Harnessing the Power of Gamma-Delta T Cells

March 2026

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Forward-looking statements are based on IN8bio's current expectations and assumptions. Because forward-looking statements relate to the future, they are subject to inherent uncertainties, risks and changes in circumstances that may differ materially from those contemplated by the forward-looking statements. Important risks that could cause actual results to differ materially from those in the forward-looking statements include, without limitation: uncertainties inherent in the preclinical and clinical development and regulatory approval processes; whether interim or preliminary results from a clinical trial will be predictive of the final results of the trial or the results of future trials; the risk that IN8bio may be unable to raise additional capital and could be forced to delay, further reduce or to explore other strategic options for certain of its development programs; and IN8bio's ability to identify business development targets or strategic partners, to enter into strategic transactions on favorable terms, or to consummate and realize the benefits of any business development transactions. Additional risks that could cause actual results to differ materially from those in the forward-looking statements are set forth under the caption "Risk Factors" in IN8bio's Annual Report on Form 10-K filed with the Securities and Exchange Commission (SEC) on March 12, 2026, and in future filings IN8bio makes with the SEC. Any forward-looking statements contained in this presentation speak only as of the date hereof, and IN8bio assumes no obligation to update any forward-looking statements contained herein, whether because of any new information, future events, changed circumstances or otherwise, except as otherwise required by law.

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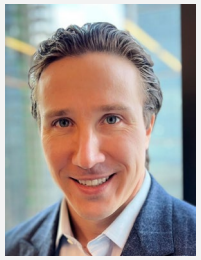
Deep Experience Across Cell Therapy and Oncology

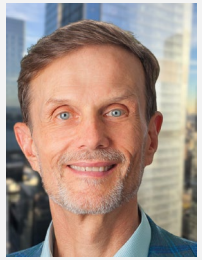
William Ho
Co-Founder, Chief Executive Officer

Lawrence Lamb, PhD
Co-Founder and Chief Scientific Officer



Patrick McCall, CPA
Chief Financial Officer

Kate Rochlin, PhD
President & Chief Operating Officer



Lou Vaickus, MD, FACP
Interim Consulting Chief Medical Officer

IN8bio's team has deep experience in gamma-delta T cells, cell therapy & oncology expertise:

- Our leadership team brings decades of extensive background in oncology discovery, business insights, franchise creation, product development, regulatory affairs, and commercialization
- Business development and licensing expertise across biopharmaceutical and biotechnology companies
- Founding of a private healthcare investment fund and management of public investments and cross-over portfolio at leading healthcare venture capital firm, New Leaf Venture Partners
- Specialization in transplantation immunology and recognized innovation in the field of γδ T cells
- Leadership of Curadigm's spin-out from Nanobiotix and platform collaborations and partnerships

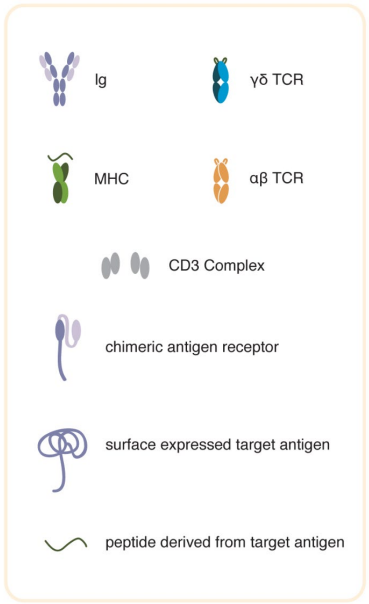
AlephPoint Capital
Bank of America
BIOTAGENICS
CATALYST BIOSCIENCES
CHUBB
COWEN
CURADIGM EXPANDING LIFE
CuraGen Corporation
Deloitte.
filament BIOSOLUTIONS INC.
IMMUNOVENT
NEW LEAF VENTURE PARTNERS
O'NEAL COMPREHENSIVE CANCER CENTER UAB THE UNIVERSITY OF ALABAMA AT BIRMINGHAM
Palmetto Health USC MEDICAL GROUP
PiperJaffray
UNIVERSITY OF SOUTH CAROLINA
TURNSTONE BIOLOGICS

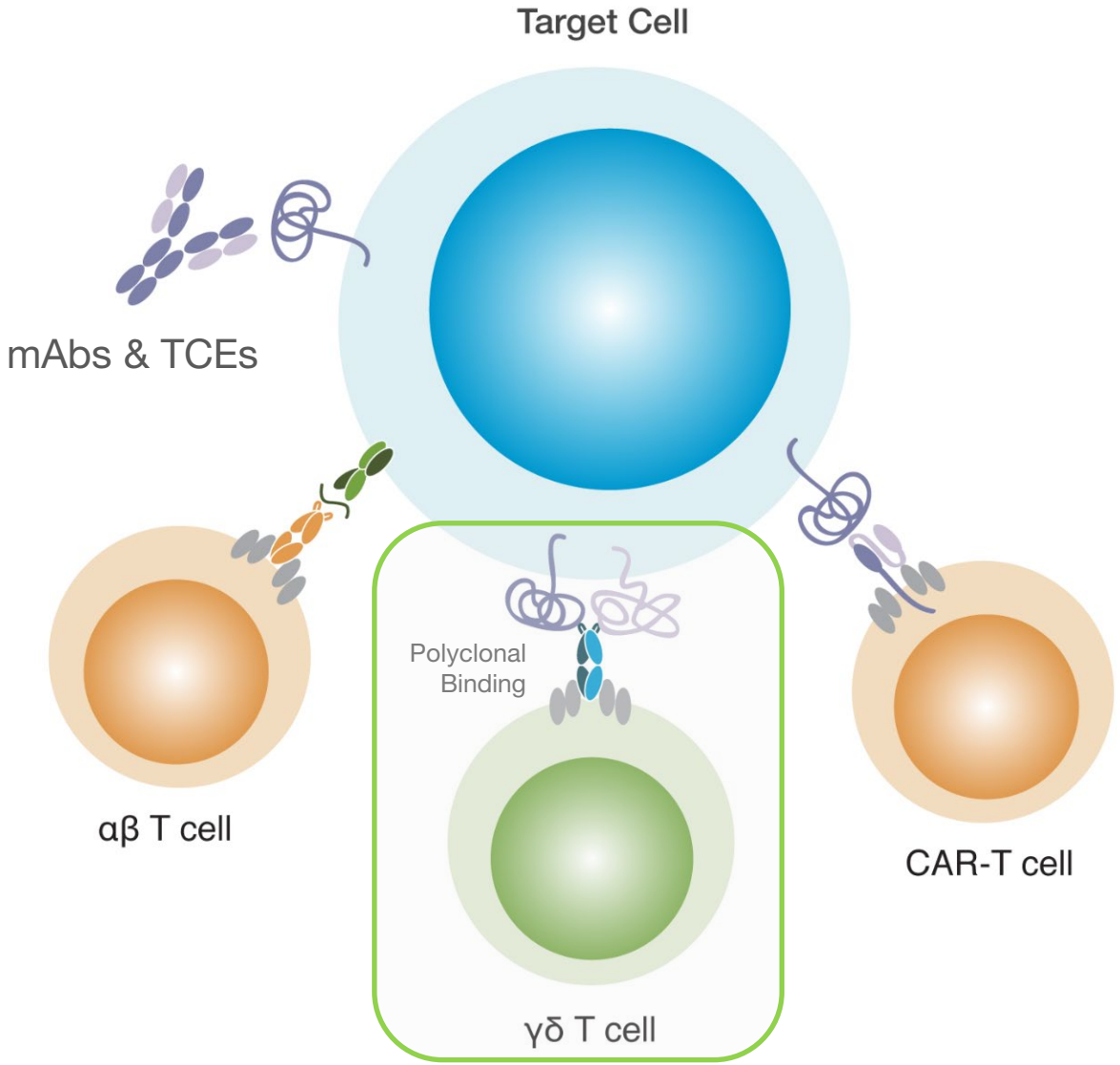
Revolutionizing γδ T cell Therapies

- **IN8bio is a leader in the development of gamma-delta (γδ) T cell therapies and T cell engagers (TCEs) in oncology and autoimmune diseases**
  - **Harnessing the Power of Immune Cells:** γδ T cells are “Nature’s CAR-T” cells that act as direct killers while orchestrating a comprehensive immune response to eliminate cancers and other targeted dysfunctional cells
  - **Durable Cancer Remissions:** IN8bio is targeting significant unmet needs by pioneering novel approaches to keep patients' progression-free longer with multiple remissions exceeding 4+ years in patients with difficult to treat cancers
  - **Precision and Safety:** γδ T cells have demonstrated in patients a better safety profile to date, with lower rates of adverse events and toxicities including cytokine release syndrome (CRS) and neurotoxicity (ICANs)
  - **Strong Capabilities:** We are translating over 30 years of expertise in γδ T cell research, our DeltEx™ platform has solved certain key biological, clinical and manufacturing issues that historically plagued the field across cell therapy and TCEs
  - **Powerful Platform Approaches**: We have developed a γδ TCE that can efficiently drive depletion of target cells, while inducing expansion of γδ T cells. This construct has unique differentiated properties to drive deeper cell depletion in cancer and autoimmune indications
- Our Mission: **Cancer Zero**™ - Driven by our goal to safely eradicate residual cancer cells. Join us in transforming cancer care

