NAYA BIOSCIENCES

Corporate Presentation

February 2025

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NAYA Biosciences (NASDAQ: NAYA)

A Portfolio Company with Multiple Bifunctional Antibody Clinical Milestones

Competitive Bifunctional Antibody Pipeline

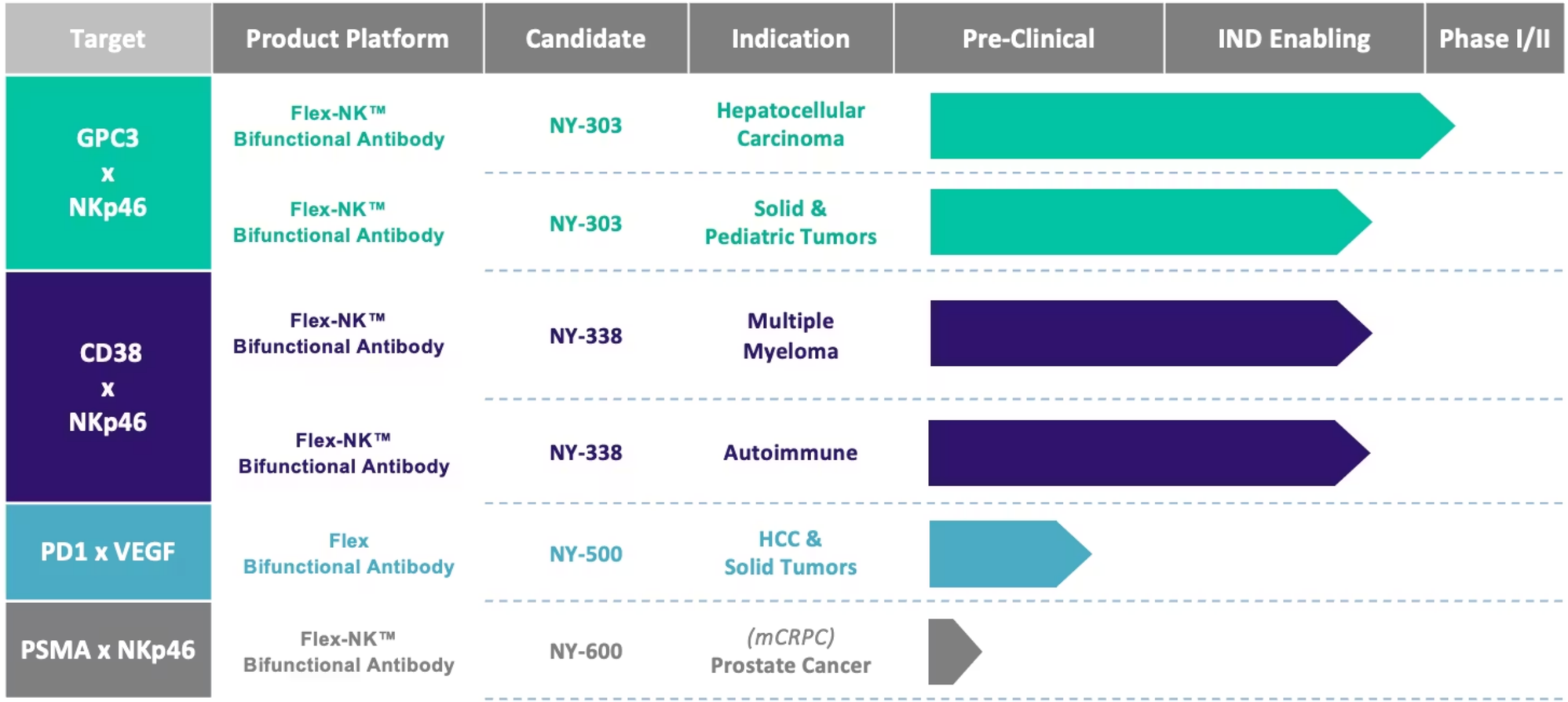
- *Multiple clinical readouts in 2025-2027*
- *Potential for leadership in hepatocellular carcinoma, multiple myeloma, prostate cancer, and autoimmune diseases*

Validated Hub & Spoke Model

- *Supports acquisition, development, and partnering of high-potential assets*

Promising Therapeutic Portfolio with 2025-2027 Clinical Milestones

Validation of Data at Major Oncology Meetings including AACR, ASH, EHA, ESMO, and SITC



Companies Engaged in the Development of Bifunctional Antibody Therapeutics

Summit therapeutics	PD1 x VEGF *(Oncology)* Phase II/III	$13.16 Billion Market Cap* *Data demonstrates superior efficacy to Keytruda®******
OTTIMO PHARMA	PD1 x VEGF *(Oncology)* Preclinical - *IND Q4 '25/Q1 '26*	$140 Million Series A Financing Led By Orbimed, Avoro, and Samsara******
CRESCENT BIOPHARMA	PD1 x VEGF *(Oncology)* Preclinical - *IND Q4 '25/Q1 '26*	Implied Valuation: $518 Million** Reverse Merger with NASADAQ: GLYC, $200 Million Financing******
礼新医药 LaNova Medicines	PD1 x VEGF *(Oncology)* Phase I	Global License Acquired by Merck & Co, $588 Million Upfront, $2.7 Billion Milestones******
Vir Biotechnology™	T-Cell Engager *(Oncology)* Phase I	$1.4 Billion Market Cap, 60% Stock Price Increase Upon Phase I Data
Merus	EGFR x LGR5 *(Oncology)* Phase II/III	$2.9 Billion Market Cap*
JANUX	PSMA T-Cell Engager *(Oncology)* Phase I	$3.1 Billion Market Cap* Secondary Public Offering ($400 Million)******
BICARA THERAPEUTICS™	EGFR/TGF-β *(Oncology)* Phase I/IIa completed	$948M Market Cap*, $315 Million IPO @ $1.3B Post-Money******

**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)*

Included for informational purposes only. The performance, capital raising and market capitalizations / valuations of the foregoing companies is neither indicative nor predicative of the results of our company. We advise you to carefully review our "Risk Factors" which are included in our filings with the SEC.

Seasoned Entrepreneurial Leadership Team

Steven M. Shum
Chief Executive Officer

Daniel Teper, PharmD, MBA
President

Andrea Goren, MBA
Chief Financial Officer

Dan Chiche, MD
Chief Medical Officer

Anna-Baran Djokovic, LLM
SVP, Investor Relations

Armin Rath, PhD
SVP, Product Development

Lyn Falconio
Head, NAYA Women's Health

Ravi Kiron, PhD
Head of Strategic Innovation

Vidisha Mohad, PhD
Entrepreneur-in-Residence,

Chorom Pak, PhD
Entrepreneur-in-Residence,

Senior Advisors To Accelerate Strategic Growth

Strategic Advisors

Laurent Audoly, PhD

pieris · KYMERA · Pfizer · MERCK · MedImmune

Prakash Raman, PhD

NOVARTIS · Flagship Pioneering

Gilles Seydoux, PharmD

gsk · Pfizer

Mark Rothera, MBA

gsk · Orchard therapeutics · Shire

Rahul Singhvi, DSc, MBA

MERCK · Takeda · Flagship Pioneering

Alexandra Urman, MPH

Stanford University · ARK INVEST · Memorial Sloan Kettering Cancer Center

Melissa Fensterstock, MPhil, MBA

COLUMBIA TECHNOLOGY VENTURES · JOHNS HOPKINS TECHNOLOGY VENTURES · YALE VENTURES · material impact

Scientific Advisors

Michael Caliguiri, MD

City of Hope · RESEARCH · TREATMENT · CURES

Ola Landgren, MD, PhD

Memorial Sloan Kettering Cancer Center · SYLVESTER Comprehensive Cancer Center · UNIVERSITY OF MIAMI HEALTH SYSTEM

