

Corporate Presentation

NAYA Therapeutics:

Advancing Oncology Outcomes with Next-Generation Bifunctional Antibodies



NY-303: GPC3 x NKp46

FLEX-NK™ Bifunctional Antibody

- First-in-Class Monotherapy with Unique MoA
- Unlocks Biology of Non-Responders (70-85% of Patients) in Hepatocellular Carcinoma (HCC)



NY-500: PD-1 x VEGF

FLEX Bifunctional Antibody

- Fast-Follower to *Ivonescimab* in PD-1 x VEGF Market (\$100 Billion)
- Aiming to Be First PD-1 x VEGF to Market in HCC



NY-338: CD38 x NKp46

FLEX-NK™ Bifunctional Antibody

- Unique MoA Ehances Efficacy & Safety
- **Differentiated Profile** to Daratumumab
- Potential for Best-in-Class in Multiple Myeloma

Bifunctional antibodies are transforming cancer immunotherapy, significantly improving clinical outcomes.

NAYA is advancing a portfolio of highly-competitive clinical candidates with differentiated target product profiles, positioning itself for leadership with each of its strategically-selected therapeutic franchises.

NAYA's versatile plug & play FLEX construct is designed to simultaneously engage tumors and the immune system, increasing avidity and harnessing the cooperative effect of combined validated targets.

Bifunctional Antibody Companies Achieving Significant Market & Partnering Valuations

PD-1 x VEGF (Oncology) \$13.16 Billion Market Cap* Summit Data demonstrates superior efficacy to Keytruda®*** Phase II/III PD-1 x VEGF (Oncology) OTTIMO \$140 Million Series A Financing Led By Orbimed, Avoro, and Samsara*** Preclinical - *IND Q4 '25/Q1 '26* PD-1 x VEGF (Oncology) Implied Valuation: \$518 Million** CRESCENT Preclinical - IND Q4 '25/Q1 '26 Reverse Merger with NASADAQ: GLYC, \$200 Million Financing*** PD-1 x VEGF (Oncology) Global License Acquired by Merck & Co, 礼新医药 \$588 Million Upfront, \$2.7 Billion Milestones*** Phase I T-Cell Engager (Oncology) Vir Biotechnology™ \$1.4 Billion Market Cap, 60% Stock Price Increase Upon Phase I Data Phase I Merus EGFR x LGR5 (Oncology) \$2.9 Billion Market Cap* Phase II/III PSMA T-Cell Engager (Oncology) \$3.1 Billion Market Cap* **JANUX** Secondary Public Offering (\$400 Million)*** Phase I EGFR/TGF-β (Oncology) \$948M Market Cap*, \$315 Million IPO @ \$1.3B Post-Money*** Phase I/IIa completed

^{*}Source: Bloomberg, Market Cap based on 12/31/24 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's Versatile Plug & Play Bifunctional Antibody Construct Promotes Avidity & Synapse Effect, Enhancing Precision Tumor Killing

Natural bivalent design

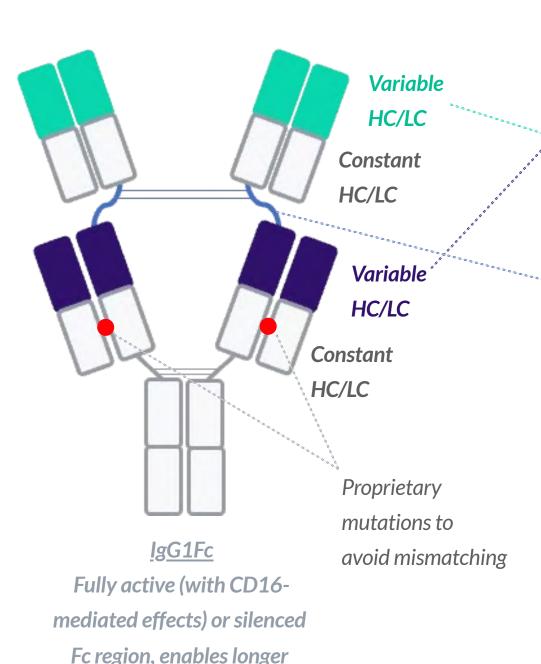
allows for binding
affinities at levels
comparable to native
monoclonal antibodies