γδ T cells are Powerful Killing Cells for Immunotherapy

γδ T Cells: Nature's CAR-T Cell





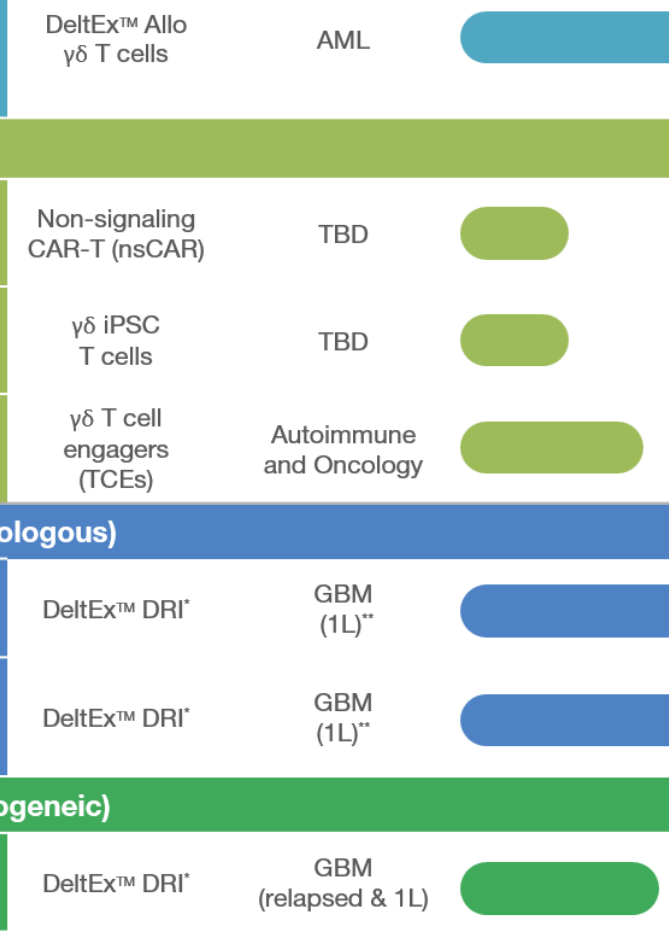
- γδ T cells **do not** require specific engineering to recognize sick cells
- γδ T cells play an **outsized role** in the immune response
- γδ T cells may drive **deep immune responses**
- Are found to be the most favorable cancer **prognostic immune cells**^ and predict responses to both checkpoint inhibition# and CAR-T$ therapy
- γδ T cells are rare but powerful cells that can **effectively identify and eradicate target cells**

Source: IN8bio – adapted with permission from Willcox et al. Immunological Reviews, 298:25-46, Sept. 2020, fig 5. and ^ Foord et al., Sci. Transl. Med. 13, eabb0192 (2021), # Widlund et al., Nature Cancer, 2024 (https://doi.org/10.1038/s43018-024-00735-y), $ Guerrero-Murillo et al., Cell Reports Medicine, 2024 (https://doi.org/10.1016/j.xcrm.2024.101803)

IN8bio Possesses a Comprehensive γδ T cell Platform

A Robust Pipeline with Multiple Near-Term Readouts

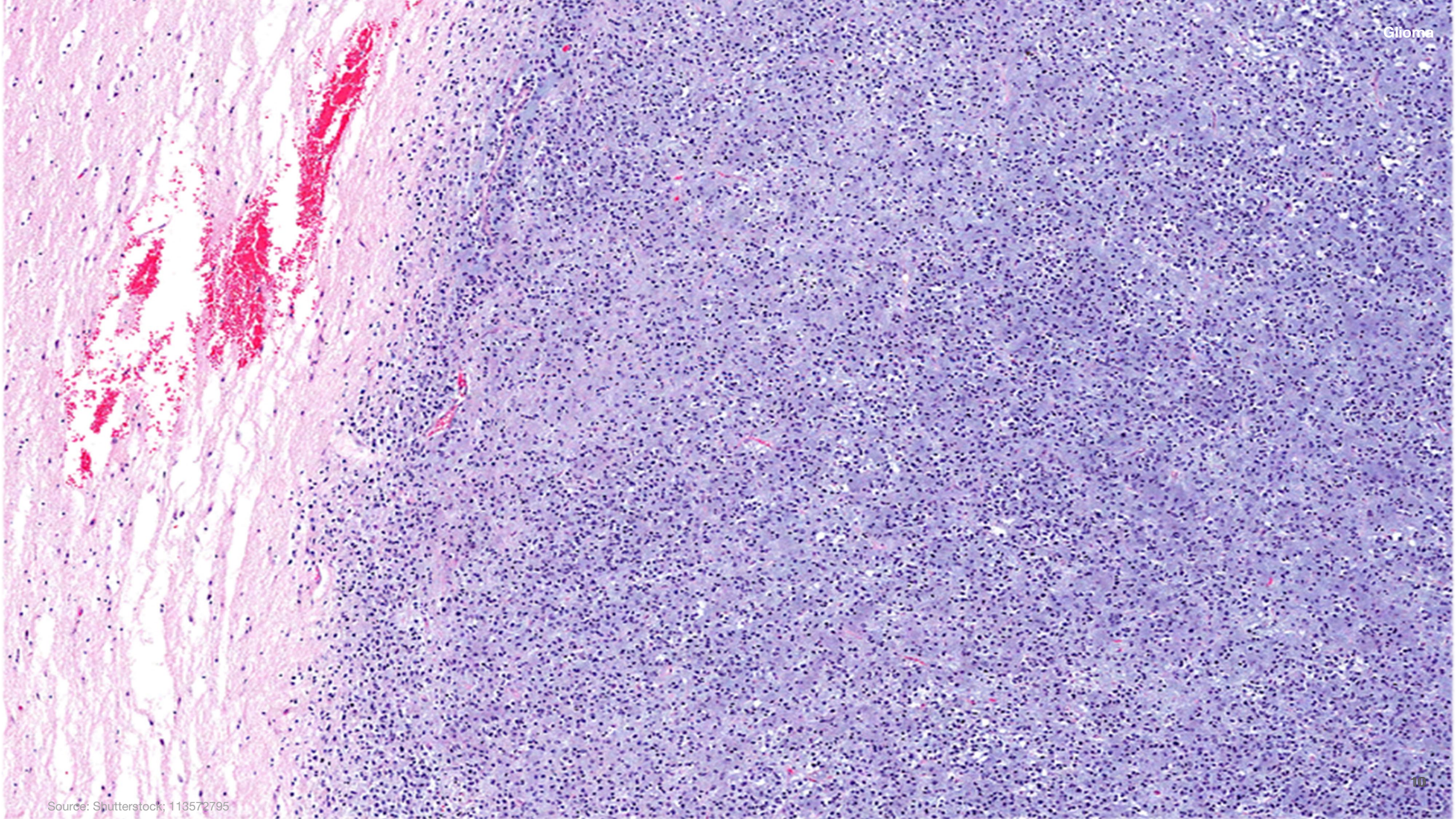
Product Candidate	Approach	Key Indications	Preclinical	Phase 1	Phase 2	Phase 3	Next Anticipated Milestone(s)^
Hematologic Malignancies (Allogeneic)							
INB-100	DeltEx™ Allo γδ T cells	AML					• Complete dosing of additional patients in the DL2 expansion cohort 1H26 • Provide clinical updates and follow-up YE 2026
In Development							
INB-300	Non-signaling CAR-T (nsCAR)	TBD					• On-hold for potential partnering
INB-500	γδ iPSC T cells	TBD					• On-hold for potential partnering
INB-619	γδ T cell engagers (TCEs)	Autoimmune and Oncology					• IND-enabling studies with initial mouse model data in 2026
Solid Tumors (Autologous)							
INB-200	DeltEx™ DRI*	GBM (1L)**					• Trial completed, pursuing peer-reviewed publication of data
INB-400#	DeltEx™ DRI*	GBM (1L)**					• Obtain FDA guidance on potential registrational pathways in 2026 • Present updated mOS data in mid- and late- 2026
Solid Tumors (Allogeneic)							
INB-400#	DeltEx™ DRI*	GBM (relapsed & 1L)					



* DRI = Drug Resistant Immunotherapy, or a chemotherapy resistant cell therapy
** 1L = First line therapy
^ Timing of next anticipated milestones are estimates based on the successful raise of additional capital to fund our programs and are subject to change
# Please refer to the Current Report on Form 10-K, filed with the SEC on March 12, 2026, for additional details about IN8bio's pipeline efforts

INB-200 & 400

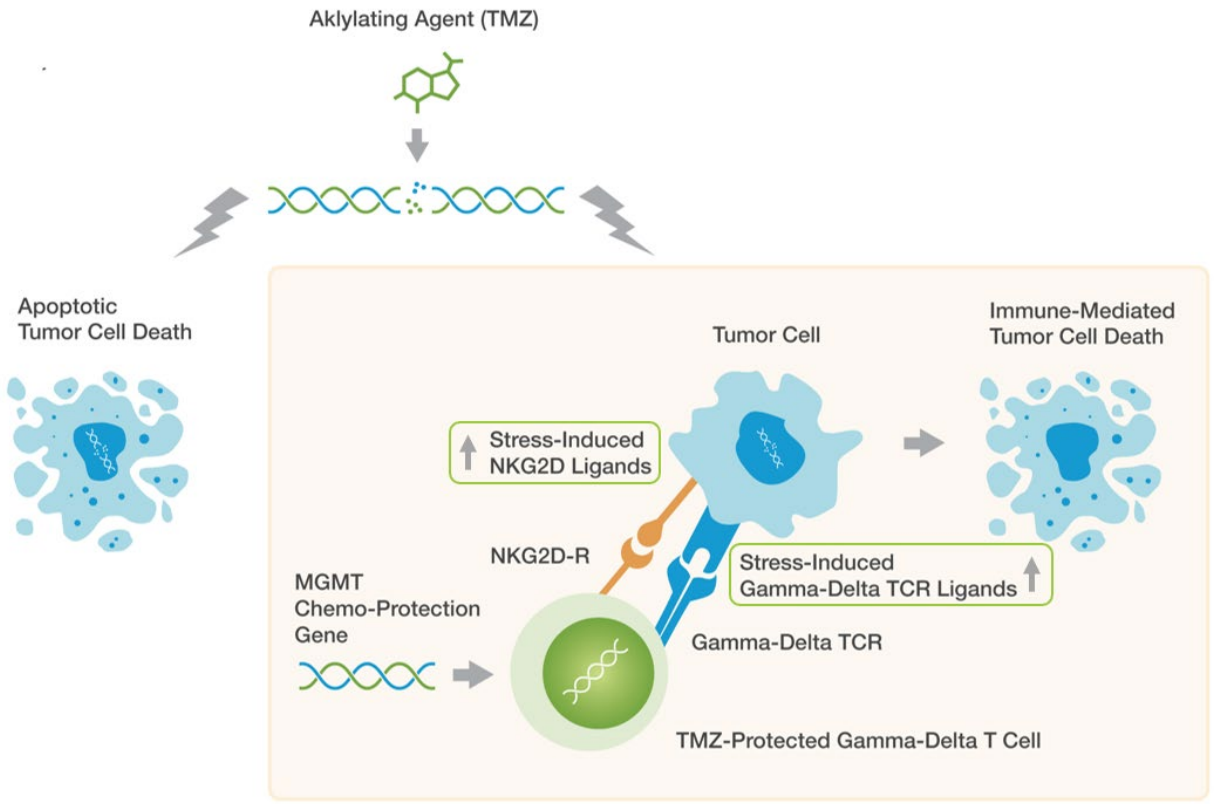
DeltEx™ Drug Resistant Immunotherapy (DRI) for Glioblastoma (GBM)



