brief description of TuHURA's other assets including the ADC and IFx-3.0 programs. The next part discusses the primary indications being pursued in MCC and AML. The epidemiology, etiology and other details of these indications are included. This is followed by a short summary of TuHURA's intellectual property (IP), Project Frontrunner and the SPA for IFx-2.0. We then discuss other competitors in the immuno-oncology space, including peers advancing innate immune agonists and anti-VISTA programs. The subsequent section reviews TuHURA's recent financial and operational history followed by an introduction to the management team and an analysis of company-specific risks. Our valuation discussion details the assumptions behind our target price. Our work generates a valuation of $9.25 per share.

Immunotherapy

Immuno-oncology

Immuno-oncology (I-O), or cancer immunotherapy, is an approach to fighting cancer that leverages the body's immune system to attack the disease. Usually, the immune system can eliminate these malignancies, but in some cases, mutant cells can adapt to hide by expressing deterrent proteins on their surface, among other mechanisms. When I-O drugs are administered, they allow the immune system to recognize harmful cells and destroy them. In many cases with fewer, shorter-lived side effects compared to surgery, chemotherapy and radiotherapy. Immunotherapy is frequently preferred because it is associated with fewer adverse events, maintains its potency, works well in conjunction with other therapies and can better target the tumors. Several classes of immunotherapy exist including therapeutic vaccines, checkpoint inhibitors, immune modulators, adoptive cell therapy (CAR-T), oncolytic viruses and antibody-drug conjugates (ADCs) to name the most prominent examples. I-O can be either active or passive. Active approaches direct the immune system to kill cancer cells by targeting tumor antigens. Passive approaches augment existing tumor responses by using a variety of proteins, lymphocytes and cytokines to enhance their activity.

Innate Immune Agonists

Innate immune agonists are a type of immunotherapy designed to activate the innate immune system to fight cancer by stimulating immune cells to begin an antitumor immune response. Some common immune agonists include toll-like receptor (TLR) agonists, NOD-like receptor (NLR) agonists, stimulator of interferon genes (STING) agonists and cytokine-based agonists among others. Innate immune agonists are molecules that stimulate the innate immune system, the body's first line of defense against pathogens. These agonists interact with specific receptors or pathways in innate immune cells, such as macrophages, dendritic cells, and natural killer (NK) cells, to activate or enhance immune responses. Their primary function is to trigger an immediate response to infections and to prepare the immune system for subsequent adaptive immunity.

The innate immune system recognizes general patterns that signal danger or infection which occurs through proteins called pattern recognition receptors (PRRs). These receptors are vigilant for specific suspicious patterns such as bacterial cell walls, viral genetic material, signals from damaged cells and other molecular patterns that are not normally present. An innate immune agonist is a substance that deliberately triggers these pattern recognition receptors. When it binds to these receptors, it activates an immune response. First, the cells release signaling molecules called cytokines and chemokines, which attract other immune cells. This causes inflammation and recruits more immune cells to the site. The activated cells also become more active in their efforts to neutralize pathogens and other non-self tissue, and they help activate the adaptive immune system to deploy a more targeted response. In medical applications, particularly cancer treatment, innate immune agonists are being studied as new therapies. By activating the innate immune system in a controlled way, researchers hope to strengthen the body's natural ability to fight cancer cells. This approach is particularly relevant because it may synergize with other cancer treatments, like checkpoint inhibitors, by creating a more immunologically active environment around tumors.

Checkpoint Inhibitors

Checkpoint inhibitors (CIs) have become one of the most visible immunotherapies in life sciences. Initial work in the early 1980s by industry luminary James Allison tested checkpoint inhibitors in animal models. This laboratory work led to the discovery of the first immune checkpoint molecule in 1987, designated cytotoxic T-lymphocyte antigen number 4 (CTLA-4). Further efforts led to the first trial for the drug class in 2000 with ipilimumab, a monoclonal antibody that binds and blocks the CTLA-4 receptor. This shift to immunotherapy developed medicines based on the tumor makeup rather than the site of the disease. After a decade of clinical trials and traversing the regulatory process, ipilimumab was approved in 2011.

While the potential for checkpoint inhibitors was understood in the oncology field, it became more widely known to the general public when Jimmy Carter announced he was cancer free in 2015. The former president's metastatic melanoma was treated with the investigational drug pembrolizumab, which was a different type of checkpoint inhibitor targeting the programmed cell death protein 1 (PD-1). The monoclonal antibody (fthe form of all checkpoint inhibitors) was able to release the brakes on his immune system, thereby allowing it to attack and shrink the tumors. The medicine allowed him to return to health without needing further treatment. This triumph put checkpoint inhibitors on the front page for those in the oncology field and for the layman.

While CIs were an impressive breakthrough in immuno-oncology, the class has weaknesses. Complete responses are frequently below 10% and most patients have no response or a partial response. Furthermore, CIs are less effective in more common cancers such as breast cancer, non-small cell lung cancer (NSCLC), prostate cancer and colorectal cancer. One of the reasons why approved therapies in the anti-PD-1, PD-L1 and CTLA-4 classes fail to stimulate clearance of the tumor is the lack of tumor-infiltrating lymphocytes (TILs) in and around the tumor. When these immune cells have not permeated a tumor, it is known as cold or immunologically suppressed.

CI's remove the restraints impeding the immune system's fighters. However, the fighters must infiltrate the tumor microenvironment for the immunotherapy to work. Other resistance mechanisms may also emerge, where cancer cells may upregulate other immune checkpoints or recruit immunosuppressive cells, such as regulatory T cells or myeloid-derived suppressor cells, to counteract the immune response induced by the checkpoint inhibitors. This has led researchers to identify combination therapies that can activate the immune system in the tumor environment and help overcome resistance mechanisms.

Therapeutic Candidates

IFx-2.0: Checkpoint Experienced Solid Tumors

IFx-2.0 is a nanoparticle complex consisting of a mammalian expression plasmid[1] encoding Emm55. Emm55 is a bacterial protein from *Streptococcus pyogenes* that has been used to stimulate the innate immune system to attack tumors. The bacterial protein is expressed on the surface of tumor cells, making them appear foreign to the body's immune system. This expression then primes a cascade of immune events that exposes the patient-specific abnormal tumor antigens to the effector mechanisms of the immune system. The immune response becomes systemic as inter-antigenic epitope spreading produces neoantigens to naïve T cells. The immune system primes, activates, and proliferates cytotoxic T cells and tumor-specific B cells to attack the tumor expressing the bacterial protein. Therefore, injected lesions are targeted along with non-injected lesions through the abscopal effect. This is especially important when the mutational phenotype varies greatly among individual lesions.

Exhibit I – IFx-2.0 Mechanism of Action

Source: TuHURA January 2025 Presentation

IFx-2.0 functions as an innate immune response agonist. It activates the toll-like receptor (TLR)-2 and pathogen-associated molecular pattern (PAMP) sentry mechanism to identify the Emm55 protein and activate the innate immune response in the tumor. This provides optimal neoantigen presentation and inter-antigenic epitope spreading.

IFx-2.0 is administered as an intralesional injection. In turn, the injection of IFx-2.0 into the lesion facilitates the expression of the immunogenic Emm55 protein by the tumor cells. The investigational drug product IFx-Hu2.0 is composed of the drug substance pAc/Emm55 (pDNA) complexed with the two excipients in vivo-jetPEI (linear polyethylenimine), a transfection reagent, and dextrose, a pDNA/polyethylenimine complex stabilizer.

[1] Plasmids are small, circular pieces of DNA that exist naturally in bacteria, separate from their main chromosome. Scientists have learned to harness plasmids and engineer them to encode proteins that can be used to produce vaccines and generate therapeutic proteins, enzymes and antigens.

Source: TuHURA January 2025 Presentation

Immune Fx (IFx) Platform

TuHURA's Immune Fx (IFx) platform employs a plasmid DNA (pDNA) or messenger RNA (mRNA) to infiltrate cancer cells that subsequently express a bacterial protein that activates the immune system. After the pDNA or mRNA is introduced into the cancer cell, it produces a highly immunogenic bacterial protein, Emm55. Bacterial protein Emm55 is a specific protein found in Group A Streptococcus (GAS), which can cause various infections in humans. In the context of the IFx platform, the expression of Emm55 makes a tumor cell appear to be a bacterium, thereby activating the innate immune response. Innate immunity is the non-specific, immediate immune response that provides rapid protection against invading pathogens. Unlike adaptive immunity, it does not require prior exposure to a specific pathogen and acts quickly to prevent infection and limit pathogen spread. It is the body's first line of defense against pathogens and protects against all microbial pathogens by immediately preventing the spread and movement of foreign pathogens throughout the body.

KVA12: Anti-VISTA Antibody

In a December 12th, 2024 press release, TuHURA announced that it will merge with Kineta, Inc. and acquire its VISTA-blocking immunotherapy KVA12. TuHURA will prepare to launch a Phase IIa/b clinical trial investigating KVA12 in mutated nucleophosmin 1 (mNPM1) acute myeloid leukemia (AML) in 2025. TuHURA had previously provided Kineta a $5 million exclusivity fee and additional operating funds to support its ongoing trial for KVA12. This trial is pursuing an indication in advanced solid tumors. TuHURA will shift the focus of the development program towards blood-related cancers such as AML.

KVA12 is a VISTA-blocking immunotherapy developed to reverse immunosuppression in the tumor microenvironment (TME). The candidate 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.

VISTA is upregulated after treatment with other checkpoint inhibitors and is associated with poor survival.[2,3] Blocking VISTA has been shown to activate an immune response in cold tumors through innate and adaptive mechanisms. Blocking high VISTA expression on myeloid cells can drive tumor inflammation, allowing T cell therapies to be more effective. Anti-VISTA can reduce T regs, specifically tumor-specific Foxp3 CD4 regulatory T cells, thereby

[2] Kuklinski, L.F., *et al*. VISTA expression on tumor-infiltrating inflammatory cells in primary cutaneous melanoma correlates with poor disease-specific survival. Cancer Immunology, Immunotherapy. July 2018.
[3] Kakavand, H., *et al*. Negative immune checkpoint regulation by VISTA: a mechanism of acquired resistance to anti-PD-1 therapy in metastatic melanoma patients. Modern Pathology, December 2017.

increasing the effectiveness of cancer immunotherapy. It can enhance the infiltration, proliferation and effector function of tumor-reactive T cells within the tumor microenvironment (TME) and reverse the suppressive environment.[4]

Exhibit III – KVA12 Structure

Source: Kineta management

Cytokine Release Syndrome

Cytokine release syndrome (CRS) is a systemic inflammatory response that can occur as a result of certain medical treatments, particularly immunotherapies such as CAR-T cell therapy or certain monoclonal antibody treatments. It is caused by the release of large amounts of cytokines into the bloodstream. This activates immune cells that can produce many symptoms similar to that of the flu. When severe, CRS can be life-threatening and cause organ dysfunction or failure.

Exhibit IV – Absence of CRS-Associated Signal in Multiple Studies

Kineta corporate presentation, June 2023

KVA12 has been designed on an immunoglobulin G1 (IgG1) monoclonal antibody backbone which binds to a unique epitope on the VISTA-expressing cells. This structure differs from the candidates of other VISTA program sponsors. It can bind to its receptor at both physiologic and acidic pH, which is critical as binding takes place in both the tumor microenvironment and the lymphoid organs. KVA12123 was also designed to avoid CRS. Some of the features that reduce the incidence of CRS when administering KVA12123 include its IgG1 backbone, presence of a mutation that changes the position of the receptor on the cell and reduced binding to Fc γ receptors involved in CRS.

[4] LeMercier, I., *et al*. VISTA regulates the development of protective anti-tumor immunity. Cancer Research, April 2014.

KVA12123: Preclinical Work

In preclinical models, KVA12 demonstrates single-agent tumor growth inhibition in cold tumors and in combination with other checkpoint inhibitors (CIs). It is well-tolerated with no CRS-associated cytokine release (no change in IL6 and TNFα levels) or neurotoxicity. Preclinical work was memorialized in a series of posters presented at scientific conferences beginning in 2020.