Josep Lovett, MD, PhD

Mount Sinai MEDICAL CENTER

Yaron Ilan, MD

HADASSAH UNIVERSITY MEDICAL CENTER

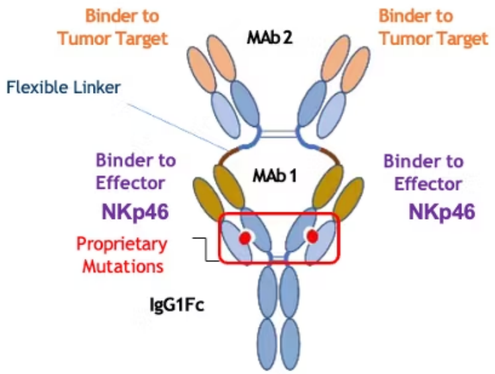
Jean Kadouche, PhD

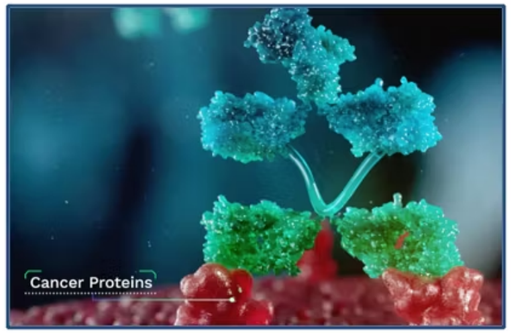
genzyme · J&J · INSTITUT PASTEUR

NAYA THERAPEUTICS

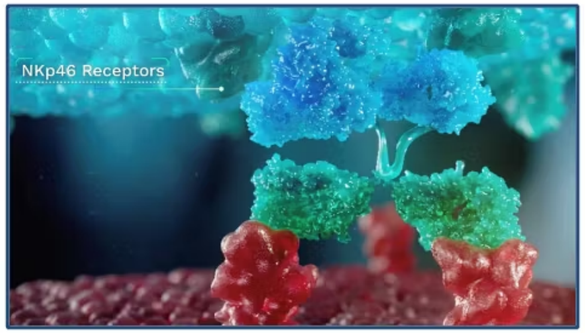
Solid Tumors Franchise

Enhanced Tumor Killing with NAYA's Bifunctional Antibodies

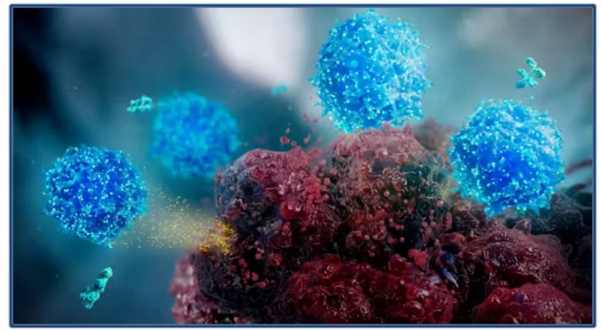




(1) Engaging Cancer Cells



(2) Engaging NK Cells Through NKp46

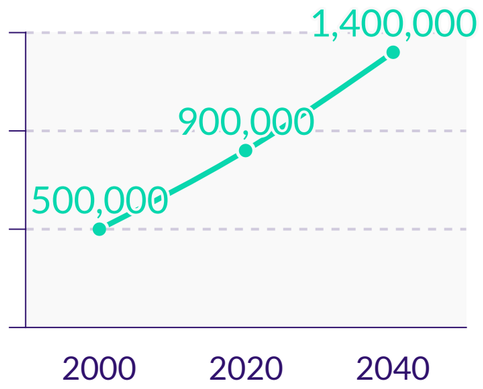


(3) Destruction of Tumor

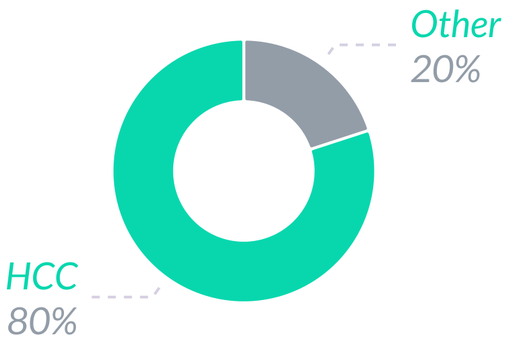
- **Proprietary, differentiated design** *(tetravalent, flexible linker, full Fc)*
- **Validated manufacturing process** *with high yields for GMP clinical trial batches*
- **Immune synapse effect** *through simultaneous attachment to both natural killer & targeted cancer cells*
- **Improved & sustained engagement** *of natural killer cells in tumor microenvironment through NKp46 activating receptors*
- **Demonstrated reversal of NK cell dysfunction**
- **Unique mechanism turns "cold" tumors "hot"**, *making them more susceptible to immunotherapy*

HCC: A Highly Prevalent Cancer Globally with Limited Therapeutic Options

Large Adressable Market for Systemic Therapy



Rapid Increase in Liver Cancer Incidence*



HCC Makes up 75-85% Liver Cancer Cases*

50-60%

of HCC Patients are Candidates for Systemic Therapy*

70%

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)*

Rapidly-Evolving HCC Treatments Favor Combination Therapy & Bifunctional Antibodies

High Unmet Medical Need for 70% of Non/Partial Responders Despite Significant Advances with New Standard of Care (Atezo-Bev)

2007

TKIs
(sorafenib)

2.3%
Response Rate
5.5 months
Progression-Free Survival
10.7 months
Overall Survival

2017

Anti-PD1s
(pembrolizumab, nivolumab)

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

2020

Anti-PDL1 + 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*

PD1 x VEGF Bifunctional Antibody

GPC3 x NKp46 Bifunctional Antibody

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

* projected

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

NY-500

PD1 x VEGF

Flex Bifunctional Antibody

Next-Generation First-Line Immunotherapy as Alternative to Current Standard of Care (Tecentriq + Avastin™)

- AI-optimized bifunctional antibody synergistically blocks PD1 *(immune checkpoint)* & VEGF *(regulation of vessel formation)*
- Benefits of PD1 x VEGF approach demonstrated by recent *ivonescimab* Phase III clinical trials in Lung Cancer
- Initiating phase I/II in HCC in 2026
- *Other potential indications include lung, colorectal, renal cancers*

NY-303

GPC3 x NKP46

Flex-NK™ Bifunctional Antibody

Next-Generation Second-Line Therapy for Non/Partial Responders to Current Standard of Care (Tecentriq + Avastin™)

- Directly addresses biology of non-responders by targeting both GPC3 & NK cells and making tumors sensitive to PD1 Checkpoint blockade, turning tumors from "cold" to "hot"
- Initiating phase I/II in HCC in 2025
- Potential phase II/III accelerated development in 2027
- *Other potential indications include ovarian, lung, pediatric cancers*

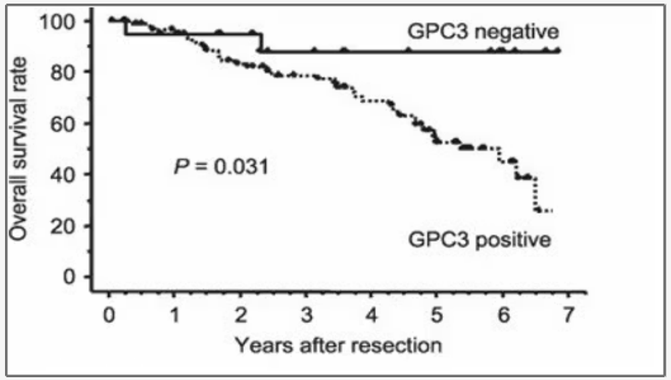
Llovet, J.M., Kelley, R.K., Villanueva, A. et al. Hepatocellular carcinoma. Nat Rev Dis Primers 7, 6 (2021).
***Han JW et al: Predicting Outcomes of Atezolizumab and Bevacizumab Treatment in Patients with Hepatocellular Carcinoma. Int J Mol Sci. 2023 Jul 22;24(14):11799.*

Targeting HCC with GPC3 & NK Cells with NKp46

GPC3*: A Promising Target

Glypican 3 (GPC3) is an oncofetal protein expressed on 80% of HCC cells & 30-50% of other solid tumors, while predominantly absent in normal adult tissue.

GPC3 blood levels correlate with severity of HCC



NKp46: A Preferred NK Cell Activator**

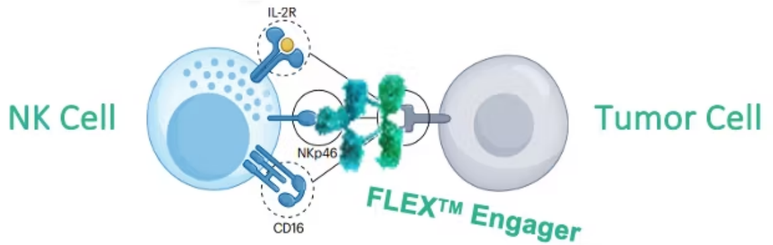
NKp46 is a preferred activating receptor to induce NK cell-mediated immunity in solid tumors.

