



NAYA
THERAPEUTICS

Pioneering the Next Generation of Cancer Immunotherapies

 **HCW**
H.C. WAINWRIGHT & CO.

**H.C. WAINWRIGHT 3RD ANNUAL IMMUNE CELL
ENGAGER VIRTUAL CONFERENCE**

JUNE 24, 2025

NAYA Therapeutics is pioneering the next generation of cancer immunotherapies with a pipeline of bifunctional antibodies & targeted alpha therapies designed to unlock deeper, more durable responses for patients not responding to current standard-of-care.

Our primary focus is on hepatocellular carcinoma (HCC), one of the largest solid tumor indications with high unmet medical need, with three candidates including NY-303, our GPC3-targeting FLEX-NK™ cell engager advancing to a monotherapy Phase I/II clinical trial.

In addition, we are developing NY-338, our CD38-targeting FLEX-NK™ cell engager, with the optionality to expand our pipeline to other hematological & solid tumors indications.



**Novel FLEX-NK™ Cell Engager
Bifunctional Antibodies**

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

Natural Bivalent Design

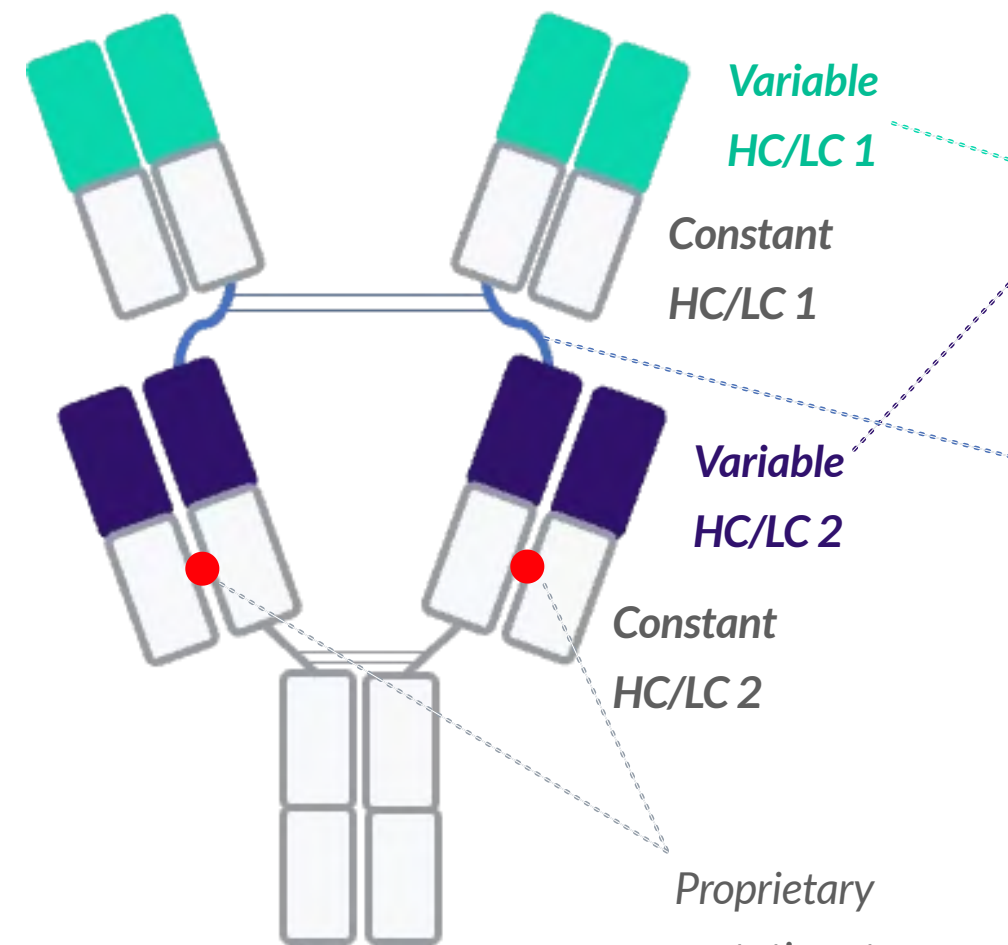
allows for binding affinities at levels comparable to native monoclonal antibodies

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

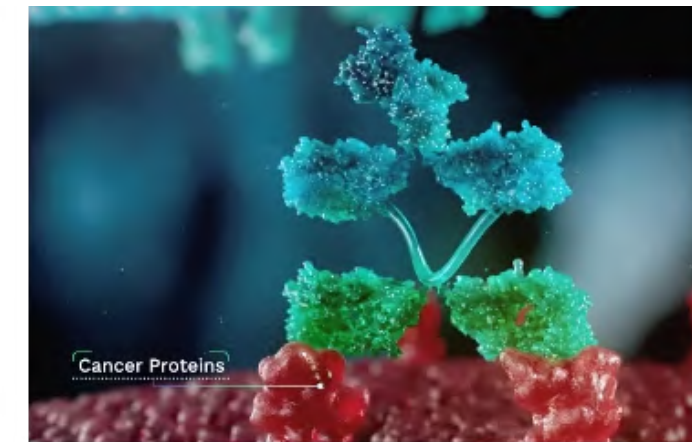
Plug & Play of Variable HC/LC parts enable faster development