Highly Potent Fully Human anti-VISTA Antibodies[5]
The 2020 American Association for Cancer Research (AACR) poster highlighted the background of the VISTA ligand and receptor, showing that it is predominantly expressed in the myeloid lineage within peripheral blood. The goal of the underlying study was to screen for specific, fully human anti-VISTA antibodies and evaluate them. Kinetic and binding studies were performed which found that the anti-VISTA antibodies bind to human VISTA but not to mouse VISTA. The poster continued with a survey of anti-VISTA tumor response both as monotherapy and in combination with other checkpoint inhibitors. In a mouse model, the candidate demonstrated a reduction in tumor growth for mouse anti-VISTA and an even greater reduction in tumor growth when mouse anti-VISTA was combined with anti-PD-L1 and anti-CTLA-4.

Conclusions from the poster found that the anti-VISTA antibodies are highly specific and can produce a strong anti-tumor response either by themselves or in combination with other checkpoint inhibitors in a mouse model. The following exhibit shows the effect on tumor volume of mouse anti-VISTA antibody with and without other checkpoint inhibitors compared to non-treated animals.

Exhibit V – VISTA Antagonist Antibody Induces Anti-Tumor Response

Kineta poster presentation, October 2020, AACR

A Fully Human anti-VISTA Antibody as a Promising Therapy Against Poorly Immunogenic Tumors[6]
This poster was presented in April 2021 at the AACR scientific conference. It sought to screen for effective anti-VISTA antibodies, evaluate and select an antagonist anti-VISTA antibody as a clinical candidate and characterize the lead candidate for investigational new drug (IND) submission.

Kineta's anti-VISTA candidates were shown to bind to cell surface human VISTA but not to other B7-family proteins, providing support for its specificity. The antibodies were further shown to enhance T cell, monocyte and natural killer cell activation. The anti-VISTA antibodies were able to reverse myeloid-derived suppressor cell (MDSC) suppression of T cells in PBMC culture.

Kineta developed 107 fully human anti-VISTA antibodies and analyzed them to identify a lead. The resulting candidate, KVA12, is highly specific, allows the activation of numerous components of the immune system and works well in combination with other checkpoint inhibitors.

[5] Poster presentation, AACR, October 2020. Highly Potent Fully Human anti-VISTA Antibodies: A New Target Checkpoint Inhibitor against Immunosuppressive Myeloid Cells.
[6] Poster presentation, AACR, April 2021. A Fully Human anti-VISTA Antibody as a Promising Therapy Against Poorly Immunogenic Tumors

VISTA expression in patients with advanced solid tumors[7]
At the AACR conference in April 2023, Kineta submitted another poster where the underlying work was conducted to better understand the expression level of VISTA in selected human tumor tissues. The poster also outlined the design of the Phase I/II open-label, combination, dose-escalating basket and solid tumor trial for KVA12.

The authors used immunohistochemistry on tumor-infiltrating immune cells to identify VISTA expression. It was identified on lung, head and neck, ovary, kidney, skin, uterus and liver tumor cells.

This indicates the sites where an anti-VISTA antibody may be able to block immune suppression by VISTA. Additional clinical trial details were provided, including plans to collect tissue and serum samples to identify biomarkers that may be significant for measuring KVA12 efficacy. These samples will also aid in the effort to understand the clinical response to KVA12 and consider VISTA expression as a potential biomarker for efficacy.

Antibody-Drug Conjugates (ADCs)

ADCs are a class of therapeutic agents that consist of a monoclonal antibody and a payload. In most cases, the payload is cytotoxic; however, a wide variety of cargo can be linked to the targeting molecule. This class of drug has commanded attractive valuations given its flexibility, precision, and intellectual property that is easier to protect compared with less complex structures. Deals have valued companies with ADC platforms anywhere from a few million to the mega-deal between Pfizer and Seagen for $43 billion. See Exhibit VII for details of the 20+ transactions that have taken place over the last few years.

TuHURA is evolving its bi-specific ADC program to target the delta opioid receptor (DOR) and carry the anti-VISTA monoclonal antibody to address AML. TuHURA's approach will use its ADC to carry a small molecule drug to target the delta receptor on MDSCs and a checkpoint inhibitor to allow the immune system to recognize the cancer cells.

Blocking the delta receptor will disrupt the production of immune suppressing factors such as Arginase-1 (Arg-1), inducible Nitric Oxide Synthase (iNOS), Cyclooxygenase-2 (COX2), indoleamine 2,3-dioxygenase (IDO), interleukin-10 (IL-10) and Transforming growth factor β (TGF-β). The checkpoint will block other immune suppressive systems used by the cancer cells allowing for a multi-pronged attack on the malignancy.

ADCs have moved to the forefront in oncology because of their precision delivery of the payload while simultaneously reducing off-target effects and reducing side effects as lower levels of toxic drug are needed. Improved conjugation technology, greater availability of payloads and expansion of the number of validated targets have increased the versatility of the class providing a helpful tool for fighting cancer.

Exhibit VI – Features of TuHURA's ADC

Source: TuHURA January 2025 Presentation

[7] AACR Podyrt, April 2023. VISTA expression in patients with advanced solid tumors: A potential biomarker in VISTA-101 clinical trial

Exhibit VII – Executed ADC Deals 2022-2024

IFx 3.0: Diffuse Large B-Cell Lymphoma

IFx-3.0 is the next generation of the IFx platform and seeks to evolve from intratumoral injection in the 2.0 iteration to intravenous delivery. It will use messenger RNA to code for Emm55 targeting CD22, which is overexpressed in cancerous B cells. The codon-optimized mRNA improves the efficiency of translation of the Emm55 bacterial protein, which is expressed on the surface of the tumor cells and primes the immune system to attack and target them. This approach allows TuHURA to expand its tumor approach from solid tumors to blood cancers. The company is designing a monoclonal antibody with high affinity and avidity to the CD22 receptor to combine with the lipid nanoparticle. The lipid nanoparticle contains emm55 mRNA which is delivered to the cancer cell.

Exhibit VIII – IFx-3.0 (mRNA)

Source: TuHURA October 2024 Presentation

Clinical Trials

IFx-2.0 in MCC

TuHURA has filed several IFx-2.0 Phase I trials on clinicaltrials.gov. The most relevant trial has been a Phase Ib that enrolled 23 patients with advanced skin cancer and who have progressed despite other standard therapies. Entitled Immunotherapy with IFx-Hu2.0 Vaccine for Advanced Non-melanoma Skin Cancers, the trial is listed under designator NCT04160065 and enrolled at four US sites.

Eligible patients had at least one injectable lesion and received IFx-2.0 as monotherapy in up to three lesions at up to three time points. The first set of three patients received a fixed 0.1 mg dose of IFx-2.0, while the second set of three received two fixed doses of 0.1 mg. The remaining subjects received 0.1 mg of drug at three time points seven days apart.

The goal of the trial was to assess safety and determine the optimal dose and schedule for maximizing immune response. A total of 23 patients were enrolled, 21 were evaluated for safety and 19 were assessed for response. Treatment related adverse events (TRAE) were favorable with eight (35%) experiencing a Grade 1 TRAE and one (4%) experiencing a Grade 3 TRAE. Two patients showed a complete response, three, a partial response and two, progressive disease. The two complete responses were in MCC. Median duration of response was 21 months.

Another study designed as a Phase Ib targeting MCC patients was entitled pDNA Intralesional Cancer Vaccine for Cutaneous Melanoma. It enrolled seven subjects and is listed under NCT03655756. Each of the enrollees had prior

treatment with checkpoint inhibitors, either single agent or multiple therapies. Primary endpoints for the study related to safety and measured the number of participants with serious adverse events and dose limiting toxicities. Secondary endpoints relate to antitumor response. Results from the study generated two (29%) complete responses and three (43%) partial responses producing an overall response rate of 71%. Median duration of response was 21 months. In some patients in this trial IFx-2.0 was followed by pembrolizumab in a rechallenge.

Exhibit IX – IFx-2.0 Weekly x 3 Followed by Pembrolizumab

Source: TuHURA January 2025 Presentation

One patient in the trial progressed through three months of pembrolizumab and was injected with IFx-2.0. The patient then received a pembrolizumab rechallenge, resulting in biopsy proven complete response. As of the date of the update, response duration was in excess of 23 months.

IFx-2.0 Phase III Pivotal Study in MCC

TuHURA is planning to launch a pivotal Phase III study for its IFx-2.0 candidate in Merkel cell carcinoma (MCC) in the second quarter of 2025. It has been granted permission to pursue 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 and is appropriate when pursuing serious indications that lack other therapies. In most cases, an accelerated approval will require post-market confirmatory trials to verify the clinical benefit. However, in the case of this trial, 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.

The trial will be designed as a 1:1 randomized, placebo and injection-controlled trial in first-line, checkpoint inhibitor naïve, advanced metastatic MCC. It plans to enroll 118 subjects at 20 to 25 clinical centers in the United States. IFx-2.0 will be administered three times per week in combination with pembrolizumab vs. placebo with pembrolizumab. The primary endpoint will be overall response rate (ORR) which will allow for accelerated approval. The secondary endpoint, which indicates clinical benefit is progression free survival (PFS). If this endpoint is achieved, it will allow for full approval and satisfy the requirement for a confirmatory trial.

For the primary endpoints to support an accelerated approval, ORR needs to be 75% compared to the 50% which is the historical rate from other trials. For full approval, based on a continuation of this same trial, PFS has to achieve a 50% or better result.

The trial is expected to start in the second quarter of 2025 with enrollment taking 12 months. ORR will be measured at six months, which indicates that top line results can be available 18 months after the trial begins. Accelerated approval will allow a rolling review and potential approval within six months of topline data.

Exhibit X – IFx-2.0 Phase III Trial Design

Source: TuHURA January 2025 Presentation

KVA12 Phase I/II Clinical Trial in Solid Tumors

The KVA12 program is collaborating with Merck through the latter's contribution of pembrolizumab (Keytruda) to the alliance. It seeks to determine the safety and efficacy of KVA12 both alone and in combination with the PD-1 inhibitor in a Phase I/II clinical trial. A summary of the trial is posted on clinicaltrials.gov under NCT05708950. The primary endpoints are adverse events. Secondary endpoints include pharmacokinetics, immunogenicity, tumor response as per Immunotherapy Response Evaluation Criteria in Solid Tumors (iRECIST) standards and objective response rate (ORR). The study will be conducted in four parts: The first two will focus on dose escalation (single-agent and in combination), and the second two will concentrate on dose expansion (single-agent and in combination). Up to 10 dose escalation cohorts will be evaluated in Phase I and maximum tolerated dose will be identified. Exploratory endpoints will examine receptor occupancy, chemokine and cytokine levels in blood, immune cell populations in blood and VISTA expression in the tumor pre-and post-treatment. Up to 60 adult patients with advanced or metastatic solid tumors that have progressed or have been non-responsive to standard-of-care therapies are eligible to enroll.

Exhibit XI – KVA12 Phase Ib Clinical Trial Design

Source: TuHURA January 2025 Presentation

SITC November 2024 Abstract and Poster
The most recent data for KVA12 was presented at the Society for Immunotherapy of Cancer of Hematology (SITC) on November 8th, 2024 in Houston, Texas. It updated the ongoing Phase I/II VISTA 101 study and announced that the monotherapy arm was fully enrolled. As of the cutoff date in mid-October 2024, KVA12 cleared all six monotherapy dose levels and the second of four cohorts in combination with pembrolizumab. KVA12 was well tolerated with no dose limiting toxicities and a favorable safety profile at all dose levels in both arms of the study. 24 patients were enrolled in the six monotherapy dose cohorts. 19 of these patients had a least one follow-up scan and 13 of the patients experienced stable disease as per iRECIST standards. Dosing began at 3 mg and increased to 1000 mg and was administered every two weeks. Combination therapy included 30 mg to 100 mg of KVA12 administered every two weeks along with 400 mg of pembrolizumab given every six weeks. Complete and partial responses have been observed and the combination has been well tolerated. The two remaining combination cohorts are expected to be fully enrolled by the end of 2024.

