

AGM PRESENTATION

Melbourne, Australia – 7 October 2026: In advance of this morning's Annual General Meeting of shareholders to be held at 10:00am, and in accordance with ASX Listing Rule 3.13.3, Percheron Therapeutics Limited (ASX: PER or 'the Company') is pleased to provide a copy of the presentation that will be made to shareholders at the Annual General Meeting.

About Percheron Therapeutics Limited

Percheron Therapeutics Limited [ASX: PER | US OTC: PERCF] is a publicly listed biotechnology company focused on the development and commercialisation of novel therapies for oncology and rare diseases. The company's lead program is HMBD-002, a monoclonal antibody targeting the immune checkpoint regulator, VISTA. HMBD-002 has completed a phase I clinical trial in patients with advanced cancer, which has shown the drug to be generally safe and well tolerated, and Percheron aims to commence further clinical trials in CY2026. For further information, please see our website at www.PercheronTx.com, or email info@PercheronTx.com.

This announcement has been authorised for release to the Australian Securities Exchange by the Board of Directors.

Annual General Meeting 2026

CEO and MD address

Dr Michael Baker | October 2026

ASX: PER

US OTC: PERCF

WWW.PERCHERONTX.COM



Forward-Looking Statements



This presentation contains forward-looking statements within the meaning of the safe-harbor provisions such as the Private Securities Litigation Reform Act of 1995. These statements do not relate strictly to historical or current facts and may be accompanied by words such as 'could,' 'would,' 'may,' 'potentially,' 'suggest,' 'believes,' 'expects,' 'should,' 'intends,' 'plans,' 'forecasts,' and similar words or expressions.

Such statements involve substantial risks and uncertainties, not all of which may be known at the time. All statements contained in this presentation, other than statements of historical fact, including without limitation statements regarding our strategy, research and development plans, collaborations, future operations, future financial position, future revenues, projected costs, pricing, prospects, plans, and objectives of management, are forward-looking statements. Not all forward-looking statements in this presentation are explicitly identified as such.

The Company does not warrant any of the forward-looking statements in this presentation, and investors are advised to interpret such statements in the context of other available sources of information and with the assistance of expert advisors as appropriate.

Many factors could cause the actual results of the Company to differ materially from the results expressed or implied herein, and you should not place undue reliance on the forward-looking statements. Drug development is inherently risky, and only a small proportion of research and development programs lead to a marketed product. Factors which could change the Company's expected outcomes include, without limitation, our ability to: advance the development of our programs, and to do so within any timelines that may be indicated herein; the safety and efficacy of our drug development candidates; our ability to replicate experimental data; the ongoing validity of patents covering our drug development candidates, and our freedom to operate under third party intellectual property; our ability to obtain necessary regulatory approvals; our ability to enter into and maintain partnerships, collaborations, and other business relationships necessary to the progression of our drug development candidates; changes in the competitive landscape pertaining to our drug development candidates; the timely availability of necessary capital to pursue our business objectives; changes in the public policy environment in one or more countries in which we operate or may seek to operate which disfavour our business; our ability to attract and retain qualified personnel; changes from anticipated levels of customer acceptance of existing and new products and services; and other factors.

Although the Company believes that the expectations reflected in such forward-looking statements are reasonable, and although they reflect our current views as at the date of this presentation, there can therefore be no assurance that such expectations will prove to be correct. The Company has no obligation as a result of this presentation to pursue any specific strategy or plan outlined herein, or to deliver any specific outcome that may be implied or inferred.

Any forward-looking statements contained in this presentation speak only as of the date this presentation is made, and we expressly disclaim any obligation to update any forward-looking statements, whether because of new information, future events or otherwise, except as may be required by law.

Percheron Investment Highlights



Strong Science

HMBD-002 targets VISTA using a differentiated mAb scaffold supported by compelling preclinical data



Interest For Investigator Led Studies

Investigator initiated clinical studies may expand the clinical data set at modest cost to Percheron and yield important data



Clinically Validated Safety

US FDA IND-enabled phase 1 study showed favourable safety profile, alone and in combination with Keytruda (α PD-1 check point)



Large Market Opportunity

VISTA is implicated across multiple tumour types, opening a broad set of potential indications to target



Multiple Partnership Opportunities

Synergy between VISTA and other anti-cancer agents may make HMBD-002 attractive to potential partners



Strong Team

Leadership with deep, hands-on experience in antibody drug development, regulatory strategy and deal-making

Corporate and Financial Overview



Corporate Fundamentals

Market Capitalisation ¹	AU\$ 7.3 million
Primary Listing:	ASX: PER
Secondary Quotations:	OTC: PERCF
Shares on Issue:	1,822 million
Cash Balance (June 30) ²	\$4.1 million

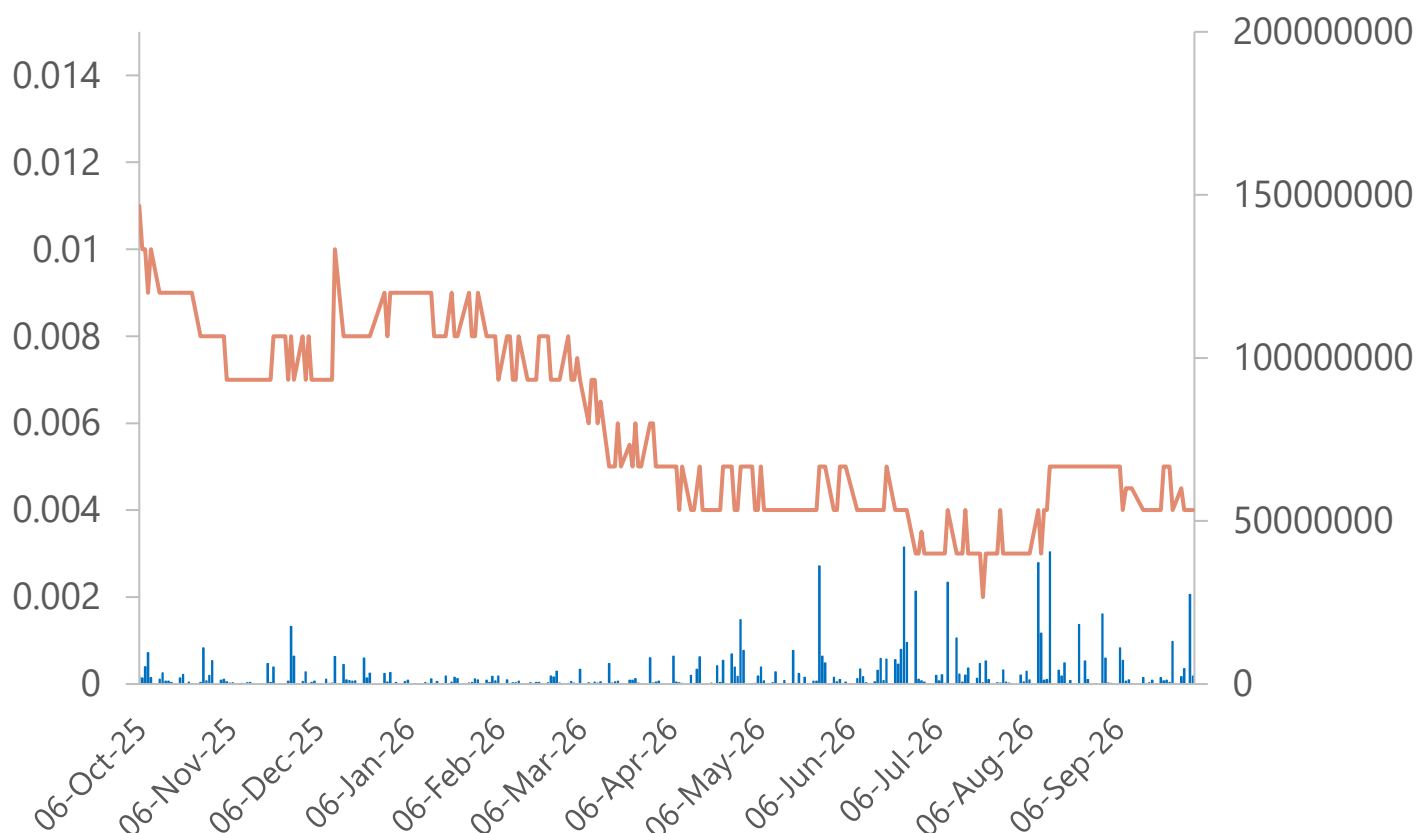
Key Shareholders

Mutual Investments Limited	4.5%
Glen Bull	4.0%
Non-correlated Capital Limited	3.8%