Distal FAB 1

Binds to Tumor or Vascular Target: GPC3, CD38, VEGF

Proximal FAB 2

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



half-life

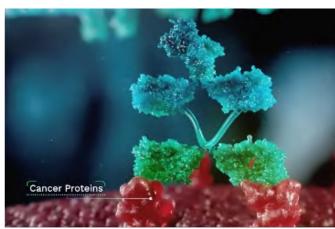
Plug & Play of variable HC/LC

parts enable faster development

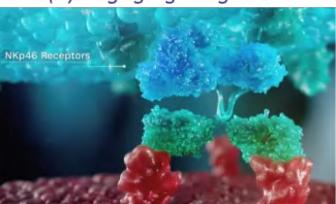
Proprietary FLEX linkers enable:

- Simultaneous binding to targets 1 & 2
- Biological synapse in TME
- Higher stability due to connecting disulfide bridges

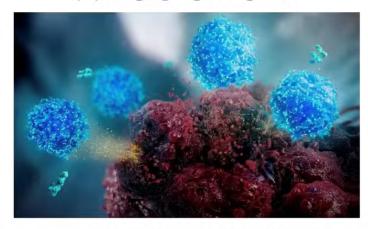
Validated Manufacturability



(1) Engaging Target 1



(2) Engaging Target 2



(3) Destruction of Tumor

Watch Full Video

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

NKp46 Activation Unlocks Immunotherapy Efficacy Across Tumors & Targets

<u>T-Cell Engagers (TCE)</u>		<u>FLEX-NK™ Engagers (FNKE)</u>	
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	

Multiple Clinical Readouts in 2026-2027

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

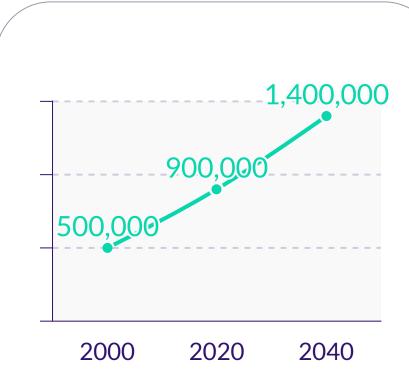
Target	Product Platform	Candidate	Indication	Pre-Clinical	IND Enabling	Phase I/II
GPC3 x NKp46	Flex-NK™ Bifunctional Antibody	NY-303	Hepatocellular Carcinoma & Other Solid Tumors			
PD-1 x VEGF	Flex Bifunctional Antibody	NY-500	Hepatocellular Carcinoma & Other Solid Tumors			
CD38 x NKp46	Flex-NK™ Bifunctional Antibody	NY-338	Multiple Myeloma, AML, Lymphoma			



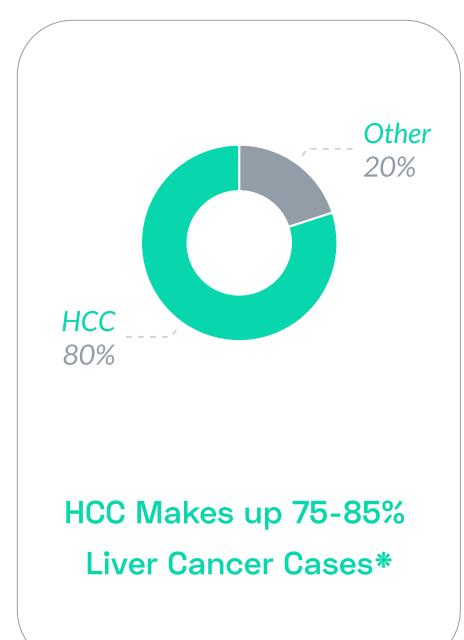
Hepatocellular Carcinoma (HCC) Franchise

HCC: A Globally Highly-Prevalent Cancer with Limited Therapeutic Options

Large Addressable Market for Systemic Therapy



Rapid Increase in Liver
Cancer Incidence*



50-60%

of HCC Patients are
Candidates for
Systemic Therapy*

70-85%

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

NAYA Positioned to be a Leading Player in HCC by 2028 with Next-Generation NY-303 & NY-500 Bifunctional Antibodies