TuHURA's Interest and Next Steps
TuHURA provided interim financing to continue Kineta's Phase I/II ascending dose trial in July 2024. At that time, the trial was only halfway through the dose escalation study. The pharmacokinetic and pharmacodynamic data suggested that receptor occupancy was linear, prompting TuHURA to surmise that at 1000 mg, 100% of the VISTA receptors would be occupied for four weeks. If confirmed, this profile would provide potency and long duration.

NPM1-mutated AML is considered a distinct genetic entity in the World Health Organization (WHO) classification of hematopoietic malignancies.[8] NPM1 mutations are acquired driver mutations essential to the pathogenesis of mutated NPM1 AML. In this subtype of AML, menin interacts with wild-type KMT2A which drives the expression of genes that contribute to leukemogenesis. Despite the advances in the treatment of NPM1-mutated AML, about 50% of patients still die of progressive disease.[9]

30% to 35% of all adult Acute Myeloid Leukemia present NPM1 gene mutations. Immune evasion of NPM1-mutated AML is stimulated by VISTA which is upregulated in these patients. This suppresses proliferation of T cells and reduces production of T cell cytokines making it a potential VISTA target. Data presented at December's American Society of Hematology (ASH) meeting evinced a correlation between NPM1 mutations and elevated VISTA expression, particularly in AML, where high VISTA levels on leukemia cells, associated with the mutations, can potentially contribute to immune evasion and disease progression reducing the treatment response. Mutated NPM1 drives leukemogenesis through menin. Administering menin inhibitors may improve treatment responses in mutated NPM1-associated AML. Menin inhibitors produce responses by blocking the oncogenesis and proliferation of leukemia cells. Combining menin with a VISTA-inhibiting antibody could produce improved response rates in AML.

Management believes that it could run a 20-patient trial in AML to demonstrate that the NPM1 mutated cancer is expressing VISTA, administer a menin inhibitor along with a VISTA inhibiting antibody to see if response rates increase materially. If KVA12 can produce this result, it could support an accelerated approval discussion with the FDA. TuHURA estimates that this 20-patient trial could cost as little as $2 million.

Anti-VISTA Phase II Proof of Concept Trial in AML

Following the acquisition of Kineta, TuHURA anticipates launching a Phase II proof of concept trial with the anti-VISTA monoclonal antibody. This is expected to begin in 2H:25. The interest in using KVA12 in AML emerged from research showing that VISTA is overexpressed on myeloid-derived suppressor cells (MDSCs). Another marker that is important in AML is the nucleophosmin 1 (NPM1) mutation which is a genetic alteration that is commonly found in 30 to 35% of AML cases.

Work done by Vanderbilt University and presented at the December 2024 ASH meeting demonstrated that AML with mutated NPM1 expresses high levels of VISTA which creates an immunosuppressive environment. A recently approved class of drug called menin inhibitors, represented by Syndax Pharmaceuticals' revumenib, can treat this group. Menin inhibitors bind to the menin protein and prevent it from binding to the KMT2A protein. This disrupts the interaction between the two, forcing leukemia cells to differentiate into normal cells. These AML cells may also express the VISTA checkpoint receptor that prevents T cells from recognizing and attacking them.

TuHURA plans to evaluate the combination of its anti-VISTA candidate KVA12 along with a menin inhibitor to identify if there is a material benefit. If the combination can block the NPM1 cascade and VISTA receptor, it can eliminate two important therapeutic choke points represented by proliferation and escape mechanism respectively. TuHURA

[8] Ranieri, R. Current status and future perspectives in targeted therapy of NPM1-mutated AML. Leukemia. August 2022.
[9] Ranieri, R. Current status and future perspectives in targeted therapy of NPM1-mutated AML. Leukemia. August 2022.

estimates that for a few million dollars they can design a study that will show if the combination of KVA12 with revumenib provides greater complete remission compared with revumenib alone.

If the trial is successful, TuHURA will consult with the FDA to pursue a similar route to approval as IFx-2.0 with a Phase III trial. The candidate would seek the accelerated approval pathway and employ a trial design that obtains approval using a primary endpoint of a biomarker and a secondary endpoint demonstrating clinical benefit. Further details on the design and pathway forward are expected to be disclosed in upcoming quarters.

Exhibit XII – VISTA Phase II Trials Under Consideration

Source: TuHURA January 2025 Presentation

TuHURA Pipeline

TuHURA's most important assets are its IFx-2.0 innate immune agonist and its soon to be acquired anti-VISTA checkpoint inhibitor designated KVA12. IFx-2.0 is in Phase I trials targeting Merkel Cell Carcinoma and KVA12 will pursue an indication in acute myeloid leukemia (AML) The company is also advancing its next generation IFx candidate as IFx-3.0 and a bi specific antibody-drug conjugate (ADC) which may target the delta opioid receptor and carry the newly acquired anti-VISTA antibody.

Exhibit XIII – TuHURA Pipeline

Source: TuHURA January 2025 Presentation

REM-001 Photodynamic Therapy

TuHURA inherited REM-001 as part of the reverse merger with Kintara Therapeutics. The candidate is a photodynamic therapy (PDT) for the treatment of cutaneous metastatic breast cancer (CMBC). It uses light sensitive compounds, or photosensitizers, that, when exposed to specific wavelengths of light, act as a catalyst to produce a form of reactive oxygen that induces local tumor cell death. The REM-001 therapy consists of three parts: the laser light source, the light delivery device and the drug REM-001. REM-001 is first administered by intravenous infusion and allowed to disperse within the body and be taken up by the tumors. Tumors are then illuminated using the light delivery device, which is attached to the laser light source, so that the accumulated REM-001 can be activated for the desired clinical effect.

Under Kintara, the REM-001 program was suspended pending additional funding. In mid-2023, the program received a grant from the National Institutes of Health which led to the initiation of treatment in four patients in November 2024. Enrollment in the study is expected to be complete before year end 2025. In connection with the merger, TuHURA must provide contingent value rights (CVRs) based on achieving multiple milestones related to the trial. If TuHURA enrolls ten CMBC patients in the REM-001 trial and determines whether a dose of REM-001 lower than 1.2 mg/kg elicits a treatment effect similar to that seen in prior studies of REM-001 at the 1.2 mg/kg dose and the patients enrolled in the study complete eight weeks of follow-up on or before December 31st, 2025, Kintara shareholders are entitled to receive approximately 1,539,918 shares of common stock. Further details of the arrangement are provided in corporate filings and in the CVR Agreement. TuHURA does not expect to advance REM-001 further and anticipates divesting the asset after completing the ten-patient trial.

Intellectual Property

TuHURA holds exclusive license agreements with H. Lee Moffitt Cancer Center and Research Institute and the West Virginia University (WVU) Research Corporation for the development, commercialization and marketing of DOR and ADC. These agreements related to the patents entitled “Conjugates for Immunotherapy” and “A Delta-Opioid Receptor Targeted Agent for Molecular Imaging and Immunotherapy of Lung Cancer.” The WVU Agreement provides commercialization rights relating to Delta receptor targeted agents for WVURC patent rights pertaining to molecular imaging and cancer immunotherapies. Under the WVU Agreement, among other rights, WVU granted TuHURA a worldwide, exclusive right, with limited sublicense rights, to develop and commercialize the WVU patents. Patent titles related to Moffett and WVU include “DNA Vector and Transformed Tumor Cell Vaccines,” “Cancer Vaccine Comprising mRNA Encoding a M-Like Protein” and “Modified mRNA for Multicell Transformation.”

FDA Project Frontrunner

Project Frontrunner is an initiative offered by the FDA’s OCE. It encourages drug sponsors to find safe and effective medicines to treat advanced or metastatic disease in an earlier clinical setting than what otherwise might occur using traditional drug approval pathways. Well worn approaches to cancer drug approval investigates using novel agents in patients that have received numerous prior lines of therapy or have exhausted existing treatment options. This limits initial use of the potentially valuable agent in earlier lines of therapy and when the drug has the potential to alter the course of the disease more effectively. Other benefits of participating in the initiative allow for a greater opportunity to characterize the drug’s safety profile while avoiding the confounding effects of disease-related complications. It also may help a safe and effective agent demonstrate its benefits relative to standard of care and may help improve treatments in the frontline setting. This is especially relevant for indications where front line treatments are non-curative or toxic.

The program’s goals are to develop a framework that identifies candidate drugs in the treatment of early meta-static disase, interact with sponsors to identify candidates and engage and collaborate with stakeholders to advance the medicines. The program asserts that the project will be most effective in the advanced and metastatic setting where candidates are not expected to be curative which is where the new therapies may have the best chance of improving outcomes.

Special Protocol Assessment

The FDA has developed a Special Protocol Assessment (SPA) with TuHURA which is a formal agreement that confirms the design and size of the clinical trial that will be required for the drug application for IFx-2.0 in MCC. The agreement is binding upon the FDA and provides certainty the agency will accept the primary endpoints, study population, statistical analyses and study size of the clinical trial. An SPA reduces regulatory and development risk but does not guarantee ultimate approval as safety and efficacy hurdles must still be met.

Primary Indications

Merkel Cell Carcinoma (MCC)

Merkel cell carcinoma (MCC) is a rare but aggressive form of skin cancer that originates in Merkel cells, which are specialized neuroendocrine cells located in the bottom layer of the skin's epidermis. The disease is also called neuroendocrine carcinoma of the skin. Despite its rarity, MCC is notable for its high potential for metastasis and significant mortality rate. It is a neuroendocrine skin cancer with high metastatic potential, with one-third to one-half of patients developing recurrence or metastasis.[10] MCC was first identified in 1972 by Cyril Toker, who was a pathologist at the Mount Sinai School of Medicine.[11] It is a very rare type of skin cancer with about 3,300 cases reported in the U.S. each year. The rate of diagnosis has increased in recent years, a change which is attributed to improved diagnostic tests.

Exhibit XIV – Merkel Cell Carcinoma on the Forearm

Source: merkelcel.org clinical photos of MCC

The cancer most commonly occurs on the head and neck and other sun-exposed skin surfaces; however, it can appear on any body part. The lesion appears as a firm, painless, rapidly enlarging, red–violet cutaneous tumor nodule that is typically dome-shaped. There are three histological subtypes including trabecular, intermediate and small cell. Treatment for MCC depends upon its stage and includes surgery, radiation therapy, chemotherapy and immunotherapy. The latter approach is frequently effective as the Merkel cell polyomavirus is recognized as foreign to the immune system. Checkpoint inhibitors are commonly used and work in more than half of cases and produce a durable response.

Immunotherapy has added a new tool to the arsenal that has improved the prognosis for MCC patients; however, the five-year survival rate hovers around 64%.[12] Resistance or contraindication for checkpoint inhibitors is common and active research priorities are seeking therapies that are able to improve the responsiveness to immunotherapy by further activating the immune system. In the minority of MCC cases where the checkpoints do not work, it may be due to an immunosuppressed tumor, the absence of sufficient neoantigens that make the tumor appear foreign or acquired resistance. All of these represent unmet needs.

[10] Paulson, K.G. *et al*. Merkel Cell Carcinoma: Current United States Incidence and Projected Increases based on Changing Demographics. Journal of the American Academy of Dermatology. March 2019.
[11] Erovic, I., Erovic, B.M. Merkel Cell Carcinoma: The Past, The Present and the Future. Journal of Skin Cancer. April 2013.
[12] Zaggana, E., *et al*. Merkel Cell Carcinoma—Update on Diagnosis, Management and Future Perspectives. Cancers. November 2022.

Merkel Cells
Merkel cells are found in the epidermis which is the outermost layer of the two main layers that make up the skin. The cells normally appear in the basal layer of the skin. The German anatomist Friedrich Merkel first described these eponymous cells in 1875.[13] He found that they occur at high density in the paws of rats and hypothesized that they may serve as touch cells. Merkel cells appear in skin all over the body but are at their highest concentration where touch is most sensitive such as on the fingertips, lips and face where touch sensation is most acute. In addition, Merkel cells produce certain hormones and are sometimes referred to as neuroendocrine cells. Neuroendocrine refers to the type of cells that make up Merkel cell carcinoma. Merkel cells are hormonal and provide sensory function related to the nervous system.