Directors and Management **4.7%**

1. As of 2 October 2026; 2. Does not include the proceeds from the R&D Tax rebate announced 31 August 2026 and the gross proceeds from the \$2.3 million Placement announced 1 September 2026; 3. Holding percentages reflect holdings post-completion of tranche 1 and pre-completion of tranche 2 of the Placement announced 1 September 2026.

PER Price and Volume - 12 months¹



Checkpoint inhibitors play a key role in cancer treatment

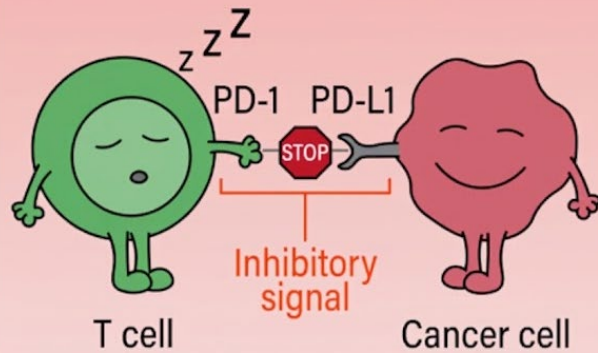


Checkpoint inhibitors can activate T cells or influence immunosuppressive myeloid cells



Myeloid derived suppressor cells (MDSCs) and tumour associated macrophages (TAMs) express VISTA and can make the TME immunosuppressive supporting tumour growth

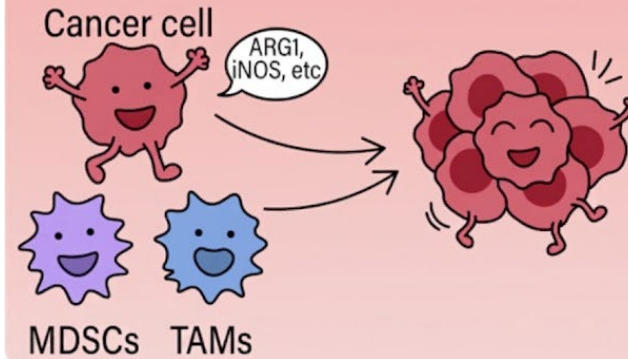
Checkpoints act as an inhibitory signal and block T cell killing



OUTCOME

- ↓ T cell activity
- ↓ Cytokine production
- ↓ Cytotoxic function
- ↑ Tumour cells survive and proliferate
- ↑ Immune evasion maintained

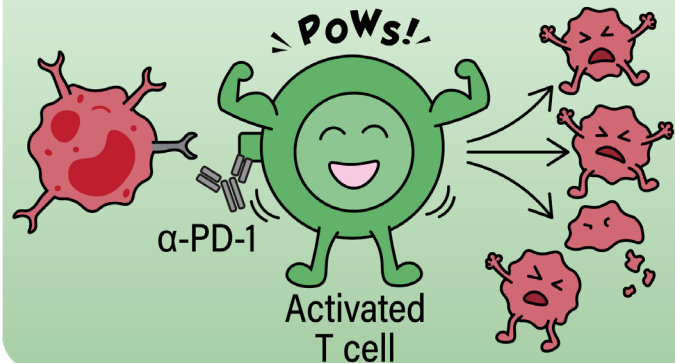
Checkpoints can contribute to an immunosuppressive tumour microenvironment (TME)



OUTCOME

- ↑ Immuno-suppression
- ↑ Tumour growth
- ↑ Angiogenesis
- ↑ Metastasis

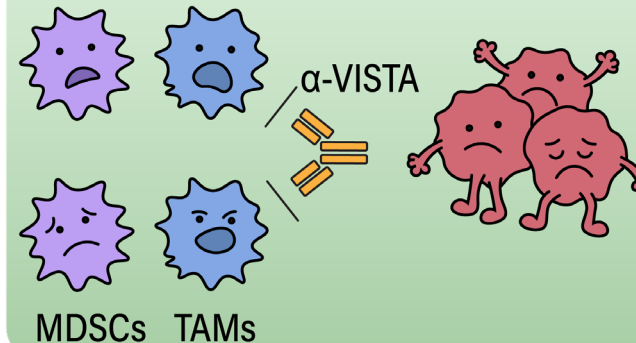
Checkpoint inhibitors (i.e. α -PD-1) reactivate T cells resulting in tumour cell killing



OUTCOME

- ↑ T cell activation
- ↑ Cytokine release
- ↑ Perforin/Granzyme mediated killing
- ↑ Tumour cell death
- ↑ Immune memory

Blocking immunosuppressive myeloid cells can re-program the TME



OUTCOME:

REPROGRAMMED TME

- ↓ Immuno-suppression, etc
- ↓ Matrix remodelling
- ↑ Tumour cell death

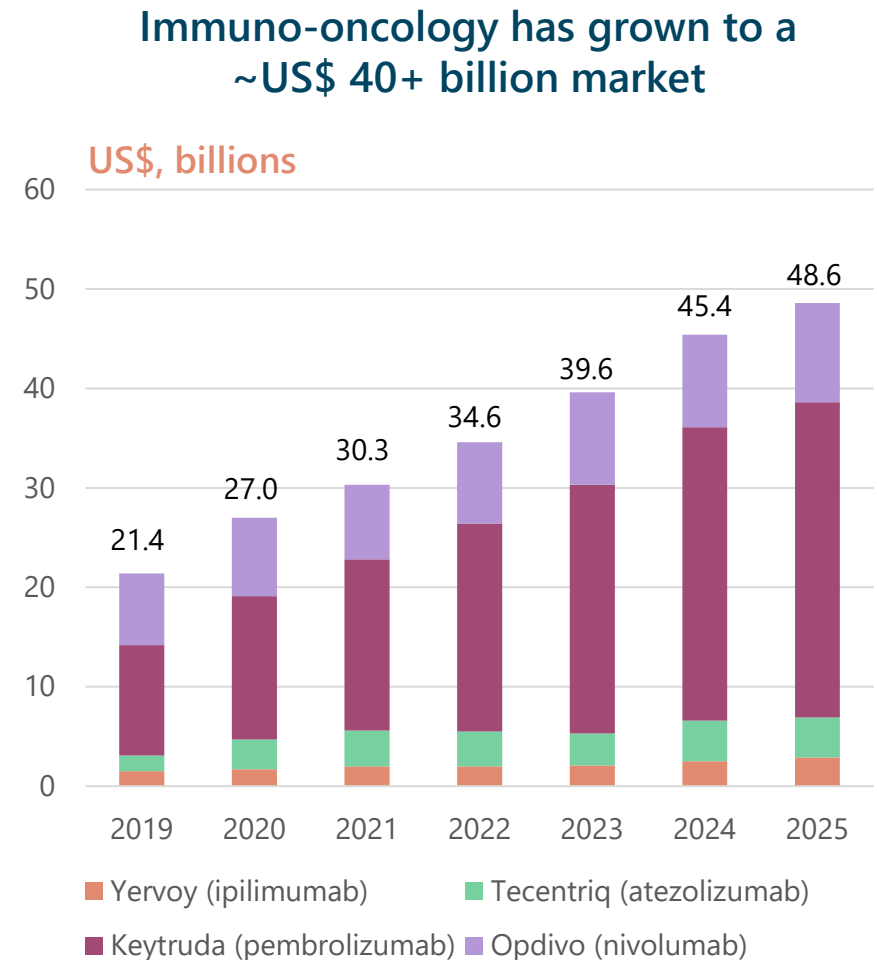
Immune checkpoint inhibitors have been one of the fastest growing classes of medicine in the immuno-oncology field, with close to US\$ 50 billion in annual sales