Rapidly-Evolving HCC Treatments Favor Combination Therapy & Bifunctional Antibodies

2007

TKIs

(sorafenib)

2.3%Response Rate5.5 monthsProgression-Free Survival

10.7 monthsOverall Survival

2017

Anti-PD1s (pembrolizumab, nivolumab)

~13%
Response Rate
~3 months
Progression-Free Survival
~15 months

2020

Anti-PDL1
+ Anti-VEGF
(atezolizumab +

30% Response Rate

6.9 months

bevacizumab)

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

Overall Survival

16.4 months

2028*

NY-303:

GPC3 x NKp46

Bifunctional

Antibody

NY-500:

PD1 x VEGF

Bifunctional

Antibody

Overall Survival

NY-303: A Next-Generation Monotherapy Enabling Response for Non-Responders to PD(L)1 Inhibitors (+/- VEGF Inhibitors)

NY-303 Combines Two Novel, Validated, and Synergistic Targets, Enabling Triple Killing of Cancer Cells



NY-303 GPC3 x NKp46

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

Unique MoA Directly Addresses Biology of Non-Responders

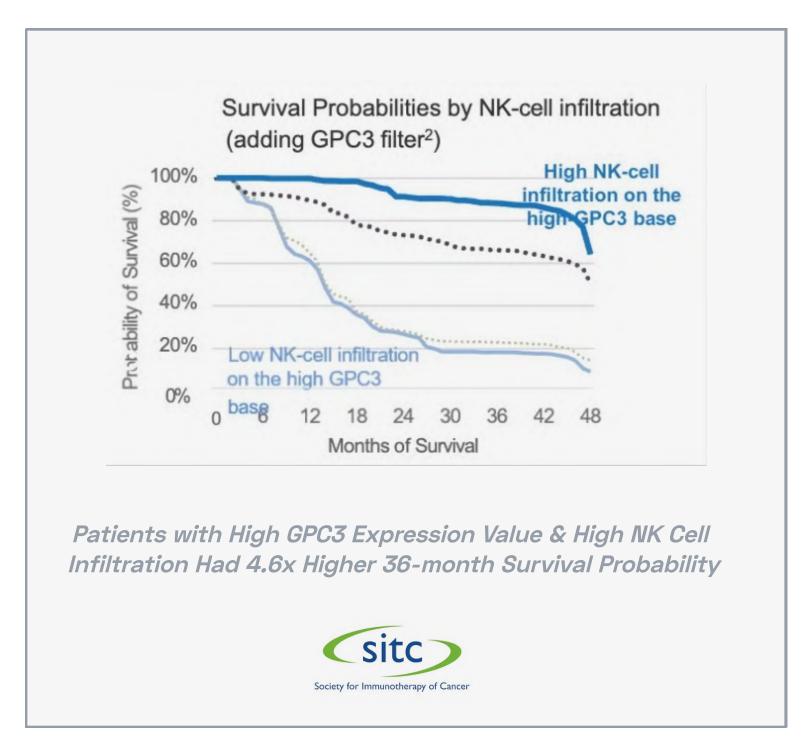
- Triple killing of cancer cells (with NK, T, and adaptive immune cells) unlocks increased response & survival
- Ability to turn cold tumors (non-susceptible to immunotherapy) hot (susceptible) through reduction of β -Catenin levels increases infiltration into TME & sensitivity to PD1 checkpoint blockade