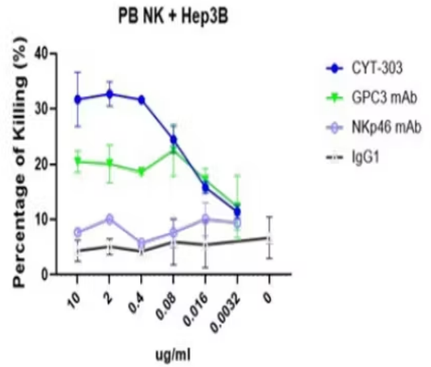
- Primary driver of natural NK cell cytotoxicity
- Shows sustained expression on NK cells in the tumor micro-environment (TME) while other activating receptors can be down-regulated
- NKp46 binding induces reversal of NK cell exhaustion
- NKp46-induced activation of NK cells drives an increase in infiltrating NK cells in the TME

**Guo M et al. Glypican-3: A New Target for Diagnosis and Treatment of Hepatocellular Carcinoma. J Cancer. 2020 Feb 3;11(8):2008-2021.*
**Glypican 3 Overexpression across a Broad Spectrum of Tumor Types Discovered …Kirsten L. et al, The American Journal of Pathology, Sept 2018*
*** Recognition and Prevention of Tumor Metastasis by the NK Receptor NKp46/NCR (hyperlinked)*
***Multifunctional Natural Killer Cell Engagers Targeting NKp46 Trigger Protective Tumor Immunity (hyperlinked)*

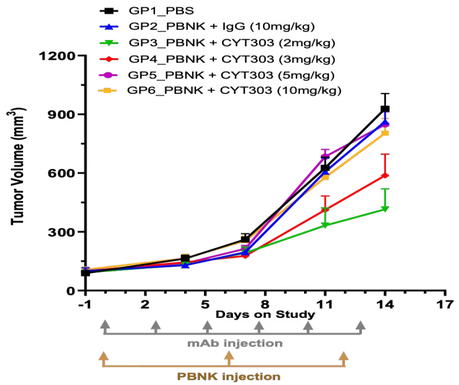
NY-303 Pre-Clinical Proof of Concept & Unique Reversal of NK-Cell Dysfunction

Immune Synapse:

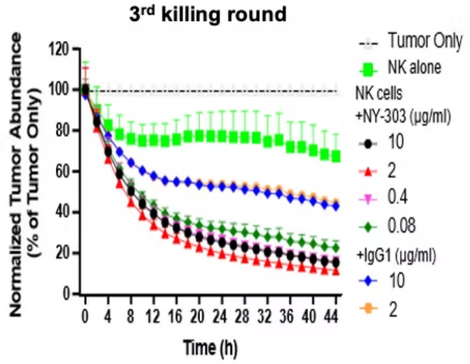




In Vitro
Killing Assay



In Vivo
Tumor Model

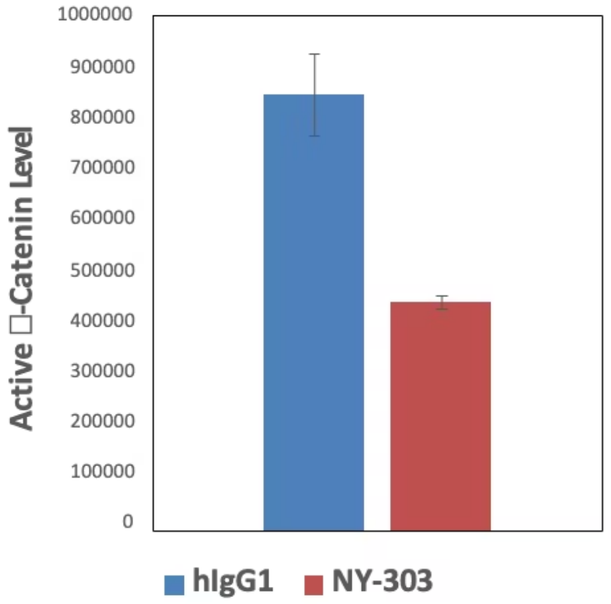


Reversal of NK Cell Dysfunction

Khairnar V, Li W, Rath A, et al, Journal for ImmunoTherapy of Cancer 2024;12:doi: 10.1136/jitc-2024-SITC2024.1074 (hyperlink)

NY-303 Turns “Cold” Tumors “Hot” by Reducing Active β-Catenin Levels

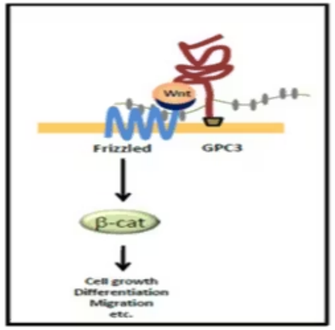
Reduced β -Catenin Levels



sitc
Society for Immunotherapy of Cancer

- GPC3 Makes HCC Tumors “Cold” (Non-Susceptible for Immunotherapy)
- By Reducing Reduced β -Catenin Level, NY-303 Makes them “Hot” (Susceptible), Facilitating Immune Cell Infiltration in Tumor Microenvironment

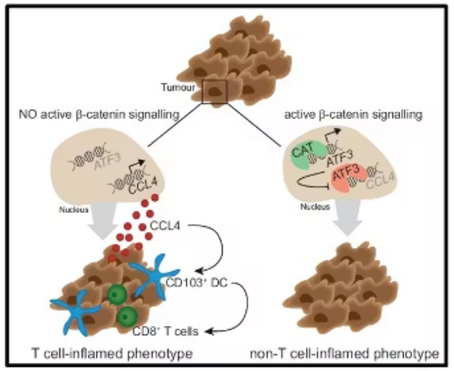
GPC3 induces HCC proliferation via Wnt/β-catenin pathway



(Ho M *et. al.*, 2011)

- High tumor GPC3 levels are known to be associated with increased Wnt/β-catenin oncogenic signaling resulting in HCC tumor proliferation

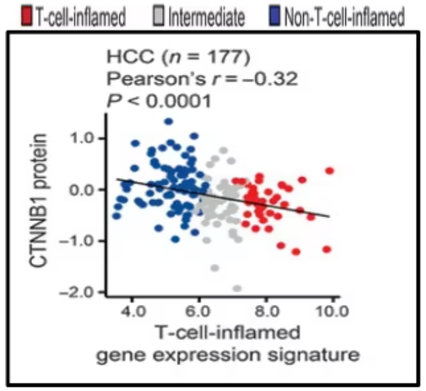
Wnt/β-Catenin activation results in T cell exclusion in TME



(Spranger T and Gajewski T *et. al.*, 2015)

- Wnt/β-catenin signaling activates ATF-3 transcription factor that <u>represses</u> chemokine (CCL4) production and DC migration to tumor resulting in T cell exclusion in TME

High β-catenin associated with non-inflamed T cells in HCC TME



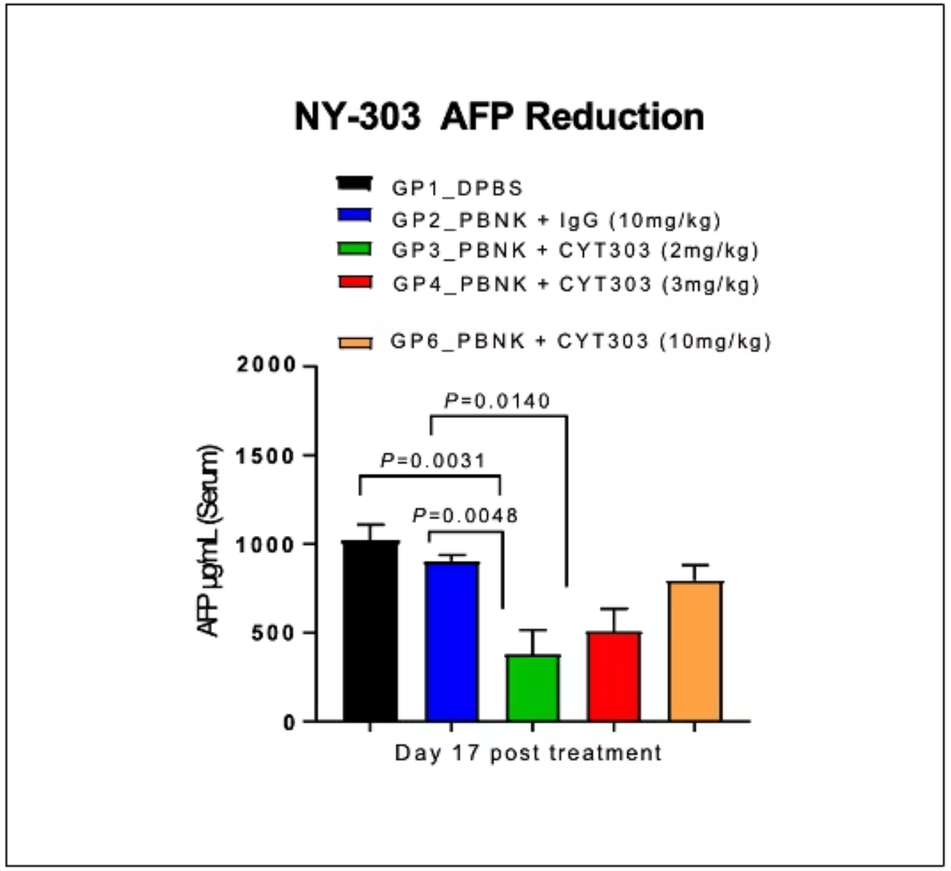
(Luke J and Gajewski T *et. al.*, 2019)

