NAYA THERAPEUTICS

Corporate Presentation

June 2025

NAYA Therapeutics:
Pioneering the Next Generation of Cancer Immunotherapies

NAYA is building a clinical portfolio of immuno-oncology assets leveraging two disruptive modalities:
bifunctional antibodies & alpha radioimmunotherapy

- **Bifunctional antibody market** expected to reach $50B by 2030 *(growing 3x faster than overall biologics market)*
  - *NAYA advancing 3 potential best-in-class clinical candidates leveraging proprietary plug & play multifunctional construct*
  - *NKp46 activation provides key differentiation from T-cell & other NK cell engagers, unlocking immunotherapy efficacy across tumors & targets*
- **Radioimmunotherapy market** estimated to top $16B by 2033[2]
  - *NAYA pioneering novel GPC3-targeted astatine-211 alpha radioimmunotherapy for HCC*

- Significant M&A/co-development appetite for both bifunctional antibodies & radioimmunotherapy, leading to multi-billion dollar valuations
- Unique opportunity for NAYA to build leadership in HCC with 3 differentiated first/best-in-class candidates in its pipeline

[2]Source: BioSpace [1]Source:QuickResearch

Building a Best-in-Class Cancer Immunotherapy Pipeline

NAYA's Candidates Join Wave of Bifunctional Antibodies & Targeted Alpha Therapies Competing to Displace Blockbuster Immunotherapies

NY-303	**GPC3/NKp46/CD16 Bifunctional Antibody for 2nd-Line HCC**	*Aims to unlock biology of non-responders to PD(L)-1/VEGF Inhibitors (70-85% of HCC patients) with strategic triple-targeting & unique MoA designed to enhance both efficacy and safety*
NY-500	**PD-1/VEGF Bifunctional Antibody for 1st-Line HCC**	*Aims to be first PD-1/VEGF to market in HCC as first-line alternative to Keytruda & Tecentriq-Avastin, leveraging FLEX construct, AI-optimization, and accelerated development strategy*
NY-700	**GPC3-Targeted Astatine-211 Alpha Therapy for 2nd/3rd-Line HCC**	*Aims to address needs of advanced metastatic, pre-transplant, & post-resection HCC patients with key safety advantages over lead TAT Actinium & enhanced targeting through GPC3*
NY-338	**CD38/NKp46/CD16 Bifunctional Antibody for 2nd-Line Multiple Myeloma**	*Aims to address limitations of daratumumab & T-cell engagers with strategic triple-targeting & unique MoA designed to enhance both efficacy and safety*

Bifunctional Antibody & Targeted Alpha Radioimmunotherapy Companies Achieving Significant Market & Partnering Valuations

Summit therapeutics	PD-1 x VEGF *(Oncology)* Phase II/III	$17.3B Market Cap* *Data demonstrates superior efficacy to Keytruda®******
OTTIMO PHARMA	PD-1 x VEGF *(Oncology)* Preclinical - *IND Q4 '25/Q1 '26*	$140 Million Series A Financing Led By Orbimed, Avoro, and Samsara******
CRESCENT BIOPHARMA	PD-1 x VEGF *(Oncology)* Preclinical - *IND Q4 '25/Q1 '26*	Implied Valuation: $518M** Reverse Merger with NASADAQ: GLYC, $200 Million Financing******
礼新医药 LaNova Medicines	PD-1 x VEGF *(Oncology)* Phase I	Global License Acquired by Merck & Co, $588M Upfront, $2.7B Milestones******
JANUX	PSMA T-Cell Engager *(Oncology)* Phase I	$1.84B Market Cap* Secondary Public Offering ($400 Million)******
Fusion Pharmaceuticals Inc.	Targeted Alpha Therapy *(Oncology)* Phase II	Acquired by AstraZeneca for $2B + $400M in Milesones
mariana ONCOLOGY	Targeted Alpha Therapy *(Oncology)* Pre-Clinical	Acquired By Novartis for $1B upfront + $750M in milesones, Prior Novartis Acquisitions of Endocyte & AAA
RayzeBio A Bristol Myers Squibb Company	Targeted Alpha Therapy *(Oncology)* Phase I	Acquired by BMS for $4.1B

**Source: Bloomberg, Market Cap based on 4/28/25 Closing Price*
*** 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 THERAPEUTICS

Novel Bifunctional Antibodies

NY-303

NY-500

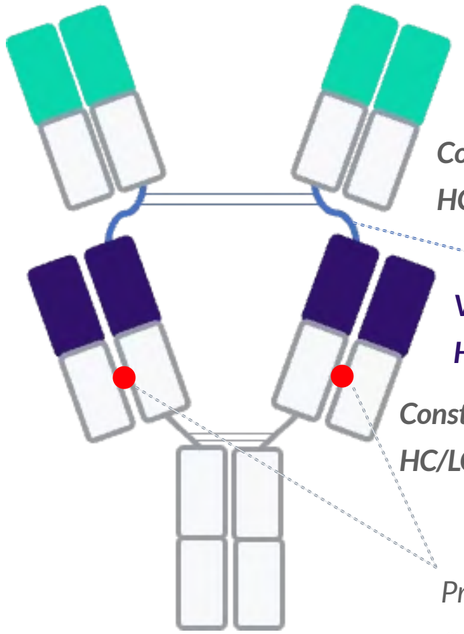
NY-338

NAYA's Plug & Play Bifunctional Antibody Construct Promotes Avidity and 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