Immuno-oncology therapies have had a profound impact on the cancer treatment landscape, but patent cliffs are looming



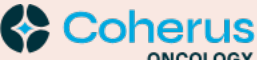
	Yervoy	Tecentriq	Keytruda	Opdivo
Approval	2011	2016	2014	2014
Target	CTLA-4	PD-L1	PD-1	PD-1
Patent Expiration*	2025/2026	2028	2028	2028



Source: Company filings; * - primary market exclusivity



Successful immuno-oncology assets are highly partnerable, with comparable deals suggesting attractive valuations¹

Date	Acquirer	Target	Surface target	Lead asset stage	Tumour types	Upfront	Milestone payments
Jan-24	 GILEAD	 ARCUS BIOSCIENCES	TIGIT	Phase 3	Lung cancer, GI cancer	\$320m equity investment	NA
Jun-23	 Coherus ONCOLOGY	 SURFACE ONCOLOGY	IL-27 & CCR8	Phase 2	Lung, liver cancer, solid tumours	Stock transaction value of \$65m	Acquisition
Mar-23	 BIONTECH	 OncoC4	CTLA-4	Phase 2 / 3	Solid tumours, NSCLC	\$200m	Development, regulatory and commercial milestones, tiered royalties
Jun-22	 REGENERON	 sanofi	PD-1	Commercial / Phase 3	Skin cancers, NSCLC	\$900m	Milestone payments and royalties
Jun-21	 GSK	 ITEOS THERAPEUTICS	TIGIT	Phase 1	Solid tumours	\$625m	Up to \$1.45 billion in milestones; tiered royalties on Ex-US sales; co-commercialisation and profit share in US
Dec-21	 NOVARTIS	 BeiGene	TIGIT	Phase 3	Solid tumours and lung cancer	\$300m	Up to \$700 million if the option is exercised; royalties
Feb-21	 Coherus ONCOLOGY	 君实生物 TopAlliance	PD-1	Commercial / BLA	Nasopharyngeal carcinoma, NSCLC	\$150m	\$380 million in milestone payments and 20% royalty on net sales
Dec-20	 GSK	 SURFACE ONCOLOGY	PVRIG	Phase 1	Advanced solid tumours	\$85m	Up to \$730 million in milestones; tiered royalties on net sales
Sep-20	 abbvie	 I-MAB BIOPHARMA 天 境 生 命	CD47	Phase 1 / 2	Solid tumours and AML	\$180m	Up to \$1.94 billion; tiered royalties on global net sales
Mar-20	 GILEAD	 FORTY SEVEN	CD47	Phase 1 / 1b	AML, DLBCL	\$4,900m	Acquisition

1. Deal references are provided on the final slide; GI – gastrointestinal; NSCLC – non small cell lung carcinoma; BLA – Biologics License Application; AML – Acute Myeloid Leukaemia; DLBCL – Diffuse Large B-cell Lymphoma.

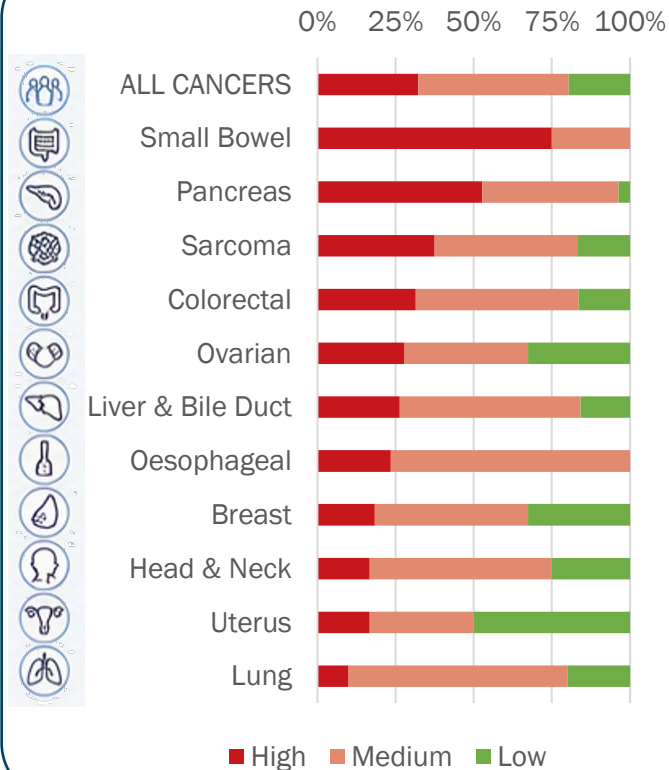
VISTA is a novel checkpoint



VISTA is an attractive target across a range of solid tumours



VISTA is expressed in tumour samples from a wide range of cancer types¹



A relevant case study: VISTA is the highest checkpoint gene expressed in late-stage glioma and is prognostic for survival²

High prevalence by IHC

82.4%

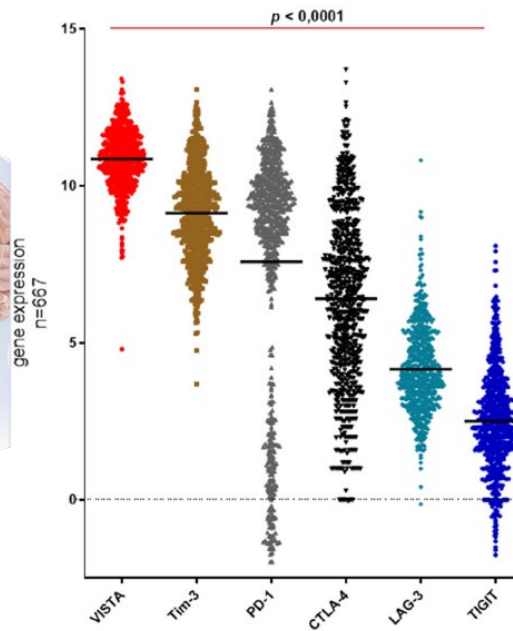
High grade gliomas (III/IV) express VISTA (14/17)