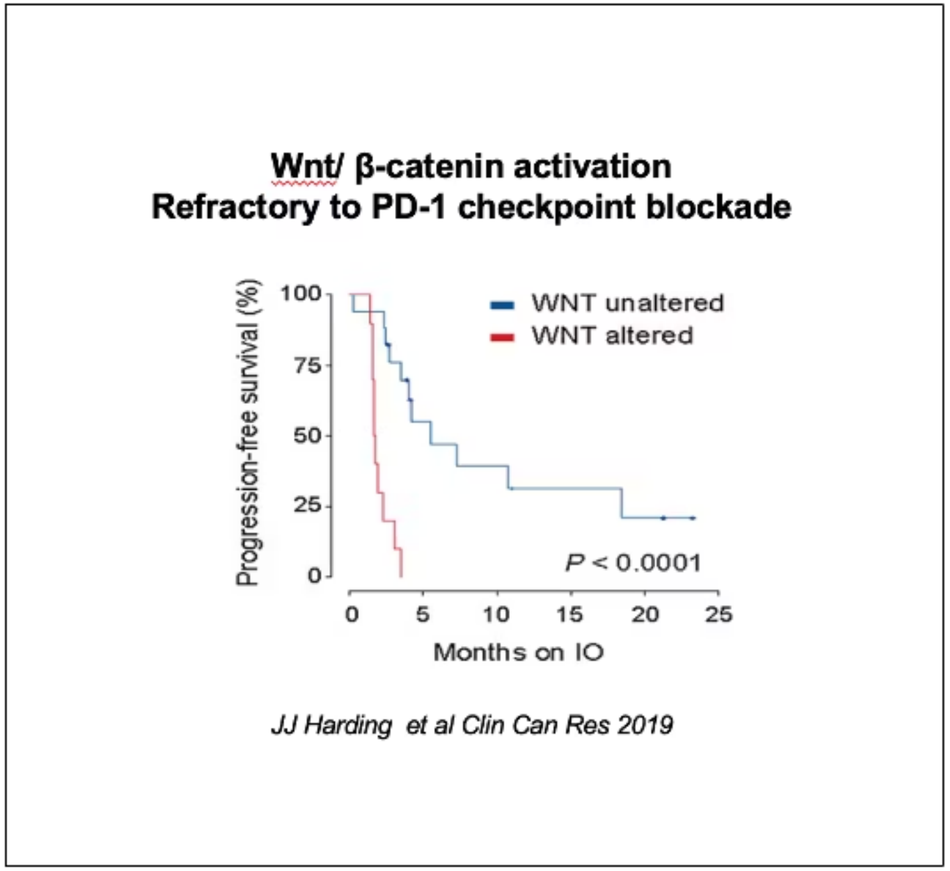
- Increased beta catenin levels show reduced T cell inflamed signatures in HCC and other solid tumors (TCGA data)

Khairnar V, Li W, Rath A, et al, Journal for ImmunoTherapy of Cancer 2024;12:doi: 10.1136/jitc-2024-SITC2024.1074 (hyperlink)

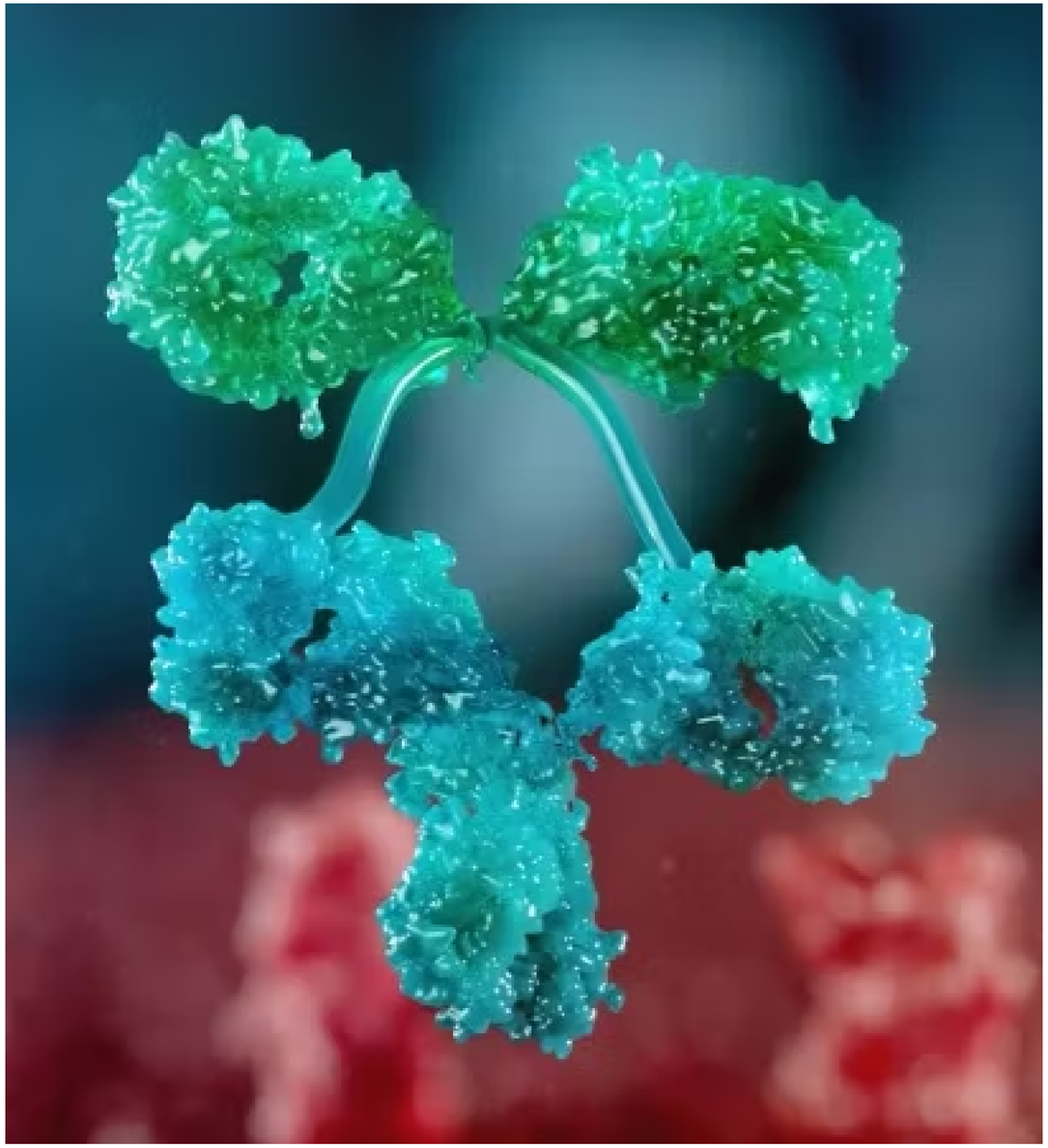
NY-303 Enables Response in First-Line Therapy (PD-1 Checkpoint) Refractory Patients

NY-303 Down-Modulates GPC3 by Inhibiting AFP & Making the Tumor Susceptible by Inhibiting β-Catenin





CYT-303 FLEX-NK™engager dose response efficacy mechanisms in HCC tumor model and safety in cynomolgus monkey toxicology studies support clinical evaluation in hepato-cellular carcinoma, Vishal et al, AACR 2022 Poster (hyperlink)