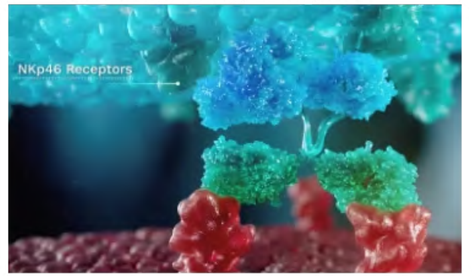
IgG1Fc
Fully active (with CD16-mediated effects) or silenced Fc region, enables longer half-life

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

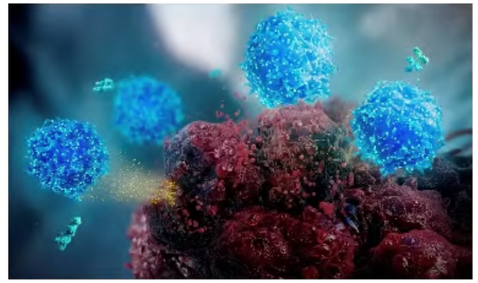
Proprietary FLEX Linkers Enable:
- *Simultaneous binding to targets 1 & 2*
- *Biological synapse in TME*
- *Higher stability due to connecting disulfide bridges*

Validated Manufacturability:
STC Biologics
(Newton, MA)

(1) Engaging Target 1



(2) Engaging Target 2


(3) Destruction of Tumor

Watch Full Video

NY-303

NY-338

Flex-NK™ Bifunctional Antibodies 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
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

NY-303

NY-338

NKp46 is Key to Depth & Durable Response in NK Cell Engagers

A Major Advance Over Legacy NK Cell Engagers

Robust and Enduring Target:

- NKp46 expression maintained on NK cells and rarely lost in tumors
- Allows NKp46-targeted engagers to continue recruiting functional effector cells long after CD16-only formats lose efficacy.

Proven Pre-Clinical Rfficacy:

- Preclinical data demonstrate survival benefit and pharmacodunamic (PD) effects lasting ≥10 days from a single dose.

Receptor Biology Drives Efficacy:

- The receptor's biology, not just the antibody's systemic half-life, is factor driving deeper, more durable responses especially in solid tumors where prolonged time-on-target within the TME is essential.

Pharmacodynamic Durability Booster

- NKp46 keeps NK cells activatable long after CD16 is shed
- NKp46 remains present on intratumoral NK cells and correlates with better survival in multiple solid tumors. pmc.ncbi.nlm.nih.gov

Adding an NKp46 Binding Arm Improves Pharmacodynamic Persistence

Durability lever	NKp46 biology	Consequence in vivo
Receptor stability	NKp46 is not cleaved by ADAM17, whereas CD16 is shed within hours of activation.	Engager keeps finding an intact activating receptor over repeated doses.
Uniform expression and polymorphism	Expressed on virtually all human NK cells with no high/low-affinity alleles.	Activates a larger NK pool with less patient-to-patient variability.
Tissue penetration	NKp46 remains present on intertumoral NK cells and correlates with better survival in multiple solid tumors.	Maintains effector function inside the TME.

NKp46 is a non-cleavable, polymorphism-agnostic ignition key for NK cells and can be layered together with CD16a for a more sustained tumor killing

NY-303

NY-500

NY-700

Hepatocellular Carcinoma (HCC) Franchise

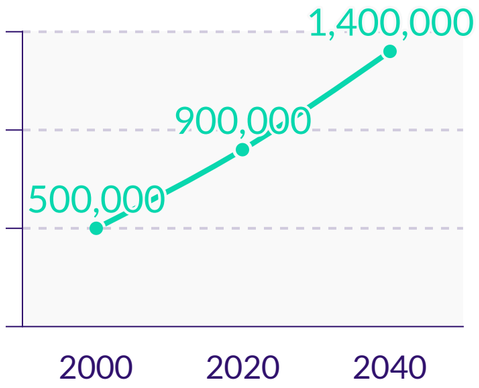
NY-303

NY-500

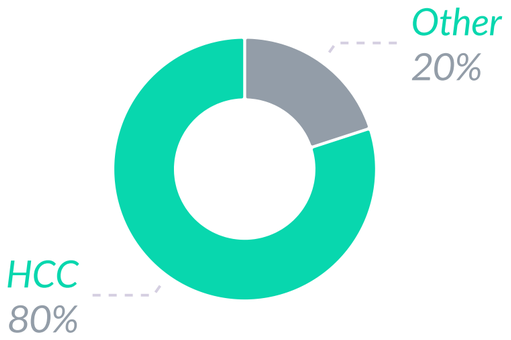
NY-700

HCC: A Globally Highly-Prevalent Cancer with Limited Therapeutic Options & High Unmet Need