Source: Shutterstock; 113572795

Targeting the DNA Damage Response (DDR) to Kill Tumors

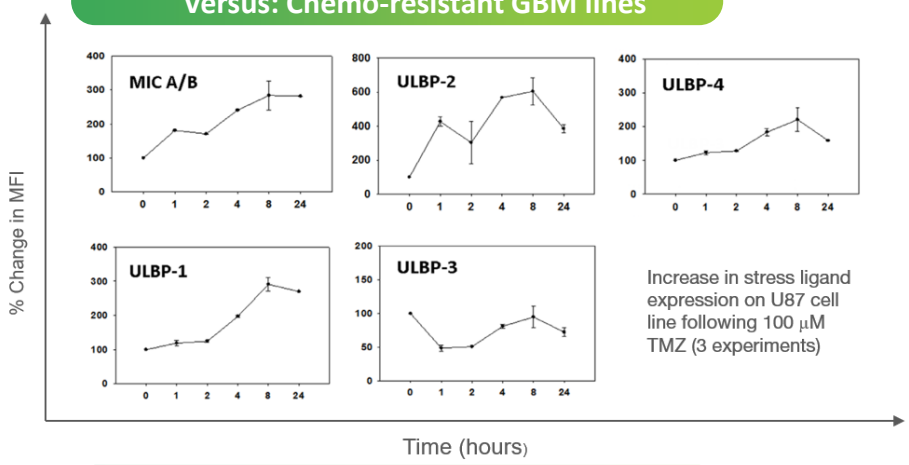
DDR is a biological process that can detect and eliminate cells with DNA damage through increased avidity



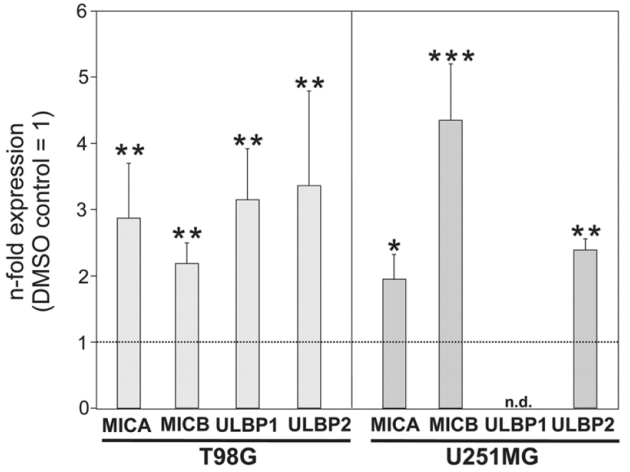
DRI gamma-delta T cell mechanism overview

TMZ Increases NKG2D-L Expression:

Versus: Chemo-resistant GBM lines

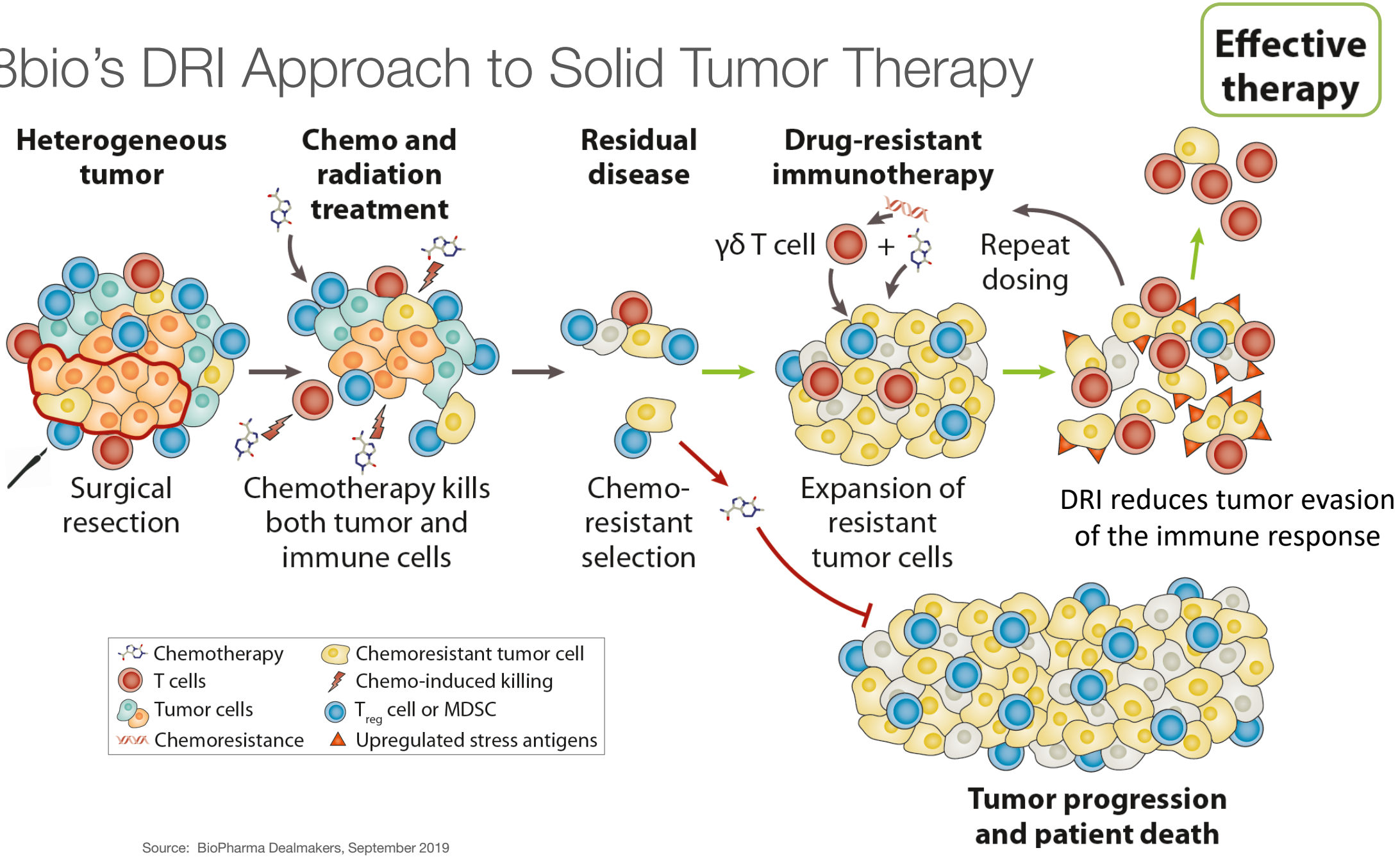


Versus: Glioma stem-like cells



Source: IN8bio, Lamb et al. Plos One, (2013) Vol 8., Issue 1, Held-Feindt et al. Histochemistry and Cell Biology, (2018) 149: 2019-233; https://doi.org/10.1007/s00418-018-1633-5

IN8bio's DRI Approach to Solid Tumor Therapy



Source: BioPharma Dealmakers, September 2019

Updated INB-200/400 Clinical Data for Year End 2025

INB-200 & 400: Phase 1/2 Study Regimen

Treatment Arms

Fixed dose level (DL) of DRI in a 3+3 design (N= ~18):

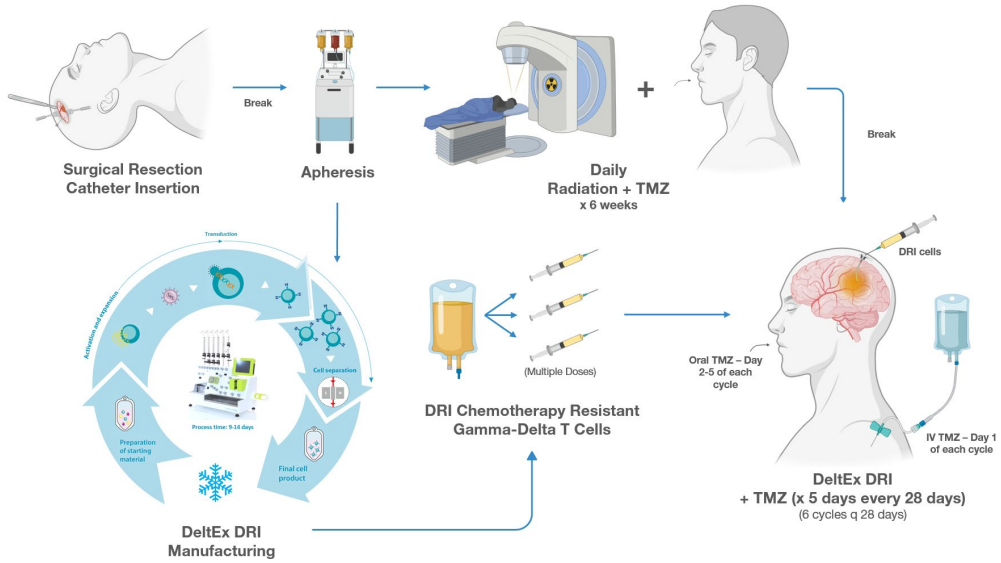
DL1: N = 3 (up to 6) patients, single dose of 1 x 10^7 cells on C1D1
DL2: N = 3 (up to 6) patients, three doses of 1 x 10^7 cells, one dose every 28 D1 of C1-C3
DL3: N = 3 (up to 6) patients, six doses of 1 x 10^7 cells, one dose every 28 days on D1 of C1-C6