NY-303 Data Supports H1 2025 Initiation of Phase I/IIa Clinical Trials

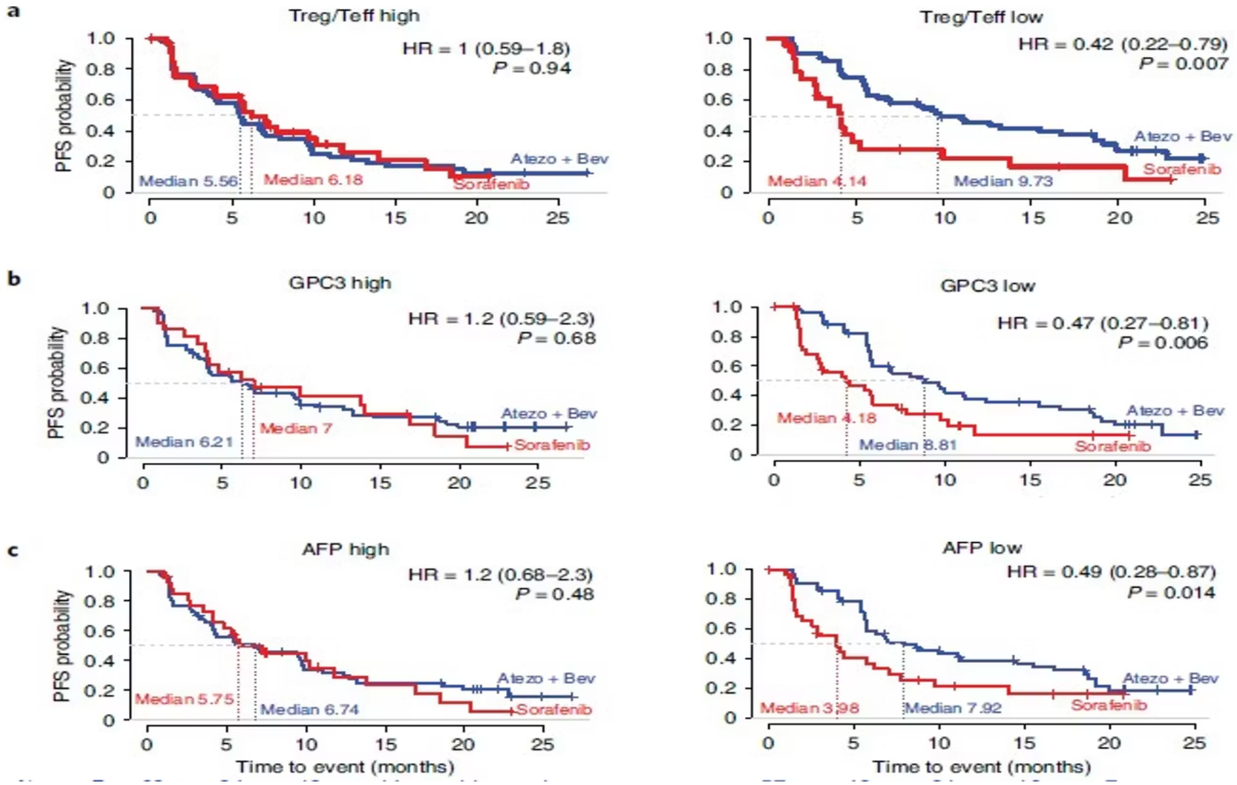
- Ability to Redirect NK Cells to Kill HCC Tumors Cells
- Enhanced Killing Ability & Reversed Dysfunction of NK Cells
- Improved Tumor Growth Inhibition & Dose-Response in HCC Tumor Models
- Turning “Cold” (Non-Susceptible) Tumors Into “Hot” (Susceptible) Tumors, Facilitating Invasion of Immune Cells to Tumors & Making Them Sensitive to PD1 Checkpoint Blockade
- No Toxicity at up to 20 Times Expected Therapeutic Dose
- Favorable FDA Feedback on CMC & Tox
- PK Supports Weekly Administration in Patients

Khairnar V, Li W, Rath A, et al, Journal for ImmunoTherapy of Cancer 2024;12:doi: 10.1136/jitc-2024-SITC2024.1074
Cancer Res (2023) 83 (7_Supplement): 5667 https://doi.org/10.1158/1538-7445.AM2023-5667

Targeting GPC3 to Enable Response in Patients Refractory to First Line Immunotherapy HCC Treatment (Tecentriq® + Avastin®)

Tecentriq® + Avastin Checkpoint Non-Responders

Tecentriq® + Avastin Checkpoint Responders



Since NY-303 can activate NK cells & reduce HCC tumor burden, high GPC3 & AFP levels can be converted to low GPC3 & AFP signatures that are repsonsive to Atezo + Bev 1st line immunotherapy

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

Phase I/IIa to Evaluate NY-303 as Monotherapy in 2nd Line HCC Patients at Leading Israeli Academic Centers, with 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 **H1 2025** and data by **H1 2026**)*
  - **Phase IIa** *to expand to at academic centers in the US, Europe and Asia starting in 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*

NAYA's AI-Optimized PD1 x VEGF Bifunctional Antibody Aiming for HCC Monotherapy Clinical Readouts in 2026

PD1 x VEGF Bifunctional Antibodies Expected to Surpass Standard Anti-PD(L)1 Antibodies in Multiple Oncology Indications

Large Market

$58B

*Forecasted Value of 2025 PD(L)1 Market**

$27.2B

*Keytruda ® (pembrolizumab) 2024 sales***

40

*FDA-approved indications for Keytruda®****

Impressive Clinical Benefits

Anti-PD(L)1 antibodies significantly prolong survival*****, shifting immunotherapy to first-line

Ivonescimab (PD1xVEGF) is the first drug to demonstrate superiority in progression-free survival over pembrolizumab *(in a randomized phase 3 in First Line (1L) Non-Small-Cell Lung Cancer)*

Opportunity for NAYA to Lead in HCC

Lead PD1 x VEGF antibodies in Phase III, with focus on Lung & Breast Cancer *(Summit Therapeutics, BioNTech)*

Rapid followers entering Phase I in 2026 *(Ottimo, Crescent, Merck & Co)*

NAYA to target clinical data readouts (2026) with NY-500 in 1L HCC

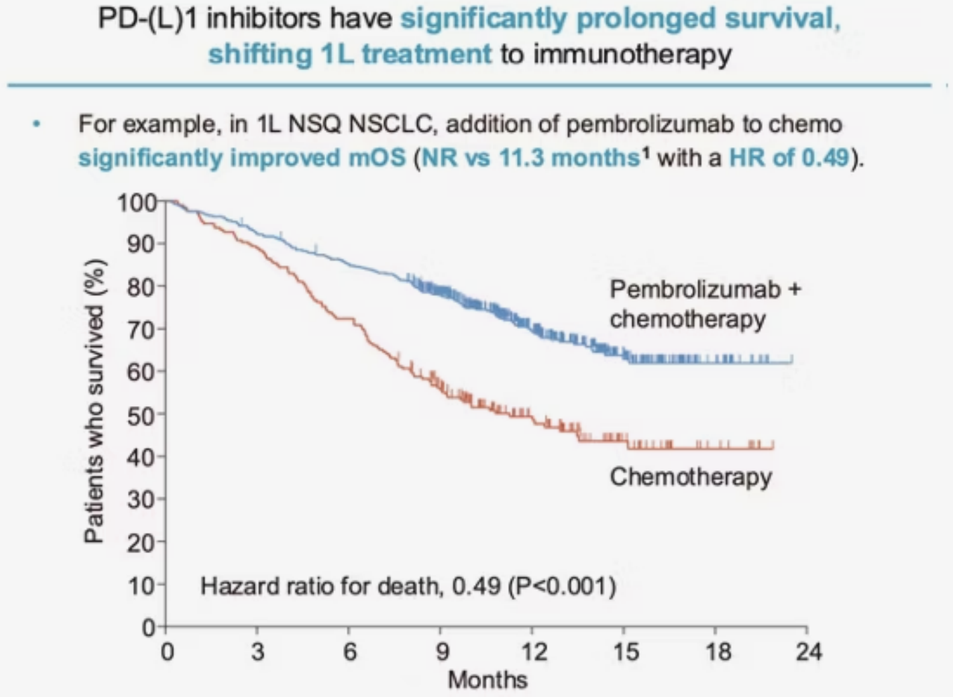
NAYA to leverage development synergies with clinical development of NY-303 in 2L HCC, non-responders to PD(L)1

** IQVIA (hyperlink)*
***Leading drugs worldwide based on projected 2024 sales (hyperlink)*
****Cancer Research (hyperlink)*
*****Annals of Oncology (hyperlink)*