Large Addressable Market for Systemic Therapy



Rapid Increase in Global Liver Cancer Incidence*



HCC Makes up 75-85% Liver Cancer Cases*

50-60%

of HCC Patients are Candidates for Systemic Therapy*

70-85%

of Candidates for Systemic Therapy are Non/Partial Responders to 1L Therapy**

**Immune Checkpoint Inhibitors in Hepatocellular Carcinoma: Current Strategies and Biomarkers Predicting Response and/or Resistance (hyperlinked)*

*** Updated efficacy and safety data from IMbrave150 (hyperlinked)*

NY-303

NY-500

NY-700

NAYA Positioned to be a Leading Player in HCC by 2028 with Best-in-Class Pipeline of Bifunctional Antibodies & Targeted Radioimmunotherapies

Rapidly-Evolving HCC Treatments Favor Bifunctional Antibodies & Radio Immunotherapeutics

2007

TKIs

(sorafenib)

2.3%

Response Rate

5.5 months

Progression-Free Survival

10.7 months

Overall Survival

2017

Anti PD-1s

(pembrolizumab, nivolumab)

~13%

Response Rate

~3 months

Progression-Free Survival

~15 months

Overall Survival

2020

Anti PD-L1 + Anti VEGF

(atezolizumab + bevacizumab)

30%

Response Rate

6.9 months

Progression-Free Survival

19.2 months

Overall Survival

2022

Anti-PD1 + Anti CTLA4

(nivolumab + tremelimumab)

20.1%

Response Rate

3.8 months

Progression-Free Survival

16.4 months

Overall Survival

2028*

NY-303 *(2nd Line)*

GPC3/NKp46/CD16 Bifunctional Antibody

NY-500: *(1st Line)*

PD-1/VEGF Bifunctional Antibody

(1st Line)

NY-700 *(2nd/3rd Line)*

Novel Targeted Radioimmunotherapy

**Immune Checkpoint Inhibitors in Hepatocellular Carcinoma: Current Strategies and Biomarkers Predicting Response and/or Resistance (hyperlinked)*

** projected*

NY-303: Next-Generation Second-Line Monotherapy Aiming to Unlock the Biology of Non-Responders to PD(L)1 Inhibitors (+/- VEGF Inhibitors)

NY-303 Combines Three Validated & Synergistic Targets, Enabling Triple Killing of Cancer Cells

NY-303

GPC3 x NKp46 x CD16

Flex-NK™ Bifunctional Antibody

GPC3: Oncofetal protein expressed on 80% of HCC cells but predominantly absent in normal tissue, enhancing precise targeting

NKp46: Activating receptor with demonstrated ability to promote NK cell migration to TME, reverse NK cell dysfunction, and enhance durable tumor killing activity

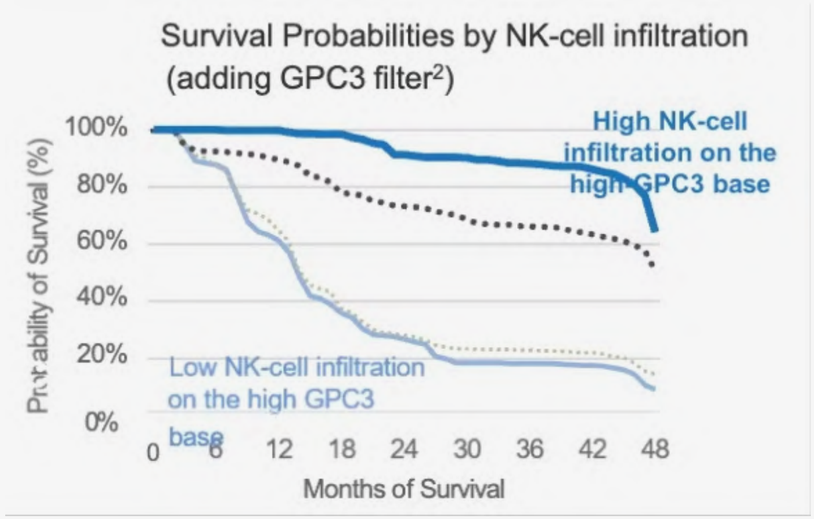
CD16: Low-affinity Fc receptor that plays a critical role in NK cell activation & drives antibody dependent cellular cytotoxicity (ADCC)

Unique MoA Directly Addresses Biology of Non-Responders

- **Dual activation of GPC3 & NKp46** simultaneously recruits NK cells while dampening Wnt- β-Catenin signaling in TME, driving ability to turn cold tumors hot & overcoming resistance to checkpoint inhibitors
- **Leverages innate immunity** (NK cells) rather than adaptive (T-cells)
- **Synergistic with checkpoint inhibitors,** allowing potential combination in first or second-line
- **Effective in patients with exhausted T-cells**
- **Lower risk of CRS** compared to CD3 T-cell engagers

