NAYA THERAPEUTICS

Pioneering the Next Generation of Cancer Therapies

Forward Looking Statement

This presentation contains forward-looking statements, including about our strategy, R&D plans, clinical and regulatory timelines, manufacturing, intellectual property, collaborations, market opportunities, and financial outlook. These statements are based on current assumptions and involve risks and uncertainties that could cause actual results to differ materially, including those related to drug development, regulatory approval, manufacturing and supply (including At-211), IP, competition, financing, and macro factors. We undertake no obligation to update these statements. This presentation is not an offer to sell or a solicitation of an offer to buy any securities.

NAYA Aims to Advance Outcomes in HCC & Multiple Myeloma Towards Cure

Despite Major Progress, Unmet Needs Remain for Deep, Durable Responses in Many Indications

Hepatocellular Carcinoma

75-85%

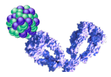
Non-Responders to Checkpoint Inhibitors

Multiple Myeloma

30-50%

of Responders to Chemo + Immunotherapy are MRD Positive

NAYA's Leadership In Transformational Astatine-211 (^{211}At) Targeted Alpha Therapies & Synergistic Immune-Cell-Engaging Bifunctional Antibodies with Early Clinical Value Inflection



NAYA Positioned as ^{211}At Leader with Execution Edge in Decentralized Supply Chain

First-in-Class Clinical Pipeline Aims to Address Major Unmet Needs in Minimal Residual Disease (MRD) and Micrometastasis.

Synergistic, First-in-Class, Immune-Cell-Engaging Bifunctional Antibodies Drive Deep & Durable Responses

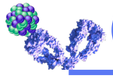
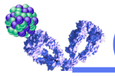
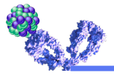
Positioned for Early Pharma Partnering With 2026-27 Clinical Value Inflection

De-Risked Clinical Pipeline With Validated GPC3 & CD38 Targets, Strong Preclinical Data, Competitive Target Product Profile

Ability to Accelerate Development Through US & European Strategic Hubs and China Access for Early Clinical Trials

Radiopharma & Bifunctional Antibodies in a Prime M&A/ Partnering Window for Early Clinical Stage Companies.

NAYA's Best-in-Class ^{211}At Targeted Alpha Therapy & Bifunctional Antibody Pipeline

NY-738 *CD38-Targeted ^{211}At Alpha Therapy for Minimal Residual Disease in Multiple Myeloma*	 *(1st gen)* **PHASE I/II**  *(2nd gen)* **PRE-CLINICAL**	*IND Approved, Initial Clinical Data H1'26* *IND/Phase 1 H2'26*
NY-703 *GPC3-Targeted ^{211}At Alpha Therapy for Residual Disease & Metastasis in Hepatocellular Carcinoma (HCC)*	 **PRE-CLINICAL**	*China IIT/ Initial Clinical Data H2 '26* *US/EU Phase 1/ 2 Initiation H1 '27*
NY-303 *GPC3-NKp46 Bifunctional Antibody for Non-Responders to Immunotherapy in Hepatocellular Carcinoma (HCC)*	 **PHASE I/II**	*Phase 1/ 2a Initiation H1'26* *Clinical Data H1'27*
NY-338 *CD38-NKp46 Bifunctional Antibody As Alternative to T-Cell Engagers in Multiple Myeloma & Autoimmune*	 **PRE-CLINICAL**	*Out-Licensing /Co-Development in H1 '26*

Optionality to Scale the Both Modalities to Other Validated Targets/Indications

NAYA To Establish France-Based European Research & Manufacturing Hub

Leverages French Excellence & Leadership in Radiopharmaceuticals & Oncology

NAYA Establishing Itself Globally with US Headquarters & French Hub,
Harnessing Leading Oncology Centers for Early Translational & Clinical Proof of Concept + GMP Manufacturing

Decentralized GMP Manufacturing Sites *to Support Clinical Trials in France & Europe*

Pipeline Development *for Alpha Therapies & Multispecific Antibodies*

Strategic Partnerships *With Leading Academic Institutions*

- ***Gustave Roussy Cancer Center****: #1 in Europe, #4 Globally, Alpha Therapy & Oncology Clinical Trial Powerhouse*
- ***Nantes University/ INSERM:*** *Pioneer & Early European Leader in Radiopharm (Chemistry, Manufacturing, Clinical Trials)*
- ***Additional Partnerships*** *in France & Europe as Clinical Development Accelerates*

université PARIS-SACLAY · GUSTAVE ROUSSY CANCER CAMPUS GRAND PARIS · Inserm · Nantes Université

Access to Non-Dilutive Capital & European Biotech Investors *to Support US Co-Lead*

NAYA To Leverage China Access to Accelerate Early Development and Clinical Data

Collaboration with Leading CDMO/CRO to Maintain Full Ownership & BD Upside

ALPHA NUCLIDE

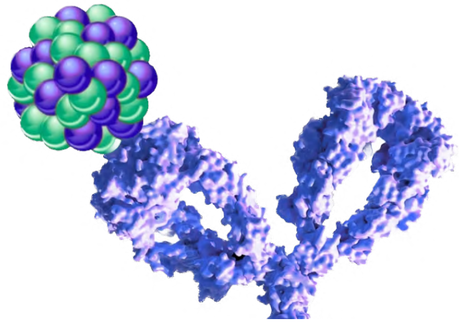
Tigermed

Alpha Nuclide Partnership Unlocks Access to China Market

- **Co-Founded by Yutian Feng, PhD** (Duke University), Leading Astatine-211 & Radiopharmaceutical Key Opinion Leader
- **Shanghai Production Center for China Clinical R&D** Ready for Operations
  - Houses TR-Alpha Cyclotron (First Specialized for Max. ^{211}At Production)
- Planning for **Network of Production Centers** for Full China Market Access