Merkel Cell Polyomavirus (MCPyV)
A primary oncogenic contributor to MCC is the polyomavirus (MCPyV) present in over 80% of MCC cases. The virus is integrated into the genome of cancer cells in these cases and is thought to play a critical role in driving the cancer's development. The cases that are not associated with MCPyV are frequently related to exposure to ultraviolet radiation. MCPyV was first discovered in 2008 and identified as a non-enveloped DNA virus. It is highly prevalent in the population with most infected in childhood and thought to be transmitted through direct skin contact. In cases where MCPyV is associated with MCC, the virus' integration is clonal and occurs early in tumor development and is present in all cancer cells. It must integrate into the host genome and is more prevalent in its association with MCC in immunosuppressed individuals.

Incidence of MCC
The global incidence of MCC ranges from 0.1 to 1.6 cases[14] per 100,000 persons per year, reaching its highest levels in regions where fair-skinned people are exposed to intense sunlight.[15] Lower levels of incidence are associated with those under 50 years of age while a higher rate is associated with those over 60. Only about 2% of cases are reported in those under age 50[16] and the cancer is very rare in children. The mean age of MCC diagnosis in the United States is 75 years.[17] The American Cancer Society estimates that about 3,000 individuals are diagnosed with MCC every year in the United States as of 2024. Another resource (Paulson 2019)[18] identified a rapid increase in prevalence with 1,500 cases per year in 2007 rising to 2,488 cases in 2014. The paper continued with estimates of 2,835 cases in 2020 and 3,284 by 2025. Delve Insight estimated an incidence of 9,000 cases of MCC in the US, top four EU countries and Japan in 2023. Incidence is expected to grow at a rate of over 4% per annum[19]

Incidence varies by region with the highest rates reported in Australia. In Europe, MCC accounts for less than 1% of all cutaneous malignancies and a number of country-specific reports show lower rates than in the United States. Many studies in the US and Europe have shown an increase in the incidence of the disease over time.[20] MCC is thought to be uncommon in Asia given the limited number of cases reported.

Men are slightly more than twice as likely to suffer from MCC than women. MCC is also more common in white populations; dark skinned individuals present the disease at about 1/8th the rate of their white counterparts.

Risk Factors
There are several risk factors for MCC that range from viral exposure and infection to sun exposure and the condition of a person's immune system. Most patients diagnosed with MCC are infected with MCPyV. High antibody titers of MCPyV were a significant predictor for progression-free survival and clinical factors such as tumor thickness, size, sex, and age are shown not to be a reliable prognostic factor for overall and disease-free survival.[21] Cancer cells in 60% to 80% of those diagnosed with MCC are MCPyV positive. Other risk factors include individuals with weakened immune systems or those who have undergone a transplant and are on immunosuppressants. This includes persons with human immunodeficiency virus (HIV). Ultraviolet radiation, sun exposure and ultraviolet light

[13] Halata, Z., *et al*. Friedrich Sigmund Merkel and his "Merkel cell", morphology, development, and physiology: review and new results. The Anatomical Record, March 2003.
[14] Fojnica, A. *et al*. An Updated Review of the Biomarkers of Response to Immune Checkpoint Inhibitors in Merkel Cell Carcinoma: Merkel Cell Carcinoma and Immunotherapy. Cancers. October 2023.
[15] Walsh, N.M., Cerroni, L. Merkel cell carcinoma: A review. Journal of Cutaneous Pathology. October 2020.
[16] Freeman, M.B. *et al*. Merkel cell carcinoma incidence, trends, and survival rates among adults aged ≥50 years from United States Cancer Statistics. Journal of the American Academy of Dermatology. April 2019.
[17] Schadendorf, D. *et al*. Merkel Cell Carcinoma: Epidemiology, prognosis, therapy and unmet medical needs. European Journal of Cancer. January 2017.
[18] Paulson, K.G. *et al*. Merkel Cell Carcinoma: Current United States Incidence and Projected Increases based on Changing Demographics. Journal of the American Academy of Dermatology. March 2019.
[19] Delve Insight. Merkel Cell Carcinoma - Market Insight, Epidemiology And Market Forecast – 2034.
[20] Schadendorf, D. *et al*. Merkel Cell Carcinoma: Epidemiology, prognosis, therapy and unmet medical needs. European Journal of Cancer. January 2017.
[21] Erovic, I., Erovic, B.M. Merkel Cell Carcinoma: The Past, The Present and the Future. Journal of Skin Cancer. April 2013.

treatment for skin conditions such as psoriasis are other risk factors as is age. The older a person is, the more likely they are to develop MCC.

Symptoms
A new, firm, painless and dome shaped nodule on the skin that ranges in color from red, purple, pink or blue is a sign of an MCC lesion. The bump can be shiny or smooth, can grow quickly over a short period and frequently measures from one to five centimeters in diameter. Lesions most commonly appear on the head, neck, face and arms and it develops rapidly over weeks or months. The malignancy can spread to lymph nodes and other body parts if not addressed quickly. The lymph nodes near the cancer site may become swollen. As the condition advances, the lesion may grow, become painful or tender and ulcerate or bleed.

Diagnosis
Diagnosis of MCC is determined through clinical examination followed by tissue biopsy which presents characteristic histopathological neuroendocrine features. Immunohistochemistry can confirm a diagnosis with the cytokeratin-20 (CK20) staining differentiating it from other morphologically similar tumors. CK20 and thyroid transcription factor 1 (TTF-1) provide the greatest sensitivity and specificity to exclude small cell lung cancer (SCLC). CK20 is positive in 75% to 100% of primary MCC tumors and rarely positive in SCLC. TTF-1 is never positive in MCC but is often positive in SCLC (>80%) and other primary pulmonary tumors.[22] Sentinel lymph node biopsy and imaging is essential for staging and determining prognosis.[23]

MCC tumors visualized by hematoxylin and eosin (H&E) staining typically contain small round cells with sparse cytoplasm, abundant mitoses, and dense core granules in the cytoplasm. MCC is similar to a variety of other widely recognized small round cell tumors, including metastatic visceral neuroendocrine carcinomas.[24]

Current Standard of Care
According to the 2023 National Comprehensive Cancer Network (NCCN) guidelines, surgery and radiation are initially recommended, with little break between the former and the latter as worse outcomes have been identified with a delay. Histologically negative margins for surgery and wide margins for radiation therapy are recommended when feasible. For primary disease, chemotherapy is not recommended. Mohs surgery is frequently appropriate, which is a precise surgical technique used primarily for skin cancer removal while preserving maximum healthy tissue.

In recurrent locally advanced disease, pembrolizumab and retifanlimab, both checkpoint inhibitors, are used. In disseminated disease, other checkpoint inhibitors may be appropriate including avelumab and nivolumab. The expression of viral oncoproteins in MCPyV-positive cancer cells, the high mutational burden in MCPyV-negative tumors and in the tumor micro-environment and the clinical association with immunosuppression make MCC a good candidate for immunotherapy.[25] In cases where checkpoint inhibitors are contraindicated or the patient has progressed, the guidelines recommend several chemotherapy regimens, oncolytic viral therapies and tyrosine kinase inhibitor approaches as appropriate.

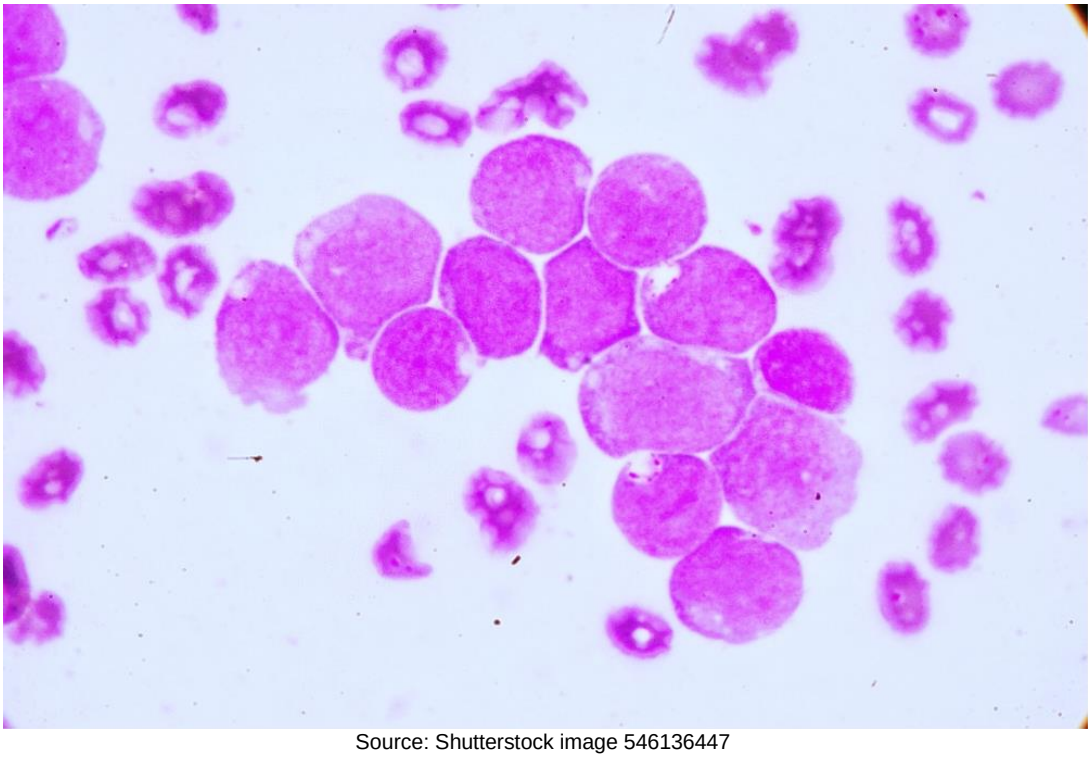
Prognosis
The prognosis for MCC depends on the stage of the disease, size of tumor, location and treatment modality in addition to the presence of the risk factors mentioned above. Metastatic disease has a poor prognosis. Outcomes are best when there is no lymph node involvement at the time of diagnosis. MCC can spread quickly, so identifying unusual symptoms is critical to an effective diagnosis and successful treatment. Age, overall health and responsiveness to treatment are other factors impacting long-term outcomes. According to the American Cancer Society, the five-year survival rate for MCC patients is 75% for localized disease, 61% for regional disease and 24% for distant or metastasized MCC. The disease can spread quickly to another body region and has high recurrence rates. A review[26] by the National Cancer Institute (NCI) found that 30 percent of patients whose cancers were caught in early or mid-stage had a recurrence on the skin. Other resources[27] found that recurrence was approximately 40% and higher than in other skin cancers such as melanoma.

[22] NCCN 2023 Guidelines, Version 1.2023.
[23] Patel, P., Hussain, K. Merkel Cell Carcinoma. Clinical and Experimental Dermatology. July 2021.
[24] NCCN 2023 Guidelines, Version 1.2023.
[25] Zaggana, E., *et al*. Merkel Cell Carcinoma—Update on Diagnosis, Management and Future Perspectives. Cancers. November 2022.
[26] NIH, National Cancer Institute. Merkel Cell Carcinoma Treatment (PDQ)–Health Professional Version.
[27] McEvoy, A.M. *et al*. Recurrence and Mortality Risk of Merkel Cell Carcinoma by Cancer Stage and Time From Diagnosis. JAMA Dermatology. April 2022.

Acute Myeloid Leukemia (AML)

Acute Myeloid Leukemia (AML) is a cancer of myeloid blood cells where the abnormal cells grow uncontrollably in the bone marrow and disperse in the blood to interfere with normal blood cell production. Hematopoietic stem or progenitor cells acquire specific mutations during early stages, providing them with a clonal advantage for expansion. The displacement of normal blood cell production by these mutated cells reduces white blood cell count making patients more susceptible to infections. Signs of AML include fatigue, weakness, shortness of breath, frequent infections, easy bruising or bleeding and weight loss. It progresses quickly and requires prompt treatment to improve the likelihood of survival. Risk factors for AML include previous exposure to chemotherapy or radiation therapy, smoking, obesity and genetic mutations. It is more likely in older adults and survival is tied to treatment which includes chemotherapy, stem cell transplantation and targeted therapies.[28,29] A common subcategory of AML is characterized by the mutated nucleophosmin (NPM1) gene, which is present in about a third of AML cases. Proteins produced by the mutated gene can move from the nucleus to the cytoplasm leading to disrupted cellular processes which contributes to leukemogenesis. In November 2024, the menin inhibitor revumenib was approved for this type of leukemia.