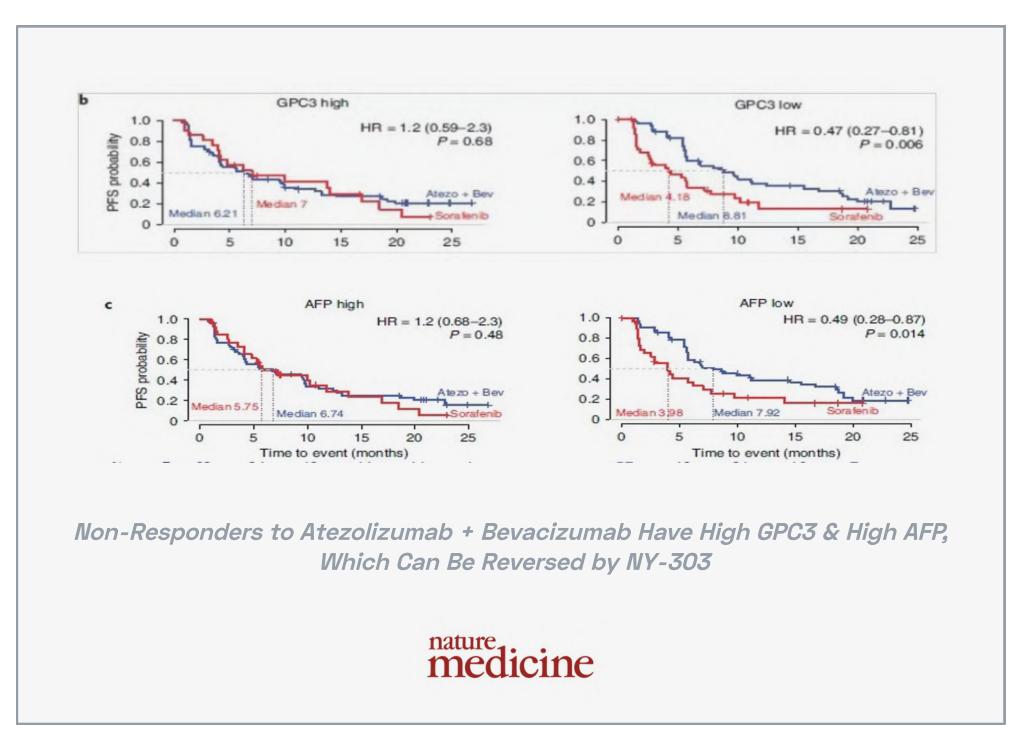
Accelerated Clinical Development with Initial Data in 2026

- Initiating phase I/II in HCC in 2025
- Potential phase II/III accelerated development in 2027
- Other potential indications include ovarian, lung, pediatric cancers

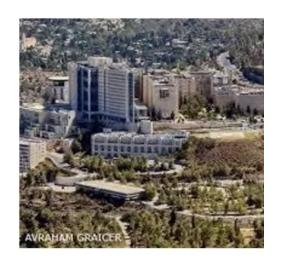
NY-303 Has Potential to Significantly Increase Survival in Patients Refractory to 1st Line Immunotherapy (Tecentriq® + Avastin®) in HCC

Translational Clinical Data Supports GPC3 x NKp46 Monotherapy in Non-Responders to PD(L)1 Inhibitors (+/- VEGF Inhibitors)





Phase I/lla 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 (check point 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 and 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 check point 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/lla for HCC: The Key to Accelerating Regulatory Pathway & Unlocking Valuation

ORR	<u>Regulatory Pathway</u>	<u>Valuation Impact + Benchmarks</u>
<20%	Early Efficacy Signal , Full Phase II/III with Progression-Free Survival/Overall Survival (PFS/OS) Required for Approval	\$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 \geq 6-9 Months	Potential Unicorn Scenario if Combined with Safety & Durability
	May Support Conditional Approvals in Ex-US Markets	Dragonfly–Sanofi NKCE: >\$175M upfront; >\$2B total

NY-500: Al-Optimized Fast-Follower to Ivonescimab in \$100B PD-1 x VEGF Market, Opportunity to Be First-to-Market in Large, Unsatisfied HCC Indication



NY-500 PD-1 x VEGF AI-Optimized FLEX Bifunctional Antibody

- Next-Generation First-Line Immunotherapy as Alternative to Current Standard of Care (Tecentriq + Avastin™)
- Leverages NAYA's Plug & Play FLEX Construct With AI-Optimized PD-1 & VEGF Binders