Tigermed CRO Collaboration Supports Early Clinical Trials in China

- **#1 Clinical CRO in China** with Global Footprint & Big Pharma Validation
- **Large Local HCC Population** (Over 200,000 New Cases Annually)
- **Opportunity for Investigator-Initiated Clinical Trials** in 2026
- **Option to Scale Patient Recruitment** as Part of Global Development Plan



Astatine-211 (^{211}At) Targeted Alpha Therapies

The Transformative Potential of Targeted Alpha Therapies

TATs Deposit Massive Amounts of Energy with Scalpel-Like Precision

<u>High-Potency = *Maximum Damage to Tumor Targets*</u>

A single alpha particle can be enough to kill a cancer cell, while it take hundreds of hits from a beta particle to do so.

<u>Short-Range = *Minimal Damage to Surrounding Healthy Tissue*</u>

Alpha particles deposit their energy within a focused 2-3 cell diameter (50-100 micrometers vs. several mm for betas), enabling highly-localized killing

<u>Targeting Vectors = *Increased Precision & Specificity*</u>

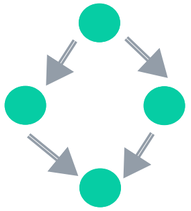
TATs combine radioisotopes with targeting vectors (such as an antibody or peptide) to help guide them towards specific cancer cells

This combination of properties makes TATs particularly well-suited to improve clinical outcomes for patients at risk of residual & metastatic cancer.

Astatine-211: The Clean, Next-Generation "Goldilocks" Emitter

^{211}At's Absence of Secondary Alpha Decay Radioactivity is Key in Maximizing Its Cancer Killing Energy & Efficiency

Short Half-Life (7.2h) → Minimal Unwanted Exposure to Radiation



Clean Decay Chain *(No Secondary Emissions)* → Minimal Off-Target Toxicity

Chelatorless Covalent Chemistry → Simplified Drug Design & Conjugation

Production from Naturally-Abundant Bismuth-209 → Reliable & Highly-Scalable Supply

Unlocking ^{211}At's Availability: The Key to Its Therapeutic Breakthrough

NAYA Partnering with Key Players to Establish Leading Decentralized ^{211}At Global Supply Network

"The perspective among many researchers and clinicians is that ^{211}At would be the most important α-particle emitting radionuclide if it were widely available. "

Prof. Michael R. Zalutsky

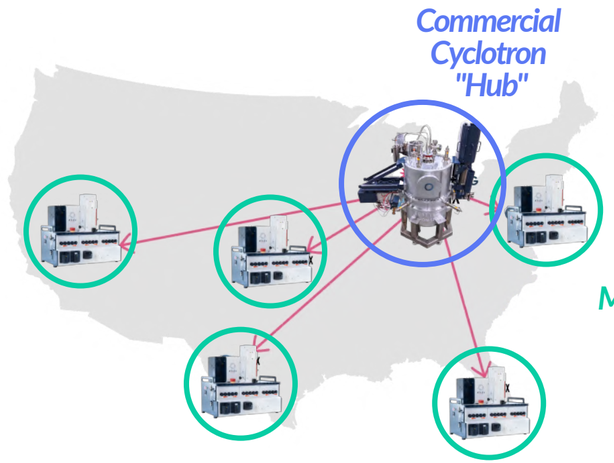
Department of Radiology, Duke University Medical Center

- ^{211}At's unique properties make it a highly-attractive alpha emitter, but supply has until now been limited to academic centers
- ^{211}At's short half-life - a key therapeutic advantage - makes centralized global production a challenge
- A decentralized, hub & spoke supply chain overcomes this hurdle
- NAYA is quickly establishing partnerships with both Astatine-211 & therapeutic-dose production sites in major metropolitan areas across the US, Europe, and Asia to ensure reliable patient access to ^{211}At therapies
- Early-mover advantage is key: securing preferred supply-chain access creates barrier to entry for followers

https://pmc.ncbi.nlm.nih.gov/articles/PMC3503149/

NAYA's Decentralized Hub & Spoke Supply Chain Unlocks Global Access to ^{211}At Therapeutics

Illustrative US ^{211}At Supply Network



~ 4-6 cyclotrons, 10-20 manufacturing sites needed for full US commercialization

NAYA Partnering with Commercial Cyclotron Companies for Reliable Global ^{211}At Supply

IONETIX

ALPHA NUCLIDE

NAYA Establishing Decentralized Drug Manufacturing Sites Using Breakthrough Atley C100 Machine

NAYA THERAPEUTICS

ATLEY SOLUTIONS

Current Supply Chain

(Specialized Academic Centers)

On-Site Cyclotron

On-Site Radiopharmacy

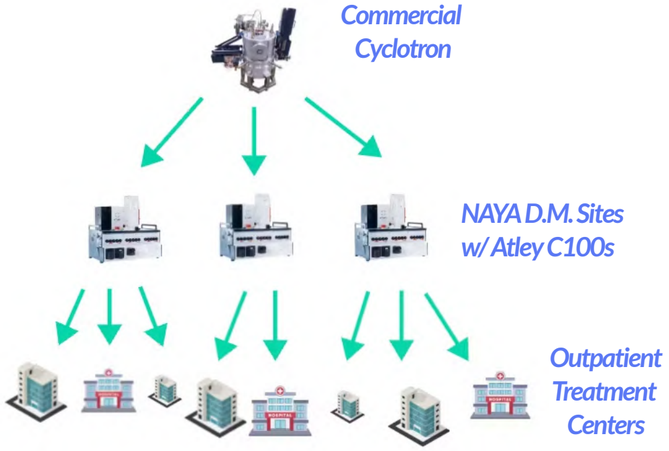
On-Site Administration

1 Cyclotron Supplies
1 Treatment Center

VS.