Exhibit XV – Blood Smear Showing AML



Source: Shutterstock image 546136447

Incidence & Prevalence

The American Cancer Society estimates that there will be about 62,800 new cases of leukemia and 20,800 of AML in the United States in 2024. According to the Surveillance, Epidemiology, and End Results (SEER) database, the rate of new AML cancer deaths per 100,000 was 4.1 in the United States in 2020. Deaths are expected to be about 11,200, heavily tilted towards the older age range of those afflicted.[30] The National Cancer Institute (NCI) estimates a 5-year survival rate of 31.7% based on 2013 to 2019 data. Males are more likely to suffer from AML in an approximate 1.3 to 1.0 ratio and African Americans present higher rates of mortality than Caucasians do.

Around the globe, a total of 474,519 cases of leukemia were reported in 2020 along with a rate of incidence of 5.4 per 100,000.[31] About a third of these are AML. Across the world, the annual number of newly diagnosed patients of all leukemias has increased by 46% in the past 3 decades while incidence for AML has increased by 15%.[32] Jani *et al*. found a global AML incidence rate of 1.85 and 1.37 per 100,000 for men and women respectively (2019). A liter-

[28] Acute Myeloid Leukemia Treatment (PDQ). National Cancer Institute. Accessed March 2024.
[29] Kristo, F., *et al*. Global Incidence and Survival of Acute Myeloid Leukemia with 20–29% Blasts: A Systematic Literature Review. Virtual ISPOR Europe. November 2021.
[30] American Cancer Society. Key Statistics for Acute Myeloid Leukemia (AML). Accessed March 2024.
[31] Huang, J., *et al*. Disease Burden, Risk Factors, and Trends of Leukaemia: A Global Analysis. Frontiers in Oncology, July 2022.
[32] Jani, C.T., *et al*. Burden of AML, 1990-2019: Estimates From the Global Burden of Disease Study. JCO Global Oncology. November 2023.

ature review of studies examining AML around the globe found an average incidence of 1.54 per 100,000. 44 studies from 39 countries provided incidence rates ranging from 2.0 per 100,000 in Sweden to 5.4 per 100,000 in Denmark. The summary of several studies conducted in the United States produced rates from 3.8 to 4.6 per 100,000. Based on its prevalence AML is considered a rare disease, falling well below the 200,000-incidence level threshold in the United States.[33]

NPM1 mutations are found in about 30% of AML cases and about half of these patients will relapse. It is the most common subcategory of AML. Those with a normal karyotype, or chromosome makeup, are about 47% of AML cases while those with an abnormal karyotype make up less than 10%.[34] Another study that reviewed studies in Asia and Europe identified NPM1 mutations in a variety of countries. In Asia, the prevalence of the mutation was in the 20% range for Thailand, China and Japan. Countries in Europe had higher prevalence of the NPM1 mutation in AML. Two studies showed ~50% prevalence in Germany while Italy and the Netherlands produced studies indicating 35% prevalence.[35]

Symptoms
The symptoms of AML that arise from the growth of abnormal blasts are many. Bleeding, bruising, fever, infections, lethargy and shortness of breath are a few. Others include chest pain, dizziness, headaches, blurred vision and joint pain. The anemia from low red blood cell counts contribute to the fatigue and weakness and the lack of normal white blood cells produces the infections. Low platelet counts cause the bruising and bleeding.

Diagnosis
After a review of a patient's medical history and exam, an AML diagnosis requires confirmation from several tests. The physical exam will pay particular attention to the eyes, mouth, skin, lymph nodes, liver, spleen, and nervous system, and will look for areas of bleeding or bruising, or possible signs of infection. A blood test measures blood cell counts and bone marrow aspiration is frequently performed with collecting tissue for a biopsy. In rare cases, cerebrospinal fluid (CSF) may be withdrawn in a lumbar puncture or spinal tap to determine if the leukemia cells have spread into the brain and spinal cord. Imaging is sometimes used to identify infections related to AML and to determine if the cancer has been spread outside of the bone marrow. When the tissue samples are evaluated in the lab, in addition to confirming the subtype of leukemia, the testing also identifies which gene mutations are present.

Standard of Care
Treatment is highly dependent on the type of AML as well as the age and health of the patient. The primary treatment for most types of AML is chemotherapy, sometimes in combination with a targeted therapy drug. This might be followed by a stem cell transplant, specifically called an allogeneic hematopoietic stem cell transplantation (allo-HSCT). For newly diagnosed AML patients who are fit enough for intensive chemotherapy, the standard induction regimen includes venetoclax in combination with cytarabine and an anthracycline in a combination known as the "venetoclax + 7+3" regimen.

In some AML patients, the leukemia cells have a mutation in the FLT3 gene. This gene helps the cells make a protein (also called FLT3) that helps the leukemic cells to grow. Drugs that target the FLT3 protein can help treat some of these malignancies. Midostaurin (Rydapt) and quizartinib (Vanflyta) are FLT3 inhibitors that can be used along with chemotherapy to treat newly diagnosed adults whose leukemia cells have a mutation in the FLT3 gene. Gilteritinib (Xospata) can treat adults whose leukemia cells have a mutation in the FLT3 gene and whose AML has not improved or recurred.

Some leukemia cells have a mutation in the IDH1 or IDH2 gene which help cells make specific proteins called IDH1 and IDH2. Mutations in one of these genes can stop blood cells from maturing normally. Targeted drugs called IDH inhibitors can block these proteins. Agents with this mechanism of action include ivosidenib (Tibsovo), olutasidenib (Rezlidhia) and enasidenib (Idhifa) which help the leukemia cells differentiate into more normal cells. Gemtuzumab ozogamicin (Mylotarg) is a combination monoclonal antibody and chemotherapy drug (antibody-drug conjugate) that attaches to the CD33 protein on AML cells and delivers the anti-neoplastic agent to them.

In younger, otherwise healthy AML patients, a stem cell transplant may be given for remission consolidation and to prevent relapse. In patients that do not respond to first line treatment and for relapsed and refractory AML, the allo-HSCT may also be appropriate. Adults over age 65 are at a higher risk from a stem cell transplant and are less of-

[33] Yi, M, *et al*. The global burden and attributable risk factor analysis of acute myeloid leukemia in 195 countries and territories from 1990 to 2017: estimates based on the global burden of disease study 2017. Journal of Hematology & Oncology. June 2020.
[34] Thiede, C. *et al*. Prevalence and prognostic impact of NPM1 mutations in 1485 adult patients with acute myeloid leukemia (AML). Blood. 2006.
[35] Thongnoppakhun, W. Nucleophosmin mutation in Southeast Asian acute myeloid leukemia: Eight novel variants, FLT3 coexistence and prognostic impact of NPM1/FLT3 mutations. Haematologica. October 2008.

ten candidates for this procedure. Higher doses of chemotherapy may be given in this circumstance. The chemotherapy kills most of the bone marrow which needs to be replaced with an HSCT. Both allogeneic and autologous transplants are given. In the allogeneic setting, which is more common, there are certain benefits including the graft vs. leukemia effect where donor cells recognize and kill remaining leukemia cells. However, this benefit may also be associated with complications related to graft vs. host disease (GvHD) where the donor immune system sees the patient's body tissues as foreign and attacks them. GvHD can be mild to severe. Autologous transplants may risk returning some of the leukemic cells to the patient.

NPM1 mutated disease receives similar treatment as AML without the mutation. Other mutations besides NPM1 drive the therapeutic approach, particularly the FLT3 mutant. In 2024, the menin inhibitor revumenib (Revuforj) was approved for translocated KMT2A. It was sponsored by Syndax Pharmaceuticals. This class of drug works by disrupting the interaction between the menin and KMT2A proteins, which forces leukemia cells to differentiate into normal cells. Another menin inhibitor in development, Kura Oncology's ziftomenib, is being investigated in NPM1-mutant AML and has shown high response rates in clinical trials.

Peers and Competitors

TuHURA participates in the immune-oncology (IO) and innate immune agonist spaces. Its IFx platform has distinguished itself as a participant in innate immune system stimulation alongside peers such as Replimune, Memgen and Innate Pharma. Several major pharmaceutical companies are developing innate immune agonists, mainly focusing on pattern recognition receptors (PRRs) like Toll-like receptors (TLRs), STING, and RIG-I. Novartis has been working on TLR7 agonists for cancer treatment. Bristol Myers Squibb, through their acquisition of IFM Therapeutics, has been developing STING agonists for immunotherapy.

TuHURA's closest peers use oncolytic viruses or mRNAs that generate tumor-associated antigens. Replimune uses an oncolytic platform that destroys tumors, which in turn releases tumor derived antigens that activate the adaptive immune system. The company is targeting indications in melanoma and other genetically defined cancers where patients have failed on checkpoint inhibitors. Memgen employs an oncolytic virus combining its CD40 ligand with interferon β and two transgenes to generate a tumor-targeted T cell immune response. BioNTech and Moderna are using a similar approach that uses a tumor biopsy to sequence the embedded oncogenes and identify the most important cancer related antigens using an internally developed algorithm. The algorithm seeks to determine which antigens are immunogenic, then develops mRNAs for each one, which are then combined into a master mRNA which is then injected into the patient as a vaccine.

Smaller biotechnology companies are also making significant contributions. Codiak BioSciences (later acquired by Lonza) developed engineered exosomes carrying STING agonists. Spring Bank Pharmaceuticals (now part of F-star) worked on STING agonists, although their lead compound SB 11285 faced some challenges in clinical development. Merck has shown interest in this space through their collaboration with Aduro Biotech (now part of Chinook Therapeutics) on STING agonists. GSK has also been active in this area, particularly in developing TLR7/8 agonists for various applications.

Antibody-drug conjugates (ADCs) have been an area of focus in an approach that links an antibody with a toxic payload to target the therapy towards cancer cells. The ADC structure has many forms which can combine targeting antibodies with other molecules to achieve varied ends. In one approach, a blocking antagonist may be used with a checkpoint inhibitor to prevent metastasis and reverse immunosuppression.

While VISTA has not achieved the name recognition of the best-known checkpoint protein receptors such as PD-1, PD-L1 and CTLA-4, there has been a substantial amount of work conducted to understand its potential. We have identified at least ten different programs advancing anti-VISTA programs ranging from preclinical to clinical stage from various sponsors in the small, mid and large market capitalization ranges. Several companies are evaluating their anti-VISTA antibody-drugs in Phase I clinical trials including Hummingbird Biosciences, Pierre Fabre and Sensei.