30.8%

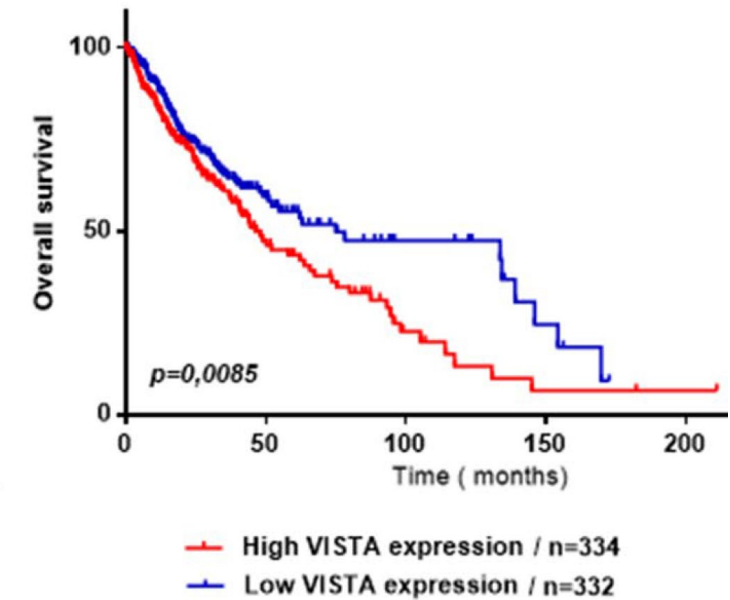
Low grade gliomas (I/II) express VISTA (4/13)



Highest expression of all check points

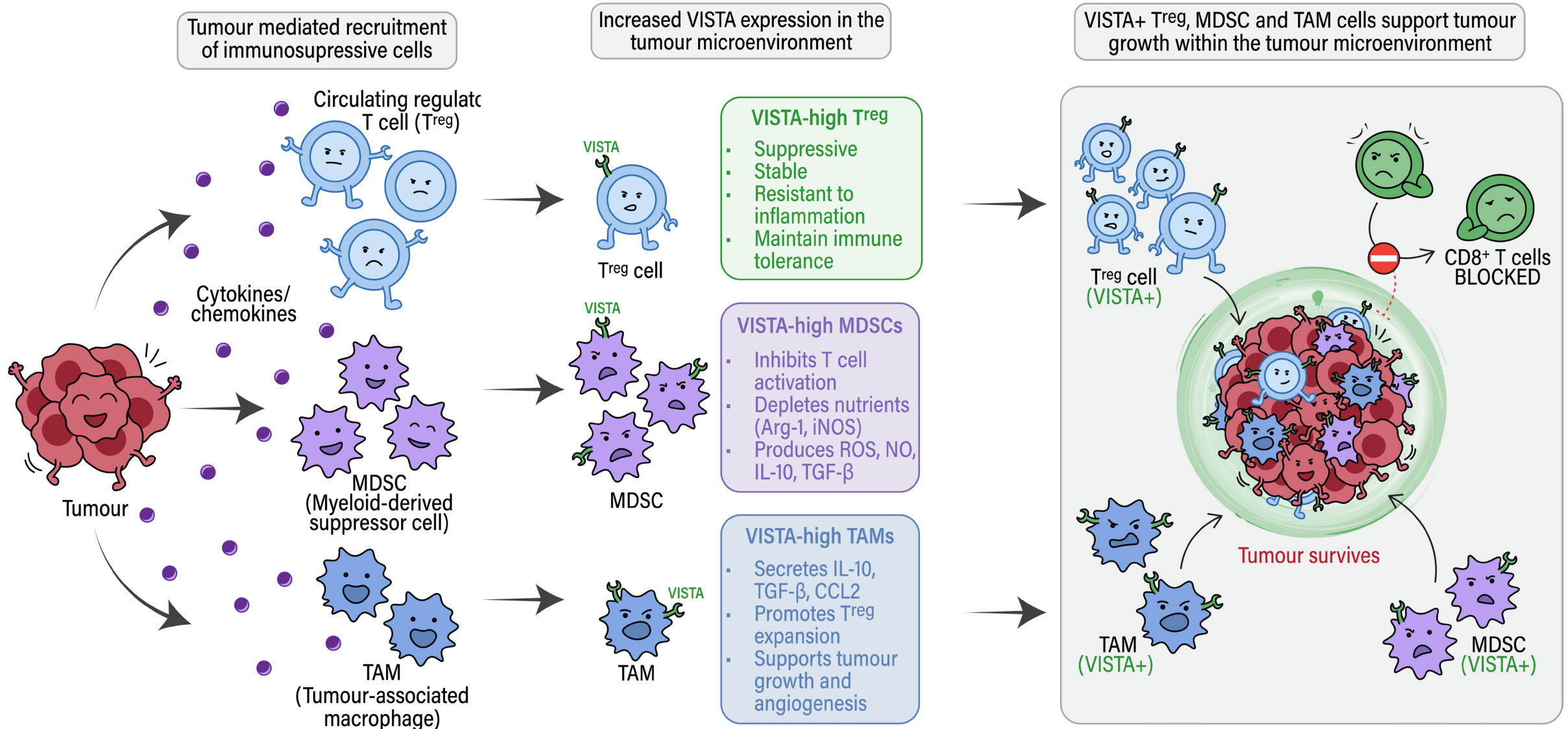


Associated with poor survival



1. <https://doi.org/10.1016/j.esmoop.2024.102942>; 2. <https://doi.org/10.1038/s41598-021-00835-0>

Targeting VISTA is expected to enhance immune cell mediated tumour killing¹

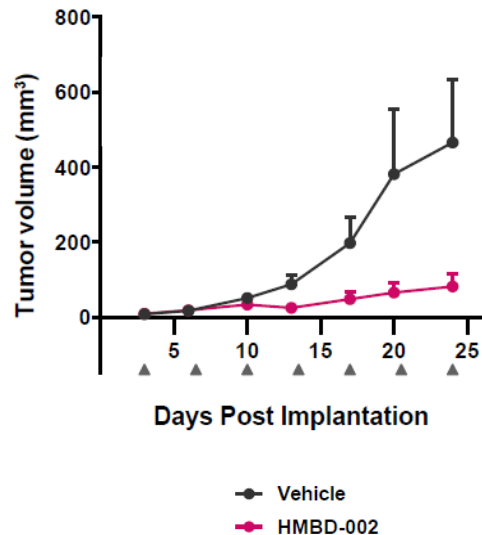


¹ VISTA mechanism of action is still being fully elucidated; iNOS – Inducible Nitric Oxide Synthase; ROS – Reactive Oxygen Species; NO – Nitric Oxide

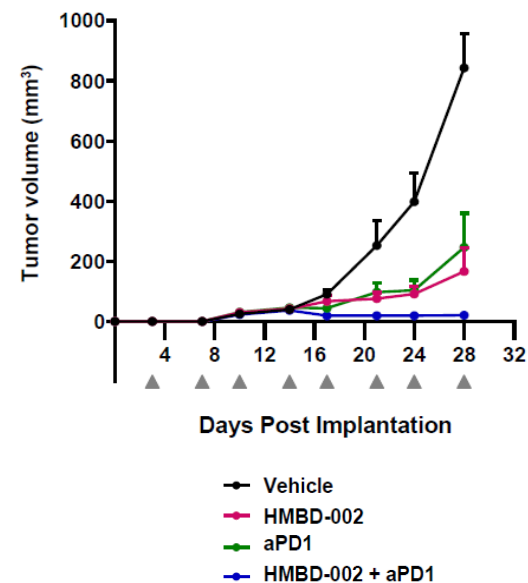
Targeting VISTA enhances the activity of other anti-tumour strategies