Decentralized Supply Chain

(Hub & Spoke Network)



1 Cyclotron Supplies
Efficient, Scalable Network of Treatment Centers

Cyclotrons Are Costliest Part of the ^{211}At Supply Chain - Turning Each One Into a "Hub" Allows for Drastically Expanded Reach

Recent Pb-212 Clinical Data Validates Power of Short Half-Life Targeted Alpha Therapies

Pb-212 Clinical Candidates Compare Favorably to Actinium & Lutetium as Best-in-Class TAT

oranomed SANOFI ESMO

61 Patients in Phase 2a

SSTR-targeted alpha therapy using Pb-212 for Neuroendocrine Tumors (GEP-NETs)

60% ORR

in RLT Naive (N=35)

34.6% ORR

in RLT Exposed (N=26)

AdvanCell ESMO

22 Patients in Phase 1b

PSMA-targeted alpha therapy using Pb-212 for Prostate Cancer (mCRPC)

80% PSA50 response

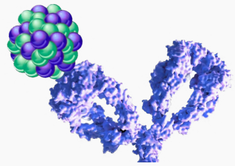
at doses ≥ 160 MBq

100% ORR

in patients with RECIST-measurable lesions, including two CR

NAYA Initiating 2026 Clinical Trials to Establish ^{211}At as Optimal, Next-Generation TAT

NAYA's GPC3 & CD38-Targeting ^{211}At Candidates Aiming for Best-in-Class in HCC & Multiple Myeloma



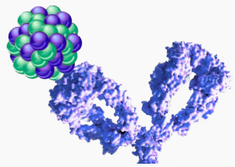
NY-703

GPC3-Targeting ^{211}At Targeted Alpha Therapy

Addressing the 50-75% Recurrence Rate
Post-Surgery/Immunotherapy in HCC

2026 Phase Ib IIT
Shanghai, China (n=20)

2027 Phase IIa
EU/US (n=60)



NY-738

CD38-Targeting ^{211}At Targeted Alpha Therapy

Addressing the Up To 50% MRD+ Rate
at Every Line of Multiple Myeloma Treatment

1st generation
US IND approved, 2026 Clinical Data

2nd generation
(highly-differentiated NAYA/INSERM antibody & linker)
2026 IND

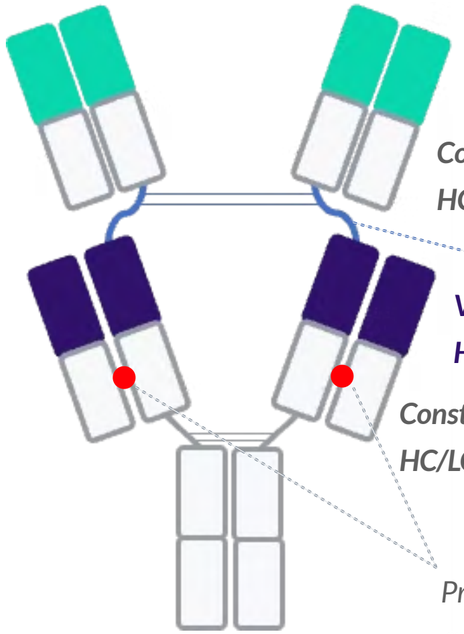
Flex-NK™ Bifunctional Antibodies

NAYA's Plug & Play Bifunctional Antibody Construct Promotes Avidity & Immunological Synapse Effect, Enhancing Precision Tumor Killing

Natural Bivalent Design
allows for binding affinities at levels comparable to native monoclonal antibodies

Distal FAB 1
Binds to Tumor or Vascular Target: GPC3, CD38, VEGF

Proximal FAB 2
Binds to Tumor or Immune-effectors: Nkp46, PD-1



Variable HC/LC 1

Constant HC/LC 1

Variable HC/LC 2

Constant HC/LC 2

Proprietary mutations to avoid mismatching

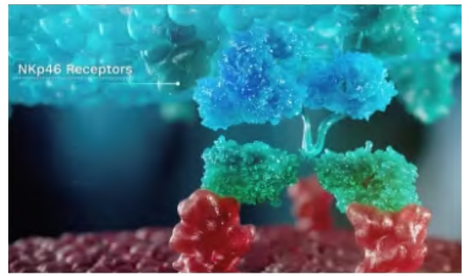
IgG1Fc
Fully active (with CD16-mediated effects) or silenced Fc region, enables longer half-life