Proprietary FLEX Linkers Enable:

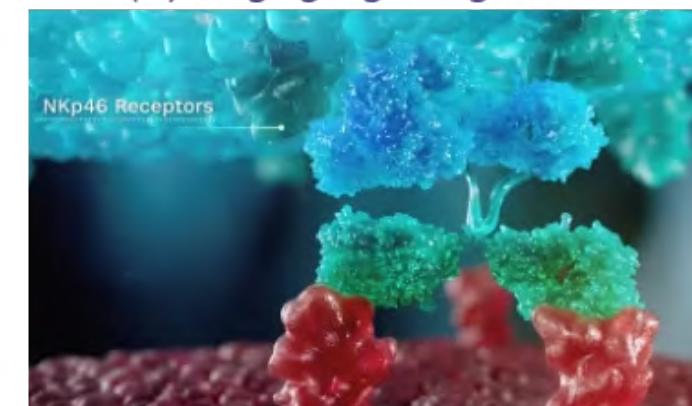
- Simultaneous binding to targets 1 & 2
- Biological synapse in TME
- Higher stability due to connecting disulfide bridges

Validated Manufacturability:

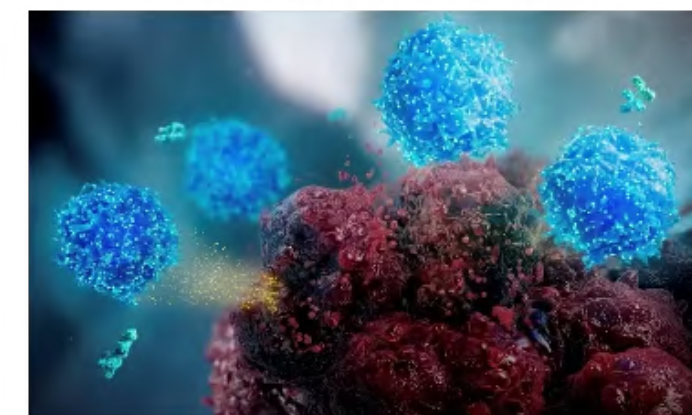
STC Biologics
(Newton, MA)



(1) Engaging Target 1



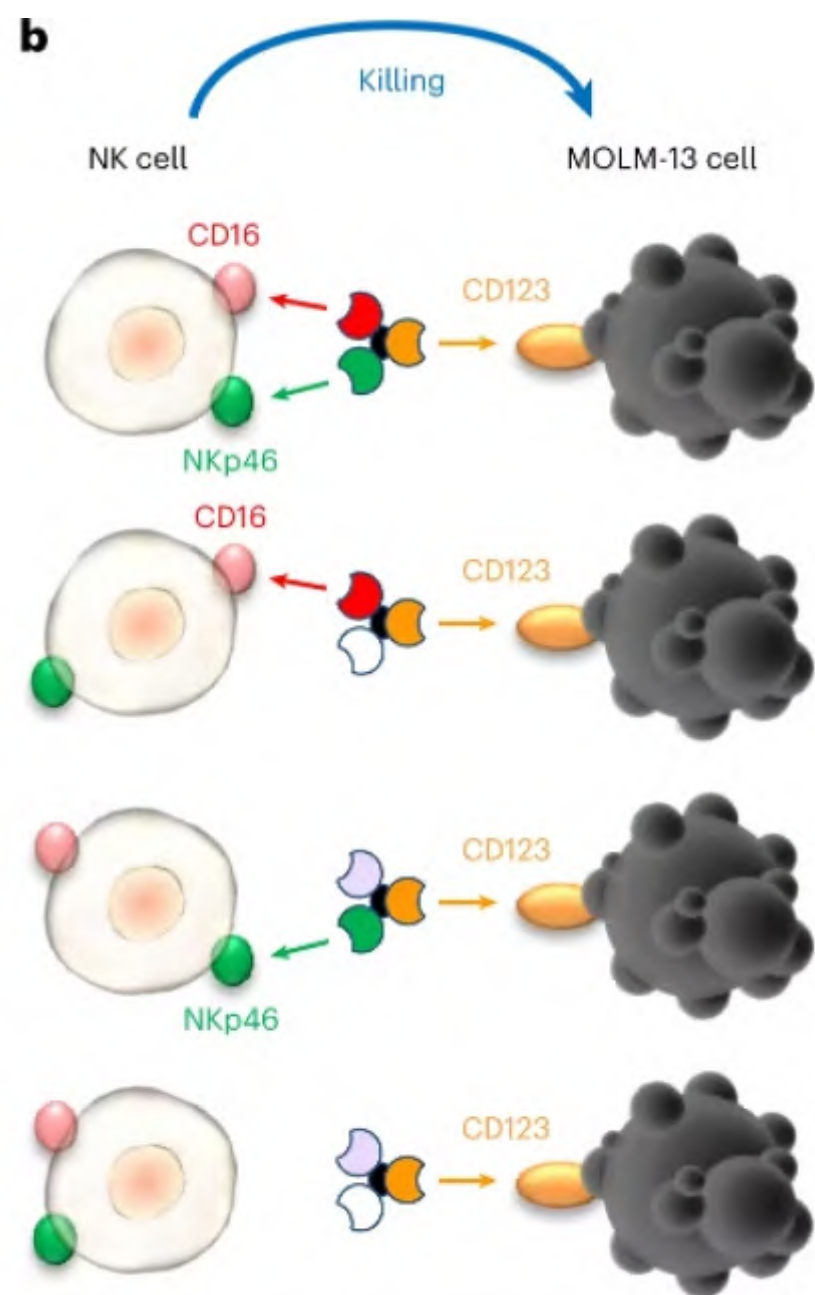
(2) Engaging Target 2



(3) Destruction of Tumor

[Watch Full Video](#)