Exhibit XVI – Peers and Competitors[36]

Ticker	Company	Price	MktCap (MM)	EV (MM)	Therapeutic Area
1801.HK	Innovent Biologics	HK$33.15	HK$53,982	HK$47,810	PD-1 Tyvyt w / 4 indications approved in China, LAG-3, TIGIT, CTLA
600276.S	Hengrui Pharma	¥44.65	¥284,822	¥262,000	PD-1 (camrelizumab), PD-L1 (adebrelimab)
ABBV	AbbVie	$190.14	$336,004	$387,228	Oncology: hematologic malignancies, broad portfolio
AGEN	Agenus	$3.51	$82	$38	Checkpoint portfolio: PD-1, CTLA-4, TIM-3, LAG-3, TIGIT
ALLO	Allogene Tx	$1.67	$350	$59	Oncology: CAR-T, liquid & solid tumors
ALXO	ALX Oncology	$1.32	$70	($69)	Checkpoint: CD47 - evorpacept
AMGN	Amgen	$288.87	$155,277	$203,120	VISTA asset through acq of "5' Tx"; no evidence pursuing
ATOS	Atossa Tx	$0.85	$107	$32	Hormone receptor breast cancer therapy
AZN	AstraZeneca	$69.86	$216,642	$240,583	Imfinzi, PD-L2, Imjudo CTLA-4
BCAB	BioAtla	$0.45	$22	($35)	Conditionally active biologics: ADCs
BCYC	Bicycle Tx	$12.66	$874	($17)	Bicyclic toxin conjugate
ONC	BeiGene	$223.37	$21,827	$19,313	PD-1 tilelizumab (China) hepatocellular carcinoma
BMY	Bristol-Myers Squibb	$59.95	$121,589	$162,169	Opdivo, Yervoy. Relatlimab, lirilumab in dev.
BNTX	BioNTech	$119.82	$28,726	$10,579	BNT411:TLR7 agonist-activate adaptive & innate immune system
CGEM	Cullinan Oncology	$10.06	$586	$8	CLN081 small molecule in NSCLC w / exon 20 insertion mutations
CRIS	Curis	$3.49	$30	$9	CI-8993 for VISTA expressing cancers & other checkpoints
FATE	Fate Therapeutics	$1.27	$145	($152)	Multiple targets (CD19/BCMA/HER2) in 5 oncology indications
GILD	Gilead Sciences	$98.38	$122,608	$140,404	Checkpoints: MB272, partnership w / Arcus anti-TIGIT & PD-L1
GSK	GlaxoSmithKline	$34.90	$72,333	$85,067	PD-1 Jemperli dMMR endometrial cancer
INCY	Incyte Corp	$73.74	$14,206	$12,460	PD-1 retifanlimab & other PD-1 & PD-L1, TIM-3, LAG-3
IPHA	Innate Pharma	$1.72	$144	$95	Checkpoint: NKG2A target w ith monalizumab
KRON	Kronos Bio	$0.98	$59	($66)	I-O for solid & liquid tumors
LLY	Eli Lilly	$810.43	$769,354	$794,881	PD-1 Tyvyt
MGNX	MacroGenics	$2.85	$179	($21)	PD-1, CTLA-4, LAG-3, HER2, ADC, bispecifics,
MRK	Merck & Co.	$99.79	$252,432	$272,821	Keytruda. TIGIT (MK-7684) & LAG-3 (MK-4280) in development
MRKR	Marker Tx	$1.79	$19	$10	I-O: multi-tumor antigen approach
MRNA	Moderna	$36.55	$14,065	$7,823	Individualized neoantigen therapies for cancer
NKTR	Nektar Tx	$0.81	$149	($95)	IL-15 receptor agonists in dev. for liquid and solid tumors
NVS	Novartis	$105.48	$215,601	$222,947	Broad oncology portfolio
ONCY	Oncolytics Biotech	$0.69	$56	$41	Oncolytic virus for BC in combo w ith IO & targeted therapies
PFE	Pfizer	$26.20	$148,475	$196,525	PD-L1 Bavencio, PD-1 sasanlimab
REGN	Regeneron	$666.85	$73,280	$66,187	PD-1 Libtayo, LAG-3 fianlimab, bispecifics
REPL	Replimune Group	$13.71	$938	$575	Oncolytic IO, RPx platform: RP1 & RP2/melanoma, CSCC
RHHBY	Roche	$39.32	$250,780	$280,810	Broad oncology portfolio: PD-L1 Tecentriq
SNDX	Syndax	$14.34	$1,224	$818	Menin inhibitor Revuforg for NPM1m AML
SNSE	Sensei Bio	$0.48	$12	($35)	SNS-101: VISTA for solid tumors. Also VSIG4 & CD39 targets
TCRT	Alaunos Tx	$1.52	$2	$1	TCR-T cell therapy (KRAS, TP53, EGFR)
pvt	Hummingbird Bio				Precision oncology, HMBD002-VISTA w / Keytruda Ph1
pvt	ImmuNext				VISTA collaboration w ith Curis
pvt	Pierre Fabre Labs				anti-VISTA W0180, K01401
pvt	Memgen				Viral IO w ith oncolytic adenovirus encoding IFNß.
pvt	Inimmune				Innate immune modulators: allergy, cancer & vaccine
pvt	IFM Tx				Small molecules targeting innate immune system
HURA	TuHURA Bio	$4.85	$205.08	$202.34	Innate immune agonists, ADCs & checkpoint inhibitors

[36] Compiled by Zacks as of February 3, 2025

Financial & Operational Results

Milestones

- TuHURA Biosciences formed – 1995
- TuHURA becomes public via reverse merger – October 2024
- Entry into definitive merger agreement with Kineta – December 2024
- Launch IFx-2.0 Phase III trial – 1Q:25
- IFx-2.0 IR basket trial – 2Q:25
- Bi-specific MDSC targeted ADC & PDC proof of concept – 3Q:25
- IFx-3.0 CD22 tumor-targeted mRNA *in vivo* proof of concept studies – 3Q:25
- VISTA inhibitor Phase II trial – 2H:25

TuHURA Origin

TuHURA was founded in 1995 by Drs. Patricia and Michael Lawman. Its IFx technology was developed at Dr. Lawman's laboratory located in the Walt Disney Memorial Cancer Institute. Michael and Patricia held director positions at the Institute and academic professorship positions in oncology at other Florida institutions.

TuHURA's ADC technology and Delta Receptor peptide antibody were developed by Dr. Mark McLaughlin at the Moffitt Cancer Center and at the West Virginia University Research Corporation.

2024 First Nine Months Financial Results

TuHURA was responsible for filing Form 10-Q for the quarter ending September 30, 2024. However, this provided financial information for Kintara as the reverse merger took place after the end of the reporting period. TuHURA's financial information was reported in other filings, including the S-4 submitted to the SEC in 2024. Based on the information found in these sources, for the nine-month period ending September 30th, 2024, TuHURA reported zero revenues. Research and development expense was $9.4 million rising 25% primarily due to increased spending on in-development assets including IFx-2.0, IFx-3.0 and the ADC program. General and administrative expenses were $2.6 million, down 23% from $3.4 million. Operating loss was ($12.0) million. Net interest expense was ($3.4) million due to interest payments for the convertible notes and expenses related to their extinguishment in conjunction with the reverse merger. Net loss was ($15.7) million for the nine-month period.

Cash as of September 30th, 2024 was $19.6 million, up from $3.7 million as of the end of 2023. Proceeds from convertible notes, equity sales and exercise of derivatives of $33.3 million were partially offset by cash burn of ($12.2) million and payment of an exclusivity rights deposit of ($5.2) million. Convertible notes were held on the balance sheet at $24.4 million. Following the reverse merger on October 18th, 2024, the convertible notes were paid down in full.

Merger Transaction with Kintara Therapeutics

Following an extensive period of review and due diligence, which began in January 2024, management for TuHURA and Kintara met to discuss a plan for a merger between the two. The results of the discussions resulted in a merger announcement in April 2024. The agreement called for an all-stock transaction allowing TuHURA to effect a reverse merger. The arrangement is subject to a shareholder vote which would have TuHURA shareholders own 97.15% of the shares outstanding as of the merger close. Contingent value rights (CVRs) were included for Kintara shareholders which provide an additional share interest if TuHURA enrolls at least 10 cutaneous metastatic breast cancer patients in a forthcoming study to determine whether a dose of REM001 <1.2 mg/kg elicits a treatment effect similar to that seen in prior studies.

On October 4th, shareholder votes allowed for the merger to proceed and two weeks later on October 18th, 2024 a reverse stock split of 1:35 was effected, the merger was completed and the corporate name was changed to TuHURA Biosciences. As opposed to conducting an initial public offering (IPO), joining with a special purpose acquisition company (SPAC) or other pathway to become public, TuHURA felt that the reverse merger with Kintara would be more cost-effective, increase the visibility of TuHURA, provide an easier path to raise capital and provide a faster route to the NASDAQ.

Acquisition of Kineta and KVA12

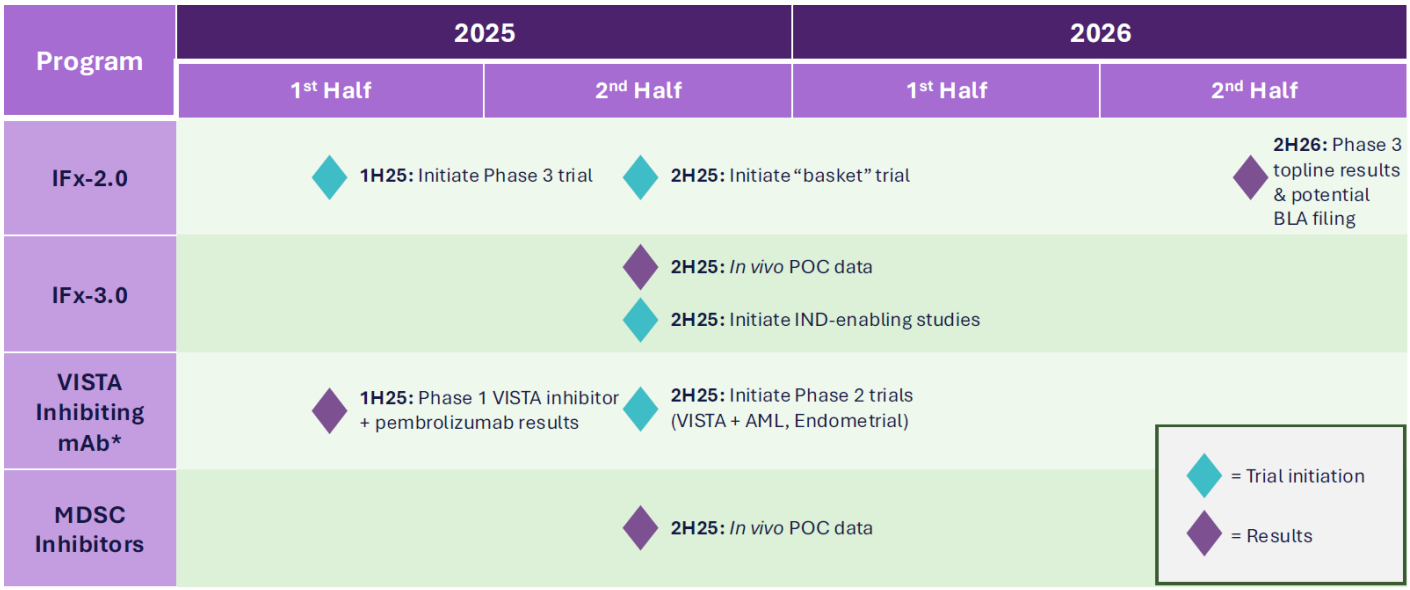
On July 8th 2024 TuHURA entered into an exclusivity and right of first offer agreement with Kineta, Inc. and its lead candidate KVA12. The offer provided a $5 million investment from TuHURA that provides rights to later acquire the company and its anti-VISTA asset. In October the arrangement was extended with additional funds paid to Kineta for the extension.

In December, TuHURA announced that it would formally acquire Kineta including the rights to KVA12 for cash and shares valued at $29.3 million. This comprised $9.0 million in cash and 3,476,568 shares of TuHURA stock, which closed at $5.84 per share the evening before the announcement was made. In connection with the merger, TuHURA and Kineta entered into a Clinical Trial Funding Agreement where TuHURA will fund clinical trial expenses for KVA12 of up to $900,000. Kineta may request the extension of up to $2,000,000 in working capital loans from TuHURA, $1,750,000 of which will be contingent on the completion of a financing transaction by TuHURA.

On Deck for 2025

We expect the IFx-2.0 Phase III trial for MCC launch in early 2025 with enrollment completion approximately one year after start. Six months after the last patient is enrolled, topline should be reported quickly followed by a new drug application filed under the auspices of accelerated approval. If the IFx-2.0 arm shows a material benefit in overall response rate and no unexpected delays, approval could come six months later for MCC. In the anti-VISTA realm, if the acquisition closes as expected, a Phase II proof of concept trial could begin in the second half of 2025 to evaluate KVA12 with a menin inhibitor against a menin inhibitor alone in mutated NPM1 AML. Success would lead to conversations with the FDA and the pursuit of accelerated approval in this indication.