Large PD-(L)1 Market

\$100B

Expected PD-1 x VEGF Market 1

\$58B

Forecasted Value of 2025 PD-(L)1 Market2

Keytruda ® (pembrolizumab) 2024 sales₃

40

FDA-approved indications for Keytruda®4

Aiming to be First PD-1 x VEGF to Market in HCC

Accelerated Development for NY-500 in HCC

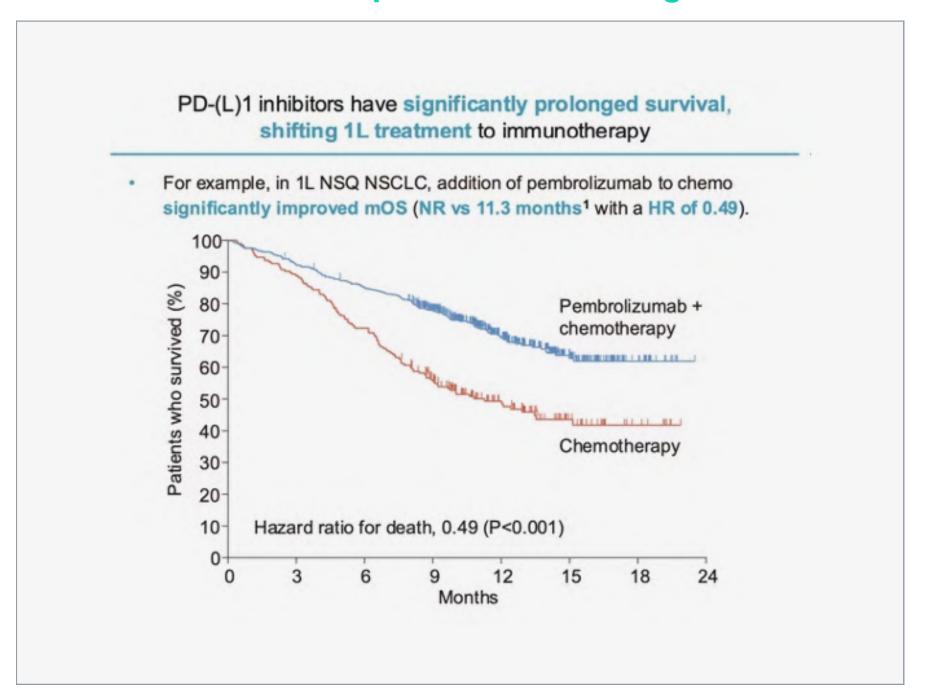
- Translational clinical & preclinical HCC data in H2 2025
- Phase I/II in First-Line HCC to be initiated in early 2026
- NAYA to leverage development synergies with clinical development of NY-303 in 2L HCC, non-responders to PD(L)1

Other PD1 x VEGF Antibodies Focus on More Competitive Lung & Breast Indications, Leaving NAYA with Opportunity to Lead in HCC

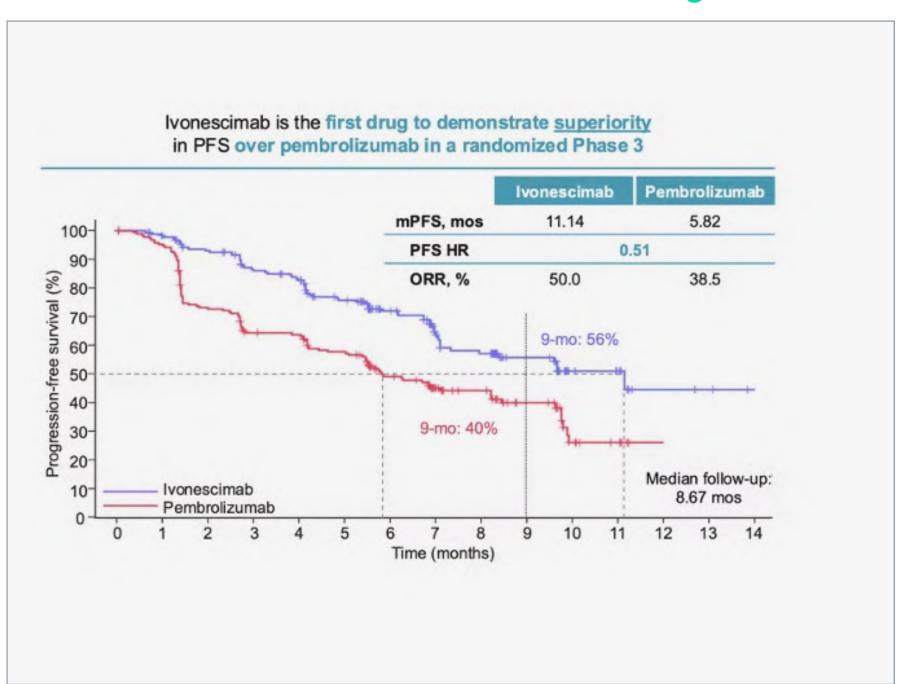
- Lead PD-1 x VEGF antibodies in Phase III (Summit, BioNTech)
- Rapid followers entering Phase I in '26 (Ottimo, Crescent, Merck & Co)