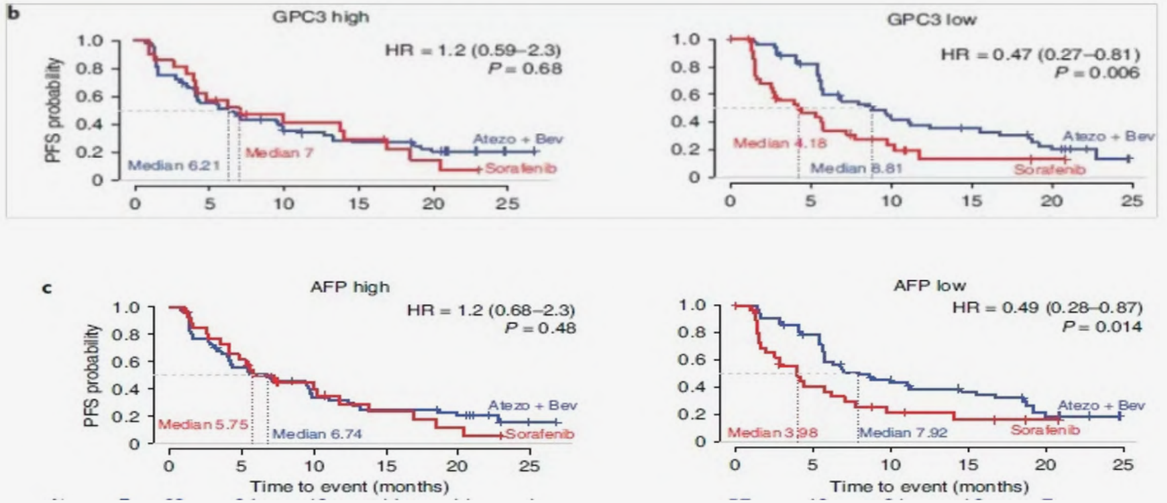
NY-303 Has Potential to Significantly Increase Survival in Patients Refractory to First-Line Immunotherapy (Tecentriq® + Avastin®) in HCC

Translational Clinical Data Supports NY-303 Monotherapy in Non-Responders to PD(L)1 Inhibitors (+/- VEGF Inhibitors)



Patients with High GPC3 Expression Value & High NK Cell Infiltration Had 4.6x Higher 36-month Survival Probability

sitc
Society for Immunotherapy of Cancer



Non-Responders to Atezolizumab + Bevacizumab Have High GPC3 & High AFP, Which Can Be Reversed by NY-303

nature medicine

Journal of Immunotherapy of Cancer, Vol. 12, 2024, 1243

Nature Medicine, A Xu et al Nat Med 2022 and GO30140 and IMbrave150

Phase I/IIa to Evaluate NY-303 as HCC Monotherapy, Phase IIa Expansion Planned in the US, EU, and Asia

Hadassah Hospital, Jerusalem

Sheba Medical Center, Tel Aviv

Sourasky Medical Center, Tel Aviv

- Initiation of phase I/IIa clinical trials cleared by Israeli ministry of health & internal review boards at leading medical academic centers
- Lead investigator: Jonathan Cohen, MD, PhD, Director of Clinical Research at the Sharett Institute of Oncology at Hadassah Hebrew University Medical Center
- Phase I/IIa monotherapy trial to enroll HCC patients not responding to first-line immunotherapy standard of care (PD(L)-1 inhibitors +/- anti-angiogenic drugs, such as Tecentriq + Avastin)
- Phase I/IIa endpoints to include safety, pharmacokinetics, activity markers, preliminary clinical efficacy (overall response rate) and time-to-progression (progression-free survival)
  - **Phase I Dose escalation** *(4 levels) with weekly administration as long as no disease progression is observed (first-patient expected in **H2 2025**, data in **2026**)*
  - **Phase IIa** *to expand to academic centers in the US, Europe, and Asia starting in H2 2026 and evaluate both monotherapy and combination with checkpoint inhibitors*
  - **Opportunity for fast-track designation** *based on phase I/IIa overall response rate & progression-free survival*

Objective Response Rate in NY-303's Phase I/IIa for HCC:
The Key to Accelerating Regulatory Pathway & Unlocking Valuation

ORR	Regulatory Pathway	Valuation Impact + Benchmarks
<20%	*Full Phase II/III with Progression-Free Survival/Overall Survival (PFS/OS) Required for Approval*	***Early Efficacy Signal***, *$30–70M Upfront, $400–800M Total (e.g. Gilead–Merus)*
20-30%	**Promising Efficacy: May be Eligible for Breakthrough Therapy Designation (BTD)**, *Confirmation of PFS in Phase IIb Required to Determine Approval Pathway*	**Potential Early BD Interest, Especially in Asia** *$75–150M Upfront (e.g. Daiichi–Arcus)*
30-40%	**Eligible for BTD & Accelerated Approval** *if Duration of Response (DoR)/PFS Compelling (6-9 months)*	**High Tier-1 Pharma Interest for Co-Dev/Acquisition** *$100–300M Upfront; $1B+ Total (e.g. Sanofi SAR443579 NKCE)*
>40%	**Strong Case for Accelerated Approval** *if Median DoR ≥ 6–9 Months* **May Support Conditional Approvals** *in Ex-US Markets*	**Potential Unicorn Scenario if Combined w/ Safety & Durability** *Dragonfly–Sanofi NKCE: >$175M upfront; >$2B total*