Exhibit XVII – Anticipated Milestones



Source: TuHURA January 2025 Presentation

Management Profiles

James Bianco, M.D., CEO and Co-Founder

Dr. Bianco has served as TuHURA's Chief Executive Officer and as a director of TuHURA since July 1, 2021. Dr. Bianco was also the founder, Chief Executive Officer and Chairman of Morphogenesis Biopharma, Inc., a biotechnology company, from its inception in November 2018 through its dissolution in January 2023, following the transfer of its assets to TuHURA. Dr. Bianco is a 30-year veteran of the biopharmaceutical industry. In 1991, Dr. Bianco founded CTI Biopharma and from 1992 to 2016 was its Chief Executive Officer. During his tenure, Dr. Bianco was responsible for strategic portfolio development and identifying, acquiring, licensing, purchasing, or acquiring through international merger and acquisition, five drug candidates, of which four have been approved by the FDA. Three have received accelerated or conditional regulatory approval in the U.S. and/or E.U. Dr. Bianco earned his M.D. from the Mount Sinai Icahn School of Medicine and completed his residency and chief residency at the Mount Sinai Medical Center in New York City. He completed his fellowship in Hematology/Oncology at the University of Washington/Fred Hutchinson Cancer Research Center (FHCRC) where he was appointed Assistant Professor of Medicine, Assistant Member FHCRC and Director of the Bone Marrow Transplant Unit at SVAMC.

Dan Dearborn, Chief Financial Officer

Dan Dearborn joined TuHURA in 2018 as its Chief Financial Officer. Mr. Dearborn is a CPA with over 25 years of finance experience exclusively with health care and biotechnology companies. Before TuHURA, from 2015 to 2017, Mr. Dearborn was Chief Financial Officer at MYMD Pharmaceuticals, an emerging biotechnology firm. Mr. Dearborn is an alumnus of Loyola University in Maryland and joined Ernst & Young early in his career. He was with Pharmerica, a long-term care pharmaceutical company, for fifteen years and advanced quickly to Director. He then moved to BioDelivery Sciences International as Controller. During his time at BioDelivery, the company signed two large commercial partnership agreements and was listed on the NASDAQ. Mr. Dearborn later joined Welldyne, Inc. as its Chief Financial Officer. Welldyne is a pharmacy benefit manager with several related health care businesses in Florida and Colorado. During his time with Welldyne, the company was sold to Carlyle Group.

Thierry Guillaudeux, Ph.D., Chief Scientific Officer

Dr. Thierry Guillaudeux has over thirty years of experience in Immuno-Oncology in France and in the U.S. Before his role at TuHURA, he held the position of Chief Scientific Officer at Kineta, Inc., a clinical-stage biotechnology company developing KVA12, an anti-VISTA antibody targeting cancer indications. Dr. Guillaudeux assumed his role as CSO at TuHURA as part of TuHURA's acquisition of Kineta. In addition to his corporate experience, he contributes a strong executive background in academia as a leader of a research laboratory of the French National Institute of Health with more than sixty employees for ten years and serving as a Vice President in Technology Transfer at the University of Rennes where he negotiated and executed numerous contracts and licenses with industry. Dr. Guillaudeux has also served as a board member of two French companies for over five years.

Dr. Guillaudeux leads the company's immuno-oncology programs from discovery and pre-clinical to IND filling. This includes all *in vitro* and *in vivo* immuno-oncological assay developments to evaluate the mechanisms of action of TuHURA's pipeline as well as toxicology studies for lead antibody selection. He also leads the scientific strategy for the selection of new innovative immuno-oncology programs.

His fields of expertise are B cell lymphoma, lung cancer and the tumor microenvironment. He has published more than 40 peer-reviewed publications and is a co-inventor of multiple patents in the field. He was principal investigator or co-investigator on numerous French government and non-profit grants for his laboratory.

Dr. Guillaudeux received his B.S. and M.S. degrees from the University of Rennes and his Ph.D. in immuno-oncology from the University of Toulouse. He earned his post-doctorate at the Fred Hutchinson Cancer Research Center and was responsible for the sequencing of the Human Major Histocompatibility Complex Class I molecule.

RISKS

All investments contain an element of risk that reflects the uncertainty of a business and what it will ultimately achieve. Some investments exhibit higher predictability, with current cash flows and established sales. These enterprises will have a lower level of perceived risk while other companies that are developing an undefined, new technology have a much higher level of perceived risk.

The biotechnology space includes companies at both ends of the spectrum, from mega-cap pharmaceutical titans with multiple products generating revenues, to small operations with a handful of employees conducting preclinical studies. Many of the risks faced by the large pharmaceutical companies and smaller biotechnology-focused firms are similar; however, some hazards are peculiar to smaller companies that have not yet established themselves or their products. TuHURA falls at the smaller end of the range and depends on continued financing as it does not generate any revenues.

For smaller, early-stage companies, investing in drug development is a lengthy process. The timeframe for conducting preclinical research to eventually commercializing a drug can take from 12 to 15 years or even longer given market and company-specific conditions. And with, on average, only one in one thousand compounds eventually making it to the market from the discovery stage, the risks are substantial. TuHURA is fortunate to have the benefit of guidance and support from several FDA programs that should accelerate the development of its candidates and submission of an application to the FDA.

Financing

Even if a company has a strong, experienced team that is developing a therapy with a high likelihood of success and a large addressable market, securing funding may be difficult. Access to financing comes and goes in cycles. During periods of improving confidence, capital may be easy to obtain; however, during a liquidity crisis or a period of heightened risk perception, even companies with bright prospects may be in trouble if they depend on the financial markets to fund their work. If capital is needed to sustain operations and it is not readily available, the company may be forced to suspend research and development activities, sell equity at a substantial discount to previous valuations and dilute earlier shareholders. A lack of funding may leave potentially promising therapies without a viable route to progress or force a company to accept onerous terms.

Increases in cost of capital can make previously attractive investments less so. Interest rates have increased over the last four years, materially changing the perceived net present value and reward to risk ratio for many investments, especially those in higher risk categories such as drug development. Early-stage life sciences companies depend on a steady flow of capital to support their research and development activities. As capital becomes more dear, previously attractive projects may become less so, making access to critical capital inflows less certain. In conjunction with the reverse merger with Kintara, TuHURA raised $31 million in a fully funded financing. In 2025, the company plans to raise further funds in a PIPE.

Partners

Contract research organizations (CROs) have assumed a more significant role in developing drug candidates as the complexity and cost of trials have increased. Finding appropriate populations to participate in clinical trials is difficult due to the shift to biomarker defined groups, screening for specific mutations, personalized medicine and orphan indications that address small clusters of patients. This shift has increased the dependence on specialized CROs for project management and clinical monitoring services that add additional risks related to third parties. TuHURA has not identified the CRO that will advance the IFx-2.0 in MCC. Kineta used PPD as its CRO for the Phase I portion of its anti-VISTA program and we expect that TuHURA will continue with the service provider for the Phase II in AML given their familiarity with the program.

A CRO partner may face competing demands which can adversely affect the work they are managing on behalf of the client. CROs and subcontractors must abide by strict execution and trial parameters that if violated can jeopardize trial execution or data validity. Patient recruitment may be difficult. Subcontractors supervise and execute research and conduct biometric and pharmaco-vigilance, which are complex tasks. Clinical investigational centers need sufficient capacity and the candidate drug needs to be manufactured in compliance with current Good Manufacturing Practices (cGMP) and be available to administer to patients. Finally, the data must produce results that achieve sufficient statistical significance and clinical relevance to justify regulatory approval.

Regulatory Environment

All drugs must navigate the regulatory approval process in the U.S., E.U., Japan and other countries before commercialization. Success is uncertain and may take years depending upon the needs and desires of the determining authority. Substantial expense is undertaken to bring a molecule or compound through clinical trials and address all regulatory agencies' concerns. Isolating companies with a history of research success in drug development, with opinion leaders and experts in the field are important factors that can help mitigate this risk. Companies and management teams that have achieved previous success with the FDA or other regulatory agencies are more attractive than those new to the process. Special programs may be available to help products be more effectively evaluated such as the FDA's Project Frontrunner which is appropriate for TuHURA's IFx-2.0 candidate in Merkel cell carcinoma (MCC). Other mechanisms include the Orphan Drug Act, Regenerative Medicine Advanced Therapy (RMAT) program and the Breakthrough Therapy designation; however, changes in sentiment or perceived safety for pharmaceuticals drugs could alter the regulatory environment to require a more thorough process and these pathways may be extended or additional requirements may be put in place. TuHURA's lead indication for its IFx-2.0 platform, MCC, meets the threshold required to qualify for the orphan disease designation.

Success of clinical trials is low and a submitted and accepted NDA or BLA does not ensure that it will receive FDA approval. Our review of several resources finds that generally a Phase I asset has an 8-10% chance of approval, a Phase II asset 15-20% chance of approval and a Phase III asset has an even chance of approval. If a BLA or NDA is accepted, it has about an 85% chance of approval. There are many factors which can either improve or reduce a candidate's chances for success including indication pursued, use of biomarkers, status as a lead candidate, use of special regulatory dispensations and other factors. Below, we provide probabilities from a reliable source on the probabilities of a candidate moving from one Phase to the next.

Exhibit XVIII – Success of Phased Trials and Regulatory Approval[37]

Phase	I - II	II - III	III - NDA/BLA	NDA/BLA - Approval	I - Approval
Probability	**52.0%**	**28.9%**	**57.8%**	**90.6%**	**7.9%**

Competitive Environment

Many firms are participating in the oncology space and products must compete against established treatments that are entrenched in the health care system. In oncology, for example, there are hundreds of approved agents available. According to Evaluate Pharma, there are 236 marketed drugs in the immuno-oncology space as of January 2025. On the development side, there are 38 I-O agents filed, 234 in Phase III and 897 in Phase II. To differentiate itself from other sponsors, TuHURA is developing both its IFx innate immune agonist candidate and the recently acquired VISTA-inhibiting antibody. It also holds an antibody-drug conjugate (ADC) asset in a preclinical stage of development.

Marketing and Commercialization Risk

Successful marketing of approved drug candidates relies on adoption by patients and providers. An approved drug must produce convincing clinical trial data and maintain a favorable reputation among prescribers. Marketing is expensive and requires an experienced sales force and a presence in the marketing region. Marketed products remain under surveillance and any unexpected adverse effects may lead to regulatory authorities revoking marketing authorization. Inclusion of the drug in insurance plan offerings is essential. Rapidly obtaining a preferred position on health plan and payor formularies is critical to achieving target penetration rates. If health plans and payors cannot agree on appropriate pricing for the drug and the compound fails to offer a significant benefit above standard of care, sales may be limited. TuHURA is several years away from marketing and commercialization activities. We anticipate that the company will hand off its assets after further development to a larger partner who will assume these responsibilities.

Manufacturing

Medical product companies can produce medicines in-house or rely on third parties to manufacture them. While there are many benefits to owning manufacturing facilities and exercising direct control, in most cases, small and medium size biopharmaceutical companies work with partners through supply agreements to make their products. Working with a partner confers several benefits including economies of scale, a management team dedicated to compliance with regulatory requirements and the flexibility of engaging other manufacturers based on changing cir-

[37] Summarized from Clinical Development Success Rates 2011-2020. Compiled by Zacks Analysts.

cumstances. Using a partner also limits the capital burden of a single product company and more closely aligns volume, costs and their respective timing. While there are numerous benefits to outsourcing manufacturing, a sponsor can also be exposed to several risks. Manufacturing partners may not prioritize client projects and run afoul of regulatory requirements. The manufacturer may experience quality control or volume constraints that could disrupt demand. Take or pay contracts could force the client to accept more product than can be sold in a reasonable time, impacting cash flow and producing excessive inventory which could expire before sale. Using outside contractors could also allow internally developed trade secrets to be compromised. Although the identity of TuHURA's manufacturing partner has not been disclosed, these operations are reported to take place domestically. TuHURA's lead product, IFx-2.0 requires the compounding of two substances prior to administration. The pDNA that instructs the cells to produce the bacterial protein is combined with the third party transfectant agent, JetPEI and glucose to enhance its entry into the cancer cell. Future formulations are expected to combine these constituents into one vial, improving ease of use and simplifying the manufacturing process. While the compounding adds another step to the process, it is well understood and presents minimal additional risk.