Treatment Regimen & Timing

Surgical resection followed by apheresis

6 weeks induction TMZ + radiation

6 cycles maintenance TMZ + DRI



Primary Endpoints

- Safety
- Maximum tolerated dose (MTD) of DeltEx DRI in two dose frequencies

Secondary Endpoints

- Time to progression
- Overall survival
- Biologic response

Site

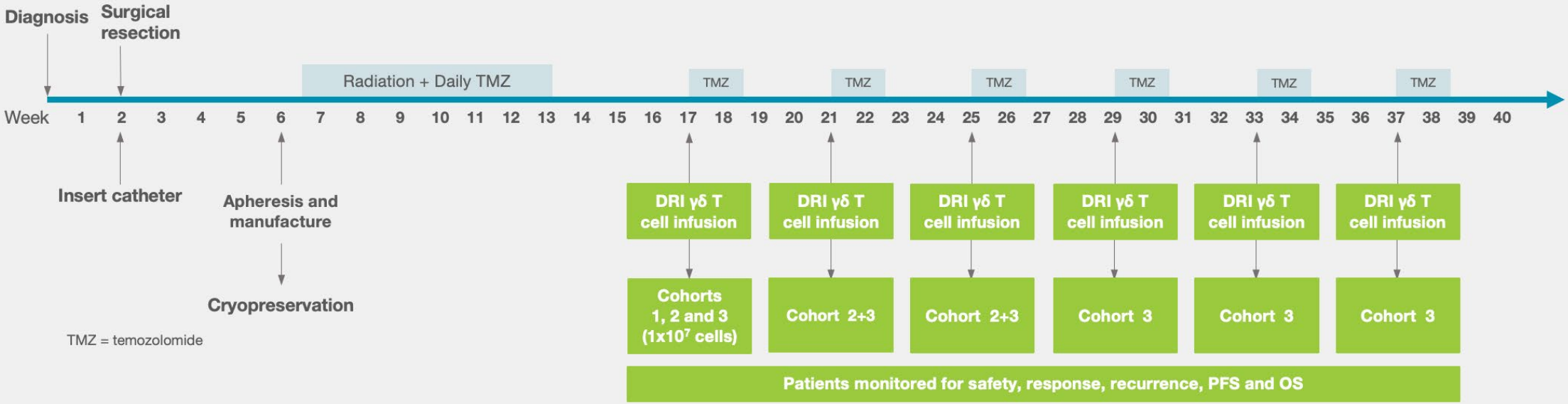
Source: IN8bio, image created with biorender.com

Repeated Dosing to Eliminate Residual Cells

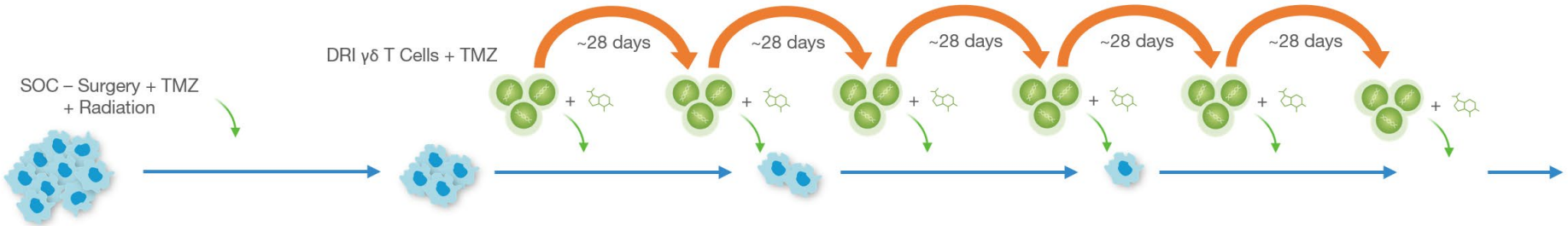
Targeting residual cells at a rate faster than the glioma doubling time to maintain remission

SOC: Stupp Regimen

IN8bio: DeltEx DRI Therapy



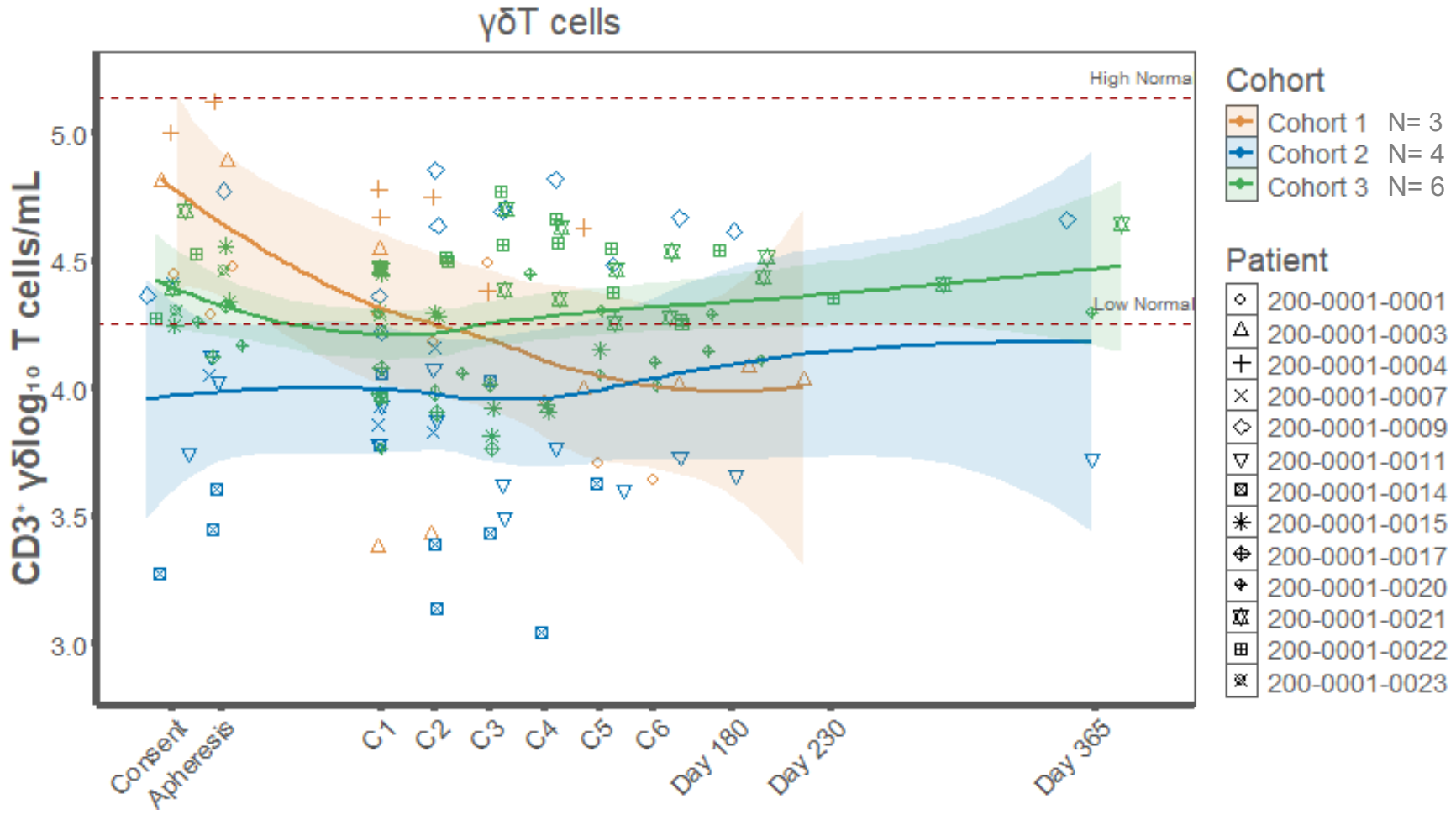
TMZ + adjuvant DRI γδ T cells multiple repeat doses



Source: IN8bio; assumptions: GBM doubling time ~50days (Berntsen et al. Neuro-Oncology, 2015), DRI kills ~50% of cells that are resistant to TMZ therapy

Repeat Doses Improves Global Patient Immunity

Peripheral γδ T cells levels correlate in a dose-dependent manner with sustained reconstitution in repeated dose patients despite multiple rounds of lymphodepleting TMZ.