NY-500: AI-Optimized Fast-Follower to Ivonescimab in $100B PD-1/VEGF Market

NY-500: AI-Optimized PD-1/VEGF FLEX Bifunctional Antibody

- *Next-Generation First-Line Immunotherapy as Alternative to Current Standard of Care (Tecentriq + Avastin™)*
- *Leverages Plug & Play Construct w/ AI-Optimized PD-1 & VEGF Binders Designed in Collaboration with MabSilico*

PD-1/VEGFs Supplanting PD-1 Only Therapies, Poised to Expand the Cancer-Immunotherapy Market

$100B *Expected PD-1/VEGF Market [1]*

$51B *Value of 2024 PD-(L)1 Market[2]*

$30B *Keytruda ® (pembrolizumab) 2024 sales[3]*

40 *FDA-approved indications for Keytruda®[4]*

A Single Molecule With Synergistic PD-1 & VEGF Mechanisms

- *Enables cooperative pharmacology*
- *Better tumor microenvironment remodeling with combined checkpoint activation & vascular normalization*
- *Antibody design supports optimal PK & binding balance*
- *Simplified administration: One molecule vs. two antibodies*
- *Potential to improve durability & ORR vs. combination regimen*

[1] Cantor Equity Research (March 21st, 2025) [2] IQVIA (hyperlink) [3] Leading drugs worldwide based on projected 2024 sales (hyperlink) [4] Cancer Research (hyperlink)

Accelerated Development Strategy Supports NY-500's Aim to Be First PD-1/VEGF to Market in Large, Unsatisfied HCC Indication

2026 Initiation of Clinical Development On Track

- **Translational clinical & preclinical HCC data** in H2 2025
- **Phase I/II in first-line HCC** to be initiated in early 2026
- *NAYA to leverage development synergies with NY-303's clinical development in 2L HCC*

De-Risking Accelerates Path to Proof-of-Concept

- **Validated Targets:** PD-1 & VEGF both well-understood and highly successful commercially
- **Validated Structure:** Similar design to ivonescimab means functionality can more quickly be de-risked
- **Validated Indication :** Demonstrated Atezolizumab-bevacizumab efficacy in HCC

Opportunity for Leadership in HCC As Other PD1/VEGFs Focus on More Crowded Indications

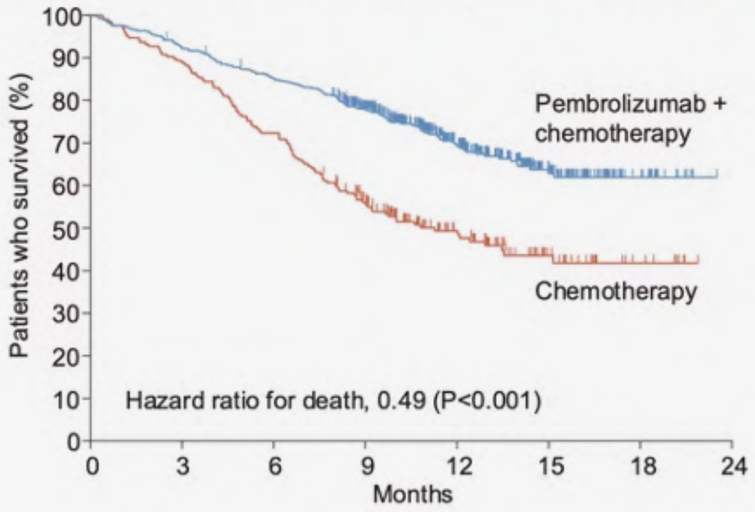
- **Lead PD-1/VEGF antibodies** currently in Phase III *(Summit, BioNTech)*
- **Rapid followers** entering Phase I in 2026 *(Ottimo, Crescent, Merck & Co)*

First PD-1/VEGF Bifunctional Antibody (Ivonescimab) Shows Striking Benefits Over First-Line Keytruda® in Lung Cancer, Cutting Risk of Progression by Half

Immune Checkpoint Breakthrough Data

PD-(L)1 inhibitors have **significantly prolonged survival, shifting 1L treatment** to immunotherapy

- For example, in 1L NSQ NSCLC, addition of pembrolizumab to chemo **significantly improved mOS** (**NR vs 11.3 months**[1] with a **HR of 0.49**).