Plug & Play of Variable HC/LC
parts enable faster development

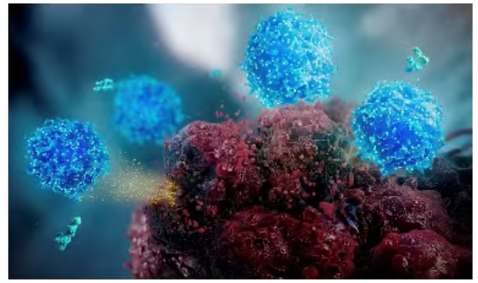
Proprietary FLEX Linkers Enable:
- *Simultaneous binding to targets 1 & 2*
- *Biological synapse in TME*
- *Higher stability due to connecting disulfide bridges*

Validated Manufacturability:
STC Biologics
(Newton, MA)

(1) Engaging Target 1



(2) Engaging Target 2



(3) Destruction of Tumor

Watch Full Video

Flex-NK™ Cell Engagers Show Significant Advantages Over T-Cell Engagers

NKp46 Activation Unlocks Immunotherapy Efficacy Across Tumors & Targets

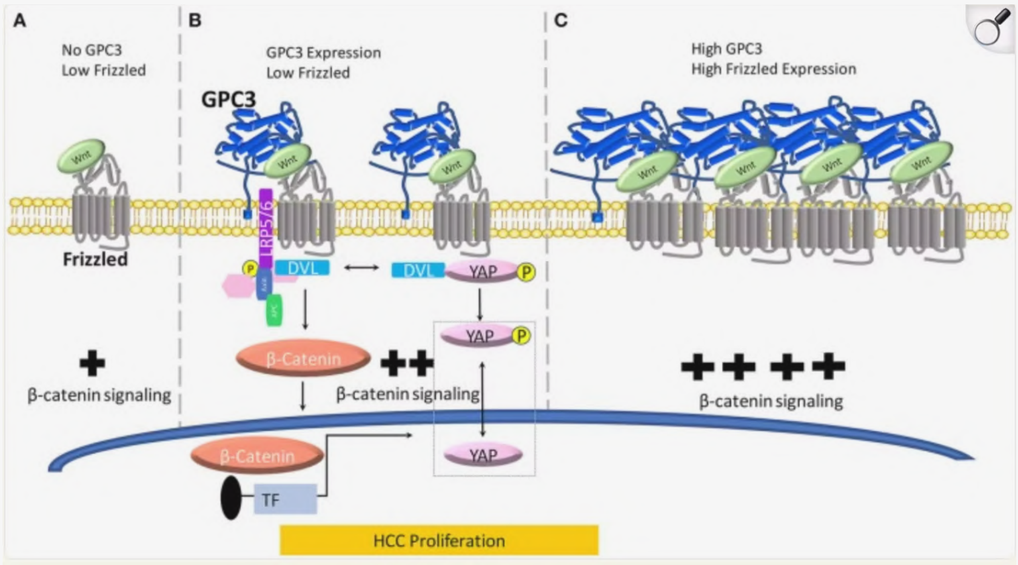
	T-Cell Engagers (TCEs)	Flex-NK™ Engagers (FNKEs)
Mechanism of Action	T-Cell Redirection via CD3	*NK Cell Redirection via NKp46 & CD16,* *Tumor Cell Apoptosis & Serial Killing via NKp46*
Safety (CRS & Neurotoxicity)	High Risk	Minimal Risk
Immune Exhaustion Risk	High T-Cell Exhaustion, *Resulting in Limited TCE Activity*	Low NK Cell Exhaustion, *FNKEs Remain Active even when T-Cells Exhausted*
Resistance via Antigen Loss	Yes	Limited, *Broad NKp46 Activity Across Tumor Types & Targets*
Activity in Solid Tumor TME	Inconsistent	Promising *(Based on NKp46 Expression & Activity)*
Stage of Development	Late Clinical Stage & Commercial	Early Clinical Stage

First-in-Class GPC3/NKp46 Bifunctional Antibody Turns Immune-Cold Tumors Hot Through GPC3, Activates Durable NK Cell Tumor Killing Through NKp46

Synergistic Effect of Targeting Highly Tumor-Specific GPC3 & Best-in-Class Immune Cell Engager NKp46

GPC3 Turns Cold Tumors Hot

Dampens Wnt-β-Catenin Signaling in TME, Overcomes CPI Resistance



NKp46 Drives Deep, Durable Response

Receptor Biology (Not Just Half-Life) Critical to Pharmacodynamic Effect

- ***Activates NK cells*** *for serial killing of tumor cells*
- ***Improves NK cell trafficking to TME***
- ***Induces reversal of NK cell functional exhaustion***
- ***Increases cytotoxicity & compensates for CD16 low-expression*** *through dual-trigger signaling with CD16*
- ***Pharmacodynamic effects*** *last 10+ days from single dose*
- ***Expressed consistently*** *across hematological & solid tumors*
- ***Validated clinical data*** *(IPH6101) supports FDA fast-track*

https://pmc.ncbi.nlm.nih.gov/articles/PMC6688162/

Global Phase I/IIa of NY-303 as HCC Monotherapy for Non-Responders to PD-1+/- VEGF Inhibitors to Evaluate Safety, Response, and Disease Progression

Hadassah Hospital, Jerusalem

Sheba Medical Center, Tel Aviv

Sourasky Medical Center, Tel Aviv

Initiation of Clinical Trials *Cleared to Enroll Patients in Israel (15-20 Patients)*

Global Expansion *to Include Up To 50 Additional Patients in US, EU, and Asia Starting in 2026*

Endpoints *to include safety, pharmacokinetics, activity markers, preliminary clinical efficacy (Overall Response Rate), and time-to-progression (Progression-Free Survival)*