NKp46 Drives Depth & Durability of Immune Response: A Major Advance Over Legacy NK Cell Engagers



- Receptor biology (not just half-life) drives deeper, more durable responses, *especially in solid tumors where prolonged time-on-target within the tumor microenvironment is essential.*
- Robust, persistent NKp46 target engagement (*for over 2 weeks*) drives serial killing.
- Consistent expression across hematological and solid tumors
- Improved NK cell trafficking to tumor micro-environment
- Reversal of NK cell functional exhaustion
- Pharmacodynamic (PD) effects lasting ≥ 10 days from a single dose in NHP.
- Dual-trigger signaling (CD16 + NKp46) increased cytotoxicity and compensates for CD16 low expression or shedding, particularly in immunosuppressive solid tumors

Flex-NK™ Cell Engagers

Show Significant Advantages Over T-Cell Engagers

NKp46 Activation Unlocks Immunotherapy Efficacy Across Tumors & Targets

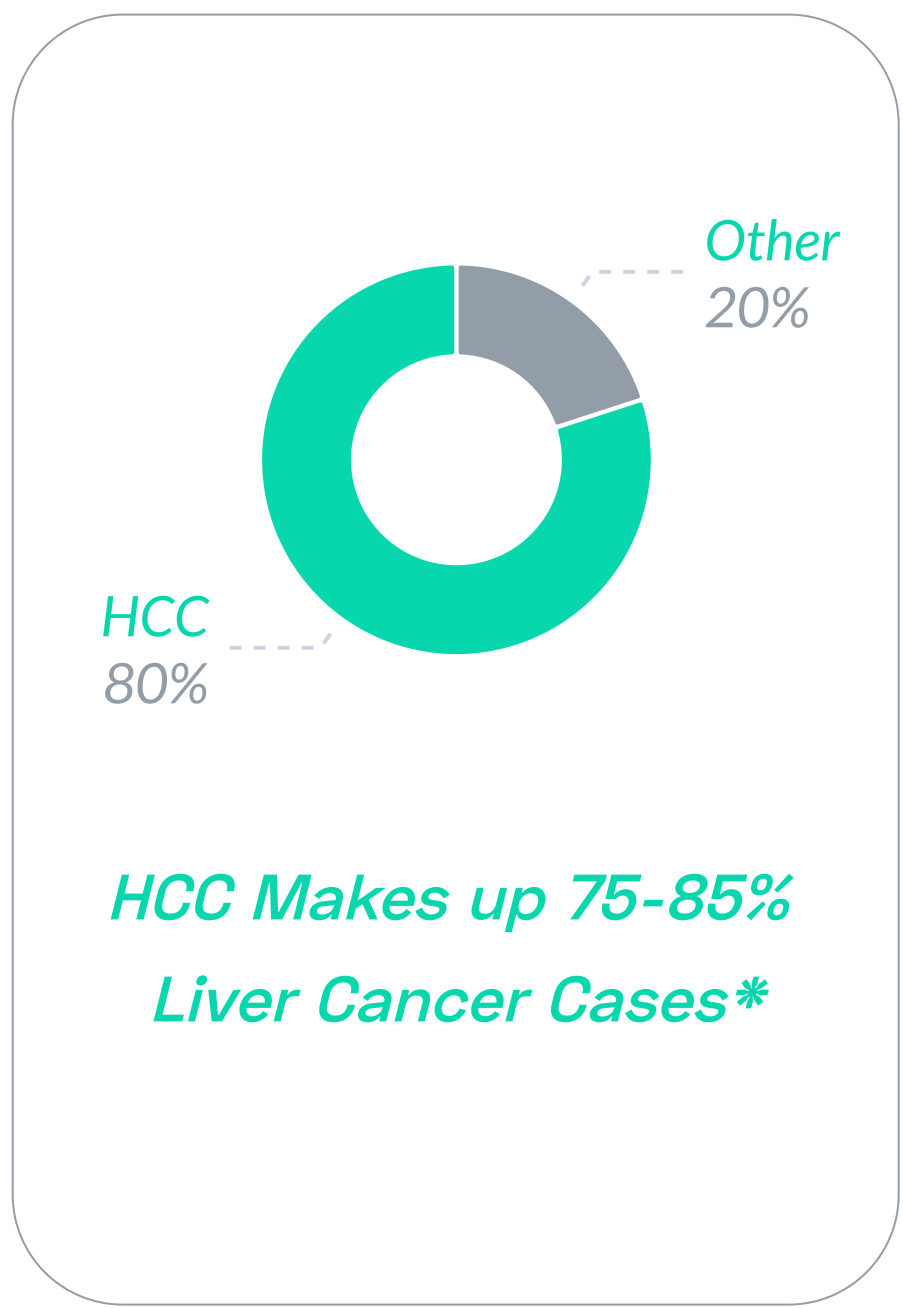
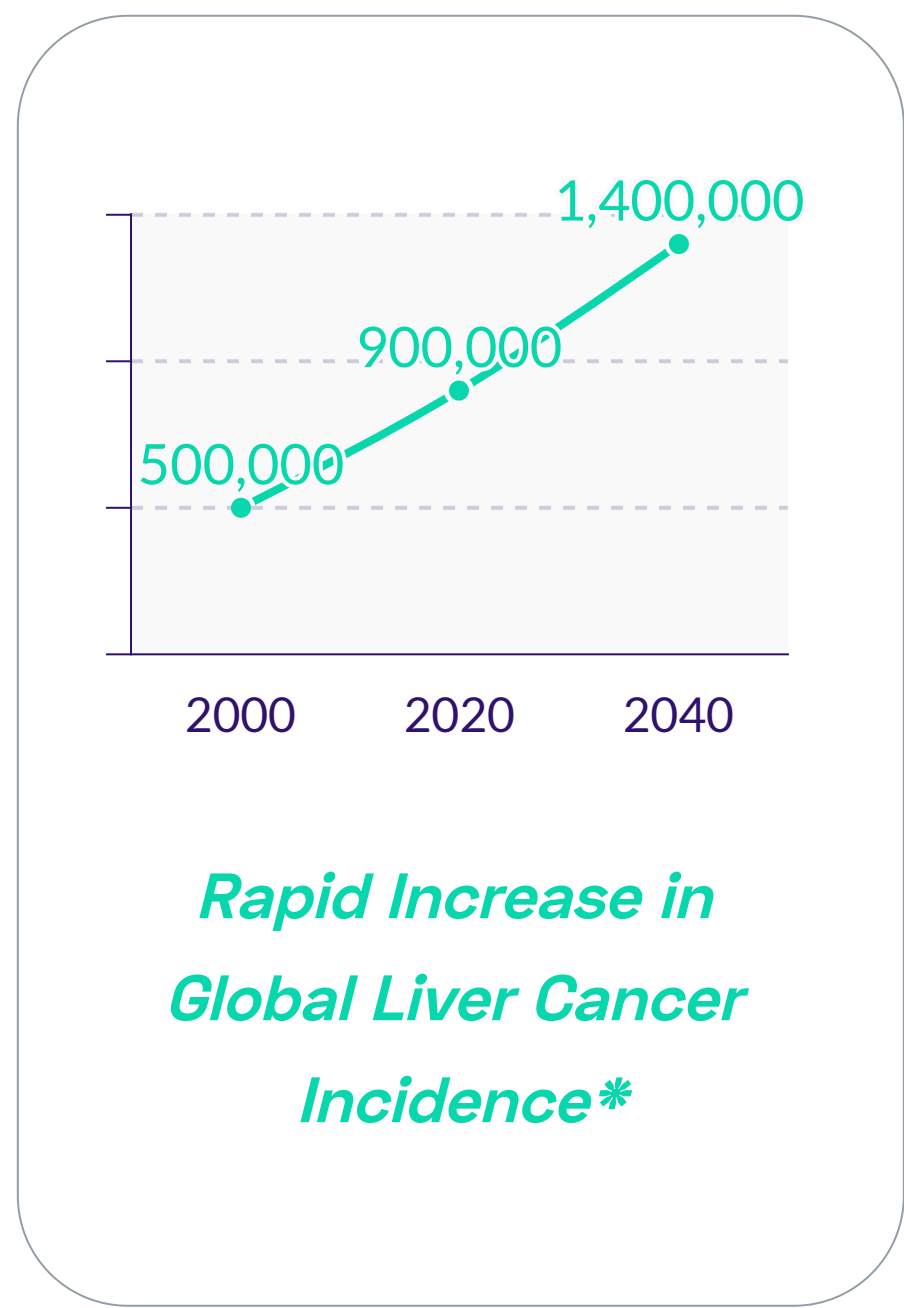
	<u>T-Cell Engagers (TCEs)</u>	<u>FLEX-NK™ Engagers (FNKEs)</u>
Mechanism of Action	T-Cell Redirection via CD3	NK Cell Redirection via NKp46 & CD16, Tumor Cell Apoptosis & Serial Killing via NKp46
Safety (CRS & Neurotoxicity)	High Risk	Minimal
Immune Exhaustion Risk	High T-Cell Exhaustion, Resulting in 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