Clinical Data Appears Consistent Across Multiple Sites

O'NEAL COMPREHENSIVE CANCER CENTER
UAB THE UNIVERSITY OF ALABAMA AT BIRMINGHAM

UAB HEERSINK SCHOOL OF MEDICINE

Cleveland Clinic

MOFFITT CANCER CENTER

THE OHIO STATE UNIVERSITY
WEXNER MEDICAL CENTER

- INB-200 is a Phase 1 investigator-initiated trial run at UAB, INB-400 is a corporate sponsored Phase 2 that enrolled patients across the three other sites
- Total of 17 patients treated with DRI γδ T cells and 10 observed in contemporaneous control arm across four centers in Phase 1 and Phase 2 clinical trial prior to suspension of enrollment in 2024
- No major toxicity signals or significant adverse events across sites or treatment arms
- Treatment activity remains consistent across centers

Active Cohort – All Repeat Dose Patients

	Subject	Age / Sex	Cytogenetics	Dose level	Resection	TMZ Maint. Cycles Received
DL2	007	74 / M	IDH-WT, MGMT-unmethylated	2	Total	2
	009	32 / M	IDH-mutant, MGMT-methylated	2	Total	12
	011^	56 / F	IDH-WT, MGMT-methylated	2	Total	6
	014	73 / F	IDH-WT, MGMT-unmethylated	2	Subtotal	6
DL3	015^	73 / M	IDH-WT, MGMT-methylated	3	Subtotal	5
	017	74 / F	IDH-WT, MGMT-methylated	3	Subtotal	3
	020	66 / M	IDH-WT, MGMT-methylated	3	Subtotal	6
	021	57 / M	IDH-WT, MGMT-unmethylated	3	Total	6
	022^	53 / M	IDH-WT, MGMT-unmethylated	3	Subtotal	6
	023	52 / M	IDH-WT, MGMT-unmethylated	3	Subtotal	1
New Patients INB-400	001	61 / F	IDH-WT, MGMT-methylated	3	Subtotal	6
	004	74 / F	IDH-WT, MGMT-unmethylated	3	Subtotal	6
	001^	52 / F	IDH-WT, MGMT-unmethylated	3	Total	3
	005	70 / F	IDH-WT, MGMT-methylated	3	Total	6

All Repeat Dose Patients (Including INB-400, N=14)

- Median age: 64
- 50% Male
- 50% **unmethylated**
- 57% **subtotal** resection
- Median KPS = 80

INB-200 Repeat Dose Patients (Excluding INB-400, N=10)

- Median age: 62
- 70% Male
- 50% **unmethylated**
- 60% **subtotal** resection
- Median KPS = 80

- ^Pts 011, 015, 022 and 001 received additional therapy following progression. No other patients received any additional therapy outside of SOC + DRI

*As of December 31, 2025; Early trial results are not indicative of future results, including the outcome of this trial.

Control Cohort – SOC and Single Dose (DL1) Patients

	Subject	Age / Sex	Cytogenetics	Dose level	Resection	TMZ Maint. Cycles Received
DL1	001	68 / M	IDH-WT, MGMT-unmethylated	1	Total	5
DL1	003	74 / F	IDH-WT, MGMT-methylated	1	Total	6
DL1	004^	21 / F	IDH-WT, MGMT-unmethylated	1	Total	3
Untreated (SOC) Patients		49 / M	IDH-WT, MGMT-unmethylated	0	Total	
Untreated (SOC) Patients		77 / M	IDH-WT, MGMT-methylated	0	Subtotal	
Untreated (SOC) Patients		66 / M	IDH-WT, MGMT-unmethylated	0	Total	
Untreated (SOC) Patients		71 / F	IDH-WT, MGMT-unmethylated	0	Total	
Untreated (SOC) Patients		75 / F	IDH-WT, MGMT-methylated	0	Total	6
Untreated (SOC) Patients		67 / M	IDH-WT, MGMT-methylated	0	Total	2
Untreated (SOC) Patients		67 / M	IDH-WT, MGMT-methylated	0	Total	
Untreated (SOC) Patients		71 / F	IDH-WT, MGMT-unmethylated	0	Total	
Untreated (SOC) Patients		65 / F	IDH-WT, MGMT-unmethylated	0	Subtotal	
Untreated (SOC) Patients		65 / M	IDH-WT, MGMT-unmethylated	0	Total	

All Control Patients (Including DL1, N=13)

- Median age: 67
- 54% Male
- 62% **unmethylated**
- 15% **subtotal** resection
- Median KPS = 80

Untreated SOC Patients (Excluding DL1, N=10)

- Median age: 67
- 60% Male
- 60% **unmethylated**
- 20% **subtotal** resection
- Median KPS = 80

- ^Pt 004 received a single dose of bevacizumab during induction for edema, Control patients unknown outside of SOC

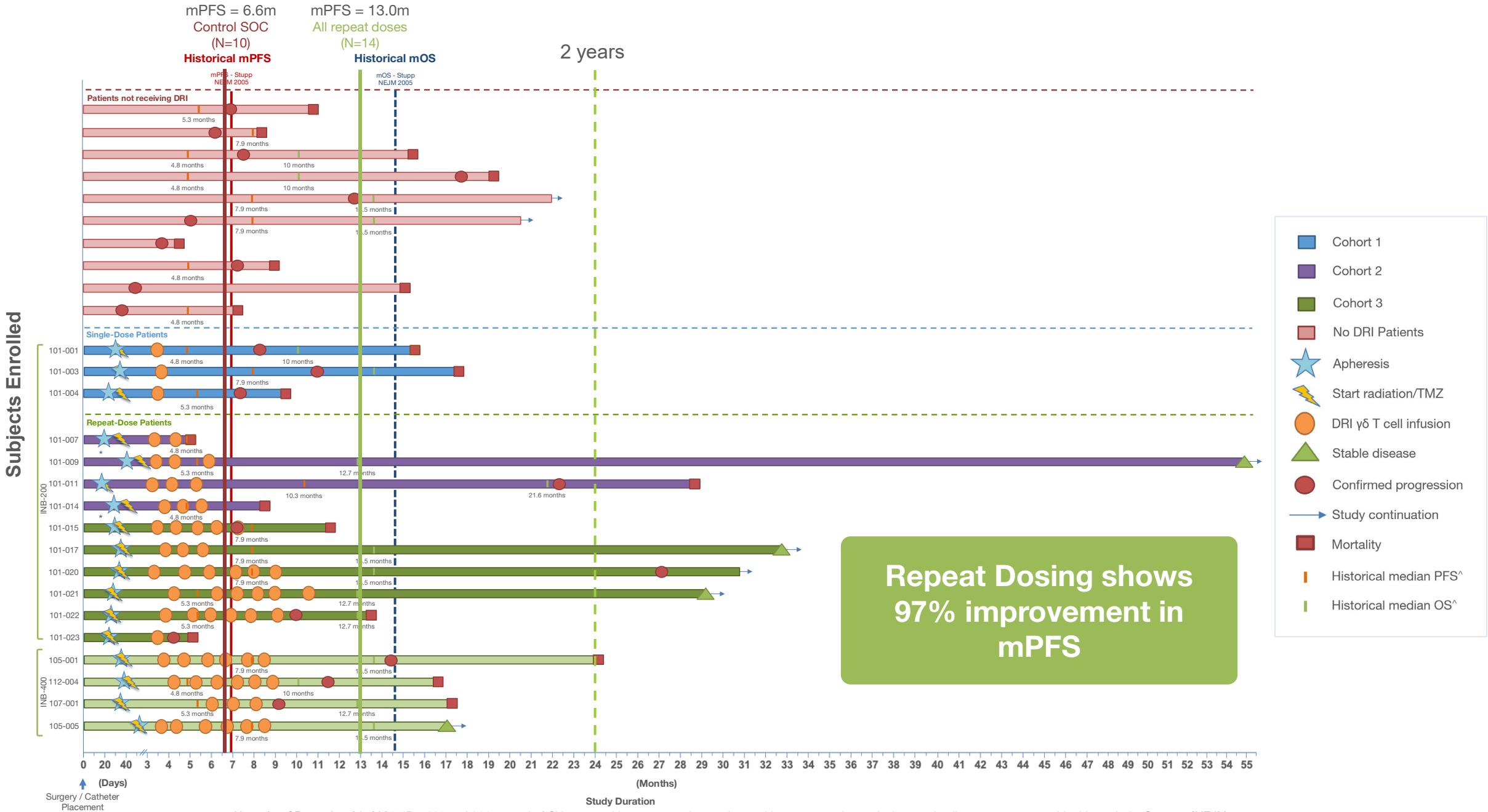
*As of December 31, 2025; Early trial results are not indicative of future results, including the outcome of this trial.

Patient Demographics Generally Comparable Across Cohorts

Exception - More gross total resections should favor the Control (SOC) & DL1 patients for better outcomes

Treatment Arm	N	Methylation Status	Resection Type		Median Age	Gender
			Subtotal	Total		
Control (SOC) Patients	10	60% Unmethylated	20%	80%	67	60% Male
INB-200 DL1 Patients	3	66% Unmethylated	0%	100%	68	33% Male
INB-200 Repeat Dose Patients	10	50% Unmethylated	60%	40%	62	70% Male
INB-400 Repeat Dose Patients	4	50% Unmethylated	50%	50%	66	0% Male
All Repeat Dose Patients	14	50% Unmethylated	57%	43%	64	50% Male

- Across all patient groups there were similar methylation status distribution, Karnofsky performance status (KPS), gender and median age; one single patient (009) enrolled before 2021 was a grade 4, IDH-mutant glioma
- Interestingly, in the SOC Control patients, the number of Total resections was much higher (80% total) compared to the Repeat Dose patients (43% total). Patients with Total resections are generally expected to have better outcomes



Note: As of December 31, 2025; *Pts 009 and 014 passed of CV events without progression, patients without progression and who remain alive are not censored in this analysis; Source: ^NEJM 2005; 352:987-996 & 352:997-1003 DOI: 10.1056/NEJMoa043330, DOI: 10.1056/NEJMoa043331; NEJM 2017; 376:1027-1037 DOI: 10.1056/NEJMoa1611977; Early trial results are not indicative of future results, including the outcome of this trial.