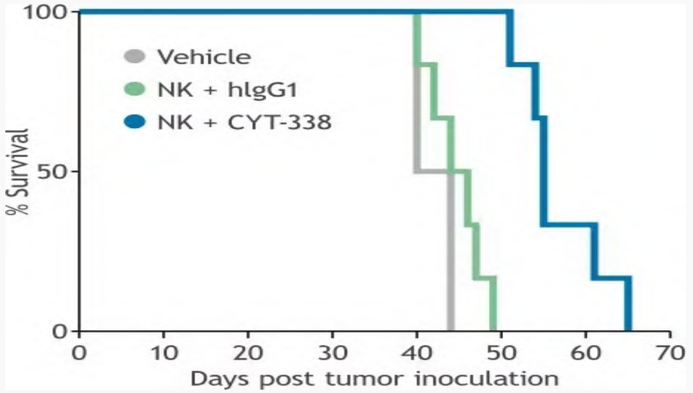
Key Opportunities *for Accelerated Regulatory Pathway, Unlocked Asset Value, Pharma Licensing/M&A with 30+% Overall Response Rate & Improved Progression-Free Survival*

NY-338 Highly-Differentiated from Daratumumab & T-Cell engagers

In Vitro & In Vivo Data Presented at ASH & EHA

- Distinct Epitopes from Daratumumab
- No NK Cell Fratricide Effect, No Effect on Other Immune Subsets
- Reverses NK Cell Dysfunction, Enhancing Killing Power
- No Cytokine Release Syndrome

Overall Survival (OS)



Liang Lin et. al. 2022 *EHA Conference*

NY-338 (CD38/NKp46) + BCMA TCE Competitively Positioned

CD38/NKp46 + BCMA TCE Reduces Both Antigen Escape & T-Cell Exhaustion Without Sacrificing Depth

	CD38/NKp46 +BCMA TCE *(NAYA)*	CD38/BCMA /CD3 *(Abbvie)*	Daratumumab + BCMA TCE *(J&J)*	BCMA TCE Alone *(J&J, Pfizer, Regeneron)*
Durable Efficacy	Very High	Very High	High	Medium/High
Orthogonal Killing (NK+T)	Strong	None	Partial	None
Fratricide/Immune Depletion	Low	Moderate	Significant	Minimal
Immune Cell Exhaustion	Lower	Higher	Moderate/High	Highest
Antigen Escape	Low	Lowest	Moderate	Highest
CRS/Neurotox	Moderate	High/ Very High	High	High

** subject to confirmation in clinical trials*

NY-338 Clinical Development Plan Targeting Initial Clinical Proof of Concept in 2027

2026 Phase 0 Translational Study

Patient samples post-Daratumumab, BCMA TCE, or CAR-T

2027 Phase 1b Monotherapy

Dose escalation with CD38/NKp46 in post-BCMA TCE

2027 Phase 1b Combo with TCE

Lead-in with CD38/NKp46, BCMA TCE Step-Up

2028 Phase 2a Expansion

Single arm combo vs. TCE alone to evaluate depth & durability (MRD negativity, preliminary PFS)

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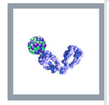
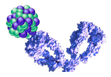
Corporate Considerations

NAYA's Unicorn Potential Supported By Strong Market Valuations of Bifunctional Antibody & Targeted Alpha Radioimmunotherapy Companies

	IGI ICHNOS GLENMARK INNOVATION / abbvie	*CD38/BCMA/CD3 T-Cell Engager Phase I*	Global License by Abbvie for $700M upfront + $1.2B milestones *Phase I Data demonstrates 79% CR in 35 heavily pre-treated patients*
	Merus / Genmab	*EGFR/LGR5 Bispecific Antibody Ongoing Phase III*	Acquired by Genmab for $8B
	BIOMUNEX pharmaceuticals / IPSEN	*GPC3 T-Cell Engager Pre-Clinical*	Global License by Ipsen for $610M
	oranomed / SANOFI	*Targeted Alpha Therapy Phase I*	$300M Investment at $1.9B Post-Money
	AKTIS ONCOLOGY	*Targeted Alpha Therapy Phase I Ongoing*	$1B+ IPO (Post-Money)
	Fusion Pharmaceuticals Inc. / AstraZeneca	*Targeted Alpha Therapy Phase II*	Fusion Acquired by AstraZeneca for $2B + $400M in Milesones
	mariana ONCOLOGY / NOVARTIS	*Targeted Alpha Therapy Pre-Clinical*	Mariana Acquired By Novartis for $1B Upfront + $750M in Milesones, *Prior Novartis Acquisitions of Endocyte & AAA*
	RayzeBio A Bristol Myers Squibb Company / Bristol Myers Squibb	*Targeted Alpha Therapy Phase I (GPC3/Ac-225)*	RayzeBio Acquired by BMS for $4.1B

*** based on Glycomimetics-Crescent post-money merger ratio of 3.1% / 96.9% & Glycomimetics closing price of of $0.25 from 12/31/24*
**** based on company press releases (hyperlinked)*

NAYA's Leadership In Transformational Astatine-211 (^{211}At) Targeted Alpha Therapies & Synergistic Immune-Cell-Engaging Bifunctional Antibodies with Early Clinical Value Inflection



NAYA Positioned as ^{211}At Leader with Execution Edge in Decentralized Supply Chain

First-in-Class Clinical Pipeline Aims to Address Major Unmet Needs in Minimal Residual Disease (MRD) and Micrometastasis.