Hepatocellular Carcinoma (HCC) Franchise

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

Large Addressable Market for Systemic Therapy



50-60%

of HCC Patients are Candidates for Systemic Therapy*

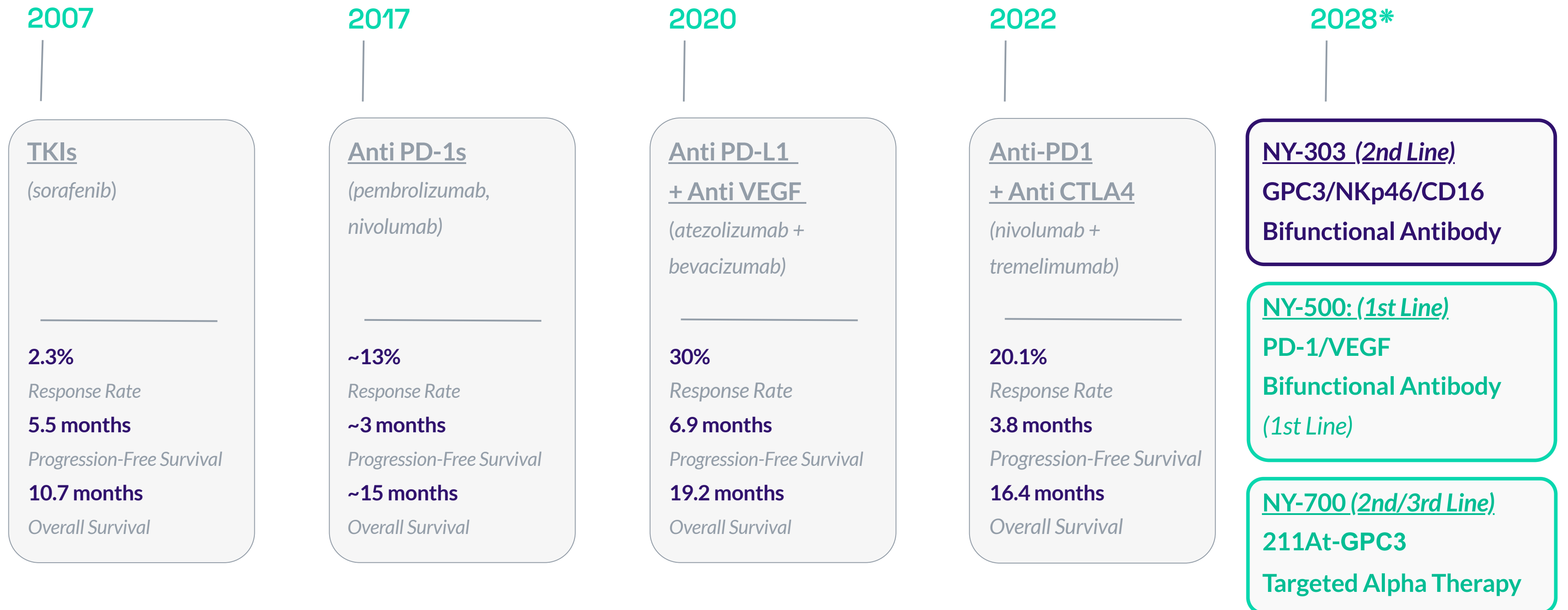
70-85%

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: First-in Class GPC3-Targeting Bifunctional Antibody Positioned as Monotherapy in Non-Responders to PD-1/VEGF Backbone Immunotherapy

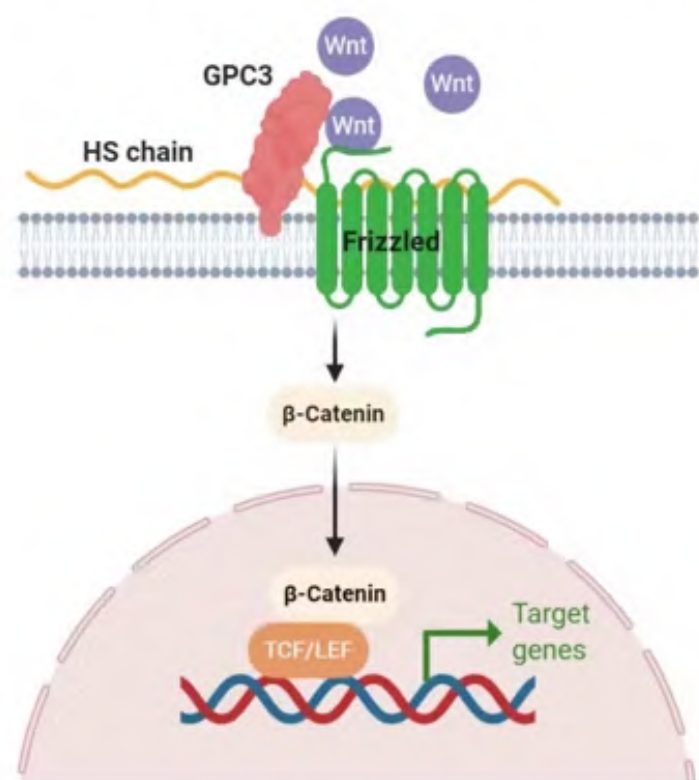


*Immune Checkpoint Inhibitors in Hepatocellular Carcinoma: Current Strategies and Biomarkers Predicting Response and/or Resistance (hyperlinked)