PFS and OS Demonstrate a Strong Treatment Effect

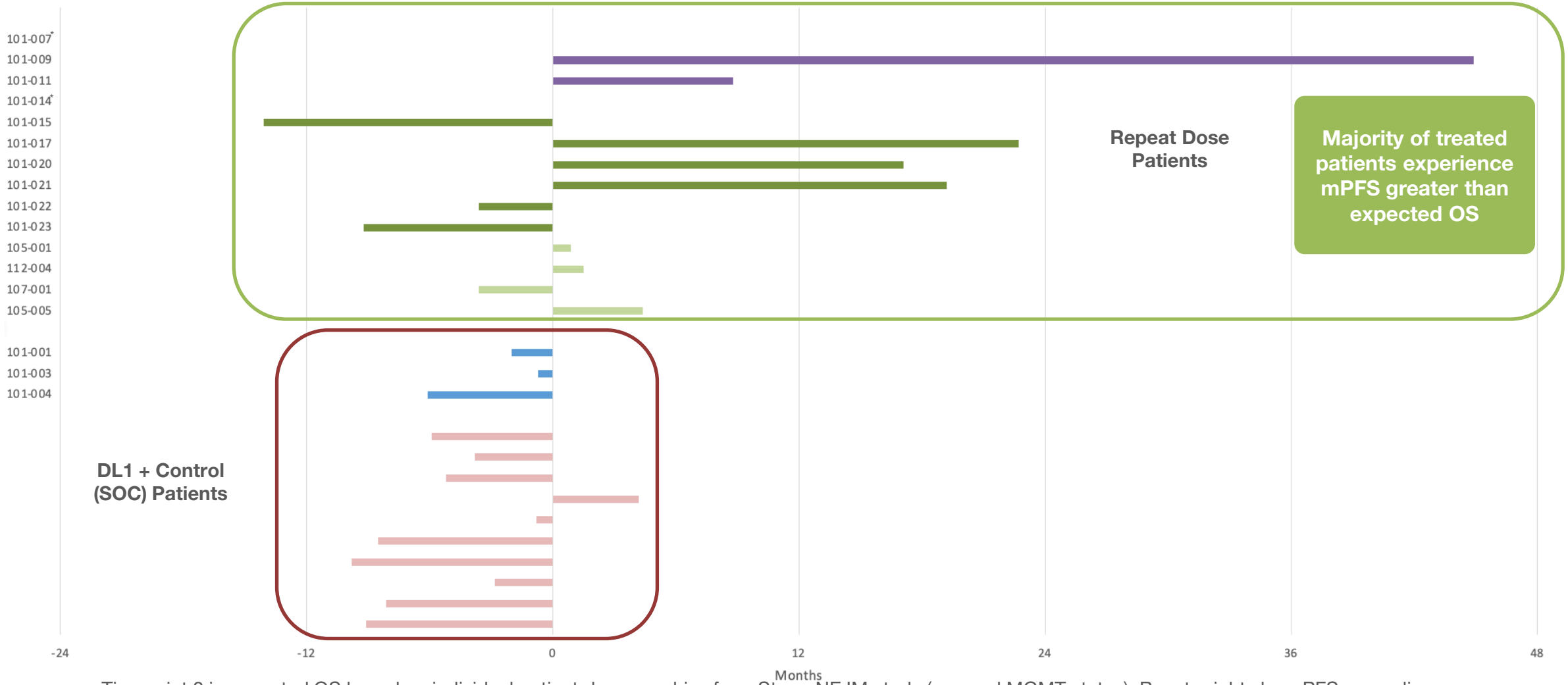
Repeat dosing of DRI γδ T cells consistently resulting in better outcomes despite fewer Total resections

Treatment Arm	Median PFS (m)	Median OS (m)	Pts without progression or PFS Exceeding Expected OS (%)
Historical NEJM Data	6.9	14.6	NA
Control (SOC) Patients	6.6	13.2	(1/10) 10%
INB-200 DL1 Patients	8.0	15.7	(0/3) 0%
INB-200 Repeat Dose Patients	16.1	21.1+	(5/10)* 50%
INB-400 Repeat Dose Patients	13.0	17.2	(3/4) 75%
All Repeat Dose Patients	13.0	17.2+	(8/14) 57%

Across Multiple Centers we Observe Extended PFS and OS Compared to Contemporaneous Controls

Note: As of December 31, 2025; *Pts 009 and 014 passed of CV events without progression, patients without progression and who remain alive are not censored in this analysis; Early trial results are not indicative of future results, including the outcome of this trial. + - OS not yet attained median and continues to increase

Comparison to Control Group Highlights Improved Outcomes



Timepoint 0 is expected OS based on individual patient demographics from Stupp NEJM study (age and MGMT status). Bars to right show PFS exceeding expected OS, bars to the left represent PFS less than expected OS

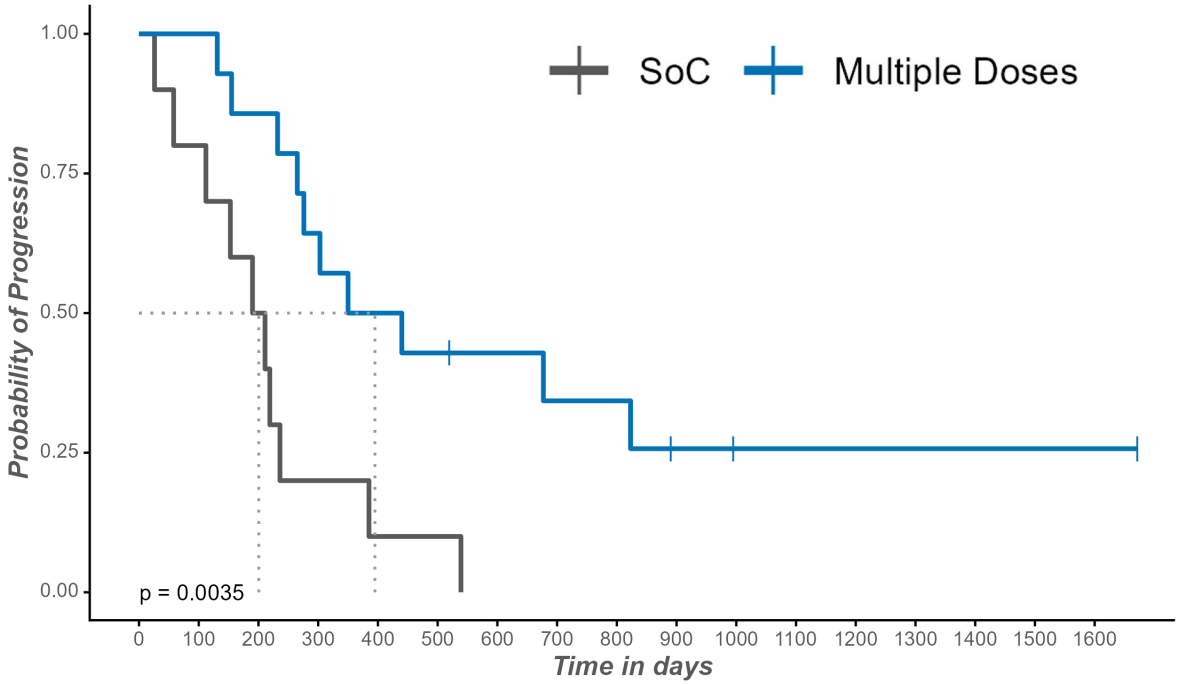
*Pts 007 and 014 passed of CV events without progression; Note: As of December 31, 2025

Kaplan-Meier Analysis Demonstrates a Treatment Benefit

Comparison of Standard-of-Care (SOC) Stupp Regimen vs. Repeated Doses of DRI γδ T cells

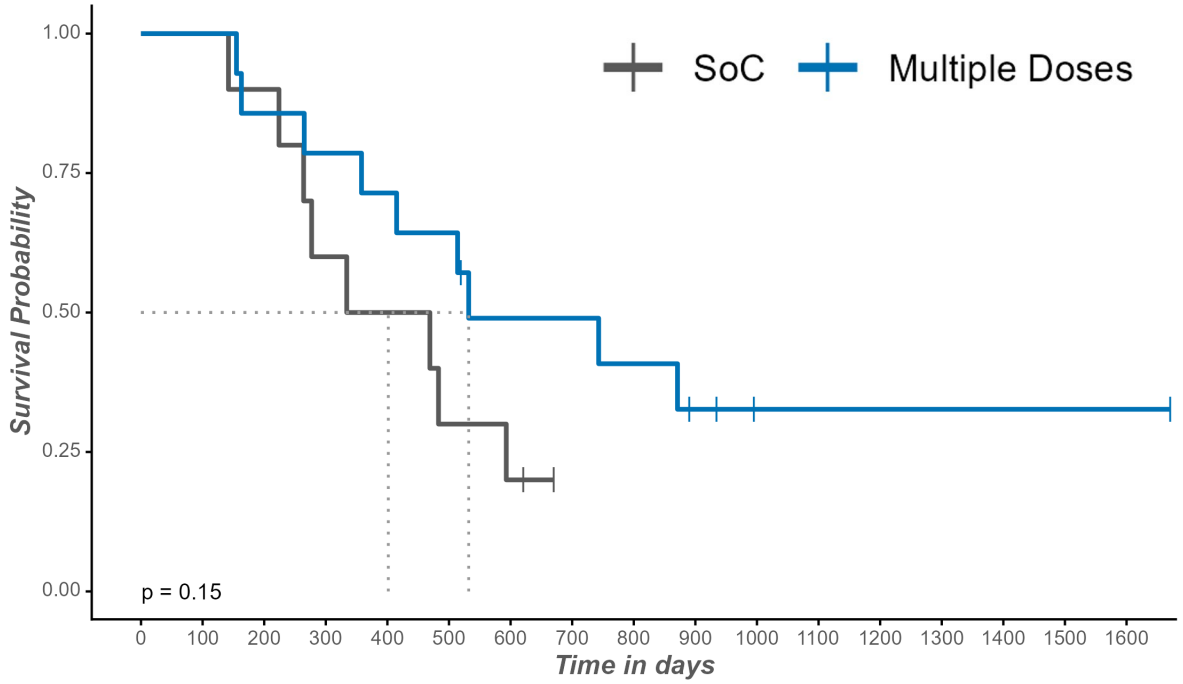
DeltEx DRI intervention; GBM Progression-free analysis