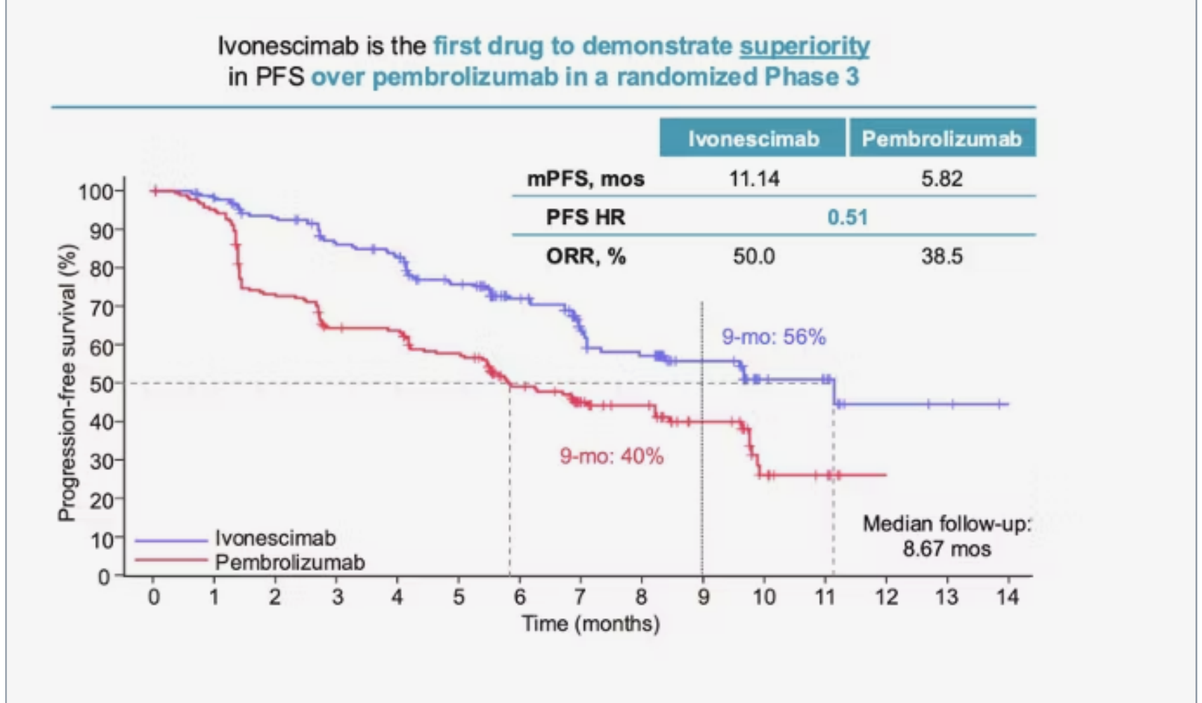
Included for information purposes to demonstrate the current market for the standard anti-PD(L)1 Antibodies cancer treatment. There is no guarantee that PD1 x VEGF bifunctional antibodies will surpass the standard of care demonstrated by Keytruda, or at all, and even if it does surpass the standard of care, there is no guarantee that sales in PD1 x VEGF bifunctional antibody products generally, or sales in our product candidate, if ever approved, and if any, will surpass those of Keytruda

First PD1 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



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

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

NAYA THERAPEUTICS

Immunotherapy Franchise

NY-338: A Promising CD38-Targeting Bifunctional Antibody for Both Multiple Myeloma & Autoimmune Diseases

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 both monoclonal & T-cell bifunctional antibodies, including:

- ☑ **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**

Emerging Opportunity for CD38 in High-Growth Autoimmune Disease Market

Use of CD38 antibodies for autoimmune diseases validated, with:

Early clinical data from Daratumumab (J&J) & Felzartamab (HI-Bio)

*$1.8B Acquisition of HI-Bio by Biogen***, based on Phase II clinical data with felzartamab in orphan autoimmune diseases*

CD38 T-cell engagers in autoimmune diseases a top development priority for IGM Biosciences

*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/

Included for informational purposes only. The performance and market capitalizations / valuations of the foregoing companies is neither indicative nor predicative of the results of our company. We advise you to carefully review our "Risk Factors" which are included in our filings with the SEC.

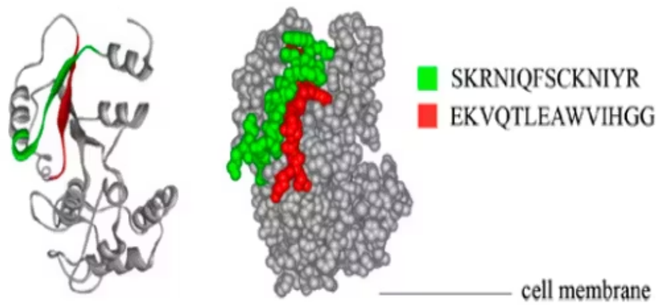
NY-338 Has Overlapping + Distinct Epitope Binding Sites and Gene Expression Profiles Compared to Daratumumab

Differentiated Epitope Binding Sites *

Only CD38 mAb binds

Only Dara binds

CD38 mAb and Dara bind



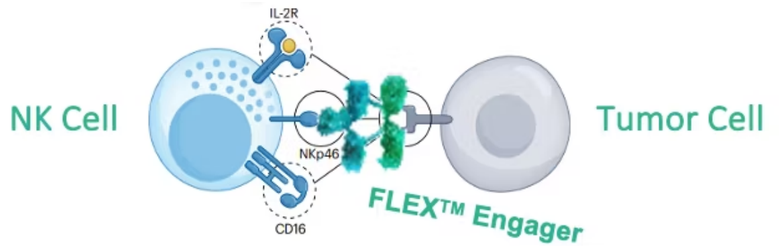
Differentiated Gene Expression Profiles **

Pathways	Genes	NY-338	Dara
Innate immunity and JAK/STAT cytokine Signaling	GZMK	+	+
	IFI35	+	+
	STAT 1,2,3,4	+	+
Inhibition receptors	TIGIT	+	+
	KIR3DL2	-	-
Lymphocyte activation Receptors	CD96	+	-
	CD244	+	-
	CD2	+	-
Leukocyte migration and chemotaxis	PECAM-1	-	+
	CCL8	-	+
	CXCL1	-	+

Source: Daratumumab from Literature, NY-338 from INternal NAYA Data
**M Weers et al. J. Immunol 2011; H Lee et al*
***Biochem Biophys Res Comm 2021*

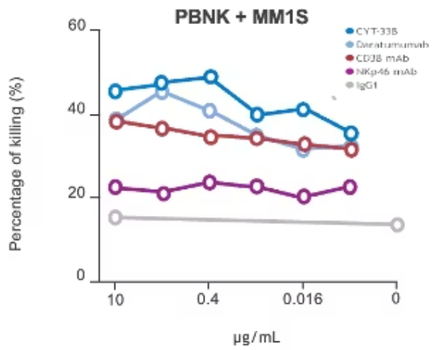
NY-338 Pre-Clinical Proof of Concept & Unique Reversal of NK Cell Dysfunction

Immune Synapse:



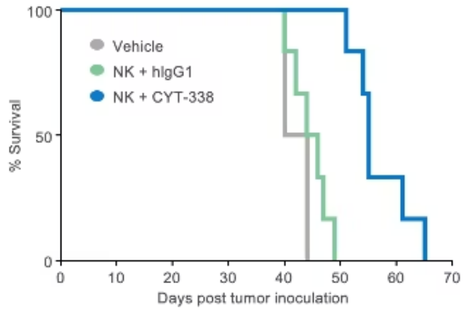
Superior Killing vs First Line

In Vitro Killing Assay



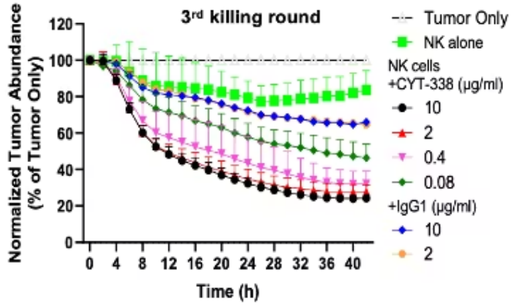
Improved Survival

In Vivo tumor model



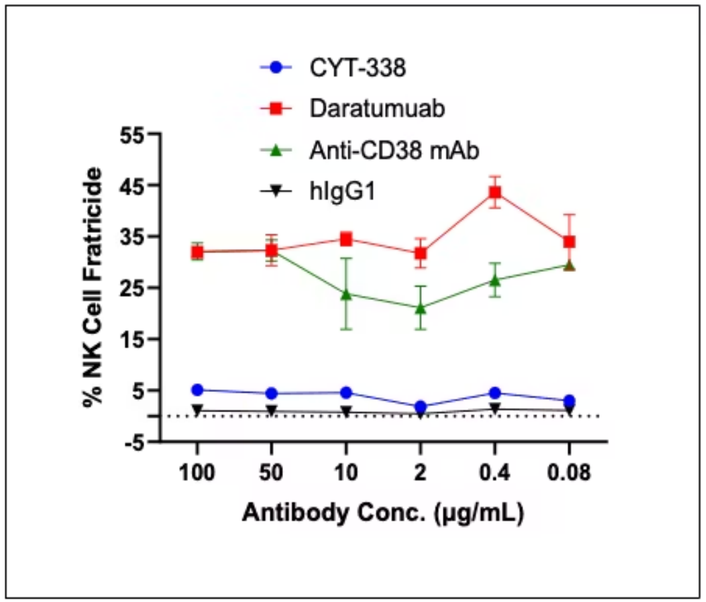
Reversal of NK Cell Dysfunction

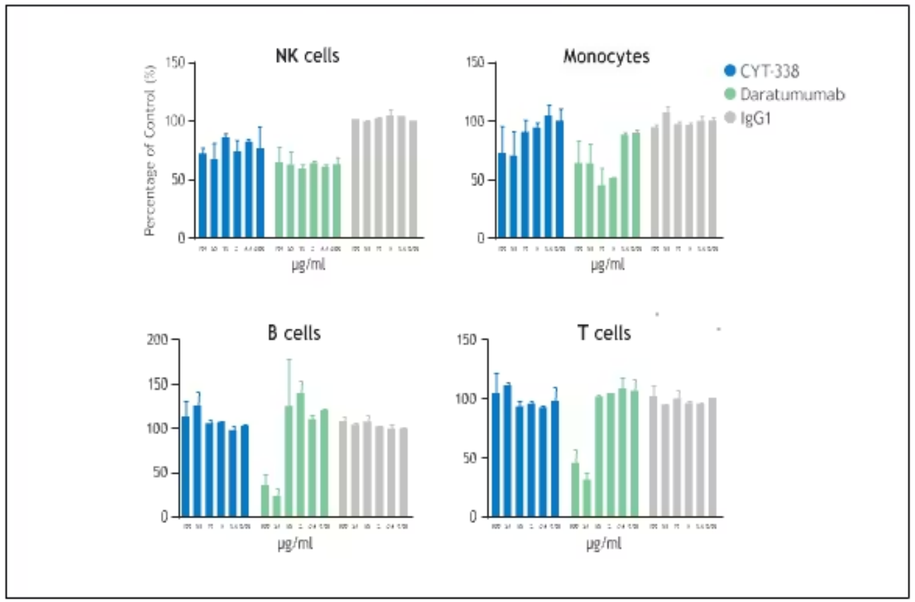
3 rounds of killing assays



Lin L, Chang H-M, Nakid C, Frankel S, Wu D, Kadouche J, et al. P842: NOVEL MULTIFUNCTIONAL TETRAVALENT CD38 NKP46 FLEX-NK ENGAGERS ACTIVELY TARGET AND KILL MULTIPLE MYELOMA CELLS. HemaSphere. 2022;6:736-7.

NY-338 Demonstrates Improved Profile Compared to Daratumumab





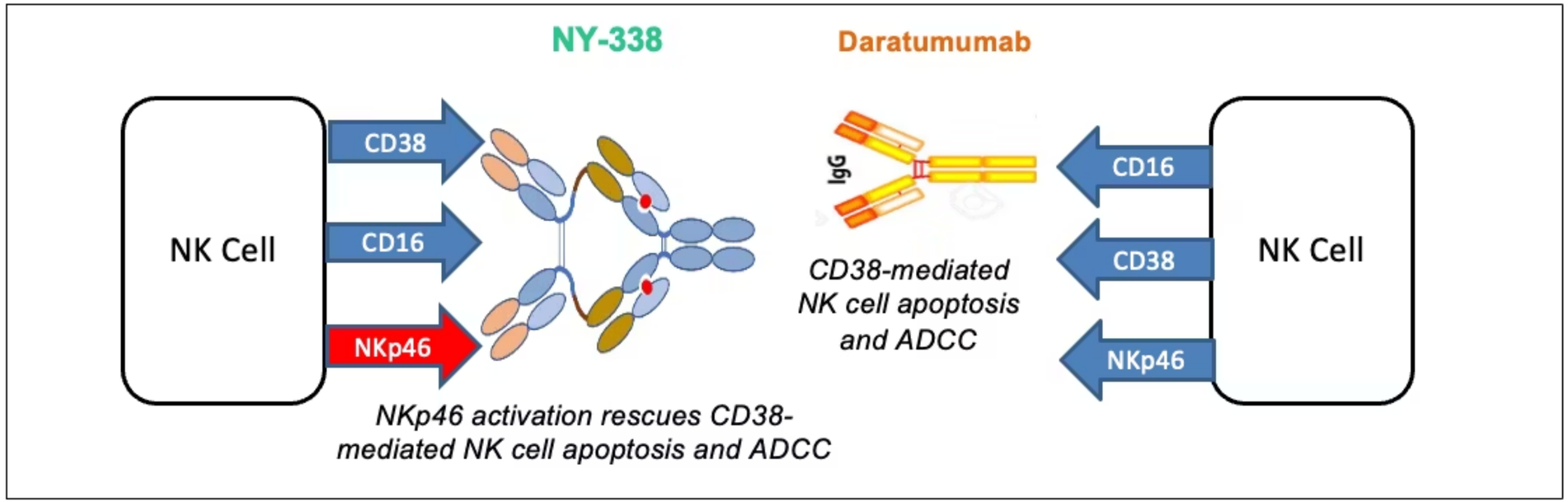
- NY-303 shows significant lower-to-no NK fratricide compared to Daratumumab
- NY-303 shows lower immune cell depletion compared to Daratumumab
- NY-303 shows low-to-no cytokine release (CRS)

Lin L, Chang H-M, Nakid C, Frankel S, Wu D, Kadouche J, et al. P842: NOVEL MULTIFUNCTIONAL TETRAVALENT CD38 NKP46 FLEX-NK ENGAGERS ACTIVELY TARGET AND KILL MULTIPLE MYELOMA CELLS. HemaSphere. 2022;6:736-7.

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

NY-338 Showed Lower Fratricide, Immune Subset Depletion, and Cytokine Release

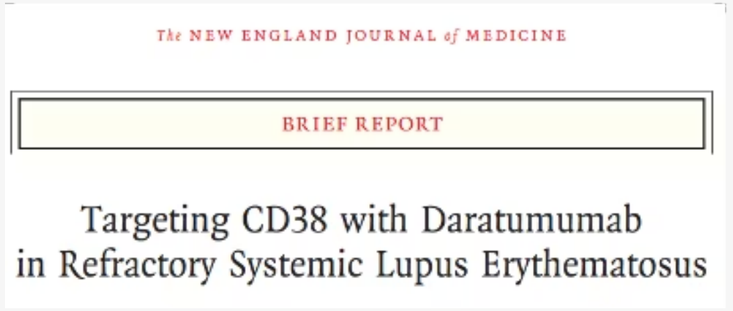
- NY-338 engagement of NK cells activates NKp46 signaling and neutralizes CD38-mediated apoptosis and ADCC of NK cells.
- NY-338 co-engagement of NKp46 & CD38 on NK cells sequesters NY-338 more to NK cells compared to other CD38+ immune cell subsets, minimizing activation and depletion of these cell types.
- NY-338 also minimizes cytokine release by these immune cells compared to Daratumumab.



Novel Multifunctional Tetravalent CD38 NKp46 FLEX-NKTMEngagers Actively Target and Kill Multiple Myeloma Cells, Liang Lin et al, AACR Poster Presentation, 2022

NY-338 Has Potential to Emerge as New Option in Multiple B-Cell Autoimmune Diseases

- **Validated B-Cell Approach**
  Therapeutics Approved in Rheumatoid Arthritis & Systemic Lupus Erythematous (Rituximab™, Belimumab™, Anifrolumab™)
- **CD38 Biology Highly-Differentiated**
  from CD19 & BCMA-Targeted approaches
- **CD38 Uniquely Inhibits Plasma Cells & Type I IFN pDCs**
  (Plasmacytoid Dendritic Cells) in Autoimmunity
- **Potential for Expansion to Multiple Indications**
  Positive Clinical Data in Multiple Orphan Autoimmune Indications with Felzartamab (CD38 mAb) leads to Biogen acquisition of HI-Bio
- **Opportunity for Unique Positioning**
  in Indications & Patient Segments where NK Activation Beneficial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BRIEF REPORT