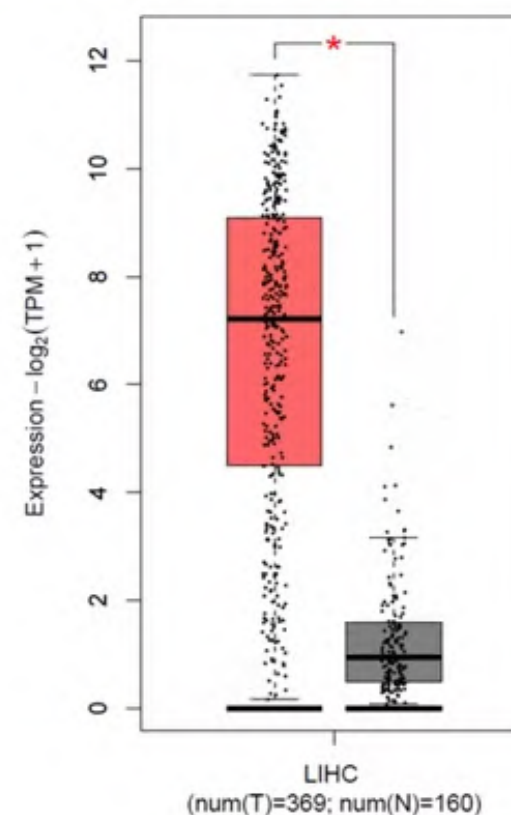
* projected

GPC3: One of the Most Promising Therapeutic Targets in HCC & Other Solid Tumors

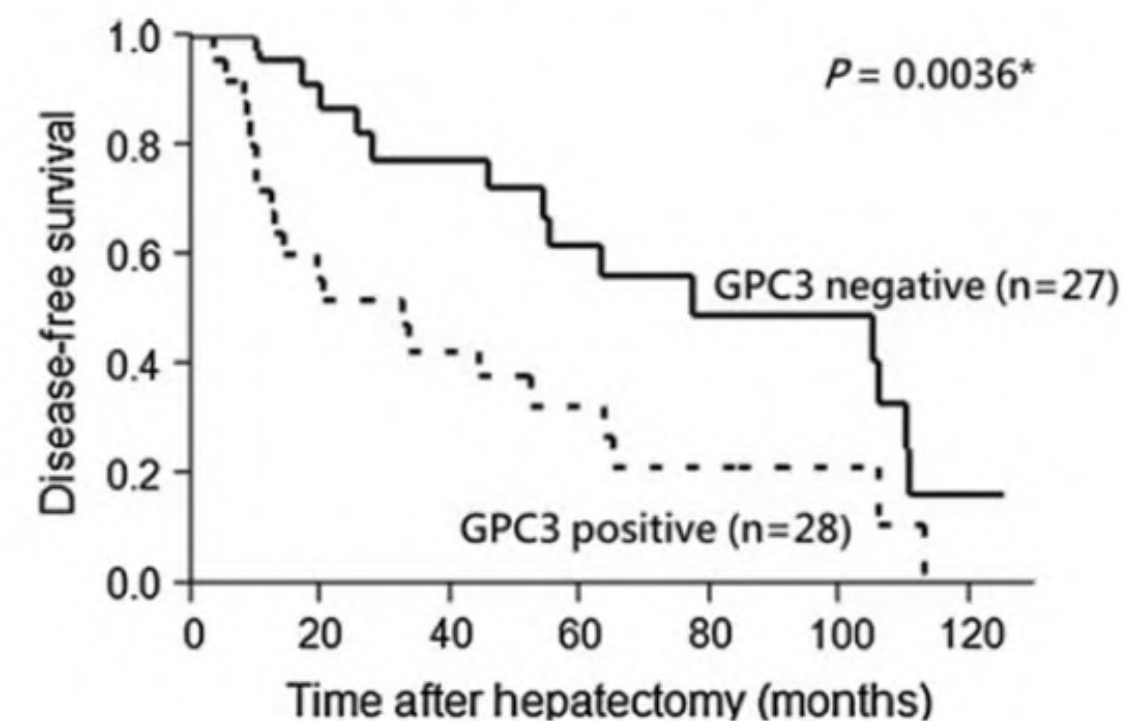
Glypican 3 (GPC3) is a cell surface protein playing a biological role in driving tumorigenesis in HCC.



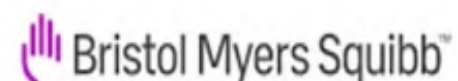
GPC3 is selectively expressed on tumor cells and is absent in normal tissue.