First PD-1 x VEGF Bifunctional Antibody Demonstrates Progression-Free Survival Benefit Over First-Line Immunotherapy (Keytruda®) in Lung Cancer

Immune Checkpoint Breakthrough Data



PD1 x VEGF Bifunctional Breakthrough Data





Multiple Myeloma Franchise

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

Continued Growth of Multiple Myeloma Market Driven by Darzalex® & Biologics

	2023	2030
Multiple Myeloma*	\$23B	\$33B
Darzalex®* (Daratumumab)	\$9.7B	\$14.7B

Differentiation Opportunity in Established, Competitive Multiple Myeloma Market

Need for new therapies

as Darzalex® now moved to first line

Bifunctional 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
Bifunctional Antibodies:

- Dual-Killing of Myeloma Cells through CD38 & NKp46 engagement
- ✓ Longer Half-Life, Increased Potency (through dual CD38/NK targeting)
- ✓ 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

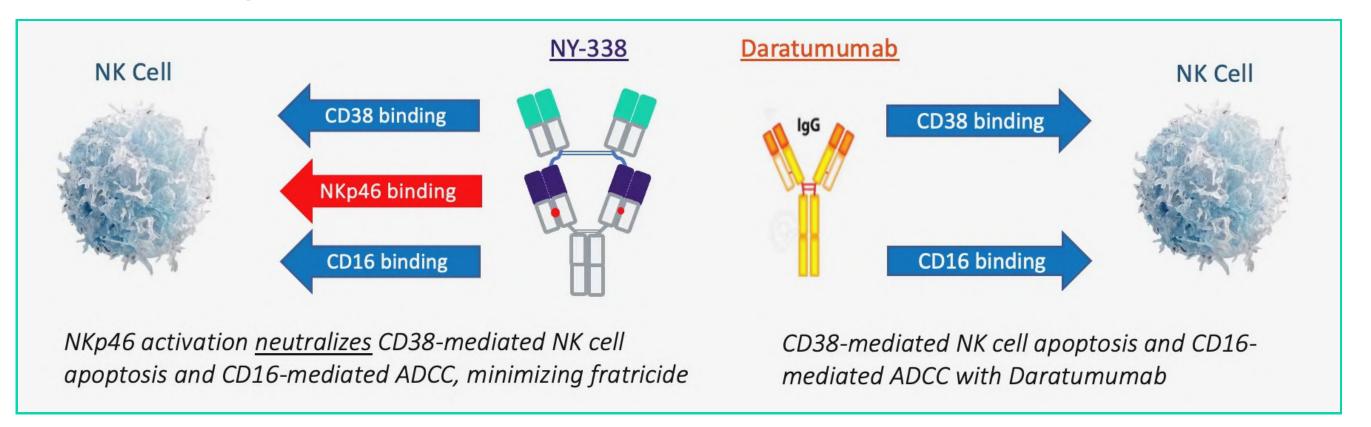
^{**}https://pmc.ncbi.nlm.nih.gov/articles/PMC10618718/

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



Data Presented at ASH 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



Investment Considerations

Seasoned Entrepreneurial Leadership Team



Daniel Teper, PharmD, MBA

Chief Executive Officer



Ravi Kiron, PhD, MBA
Chief Business Officer





Lyn FalconioChief Communications Officer





Rahul Singhvi, PhD, MBA Chief Technology Officer





Dan Chiche, MD
Chief Medical Officer





Armin Rath, PhD
Chied Product Development Officer





Arthur Levine, CPA
Chief Financial Officer





Vidisha Mohad, PhD Executive-in-Residence,



BoD Combines Executive & Investment Experience in Pharma & Biotech



Scientific Advisors To Accelerate Strategic Growth