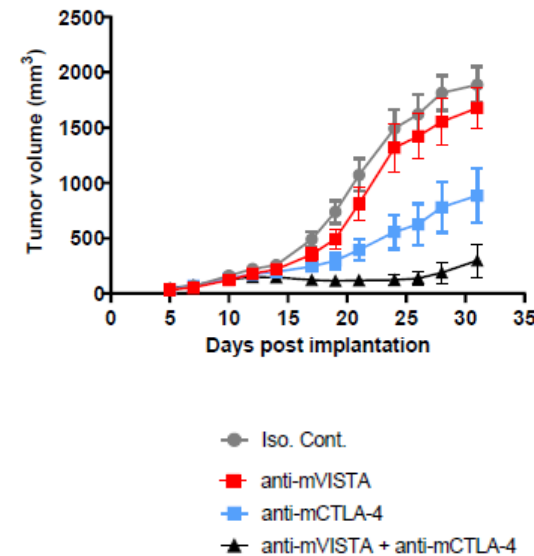
CT26 (colon cancer) – Monotherapy¹



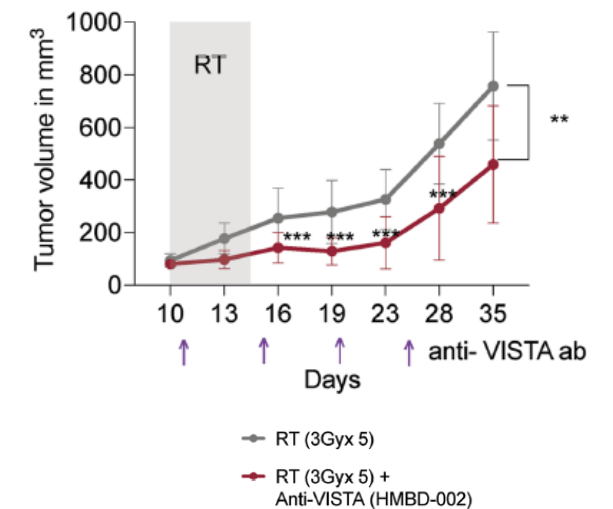
CT26 (colon cancer) – α VISTA + α PD-1²



CT26 (colon cancer) – α VISTA + α CTLA-4³



MOC2 (oral carcinoma) – α VISTA + Radiotherapy⁴

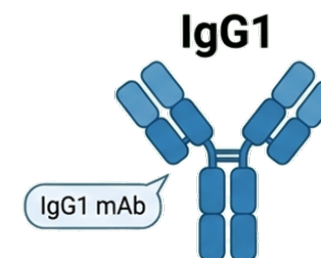


RT – Radiotherapy; 1. <https://jitc.bmj.com/content/10/2/e003382>; 2. https://jitc.bmj.com/content/10/Suppl_2/A558; 3. <https://doi.org/10.3389/fimmu.2023.1311658>; 4. [10.1016/j.celrep.2025.115893](https://doi.org/10.1016/j.celrep.2025.115893)

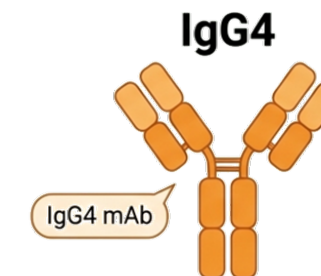
HMBD-002 is arguably the most advanced VISTA antibody in clinical development, and likely the only active member of the IgG4 class



Program	HMBD-002	TBS-2025	CI-8993	JNJ-61610588	W0180
Company					
Isotype	IgG4	Optimised IgG1	IgG1	IgG1	IgG1
Stage	Phase 1 Complete	Phase 1 Phase 1b for AML	Phase 1/2 Terminated	Phase 1 Terminated	Phase 1 Terminated
Exposure to Date	48 patients	41 patients	64 patients	12 patients	33 patients
Combination Data Available	Monotherapy & Combination with Pembrolizumab	Monotherapy & combination with Pembrolizumab	Monotherapy & Combination with Cemiplimab	Monotherapy	Monotherapy & Combination with Pembrolizumab
Status	Ongoing	Ongoing	Discontinued	Discontinued	Discontinued
Notes	Starting an IST with Vanderbilt Health for AML patients	Commencing phase 1b trial in AML in CY 2026	pH-sensitive Antibody		



- ✓ Active Cell Killing (ADCC)
- ✓ Strong Effector Function
- ✗ Cytokine Release Syndrome (CRS)
- ✗ Safety Issues



- ✓ Signaling Blockade
- ✓ Minimal Cell Killing
- ✓ Less CRS
- ✓ Better Safety Profile
- ✗ Effector Recruitment

HMBD-002

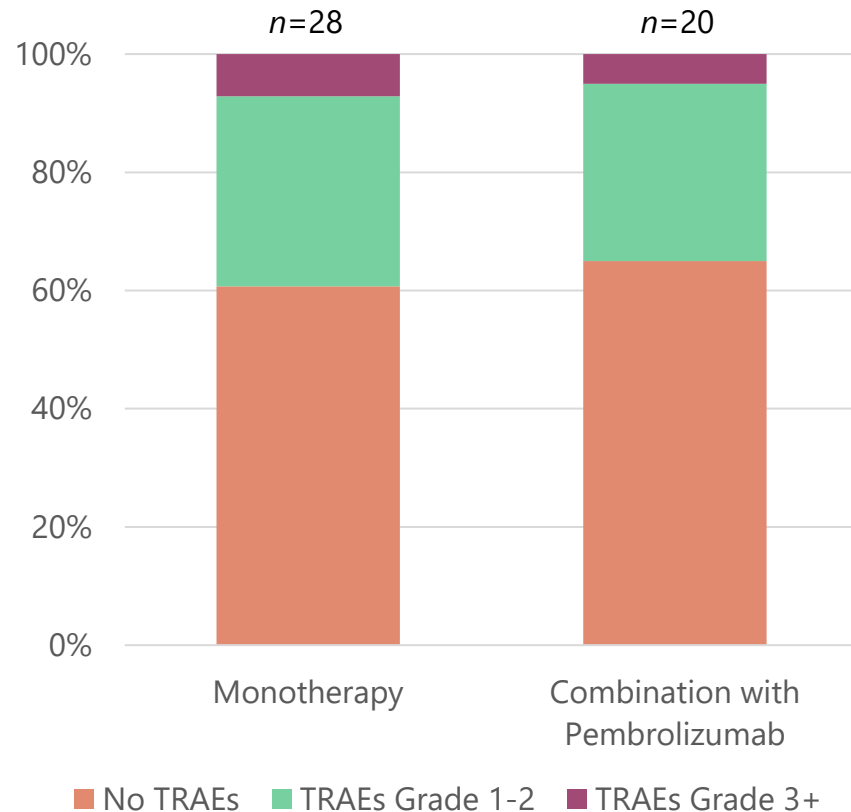
Development to date



Phase 1 data revealed that HMBD-002 has a compelling safety profile



Few patients with TRAEs at Grade ≥ 3 ;
no incremental toxicity in combination



- Clinical experience comprises 48 patients receiving doses up to 1,400mg QW, for up to ~1 year, as monotherapy or in combination
- Most common TRAEs were infusion reactions, gastrointestinal disorders (e.g. nausea, diarrhea), fatigue, and rash
- 1 dose-limiting toxicity (DLT) observed at 360mg dose: a patient with grade 3 hepatitis which resolved with corticosteroids
- Only one treatment-related discontinuation: a combination therapy patient with pneumonitis (a common side effect of PD-1 inhibition)
- **No cases of cytokine release syndrome (CRS) seen in monotherapy or combination therapy cohorts**

Key Takeaways

- HMBD-002 safety profile is favourable for an anti-cancer therapeutic.
- Only few patients experienced treatment-related adverse events $\geq$ grade 3 in severity.
- Less than half of patients in the study experienced any treatment-related adverse events at all.
- HMBD-002 and pembrolizumab combination appeared to cause no additional toxicity or potentiation of anti-PD1 toxicity.