Kaplan-Meier Estimates



DeltEx DRI Intervention: GBM Survival Analysis

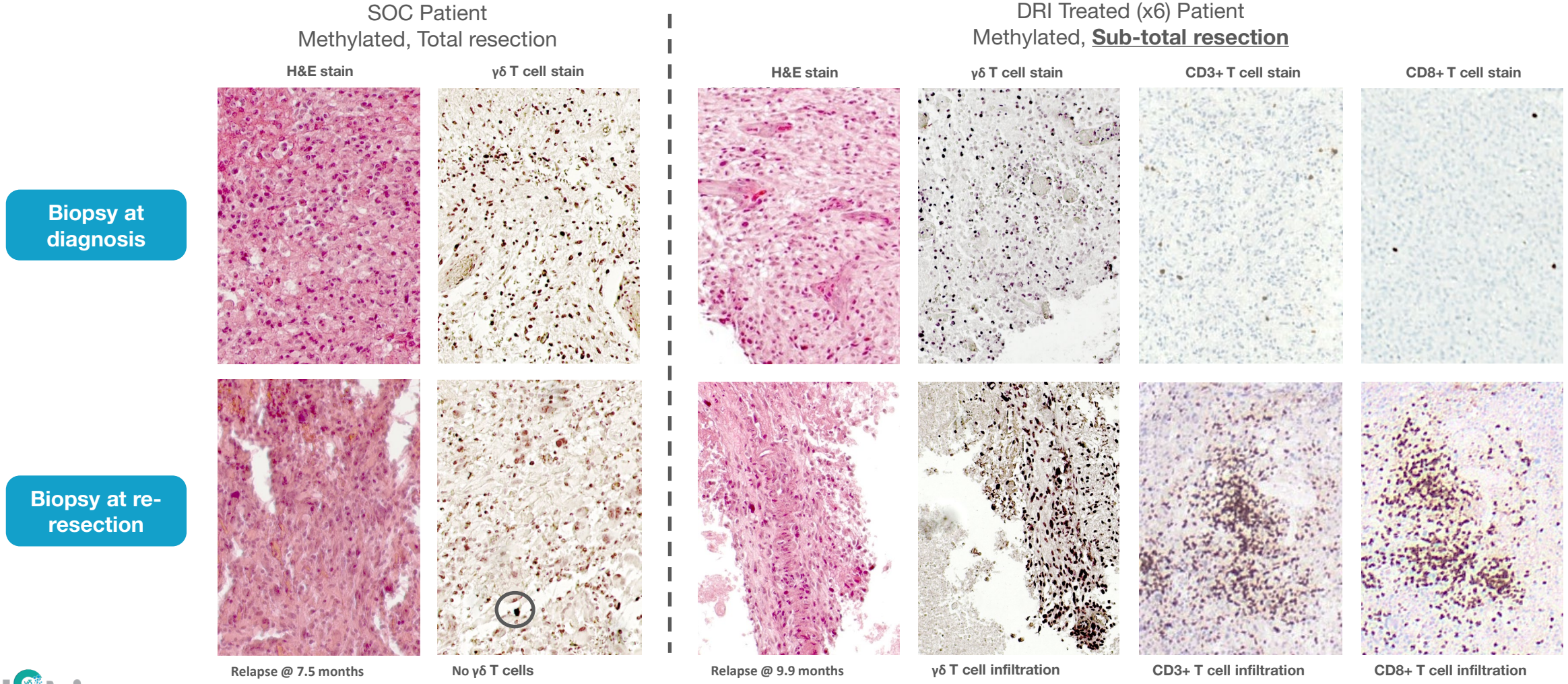
Kaplan–Meier Estimates



Note: As of December 31, 2025; Data censored for patients who have no progression and/or remain alive or lost to follow-up (LTFU, control group only); mOS not yet attained in multiple dose patients with median continuing to increase, Early trial results are not indicative of future results, including the outcome of this trial.

Histopathology Supports the PFS and OS Observations

Broad immune cell infiltration demonstrate biological mechanism for activity and duration of responses



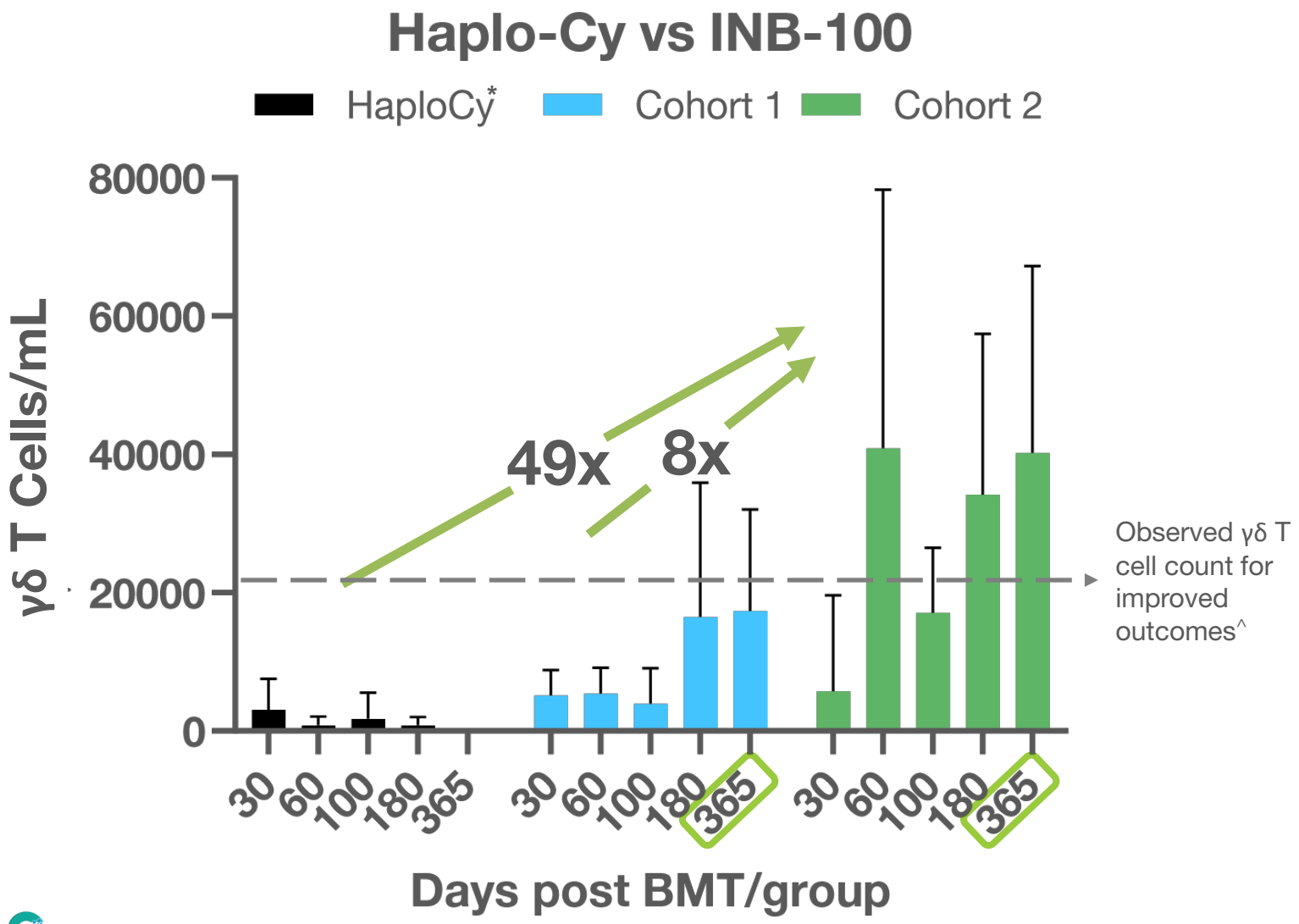
Source: IN8bio and UAB - Early trial results are not indicative of future results, including the outcome of this trial.

INB-100

DeltEx™ Allo for Leukemias

IN8bio is Increasing γδ T cell Levels

IN8bio's INB-100 Results in Higher γδ T cells



- Higher γδ T cell levels at 365 days suggests ***in vivo*** **expansion** and **persistence** enabling continued surveillance against residual cancers
- IN8bio's INB-100 results in a **dose-dependent** rise in circulating γδ T cells above the threshold associated with improved progression-free survival (PFS) and overall survival (OS)

Source: IN8bio, Inc. and UAB *previously unpublished data from laboratory of Dr. Lawrence Lamb; as of September 30, 2024 following completion of all time points by patients in dose-level 2;
^ Sengelov et al. Front. Immunol. 2019; DOI: 10.3389/fimmu.2019.01997

Relapse is the Biggest HSCT Challenge

Haploidentical Stem Cell Transplantation (HSCT) increases the market size but also results in more relapses and greater mortality

- **Our Goal:** Increase progression-free survival by providing **DURABLE** immune surveillance with γδ T cells
- Up to **25% of patients relapse within 100 days** and up to **50% relapse within 1 year**
- IN8bio's γδ T cells show **reduced relapse rates** and better **survival outcomes** to date, which may significantly expand the market

US AML Incidence

~ 21,000

~ 5,000

HSCTs per annum

1-year relapse-free survival post-HSCT

~35-50%

Source: CIBMTR summary slides - Cusatis et al. Current trends and outcomes in cellular therapy activity in the United States, including prospective Patient Reported Outcomes data collection within the CIBMTR registry.Transplant Cell Ther. 2024 Jun 27:S2666-6367(24)00482-2. doi:10.1016/j.jtct.2024.06.021; Luznik L,et al. HLA-haploidentical bone marrow transplantation for hematologic malignancies using nonmyeloablative conditioning and high-dose, post-transplantation cyclophosphamide. Biol Blood Marrow Transplant. 2008 Jun;14(6):641-50.