Synergistic, First-in-Class, Immune-Cell-Engaging Bifunctional Antibodies Drive Deep & Durable Responses

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De-Risked Clinical Pipeline With Validated GPC3 & CD38 Targets, Strong Preclinical Data, Competitive Target Product Profile

Ability to Accelerate Development Through US & European Strategic Hubs and China Access for Early Clinical Trials

Radiopharma & Bifunctional Antibodies in a Prime M&A/ Partnering Window for Early Clinical Stage Companies.

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Appendix

Astatine-211 Shows Significant Advantages Over Actinium-225 & Lead-212

Recent Clinical Data With OranoMed's SSTR-targeting Lead-212 TAT Supports Comparable Efficacy for Short Half-Life Emitters Compared to Actinium-225

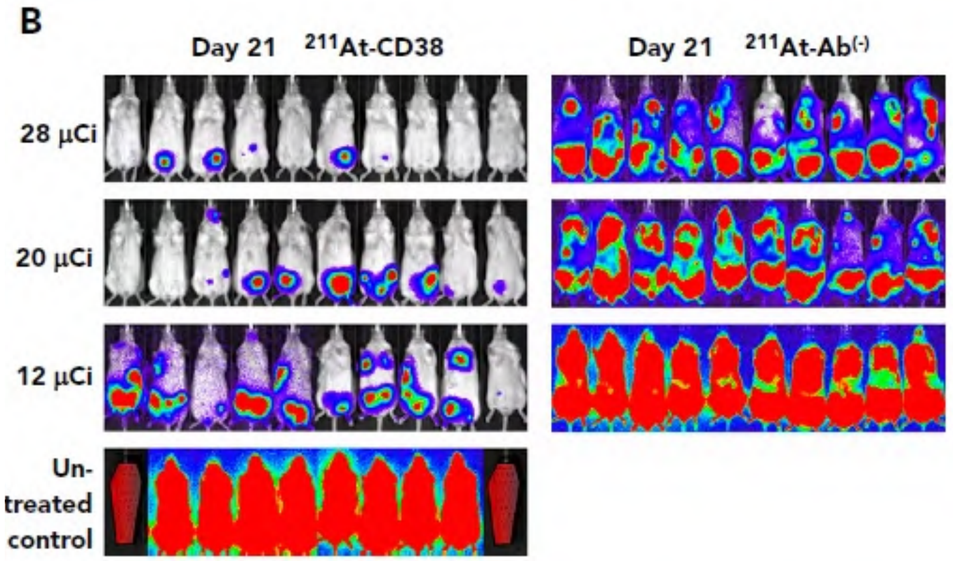
	Actinium-225	Lead-212	**Astatine-211**
Half-Life	9.9 days	10.6h	**7.2h**
Decay	Unfavorable (4 Daughters)	Moderate (2 Daughters)	**Favorable** *(No Daughters)*
Risks	Significant	Average	**Minimal**
Chemistry:	Complex, Bulky	Complex, Bulky	**Chelatorless**
Production Source	Limited Supply	Limited Supply	**Naturally-Abundant** *(209-Bi)*
Cost Effectiveness	Medium	Medium/ High	**High**
Dependable, Scalable Supply	Medium	Medium	**High**
Theranostics/Imaging	Challenging	Yes	**Yes**

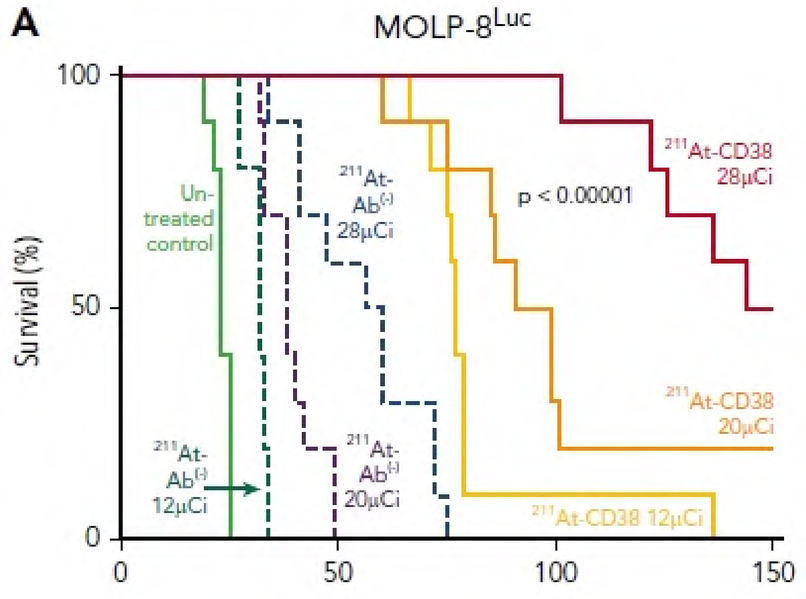
^{211}At-CD38 Therapy Demonstrates In Vivo Efficacy in Disseminated Multiple Myeloma

^{211}At-CD38 Antibody Therapy Demonstrates Potent Anti-Tumor Responses In Vivo With Improved Survival in Murine Models

Fred Hutch Cancer Center

blood





Methods: Mice bearing disseminated MOLP-8Luc xenografts were treated on day 0 with 211At-CD38 or 211At-Ab– at 12, 20, or 28 micro-Ci; n = 10 mice per group. Disease progression was monitored via weekly BLI and thrice weekly observations of mouse weight, condition, and mobility. Mice were euthanized when they experienced hind-limb paralysis or met IACUC weight loss or condition requirements. BLI of all groups at 21 days post-therapy. Red coffins indicate deceased mice.