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



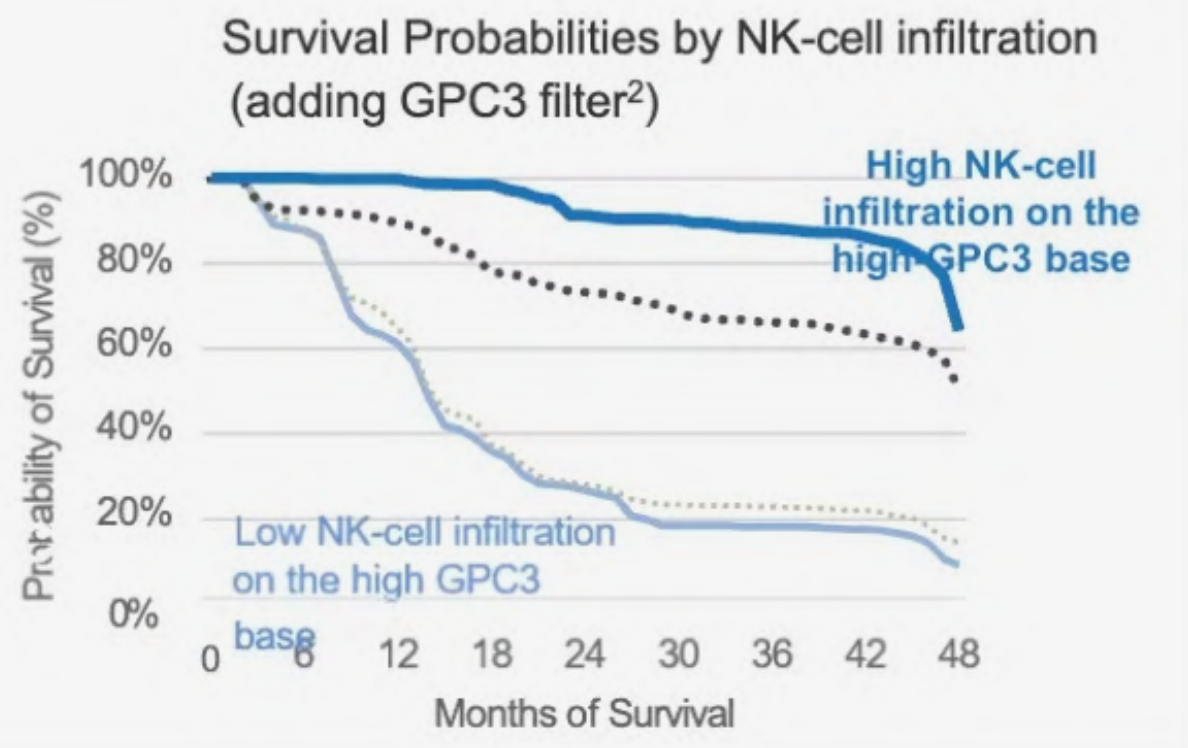
Major pharma companies with GPC3 targeted therapies in clinical development include:



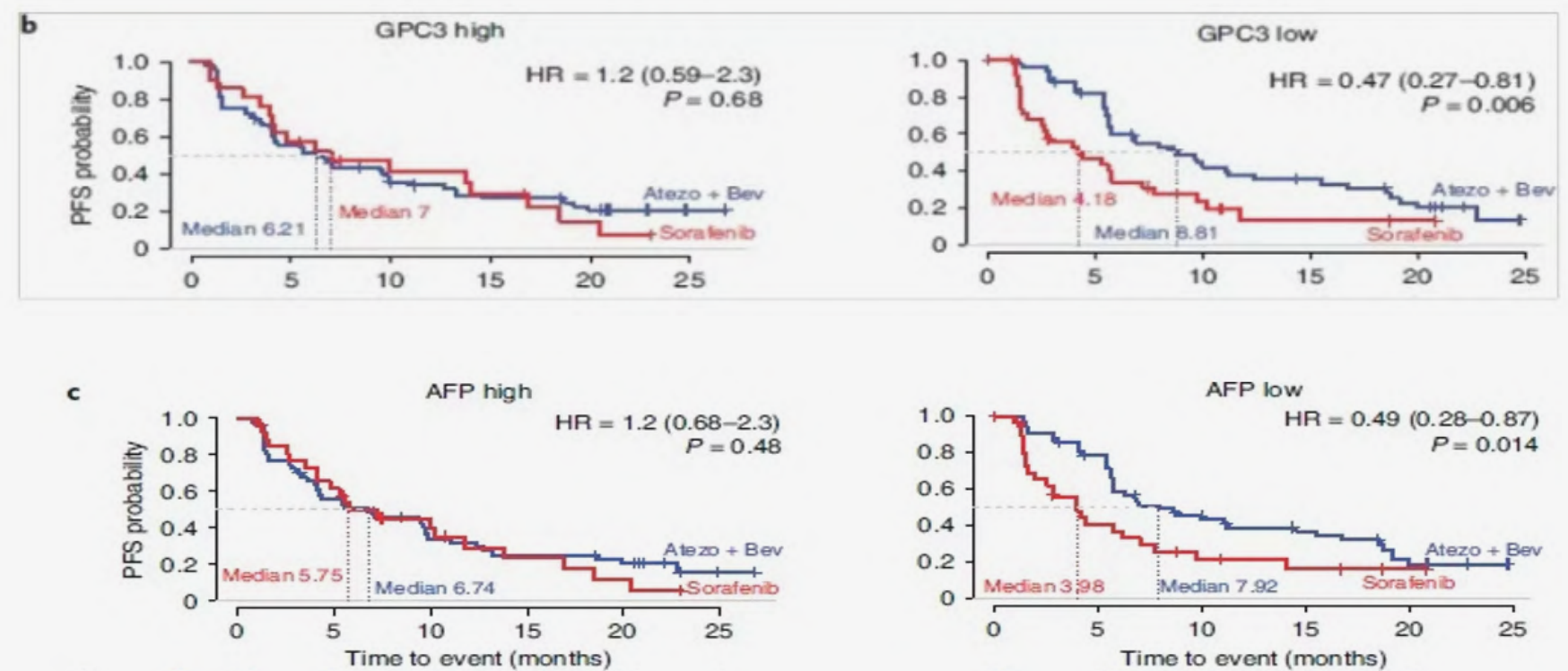
Additional indications for GPC3-targeting therapies NSCLC, Hepatoblastoma, Wilms Tumor, Malignant Rhabdoid Tumor, Yolk Sac Tumor, Rhabdomyosarcoma, Liposarcoma.