Source: Unpublished data; TRAE – Treatment Related Adverse Event, QW – Once Weekly; DLT – Dose Limiting Toxicity; CRS – cytokine release syndrome

Patients treated with HMBD-002 showed evidence of clinical benefit, providing a positive signal of potential future efficacy



Patient	Diagnosis	Time Since Diagnosis	Prior Lines of Therapy	Treatment Group	Maximum Tumour Reduction	Time on Therapy
27M	Metastatic dedifferentiated liposarcoma	5.1 years	3	360mg Monotherapy	↓ 17.2%	53 weeks
67F	Metastatic triple-negative breast cancer	2.8 years	3	360mg Monotherapy	↓ 6.2%	26 weeks
49M	Metastatic non-small-cell lung cancer	2.5 years	5	360mg Combination	↓ 1.1%	30 weeks
45F	Metastatic triple-negative breast cancer	2.7 years	5	720mg Combination	↓ 26.9%	5 weeks
59M	Metastatic squamous cell carcinoma of the head & neck	2.9 years	4	720mg Combination	↓ 13.2%	15 weeks

Patients in the study were very late-stage and heavily pre-treated

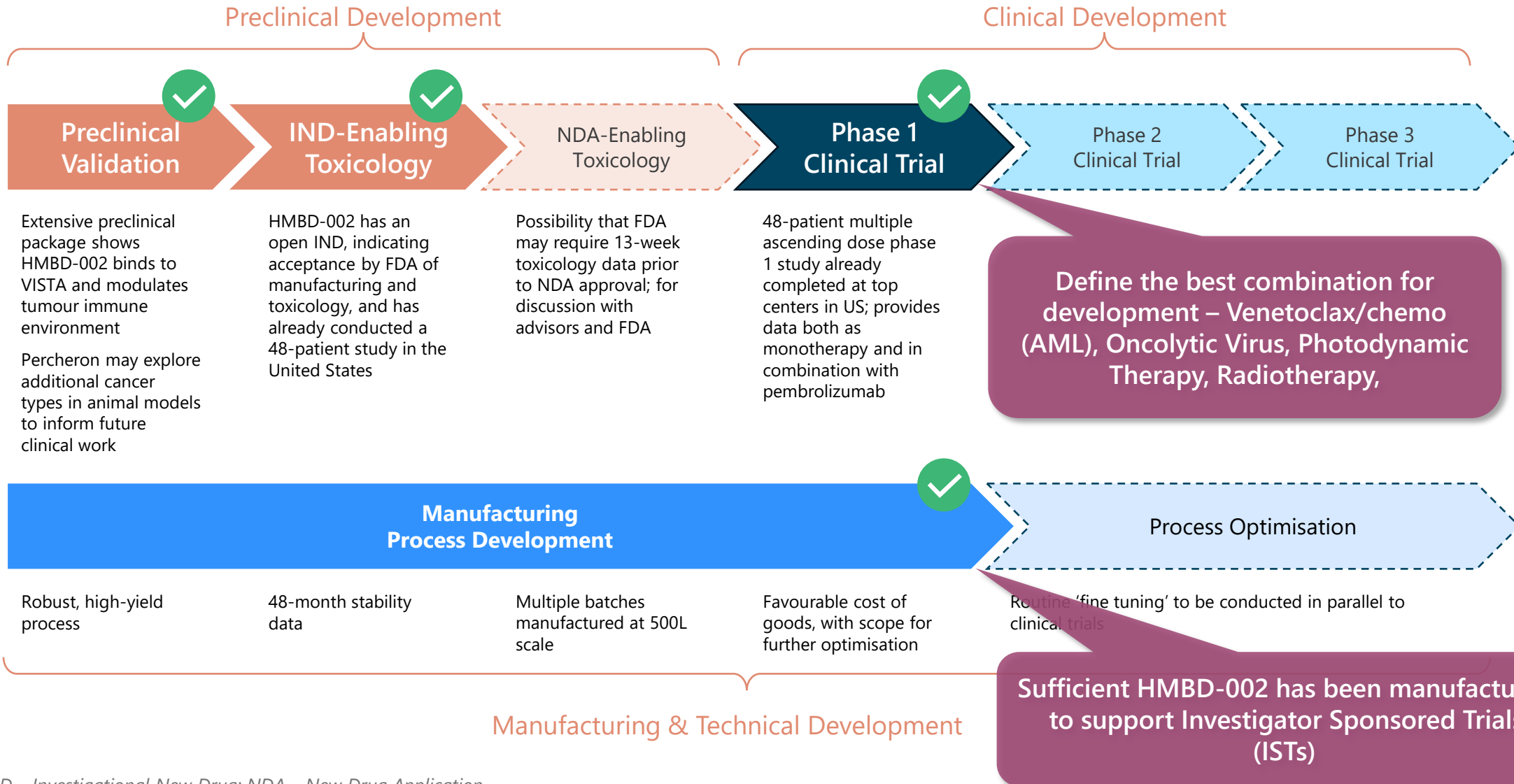
Tumour shrinkage and prolonged periods of stable disease are positive markers of potential future efficacy

HMBD-002 Development Strategy





HMBD-002 has excellent potential to enhance the activity of a range of anti-cancer therapeutics

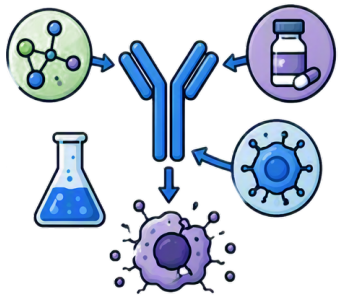


Strategies to determine the best phase 2 study to test HMBD-002



Initiate Investigator Sponsored Trials (IST) to find best combination

- Identify leading cancer centres interested in combining HMBD-002 with standard of care treatments.
- Support completion of clinical studies and assess the efficacy of the combination
- If the data supports it, commence phase 2 study
- i.e. Vanderbilt – HMBD-002 + venetoclax + chemo for AML



Complete preclinical combination studies to define best combination approaches

- Identify companies with promising anti-cancer therapies that are expected to be synergistic with HMBD-002
- Enter into a research collaboration and perform preclinical studies to assess the synergistic benefit
- i.e. oncolytic viruses, kinase inhibitors, RAS inhibitors, photodynamic therapy, cell therapy



Review the landscape for new technologies to in-license

- Engage with research institutes and universities across the globe to screen for technologies expected to be synergistic with HMBD-002
- Enter into Option Agreements to assess the activity of the combination
- If promising, execute license agreement
- Focus on cytotoxic therapies and biologics

What are the best combination strategies for HMBD-002



Checkpoint Inhibitors (CPIs)

- i.e. PD-1/PD-L1, CTLA-4
- VISTA blocks T cell activation, PD-1 promotes T cell exhaustion
- VISTA upregulation is implicated in resistance to PD-1 and CTLA-4 therapy
- Preclinical data supports approach for combination CPIs^{1,2}
- Opens up commercial opportunities with large pharma



Radiotherapy (RT)