Intellectual Property

Despite patents, exclusivity and trade secrets, infringement of intellectual property is a risk. TuHURA's portfolio consists of 33 issued patents in 13 jurisdictions. There are nine pending applications, comprising two U.S. utility patent applications and seven foreign patent applications. The patents protecting the IFx technology platform are classified as composition of matter and method of use. TuHURA licenses the intellectual property rights relating to its tumor microenvironment modulator technology platform under exclusive license agreements with H. Lee Moffitt Cancer Center and Research Institute worldwide. A component of TuHURA's intellectual property comprised know-how and trade secrets related to manufacturing. This information has not been codified or shared publicly. While this presents a risk if employees leave and take the knowledge with them, this IP also does not expire, which could provide benefits limiting the impacts of competing products.

Geopolitical

Trade tensions between the US and China were exacerbated by the COVID pandemic. There had been a cross-pollination of capital and drug development between the two countries which has slowed due to trade and political disputes. This conflict may reduce the availability of capital, partnerships and future development and commercialization deals between companies in the two nations. The UK withdrew from the European Union in 2020, creating new trade, transportation and other barriers between the UK and mainland Europe. The European Commission Decision Reliance Procedure, which allows the UK regulatory apparatus to rely on EU centralized marketing authorization decisions, has expired. Following this date, sponsors must obtain UK marketing authorization for product sales in the UK from the Medicines and Healthcare products Regulatory Agency (MHRA). Conflict between Ukraine and Russia and in Israel has led to disruptions in these countries. In Russia, sanctions have been imposed on many businesses domiciled there which may lead to product shortages. Refugees fleeing the war in Ukraine may also impact nearby nations and their productivity which could affect clinical trials and commercialization there. With the new US administration, the threat and possible implementation of tariffs could lead to trade wars that limit international sales. TuHURA has no exposure to China, Israel, Ukraine or Russia.

Cost Burden and Inflation

Drug price inflation has gained attention as it and other health care costs have risen at a materially faster pace than the consumer price index. As new therapies have been approved, drug prices have increased to reflect higher development costs and improved pricing power of pharmaceutical and biotech companies. On the demand side, deductibles have been steadily rising over the last decades, and in some cases, individuals and families must cover several thousand dollars in costs before the benefits of insurance begin. Cost sharing or co-insurance is another component of insurance plans that directly increases the burden on patients. This has resulted in greater elasticity in demand for drugs than was previously the case. Individuals with high deductibles or no insurance may be sensitive to price and avoid expensive treatments.

While we have discussed a broad variety of risks above, we believe that our forecast parameters, discount rates, success probabilities and valuation metrics address these potential outcomes and our target price reflects an assumption of these risks faced by biotechnology companies.

VALUATION

TuHURA is advancing several clinical programs built on multiple platforms. We expect the IFx-2.0 program in MCC and KVA12 in AML to advance. We also see value in the differentiated ADC program which targets the delta opioid receptor. We think that established biopharmaceutical companies will be interested in this asset and willing to execute a transaction with valuation guided by recent deals.

We expect IFx-2.0 to begin a Phase III trial in MCC in 2Q:25 that will take advantage of several FDA programs to obtain approval for candidates that address a serious unmet need. We anticipate that it will take one year to enroll and another six months to gather the necessary data to submit a biologics license application (BLA) to the FDA. The accelerated approval grant could respond within six months of submission. Based on this timeline we could conservatively see marketing of IFx-2.0 by the start of 2028. The newly acquired anti-VISTA antibody, KVA12, is slated to begin a proof-of-concept trial in the second half of 2025 in AML. This trial could generate data that may allow KVA12 to advance with a similar timeline to IFx-2.0.

Our literature review identified about 3,300 cases of MCC per year in the United States with about 90% of these patients appropriate for combination therapy with checkpoint inhibitors and the innate immune agonist IFx-2.0. Growth in this group is estimated to be 3.0%, a faster clip than population growth due to rising awareness of MCC and enhanced diagnostic capabilities. Given the small size of the targeted group, we see a relatively high initial penetration rate here, especially if IFx-2.0 can generate a meaningful increase in survival. In the first year (2028) we model 11.3% penetration rising to 64.5% by year seven (2034). This equates to 370 patients treated in year one and 2,500 treated in year seven. Pricing is expected to be at a modest discount to checkpoint inhibitors and we model $120,000 of revenue per treatment and inflation of 3% per year. This generates revenue of $48 million in year one and $396 million by year seven. We assume that TuHURA uses an established biopharma partner to commercialize IFx-2.0 and receives upfronts, milestones and royalties equal to 33% of net product sales.

For markets outside the US, particularly Australia and the EU, we estimate approximately 6,700 MCC patients per year and apply the same addressable market proportion used in the US. This produces approximately 6,100 patients in a group growing at 3.5% per year. We estimate that commercialization of IFx-2.0 in these developed markets one year after first sales in the United States. We assume a somewhat lower penetration rate in overseas markets with 7.5% of the addressable market served in year one (2029) rising to 43.0% in year seven (2035). This assumption treats 520 patients in year one and 3,700 in year seven. Pricing is expected to be at $60,000 per treatment, half the amount in the US and inflating at 1.5%. These assumptions generate revenue of $33 million in year one and $257 million in year seven. Similar to the arrangement in the US, we assume that TuHURA gains economic value of 33% of net product sales.

KVA12 is expected to follow an expedited pathway following successful proof of concept data from the trial anticipated to begin in 2H:25. If the upcoming study is successful, this candidate could enter pivotal trials in 2027 and produce data sufficient to support a BLA in 2029 and obtain approval and first sale in 2030 in the United States. There are an estimated 63,000 cases of AML in the US in 2024 which we estimate will grow at a 0.8% annual rate. About a third of this population is positive for NPM1 and appropriate for combination therapy with menin inhibitors and represents the addressable market. This equates to 22,000 patients. Due to the larger patient population and the greater number of therapies for AML, we estimate a 90-basis point penetration into the addressable population in year one (2030) which equates to approximately 200 patients. By year seven this is estimated at 12% penetration or 2,900 patients. Pricing for KVA12 is estimated to match other checkpoint inhibitors such as Keytruda at $160,000 per treatment. In year one, this produces product revenues of $37.3 million and in year seven, $605 million. Price inflation is 3% per year. We also assume that TuHURA will negotiate an economic value of net product revenues equal to 33%.

Outside of the US we anticipate that KVA12 will begin first commercialization in 2031. The global incidence of AML is 145,000 in 2024 and about 35% of this population expresses the NPM1 mutation, generating an addressable market of 51,000. Growth in this population is estimated to be 1.6%. Penetration in the first year of commercialization (2031) is estimated to be 80 basis points or 447 patients. By year seven (2037), we anticipate 7.0% penetration, equivalent to 4,300 patients. Pricing is estimated to be 50% of US levels. These assumptions generate revenue of $39 million in year one and $411 million by year seven. Economic value realized by TuHURA is in line with our other forecasts at 33% of net product revenues.

ADC Valuation

Over the last several years there have been numerous deals for ADCs in all stages of development which we discuss in an earlier section of this report. One of the most visible was the $43 billion acquisition of Seagen by Pfizer which closed in December 2023. This followed two significant years of ADC deals of 26 transactions each year[38] in 2021 and 2022. Most of the deals were for early-stage assets as established biopharmas jockeyed for position. A research piece compiled by Back Bay Life Science Advisors found that ADCs received the highest deal value of any other drug class, with an average upfront of $851 million (median $848 million) across all stages of development. Phase I and Phase II average upfronts were $520 million and $893 million respectively. The value attributed to this modality is justified by the greater ease of competitive protection afforded by an ADC which has multiple components which can obtain patent protection as well as biologics protection for the monoclonal antibody portion of the drug.

Given the substantial interest in ADCs by established biopharmaceutical companies, we value TuHURA's differentiated bi-functional ADC platform. It offers novel capabilities including binding the delta opioid receptor and bifunctional capabilities that can bring novel monoclonal antibodies such as checkpoint inhibitors into the mix. Based on our review of completed deals we apply a 25% probability of a deal getting done with a value of $400 million which adds $100 million to the company's valuation.

Estimates for operational costs call for $27 million in cash expense for 2025, $31 million in 2026 and $36 million in 2027 after which costs are estimated to grow at a low-single-digit per annum rate. After approval of KVA12 outside the US, we estimate R&D expenses to fall to zero over the next two years. General and administrative expenses will continue at low single-digit inflation rates. Initial profits will be offset by net operating losses (NOLs). After exhausting NOLs, we forecast a 25% cash tax rate on earnings. After-tax cash flows to the company are discounted at a 15% rate with a terminal growth rate of -10% beginning in 2047. We apply an overall weighted 50% likelihood of success of TuHURA's programs given the stage of development, access to FDA expedited programs, early results and data generated by other candidates in the class.

Stock options are assumed to be exercised if below our target price with proceeds added to cash and exercised instruments added to shares outstanding. In addition to the approximately 42 million shares of common stock outstanding, we add 18.5 million exercised options, 1.5 million CVR shares, 5.6 million shares related to the acquisition of Kineta and 11 million other shares to reflect potential capital raises over the next 18 months. Shares used to calculate our target price are approximately 79 million.

Despite the conservative stance of our assumptions, penetration into addressable markets could be different for TuHURA's candidates if study results deviate from expectations. The valuation does not consider other indications for IFx-2.0 and KVA12 outside of what is explicitly modeled. If these candidates are successful in the initial indications, TuHURA can seek additional indications on their behalf, which could increase the valuation. We note that the determinant for many of the variables in our model will be the ultimate performance of TuHURA's candidates in pivotal studies and partnership activities. We will update our model accordingly as data is made available and if other assets in the pipeline are advanced toward a new drug application.

Based on the assumptions identified in our discounted cash flow model, we generate a valuation of $9.25 per share.

[38] This included option, license, collaboration/co-development and acquisition deals.

PROJECTED FINANCIALS

TuHURA Biosciences, Inc. - Income Statement

TuHURA Biosciences, Inc.	Q1 A	Q2 A	Q3 A	Q4 E	2024 E	Q1 A	Q2 E	Q3 E	Q4 E	2025 E	2026 E	2027 E
Total Revenues ($USD)	**$0**	**$0**	**$0**	**$0**	**$0**	**$0**	**$0**	**$0**	**$0**	**$0**	**$0**	**$0**
Research & Development	$3,206	$3,206	$2,947	$3,000	$12,359	$5,125	$5,125	$6,125	$6,125	$22,500	$27,000	$30,000
General & Administrative	$906	$906	$783	$850	$3,446	$1,125	$1,125	$1,125	$1,125	$4,500	$4,200	$5,500
Other Operational Items												
Income from Operations	($4,112)	($4,112)	($3,730)	($3,850)	($15,805)	($6,250)	($6,250)	($7,250)	($7,250)	($27,000)	($31,200)	($35,500)
Restructuring & Impairments			$0	$0	$0	$0	$0	$0	$0	$0	$0	$0
Other Items	$0	($335)	$21	$0	($314)	$0	$0	$0	$0	$0	$0	$0
Net Interest Expense	$0	($1,548)	($1,870)	$0	($3,418)	$0	$0	$0	$0	$0	$0	$0
Pre-Tax Income	($4,112)	($5,995)	($5,579)	($3,850)	($19,536)	($6,250)	($6,250)	($7,250)	($7,250)	($27,000)	($31,200)	($35,500)
Provision for Income Tax	$0	$0	$0	$0	$0	$0	$0	$0	$0	$0	$0	$0
Tax Rate												
Net Income	**($4,112)**	**($5,995)**	**($5,579)**	**($3,850)**	**($19,536)**	**($6,250)**	**($6,250)**	**($7,250)**	**($7,250)**	**($27,000)**	**($31,200)**	**($35,500)**
Net Margin												
Reported EPS	**($1.79)**	**($2.60)**	**($2.42)**	**($0.09)**	**($1.60)**	**($0.15)**	**($0.15)**	**($0.17)**	**($0.17)**	**($0.73)**	**($0.65)**	**($0.60)**
YOY Growth												
Basic Shares Outstanding	2,302	2,302	2,302	42,030	12,234	42,250	42,300	42,738	43,015	36,825	48,000	59,000

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HISTORICAL STOCK PRICE

TuHURA Biosciences, Inc. – Share Price Chart[39]



[39] 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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