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



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



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 \geq 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

Redefining Standard of Care With NAYA's GPC3-Targeting NK Cell Engager and Comprehensive HCC Pipeline

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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



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>

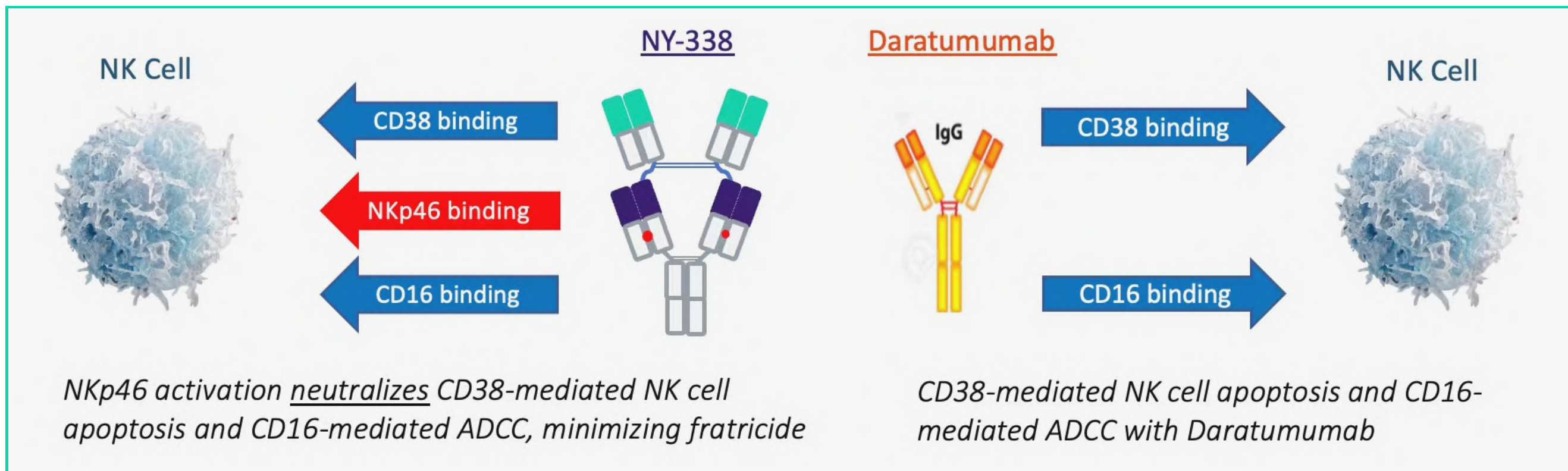
**<https://pubmed.ncbi.nlm.nih.gov/articles/PMC10618718/>

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



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

	<u>NY-338</u>	<u>Daratumumab</u>	<u>BCMA T-Cell Engagers (TCEs)</u>
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

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"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](#)



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	[Progress bar spanning Pre-Clinical, IND Enabling, and Phase I/II]		
NY-500	Hepatocellular Carcinoma (HCC) & Other Solid Tumors	PD-1 VEGF	FLEX Bifunctional Antibody	[Progress bar spanning Pre-Clinical and IND Enabling]		
NY-700	Hepatocellular Carcinoma (HCC) & Other Solid Tumors	At211-GPC3	Targeted Alpha Therapy	[Progress bar spanning Pre-Clinical and IND Enabling]		
NY-338	Multiple Myeloma, AML, Lymphoma	CD38 NKp46 CD16	FLEX-NK™ Bifunctional Antibody	[Progress bar spanning Pre-Clinical, IND Enabling, and Phase I/II]		

Seasoned, Entrepreneurial Management 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











Alexandra Urman, MPH



Bifunctional Antibody & Targeted Alpha Radioimmunotherapy Companies

Achieving Significant Market & Partnering Valuations

	PD-1 x VEGF (Oncology) Phase II/III	\$17.3B Market Cap* Data demonstrates superior efficacy to Keytruda®***
	PD-1 x VEGF (Oncology) Preclinical - IND Q4 '25/Q1 '26	\$140 Million Series A Financing Led By Orbimed, Avoro, and Samsara***
	PD-1 x VEGF (Oncology) Preclinical - IND Q4 '25/Q1 '26	Implied Valuation: \$518M** Reverse Merger with NASDAQ: GLYC, \$200 Million Financing***
	PD-1 x VEGF (Oncology) Phase I	Global License Acquired by Merck & Co, \$588M Upfront, \$2.7B Milestones***
	PSMA T-Cell Engager (Oncology) Phase I	\$1.84B Market Cap* Secondary Public Offering (\$400 Million)***
	Targeted Alpha Therapy (Oncology) Phase II	Acquired by AstraZeneca for \$2B + \$400M in Milesones
	Targeted Alpha Therapy (Oncology) Pre-Clinical	Acquired By Novartis for \$1B upfront + \$750M in milesones, Prior Novartis Acquisitions of Endocyte & AAA
	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 \$0.25 from 12/31/24

*** based on company press releases (hyperlinked)

NAYA's Strategic Pathway Towards Achieving Significant Valuation

**Innovative
Bispecific Antibody
& Targeted Alpha
Therapy
Modalities**

**Strategic HCC-
Focused Franchise
with Robust,
Differentiated
Pipeline**

**Lean, Accelerated
Execution Led By
Industry Veterans**

**Early Partnering
Optionality &
Scalability to
Additional
Therapeutic Areas**

**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*

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