- Causes immunogenic cell death and increases antigen release
- Induces suppressive myeloid cell populations, where a VISTA-targeting mAb should be most effective³
- Preclinical data supports the approach⁴
- Cost effective approach given RT is widely adopted

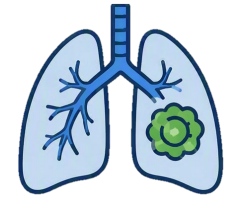
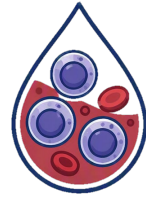


Immunogenic Cell Death Modalities

- i.e. Oncolytic viruses, some antibody drug conjugates, photodynamic therapy
- Therapeutics that are directedly cytotoxic to tumour cells, and also have the potential to illicit an anti-tumour response through the patient's immune system

1. https://jitc.bmj.com/content/10/Suppl_2/A558; 2. <https://doi.org/10.3389/fimmu.2023.1311658>; 3. [10.1016/j.ijrobp.2023.11.05](https://doi.org/10.1016/j.ijrobp.2023.11.05) 4. [10.1016/j.celrep.2025.115893](https://doi.org/10.1016/j.celrep.2025.115893)

What are the most promising tumour types to target



	Acute myeloid leukemia (AML) ¹	Triple negative breast cancer (TNBC) ²	Late-stage glioma ³	Non-small cell lung carcinoma (NSCLC) ^{4,5}
VISTA expression on immunosuppressive cells in the TME	Yes, highly expressed on myeloid cells, including blast cells	Yes, expressed on infiltrating macrophages and neutrophils	Yes, and was the highest checkpoint gene expressed	Yes, expressed on myeloid cells and also expressed on some lymphocytes
Prognostic indicator	Yes, increased expression correlates with disease progression and reduced survival	No, but blocking VISTA reverts immunosuppressive macrophages, increases T cell infiltration and enhances an anti-tumour response	Yes, increased VISTA transcripts was associated with poor prognosis of glioma patients	Varied, but data suggests that the VISTA pathway may contribute to adaptive resistance mechanisms in the tumour microenvironment
Market Size (US p.a.)	20,000	30,000-40,000	14,000-20,000	190,000-200,000

1. TK Kim et al. (2024) *J Clin Invest.* 134(3):e164325; 2. <https://doi.org/10.1038/s41416-025-03013-5>; 3. [10.1038/s41598-021-00835-0](https://doi.org/10.1038/s41598-021-00835-0); 4. [10.17305/bjbm.2021.6531](https://doi.org/10.17305/bjbm.2021.6531); 5. [10.1158/1078-0432.CCR-17-2542](https://doi.org/10.1158/1078-0432.CCR-17-2542)

Investigator Sponsored Trial (IST) for AML

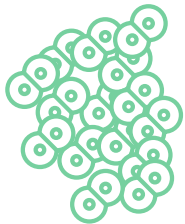
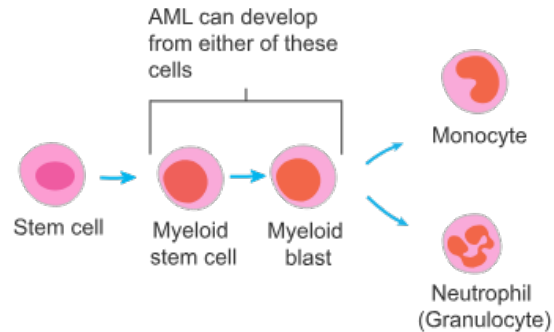
Acute myeloid leukaemia (AML) is the most common form of leukaemia in adults



Pathology



AML is a cancer of the progenitor cells which ordinarily give rise to myeloid immune cells in the blood

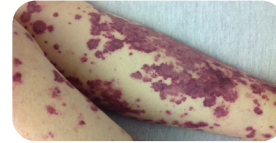


The cancerous cells multiply in an uncontrolled way and 'crowd out' healthy cells in bone marrow and blood

Clinical Picture



Common symptoms of AML include fatigue, bruising, bleeding, and infection



Untreated, AML is typically fatal within months



MALES 60+

Approximately 20,000 patients are diagnosed annually in the US; most common in males over 60 years of age



10% – 35%

Five-year survival is estimated at 10% - 35%, depending mainly on the general health of the patient at diagnosis

Standard of Care



First-line treatment is with intensive chemotherapy.

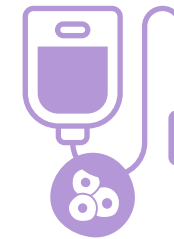


50% RELAPSE

Approximately 60-80% of patients are initially responsive. However, roughly half of those will eventually relapse.



In patients who relapse, or who are unfit for intensive chemotherapy, a range of other agents are used, including venetoclax (discovered at the Walter & Eliza Hill Institute in Melbourne, Australia)

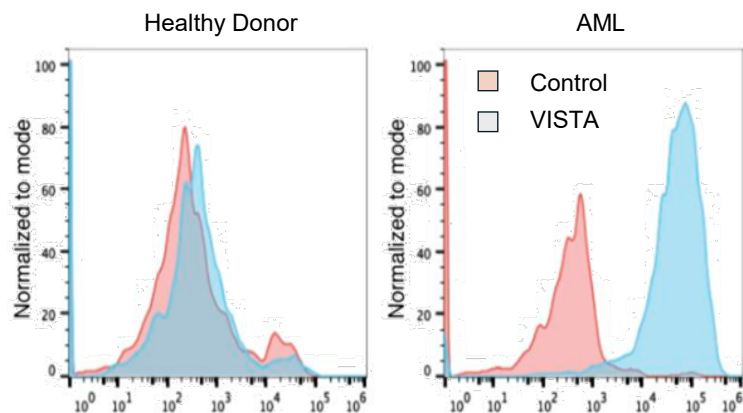


Stem cell transplants are increasingly used in patients who are not suitable for pharmacological therapies

VISTA is a highly relevant and attractive target for treatment for AML¹

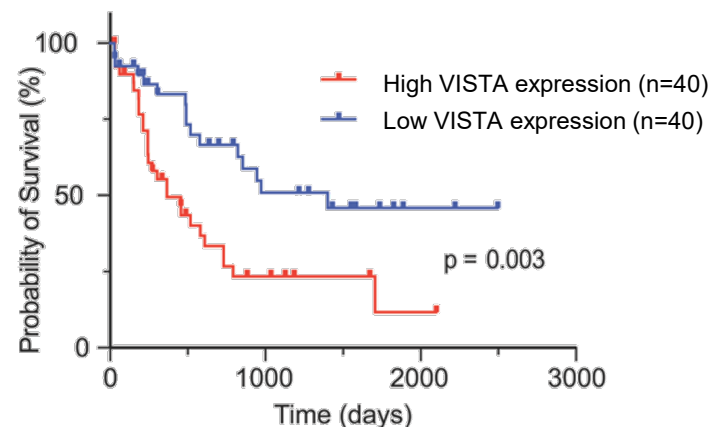


VISTA is highly expressed on AML blasts



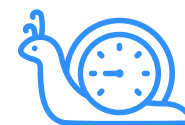
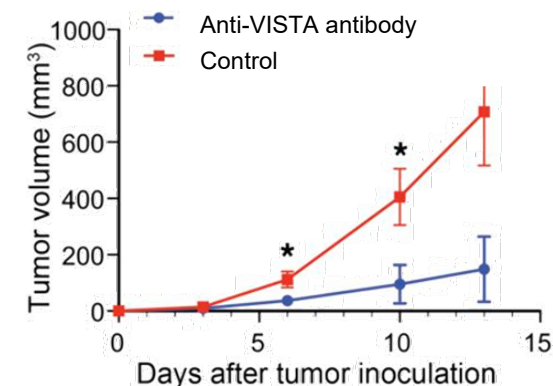
Tumour cells express much higher levels of VISTA than those drawn from healthy donors