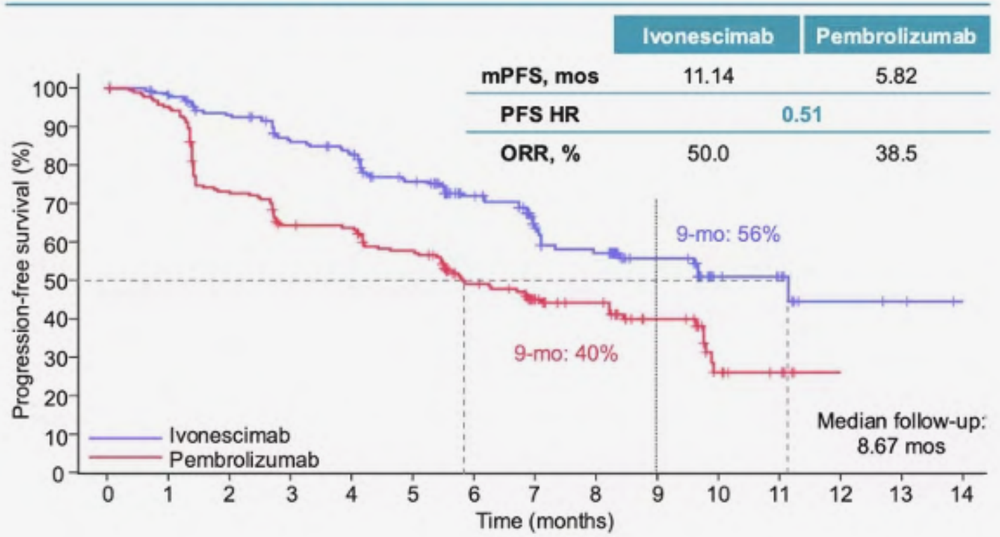


Summit lung cancer drug shows 'striking' benefit over Keytruda: cut the risk of lung cancer progression or death by half compared to Keytruda,

Sources: 2018 Gandhi (NEJM); 2023 Garassino (J Clin Oncol); GlobalData; FactSet; Pembrolizumab FDA Label

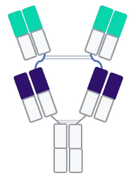
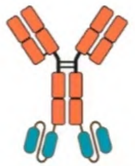
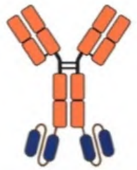
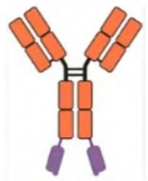
PD-1/VEGF Breakthrough Data

Ivonescimab is the **first drug to demonstrate superiority** in PFS **over pembrolizumab in a randomized Phase 3**



Sources: 2024 Zhou (WCLC Presentation on HARMONi-2); Summit Therapeutics; 2018 Paz-Area (NEJM); 2019 Mok (Lancet); 2022 De Castro Jr (J Clin Oncol); Avastin Label

NY-500 Designed to Have Similar Cooperative Pharmacology to Ivonescimab, Differentiated By HCC Clinical Focus & AI-Optimization

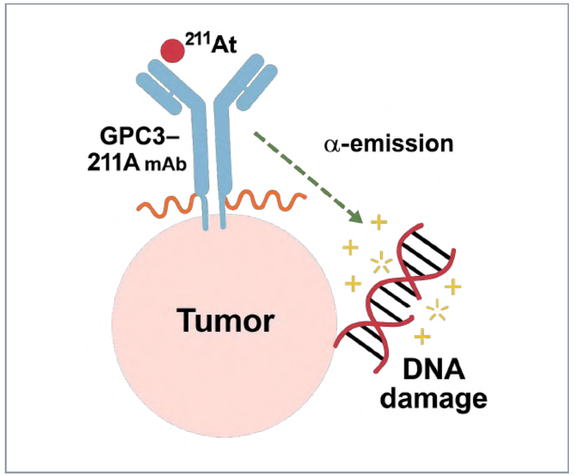
	NY-500 (NAYA)	CR-001 (Crescent)	Ivonescimab (Summit/Akeso Bio)	BNT327/PM8002 (BioNTech/Biotheus)	LM-299 (Merck/LaNova)	Jankistomig (Ottimo)
Lead Indication	HCC	NSCLC & Other Solid Tumors	NSCLC	Breast Cancer / PROC, Cervical Cancers	N/A	N/A
Development Phase	Preclinical	Preclinical	Phase 3 (Global)	Phase 2 (Global) Phase 3 (China)	Phase 1/2 Initiation (China)	Preclinical
Anti VEGF IgG	AI-Optimized VEGF scFvs	Bevacizumab	Bevacizumab	Bevacizumab	Bevacizumab	Anti-VEGF R2
Anti PDL-1	AI-Optimized PD-1 scFvs	Anti PD-1 scFvs	Penpulimab scFvs	Novel anti-PD-L1 VHHs	Novel anti PD-1 VHHs	Anti PD-1
Fc Function	Fc null, to avoid potential AEs	Fc null, to avoid potential AEs	Fc null, to avoid potential AEs	Fc null, to avoid potential AEs	Fc null, to avoid potential AEs	N/A
Cooperative Pharmacology	Yes	Yes	Yes	Expected (not disclosed); unclear impact of PD-L1 VHH	Expected (not disclosed); unclear impact of VHH structure	N/A
Design						

NAYA to Pioneer Astatine-211 as Novel Target Alpha Radioimmunotherapy

First-in-Class GPC3-Targeted 211At α-Therapy for HCC to Address Key Radioimmunotherapy Toxicity & Supply Challenges

Astatine 211, a Game-Changing Alpha Emitter Radionuclide

- **Limitations of existing alpha emitters with long half-lives** such as *Actinium 225* (up to 19 days) include bone marrow toxicity & decay management
- **Short half-life (7.2 hours) with high tumor specificity & short-range cytotoxicity** uniquely position Astatine-211 to address residual disease, micro-metastasis, and post-immunotherapy failures
- **NAYA committed to leveraging rapidly-scaling global supply networks** for Astatine 211