O'Steen S. et. al., 2019, Blood.

CD38-Targeted ^{211}At Alpha Therapies Aim to Eradicate MRD in Multiple Myeloma

MRD+ Consolidation Post-Dara and/or TCE Response is a Wide-Open Opportunity

- MRD (Minimal Residual Disease) is an FDA-recognized clinical endpoint, increasingly used in trials & clinical practice
- Deeper MRD negativity correlates with longer PFS/OS

- Alpha-radioimmunotherapy with ^{211}At has preclinical evidence of eradicating residual MM clones in MRD settings
- Uses short-path, high-LET killing that delivers localized killing with minimal marrow exposure

- Does not compete directly with daratumumab, immune cell engagers, or CAR-T cells *(alpha consolidation where mAbs plateau)*
  - *MRD consolidation post-quadruplet therapy (including CD38 mAb)*
  - *post-relapse MRD/low-burden windows (after TCE bispecifics)*
  - *Delays or avoids CAR-T, option post BCMA CAR-T response but MRD+ and T-cells exhausted*

- $2B sales potential in Myeloma MRD consolidation + $1B in AML/Lymphoma MRD

NY-738

First in Human Study of a Marrow-Sparing CD38-Targeted ^{211}At for MRD+ Consolidation

Post-Daratumumab/TCE in Multiple Myeloma - Data Readout by End of 2026

1st-Generation Product: ^{211}At-CD38

- **IND-Cleared, Phase 1 to Initiate in H1 2026**
  - **Target Patients:** *15*
  - **Daratumumab Partial or Complete Responders** *with MRD+ by NGS or high-sensitivity flow, pre-CAR-T or ASCT*
  - **Low disease burden, adequate marrow reserve, CD38+ residual clone expression**
  - **Regimen:** *Outpatient IV administration, Single dose escalation (option for second dose)*
  - **Primary Endpoints:** *Safety/ marrow tolerability (DLT-driven RP2D), MRD negativity rate*
  - **Secondary/Exploratory Endpoints:** *Sustained MRD-, Progression Free Survival, Time-to-CAR-T*

2nd-Generation Product: ^{211}At-CD38 (Fab')2

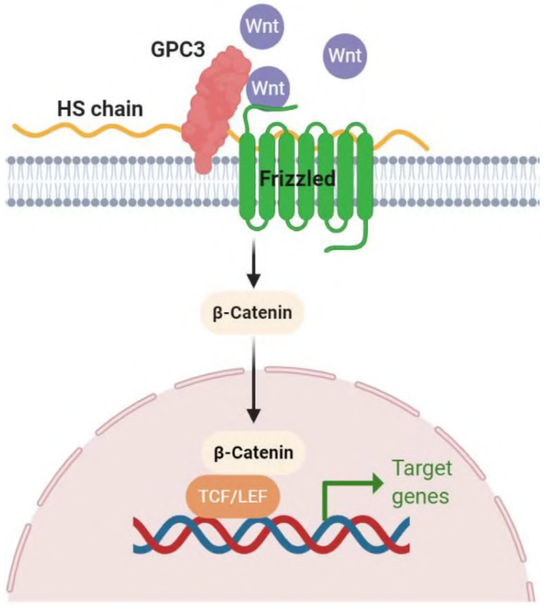
- ***Targeting IND & phase 1 initiation in early 2027***
- *Highly-differentiated CD38 fragment targeting different epitopes than Daratumumab, no fratricide or immune impact*
- *Format further minimizes marrow exposure and preserves kidney function*
- *Proprietary linker to ensure stability*

NY-703

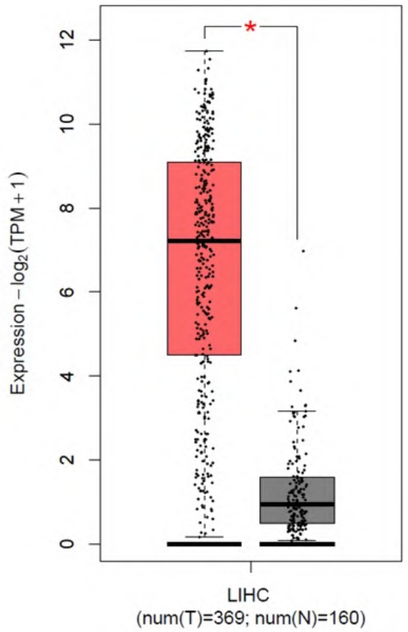
NY-303

GPC3 is a Therapeutic Target in Hepatocellular Carcinoma Currently Under Investigation in Clinical Trials

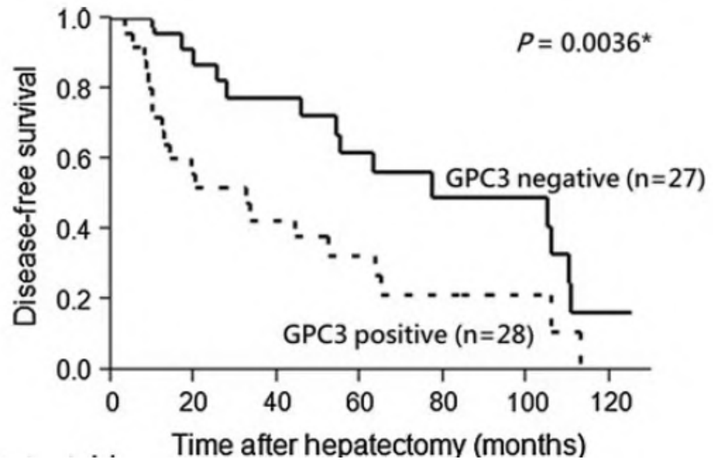
Glypican 3 (GPC3) is a cell surface protein playing a biological role in driving tumorigenesis in HCC