VISTA expression correlates with survival in AML models



Tumours with high VISTA expression have a worse prognosis than those with low VISTA expression

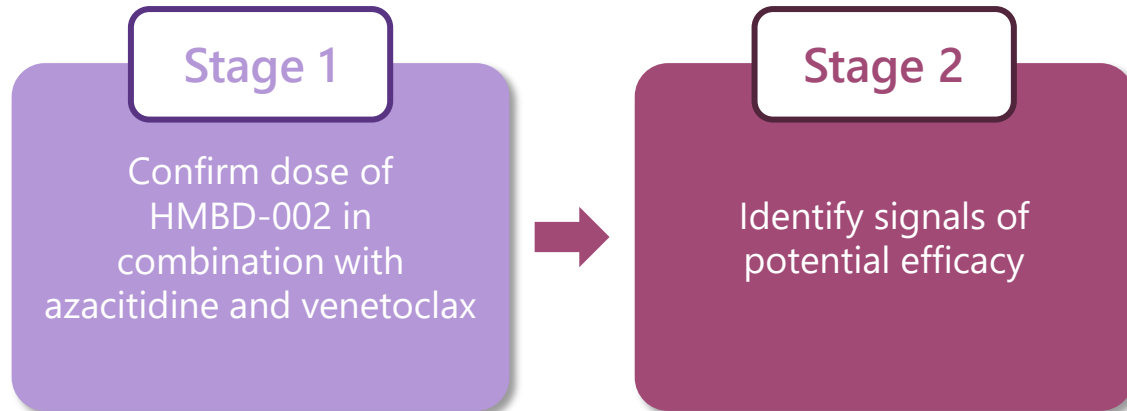
Blocking VISTA improves clinical outcome in AML models



An anti-VISTA antibody significantly slows tumour growth in an animal model of AML



The study is planned to be conducted in two stages, under an open label design¹



Venetoclax

- Inhibits Bcl-2 protein, implicated in several blood cancers
- Discovered at Walter & Eliza Hill Institute (Melbourne, VIC)
- Global sales ~US\$ 1.4 billion (2025)

Expected recruitment: 29 – 38 patients

Patient population: initially relapsed / refractory high-risk AML / MDS patients, with potential extension to newly-diagnosed patients in Stage 2



Azacitidine

- Chemotherapy agent that impedes DNA synthesis in tumours

1. Final clinical study design will incorporate FDA feedback

Percheron is focused on moving its pipeline forward expeditiously, with potential for rich, value-generating news flow over FY2027



CY2026

Release of completed HMBD-002 drug substance (DS)	2H CY2026	✓
New CEO and MD commenced	2H CY2026	✓
Release of completed HMBD-002 drug product (DP) for use in clinical trials	2H CY2026	
IND Study May Proceed for Vanderbilt study	2H CY2026	
Potential further clinical trial(s) in additional patient populations	2H CY2026	

CY2027

First patient recruited into the Vanderbilt AML study	1H CY2027	
Initial data from AML study	CY2027	
Formation of new partnerships to test combinations with HMBD-002	CY2027	
Assessment of additional technologies to in-license for combination with HMBD-002	CY2027	

Note: All timelines are indicative and subject to ongoing review

Thank you

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THERAPEUTICS



Cold Tumours	Tumours with few immune cells in the tumour microenvironment. They are typically relatively resistant to immuno-oncology therapies. Some cancers, such as ovarian cancer and brain cancer, are usually considered to be generally 'cold' tumours. By implicit contrast, a 'hot tumour' or inflammatory tumour is one with a large and active population of immune cells in the tumour microenvironment.	Overall Survival (OS)	A measurement of the average time to death from any cause in a clinical trial. When treated patients are compared to a control group, an improvement in OS is considered the 'gold standard' for FDA approval.
Expression (of an immune checkpoint)	In the context of immuno-oncology, a tumour with, on average, a lot of a given immune checkpoint on the surface of its cells is said to highly express that immune checkpoint. For example, a tumour with high PD-1 expression has a lot of PD-1 on the surface of its constituent cancer cells.	Progression-Free Survival (PFS)	A measurement of the average time to disease progression (20% increase in size of tumour) or death in a clinical trial. When treated patients are compared to a control group, PFS is often a swifter and more sensitive measure of efficacy than OS, and can often provide a basis for approval.
Immune Checkpoint	A control mechanism that allows cells, including tumour cells, to inhibit the local activity of the immune system. Analogous to the brake pedal on a car. Examples of immune checkpoints include PD-1, CTLA-4, LAG3, and VISTA.	RECIST / Overall Response Rate (ORR)	A standardised way of measuring the early activity of cancer therapies. A complete response (CR) describes 100% reduction in a specific lesion on MRI or CT scan. A partial response (PR) means a 30% or greater reduction in size. Stable disease (SD) means no material change in the size of the tumour. Progressive disease (PD) occurs when a lesion grows by more than 20%. ORR is traditionally a useful way to assess early-stage cancer therapies but rarely provides a basis for FDA approval and is often less sensitive for targeted therapies such as immune checkpoint inhibitors.
Immune Checkpoint Inhibitor (ICI)	A drug which inhibits an immune checkpoint, releasing the 'brake' on the local activity of the immune system. Examples of immune checkpoint inhibitors include Keytruda(R) (pembrolizumab), Opdivo(R) (nivolumab), Yervoy(R) (ipilimumab), and Tecentriq(R) (atezolizumab).	Resistance	In oncology, a tumour is said to be resistant to a given therapy if it does not meaningfully respond to that therapy. Resistance may be primary, if the tumour is unresponsive from the outset, or secondary / acquired if the tumour becomes unresponsive after a period of initially successful treatment.
Ligand	Generally, a molecule which connects to another molecule to bring about a biological effect. In the context of immuno-oncology, a ligand is typically the molecule on the immune cell to which the immune checkpoint binds (e.g. PD-1 to PDL-1, CTLA-4 to CD80, LAG3 to Gal3, and VISTA to VSIG-3).	T-Cell / T-Lymphocyte	T-cells are one type of cells involved in the immune system. There are many subtypes, and their effects range from control and coordination ('helper T-cells') to direct attack of infectious organisms and cancer cells ('cytotoxic T-cells').
Monoclonal Antibody	A class of genetically engineered drugs which utilise the structure of natural human antibodies to generate new molecules which can either block signalling or attack cells. Approximately 40% of cancer drugs are monoclonal antibodies. Monoclonal antibodies are manufactured in living cells under carefully controlled laboratory conditions. They are more challenging to manufacture than 'small molecule' (chemical) drugs but generally simpler than cell therapies.	Tumour Microenvironment (TME)	The TME includes the tumour itself, along with the surrounding tissues, including structural support ('stroma'), small blood vessels, etc. The TME is typically also infiltrated with immune cells. When considering the immune cells specifically, it is sometimes referred to as the tumour immune microenvironment (TIME).
Myeloid Cells	Myeloid cells are one type of cells involved in the immune system. They exert a range of effects, some of which can be immunosuppressive.	VISTA	V-domain immunoglobulin suppressor of T-cell activation. A novel immune checkpoint, first identified in the 2010s, and the target of HMBD-002.

Deal References



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