Targeting CD38 with Daratumumab in Refractory Systemic Lupus Erythematosus


nature medicine

Article

https://doi.org/10.1038/s41591-023-02479-1

Daratumumab monotherapy for refractory lupus nephritis


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ORIGINAL ARTICLE

A Randomized Phase 2 Trial of Felzartamab in Antibody-Mediated Rejection

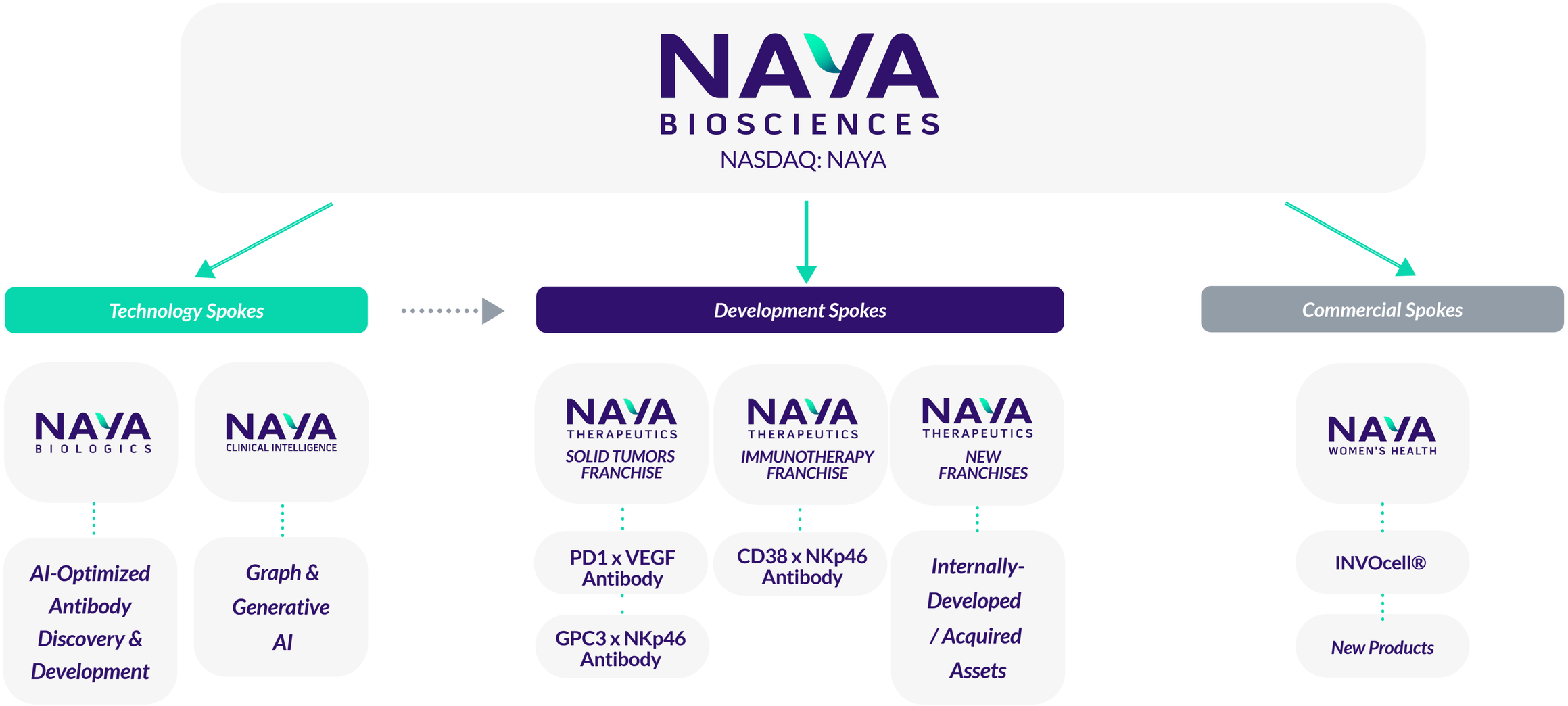
N Engl J Med 2020;383:1149-1155 DOI: 10.1056/NEJMoa2023325
Roccatello, D., Fenoglio, R., Caniggia, I. et al. , Nat Med 29, 2041–2047 (2023). https://doi.org/10.1038/s41591-023-02479-1
N Engl J Med 2024;391:122-132 DOI: 10.1056/NEJMoa2400763

NAYA BIOSCIENCES

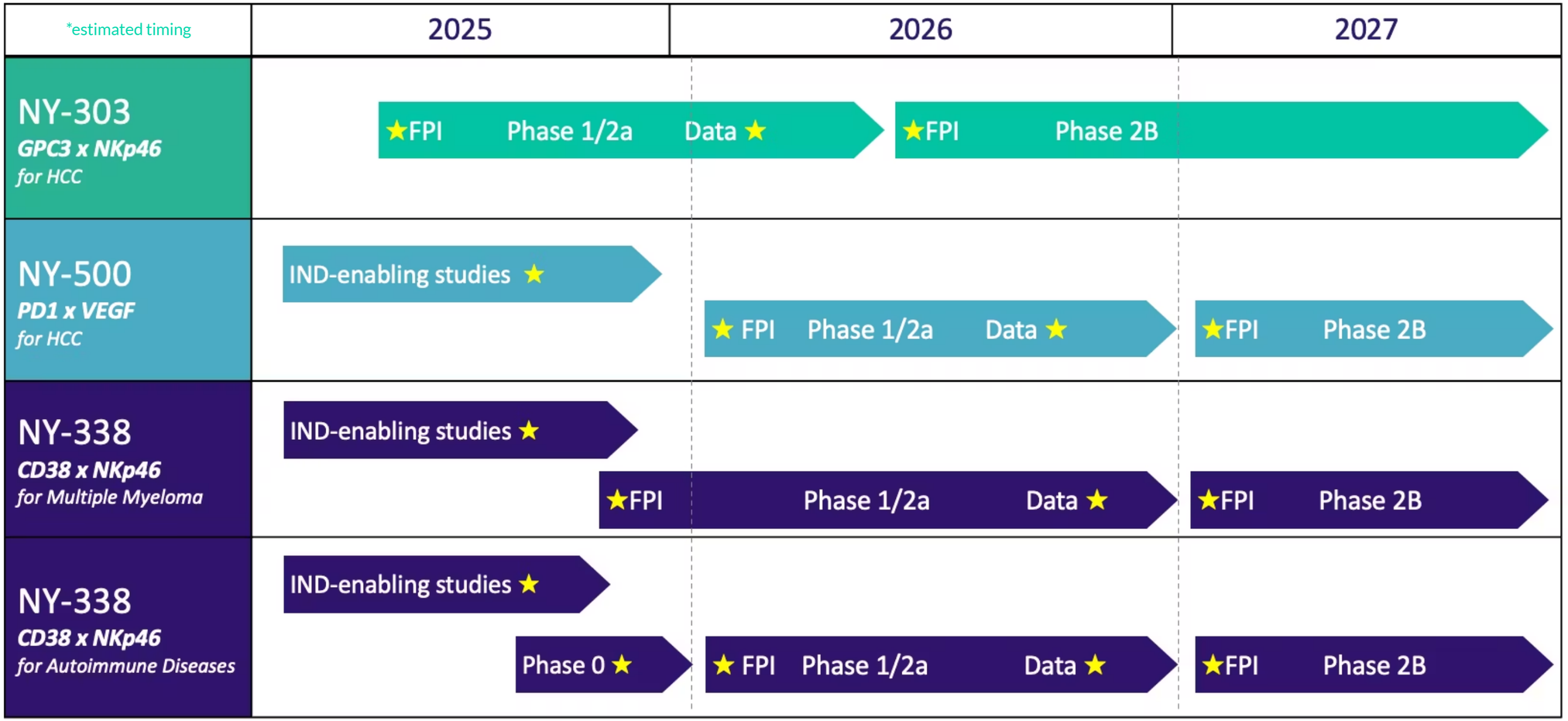
Investment Considerations

Hub & Spoke Model Leverages Shared Resources, Leadership, & Access to Capital

Enables Rapid Acquisition, Development, and Partnering of Assets



Multiple Anticipated Clinical Milestones in 2025-2027



Achieving Competitive Product Profiles with 4 Bifunctional Antibodies in 2025-27

NY-303

GPC3 x NKp46 Bifunctional Antibody

Second-Line Monotherapy in HCC for Patients Not Responding to First-Line Checkpoint Inhibitors

NY-500

PD1 x VEGF Bifunctional Antibody

AI-Optimized, US-Developed Monotherapy with Clinical Data in First-Line HCC

NY-338

CD38 x NKp46 Bifunctional Antibody

Potential for Differentiation to Darzalex & T-Cell Engagers in Multiple Myeloma & Autoimmune Diseases

NY-600

PSMA x NKp46 Bifunctional Antibody

Potential for Differentiation to T-Cell Engagers, Antibody Drug Conjugates, and Radio Immunotherapeutics in metastatic Castration Resistant Prostate Cancer (mCRPC)

NAYA Biosciences (NASDAQ:NAYA)

A Portfolio Company with Multiple Bifunctional Antibody Clinical Milestones

Competitive Bifunctional Antibody Pipeline

- *Multiple clinical readouts in 2025-2027*
- *Potential for leadership in hepatocellular carcinoma (HCC), multiple myeloma, prostate cancer, and autoimmune diseases*

Validated Hub & Spoke Model

- *Supports acquisition, development, and partnering of high-potential assets*