NY-700, NAYA's First-in-Class GPC3-Targeting Astatine-211 Alpha Therapy

- *Consists of Astatine-211 isotope conjugated to a GPC3-internalizing antibody fragment*
- **Potentially best-in-class** TAT vs. Phase I *Actinium 225-GPC3* radioligands (Bayer, Rayze Bio/BMS)
- **GPC3 enhances precision targeting** (expressed in 70-90% of HCC cells but not on adult healthy cells)
- Access to **proprietary linker radiochemistry** & **global astatine-211 supply chain**

- **Opportunity for NAYA to further build leadership in HCC**
- **Opportunity to expand astatine-211 platform with other targets such as PSMA**

Redefining Standard of Care With NAYA's Comprehensive HCC Pipeline

The outcome of the Phase I/II clinical trial for NAYA's GPC3/NKp46 antibody, evaluating patient response and time-to-progression, has the potential to establish a path towards a new standard of care for the 70% of unresectable HCC patients not responding to checkpoint inhibitors. Harnessing the GPC3-Wnt axis is an innovative approach that kills two birds with one stone by recruiting NK cells while dampening Wnt signaling - a key regulator of tumor microenvironment - with the goal of turning immunological cold tumors hot and overcoming resistance. Additionally, we are awaiting further development of NAYA's comprehensive HCC pipeline, including a PD-1/VEGF bifunctional antibody as a potential alternative to atezolizumab-bevacizumab and a GPC3-targeted Astatine-211 alpha radioimmunotherapy aiming to address the needs of advanced metastatic, pre-transplant, and post-resection patients, offering new hope towards long-term remission of HCC.

Yaron Ilan, MD
Professor of Medicine, Faculty of Medicine, Hebrew University
Chairman, Department of Medicine, Hadassah Medical Center

NAYA THERAPEUTICS

Multiple Myeloma Franchise

NY-338: A Breakthrough CD38/NKp46/CD16-Targeting Bifunctional Antibody for Multiple Myeloma

Continued Growth of Multiple Myeloma Market Driven by Darzalex® & Rise of Bispecifics

	2024	2032
Multiple Myeloma*	$28B	$44B
Darzalex®* *(Daratumumab)*	$12B	$14B
Bispecifics (TCEs & NKEs)	$400M	$11B

Differentiation Opportunity in Established, Competitive Multiple Myeloma Market

Need for new therapies
as Darzalex® moves to first line

Multifunctional antibodies positioned to address non-responders & eventually challenge Darzalex®
*(3 recent approvals from J&J, Pfizer)***

NY-338 Shows Key Differentiation Compared to Monoclonal & T-Cell Engagers:

- ☑ **Triple-Killing of Myeloma Cells** *through CD38/NKp46/CD16 engagement*
- ☑ **Longer Half-Life, Increased Potency**
- ☑ **Improved Safety** *(no CRS / undesirable effects on immune cells)*
- ☑ **Low-to-no Fratricide on NK cells**

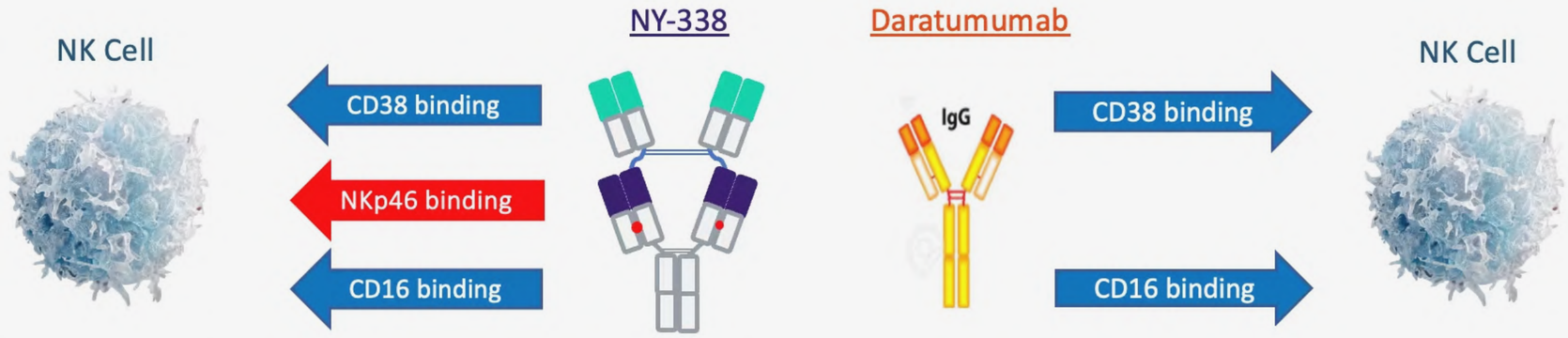
*https://www.fiercepharma.com/marketing/analysts-predict-myeloma-market-will-hit-33b-2030-and-tip-1-company-take-lions-share
***https://investors.biogen.com/news-releases/news-release-details/biogen-completes-acquisition-human-immunology-biosciences
**https://pmc.ncbi.nlm.nih.gov/articles/PMC10618718/

Unique Mechanism of Action for NY-338's Differentiated Profile vs. Daratumumab

NY-338's Unique Mechanism of Action Enables:

- ***Activation*** *of NKp46 signaling*
- ***Neutralization*** *of CD38-mediated apoptosis & ADCC of NK cells*
- ***Minimized fratricide & depletion*** *of CD38+ immune cell subsets compared to daratumumab*
- ***Minimized cytokine release*** *compared to T-cell engagers*

NKp46 Activation Reduces NK Cell Fratricide vs. Daratumumab



NKp46 activation neutralizes CD38-mediated NK cell apoptosis and CD16-mediated ADCC, minimizing fratricide

CD38-mediated NK cell apoptosis and CD16-mediated ADCC with Daratumumab

NY-338 Shows Potential to Address Limitations of Both Daratumumab & BCMA T-Cell Engagers Through Combination and/or Sequential Use

	NY-338	Daratumumab	BCMA T-Cell Engagers (TCEs)
CD38 Epitope Profile	Binds to 3 Distinct Epitopes, Including 2 Non-Overlapping with Daratumumab	Confirmational on CD38 Extra-Cellular Domain	Non-Relevant
Effective in Dara-Resistant and/or CD38 Downregulation	Yes	No	Yes
Cytokine Release Syndrome	Minimal	Low	High
Fratricide	Minimal	Yes	No
Combination and/or Sequential Use	Sequential with Daratumumab, Combination/Sequential w TCEs	Combination/Sequential w TCEs	Combination/Sequential with both NY-338 & Daratumumab

Data Presented at American Society of Hematology Supports Initiation of Clinical Trials, Establishes NY-338 as Potential Best-in-Class Therapeutic for Myeloma

> *"The synergistic engagement of NK cells through NKp46 greatly enhances the immunotherapeutic effects of FLEX-NK™ bispecific antibodies, reducing NK cell fratricide, maintaining NK cell levels, and enhancing potency including reversal of NK cell dysfunction. The data supports initiation of clinical trials to evaluate this promising new therapy and makes it a potential best-in-class anti-CD38 therapeutic for multiple myeloma."*

Ola Landgren, MD, PhD

Professor of Medicine, Chief of the Myeloma Division, and Leader of the Experimental Therapeutics Program at the University of Miami's Sylvester Comprehensive Cancer Center

Full ASH Press Release

NAYA THERAPEUTICS

Investment Considerations

Multiple Clinical Readouts in 2026-2027

Validated Preclinical Data Presented at Major Oncology Meetings including AACR, ASH, EHA, ESMO, and SITC

Candidate	Indication	Targets	Product Platform	Pre-Clinical	IND Enabling	Phase I/II
NY-303	Hepatocellular Carcinoma (HCC) & Other Solid Tumors	GPC3 NKp46 CD16	FLEX-NK™ Bifunctional Antibody			
NY-500	Hepatocellular Carcinoma (HCC) & Other Solid Tumors	PD-1 VEGF	FLEX Bifunctional Antibody			
NY-700	Hepatocellular Carcinoma (HCC) & Other Solid Tumors	At211-GPC3	Targeted Alpha Therapy			
NY-338	Multiple Myeloma, AML, Lymphoma	CD38 NKp46 CD16	FLEX-NK™ Bifunctional Antibody			

Seasoned Entrepreneurial Leadership Team

Daniel Teper, PharmD, MBA

Chief Executive Officer

& Chief Financial Officer

Michael G. King

Executive Vice President

Ravi Kiron, PhD, MBA

Chief Business Officer

Lyn Falconio

Chief Communications Officer

Dan Chiche, MD

Chief Medical Officer

Vidisha Mohad, PhD

Head of Product Development

Board of Directors Combines Executive & Investment Experience in Pharma & Biotech

Laurent Audoly, PhD

Daniel D'Agostino, MBA

Ely Benaim, MD

Melissa Fensterstock, MPhil, MBA

Daniel Teper, PharmD, MBA

Alexandra Urman, MPH

Scientific & Strategic Advisors To Accelerate Growth

Michael Caliguiri, MD

Ola Landgren, MD, PhD

Josep Lovett, MD, PhD

Yaron Ilan, MD

Jean Kadouche, PhD

Rahul Singhvi, PhD, MBA

NAYA Therapeutics:
Pioneering the Next Generation of Cancer Immunotherapies

NAYA is building a clinical portfolio of immuno-oncology assets leveraging two disruptive modalities: bifunctional antibodies & alpha radioimmunotherapy

- **Bifunctional antibody market** expected to reach $50B by 2030 *(growing 3x faster than overall biologics market)*
  - *NAYA advancing 3 potential best-in-class clinical candidates leveraging proprietary plug & play multifunctional construct*
  - *NKp46 activation provides key differentiation from T-cell & other NK cell engagers, unlocking immunotherapy efficacy across tumors & targets*
- **Radioimmunotherapy market** estimated to top $16B by 2033[2]
  - *NAYA pioneering novel GPC3-targeted astatine-211 alpha radioimmunotherapy for HCC*

- Significant M&A/co-development appetite for both bifunctional antibodies & radioimmunotherapy, leading to multi-billion dollar valuations
- Unique opportunity for NAYA to build leadership in HCC with 3 differentiated first/best-in-class candidates in its pipeline

[2]Source: BioSpace [1]Source:QuickResearch