GPC3 is selectively expressed on tumor cells and is absent in normal tissue



GPC3 expression in early HCC is associated with poor 5-year disease free survival (27% vs 62%)



Major Pharma Companies with GPC3-Targeted Therapies in Clinical Development Include:

AstraZeneca Bristol Myers Squibb BAYER BeOne

Additional indications for GPC3 include: *NSCLC, Hepatoblastoma, Wilms Tumor, Malignant Rhabdoid Tumor, Yolk Sac Tumor, Rhabdomyosarcoma, Liposarcoma.*

Tsung-Chieh Shih et. al 2020. *Liver Research.* I-Pei Chen et. al 2013. *Journal of Gastroenetro.*

NY-703

NY-303

NAYA Harnesses Early Validation of GPC3 as Optimal HCC Target While Creating First/Best-in-Class Opportunities with Highly-Differentiated Candidates

Modality	Leaders	Target Indication	Clinical Setting	Additional Comments
CAR-Ts	AstraZeneca	*Late-Stage Refractory HCC*	*Academic Centers*	*High Response Rate (75% @ DL4) & Disease Control (91.4%), But Unfavorable Safety Profile*
ADCs	zymeworks	*Bulky Tumors Post-Immunotherapy*	*Outpatient*	*Payload Toxicity*
T-Cell Engagers	IPSEN Innovation for patient care; BeiGene	*Non-Responders to First-Line Immunotherapy*	*CRS Surveillance*	*Limited Efficacy to Date, Restricted to Low CRS Risk Patients*
Actinium-225 TAT	Bristol Myers Squibb; BAYER	*Risk of Metastasis & Residual Disease*	*Radiation Surveillance*	*Strong Pre-Clinical Data, Marrow-Hepatic Toxicity Risk, Limited Supply*
Astatine-211 TAT	NAYA THERAPEUTICS	*Risk of Metastasis & Residual Disease*	*Outpatient*	*Optimal Safety & Patient Access. Best-in-Class Potential Harnessing Unique ^{211}At Properties*
Bifunctional NK Engager	NAYA THERAPEUTICS	*Non-Responders to First-Line Immunotherapy*	*Outpatient*	*Optimal Safety & Patient Access. First-in-Class Potential Harnessing NKp46 + Unique MoA*

NY-703 Designed to Address HCC Residual Disease and Micro-metastasis

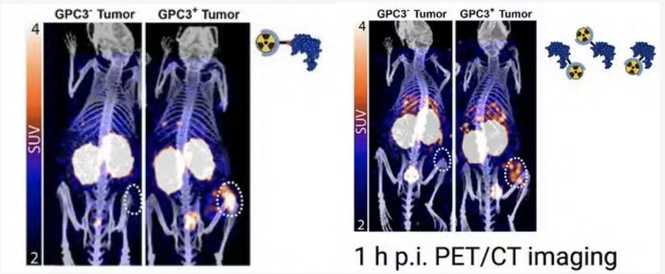
2026 Clinical Data to Validate Early-Mover ^{211}At TAT Leadership

50-75% Recurrence Rate with Surgery or Immunotherapy

- *Adjuvant/"MRD-focused" HCC after resection/ablation/TACE, GPC3-positive*
- *2L+ Advanced HCC (post-IO), GPC3-high*
- *Pediatric expansion: relapsed/refractory hepatoblastoma (HB), GPC3-positive*

Bayer & BMS Initiating Phase I Clinical Trials With ^{225}Ac-GPC3 in HCC, Supported By Strong Pre-Clinical Data

GPC3-Targeted Alpha Therapy Paired With ^{89}Zr Imaging Enables Precise Tumor Targeting & Low Off-Target Uptake



- ☑ *Planning for **Accelerated Development & 2026 Initial Clinical Data***
- ☑ ***Best-in-Class Potential** vs. Phase I Actinium-225/GPC3 Radioligands (Bayer, RayzeBio/BMS)*
- ☑ ***$3+ Billion Peak Sales Potential***

Combination/Sequencing of CD38 Antibodies & BCMA T-Cell Engagers Required for Deep & Durable Response

Builds & Expands on Established Clinical Proof of Concept for Daratumumab + TCE (ASH '25)

"Debulk then Redirect" Sequencing Approach (NK-Engager followed by T-Cell Engager)

Provides Immediate NK Killing Without Continuous T-Cell Pressure

- ***Reduces suppressive pressure of CD38 mAb maintenance*** *(distinct CD38 epitope with no fratricide and no effect on immune subset)*
- ***Improves immune synapse quality*** *for subsequent CD3 redirection,*
- ***Provides immediate innate killing*** *while T cells expand/activate under BCMA×CD3.*
- ***Complementary resistance coverage :***
  - *BCMA×CD3 resistance: antigen loss, T-cell exhaustion, impaired synapse, inhibitory cytokine milieu.*
  - *NKp46 activation reactivates exhausted NK cells; limited shedding unlike NKG2D and CD16*