INB-100: A Phase 1 Trial to Reduce Leukemic Relapse

Dose-escalation trial of DeltEx™ Allo gamma-delta T cells post-haploidentical HSCT

Treatment Arms

Single, ascending dose levels in a 3+3 design:

1. N = 3 (up to 6) patients, single dose of 1 x 10^6 cells/kg
2. N = 3 (up to 6) patients, single dose of 3 x 10^6 cells/kg ← Recommended Phase 2 Dose (RP2D)
3. N = 3 (up to 6) patients, single dose of 1 x 10^7 cells/kg

Treatment Regimen & Timing

Fludarabine + cyclophosphamide + TBI = 6 days → Haploidentical HSCT* → INB-100 infusion within 7 days after engraftment

*Neutrophil engraftment is ~15-20 days following HSCT

Key Eligibility Criteria

- Adult patients with a haploidentical donor identified
- KPS ≥70
- AML in mCR with intermediate/high-risk features or relapsed disease
- CML in any chronic phase
- MDS with intermediate/high-risk features
- ALL in mCR with high-risk features or relapsed disease

Primary Endpoints

- Safety
- RP2D of DeltEx™ Allo gamma-delta T cell infusion
- Dose limiting toxicity (DLT)

Secondary Endpoints

- Incidence of acute and chronic graft versus host disease (aGVHD), relapse, and overall survival

Site

The University of Kansas Cancer Center

Currently on-boarding additional centers

We Treated Difficult, High-Risk Leukemia Patients

Patient	Dose Level	Age / Sex	Prior Therapies	Disease	Acute / Chronic GvHD	CR (mos)	Survival
002	1	63 / female	Idasanutlin + 7+3	High-risk AML trisomy 8+ and del7, FLT3 TKD	Acute G2 GvHD Chronic limited GvHD	57.2+	Alive
003	1	44 / female	7+3	High-risk AML trisomy 8+ and del7, IDH2	Acute G2 GvHD	42.4** LTFU	Alive
006	1	66 / male	7+3 IDAC	High-risk relapsed AML	Acute G2 GvHD Chronic extensive GvHD	43.1+	Alive
007	1	71 / male	Ven/Aza+Pembrolizumab	AML	Acute G2 GvHD Chronic limited GvHD	15.5	15.5m died due to IPS
009	2	68 / male	R-CHOP, Blinatumomab, Inotuzumab, Flu/Mel/TBI, Vincristine/steroids, Flu/cy/brentuximab, CAR-T with Tecartus	Relapsed Ph- ALL; TP53 mutated	Acute G2c GvHD	14.7	20.2m
010	2	63 / female	7 cycles Venetoclax/Aza	AML	Acute G2b GvHD	26.5+	Alive
011	2	68 / male	Hydrea/Peg-IFN	ET with MDS/MPN overlap; TP53 mutated		12.4	18.3m
012	2	69 / male	2 cycles Venetoclax/Aza	AML		20.1+	Alive
013	2	71 / female	1 cycle Ven/aza/gliteritinib 2 cycles Venetoclax/Aza	AML, FLT3		19.8+	Alive
014	2	71 / male	Venetoclax/Dacogen	AML, del20, -Y		19.4+	Alive
015	2	69 / female	Aza	MDS, Complex cyto, FISH -17p, del -7, -5. NGS VUS: NSD1	Acute G1 GvHD Chronic limited GvHD	11.4	Alive @ 12.2m+
017	2	69 / male	Revlimid, Azacitidine+CD70 (SGN70 trial), Decitabine +Venetoclax	MDS,5q deletion		8.5+	Alive
018	2	64 / male	Ven/Aza	MDS high-risk, asxl1, kras, srsf2, and tet2 variants	Acute G1 GvHD	8.1+	Alive
019	2	45 / female	Daunorubin /Visticrine, Cytarabine /Methotrexate, Cyclophosphamide/Methotrexate	B-ALL		5.4+	Alive
020	2	61 / female	Ven/Aza	AML -expression of dim CD45, CD33, CD38, CD117 and myeloperoxidase	Acute G1 GvHD	4.7+	Alive
021	2	71 / female	3 cycles Decitabine/Cedazuridine, Investigational drug	MDS, del 5q and del 7q		1.0+	Alive
023	2	68 / male	Ven/Aza	AML – intermediate risk			

Median patient age ~68 y/o

Majority have AML

Received up to 7 prior therapies

23 enrolled, n=16 dosed and evaluable for safety

- 1 manufacturing failure
- Others disqualified for medical reasons
- 1 patient waiting to be dosed

- **Median follow-up = 18.8m**

- **Median follow-up of AML patients = 20.1m, 23.3m ex-expansion cohort**

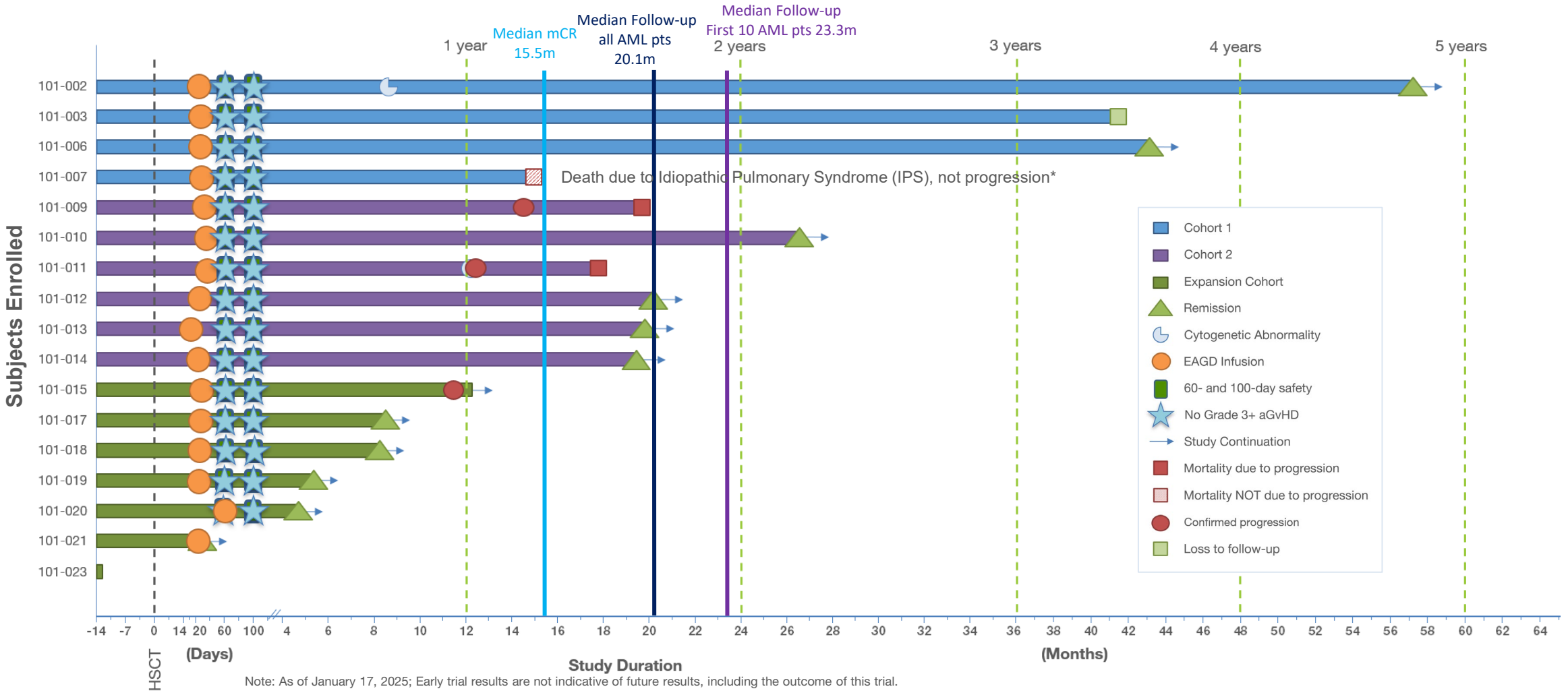
All patients with intermediate or high-risk disease

Note: As of January 17, 2025; Early trial results are not indicative of future results, including the outcome of this trial.

INB-100 is Demonstrating Durable Remissions in Complex AML Patients

INB-100 γδ T cells Show Durable AML Control

Three patients with high-risk disease remain relapse free for > 3 years with median follow-up of all patients of 20.1 months; No AML patients have relapsed to date with median follow up of 20.1 months (23.3 months, ex-expansion cohort)



Note: As of January 17, 2025; Early trial results are not indicative of future results, including the outcome of this trial.

INB-100 Patients have Improved PFS and OS at 1-year

	IN8bio INB-100 all haplo (N=11)	**IN8bio** INB-100 AML, (N=8)	**KUCC** All haplo, (N=98)*	**KUCC** AML, (N=54)*	**CIBMTR** AML, (N=684)^
Age (median)	68 (44-72)	68 (44-72)	64.3 (21-74)	64.1 (21-74)	65.6 (19.2-80.8)
Sex, female %	45.5% (5)	50% (4)	31.6% (31)	33.3% (18)	45.9% (314)
Karnofsky performance Status ≥ 90%	18.2% (2)	12.5% (1)	22.4% (22)	14.5% (8)	40.9% (280)
HCT-specific comorbidity index ≥3, n (%)	54.6% (6)	75% (6)	57.1% (56)	63% (34)	58.9% (403)
Female donor %	27.3% (3)	25% (2)	45.9% (45)	42.6% (23)	39.7% (271)
Day-100 acute graft-vs-host disease (GVHD), n (%)	72.7% (8)	62.5% (5)	61.2% (60)	64.8% (35)	NA
Non-relapse mortality (NRM), % (N)	9.1% (1)	12.5% (1)	30.6% (30)	25.9% (14)	NA
Outcomes at 1 year					
Progression Free Survival (PFS)	90.9% (10)	100% (8)	59.2% (58)	57.4% (31)	67.8% (679)
Overall Survival (OS)	100% (11)	100% (8)	69.4% (68)	66.7% (36)	74.7% (684)

^ CIBMTR CM24-35, "Retrospective analysis of RFS and OS in AML patients undergoing haploidentical transplant with 62% (N=424) receiving reduced intensity conditioning (RIC) regimen."
* Patient and transplant-related characteristics after FluCyTBI non-myeloablative RIC haploidentical HCT with PT-Cy-based GVHD prophylaxis in AML patients 2016-2024 (KUCC)

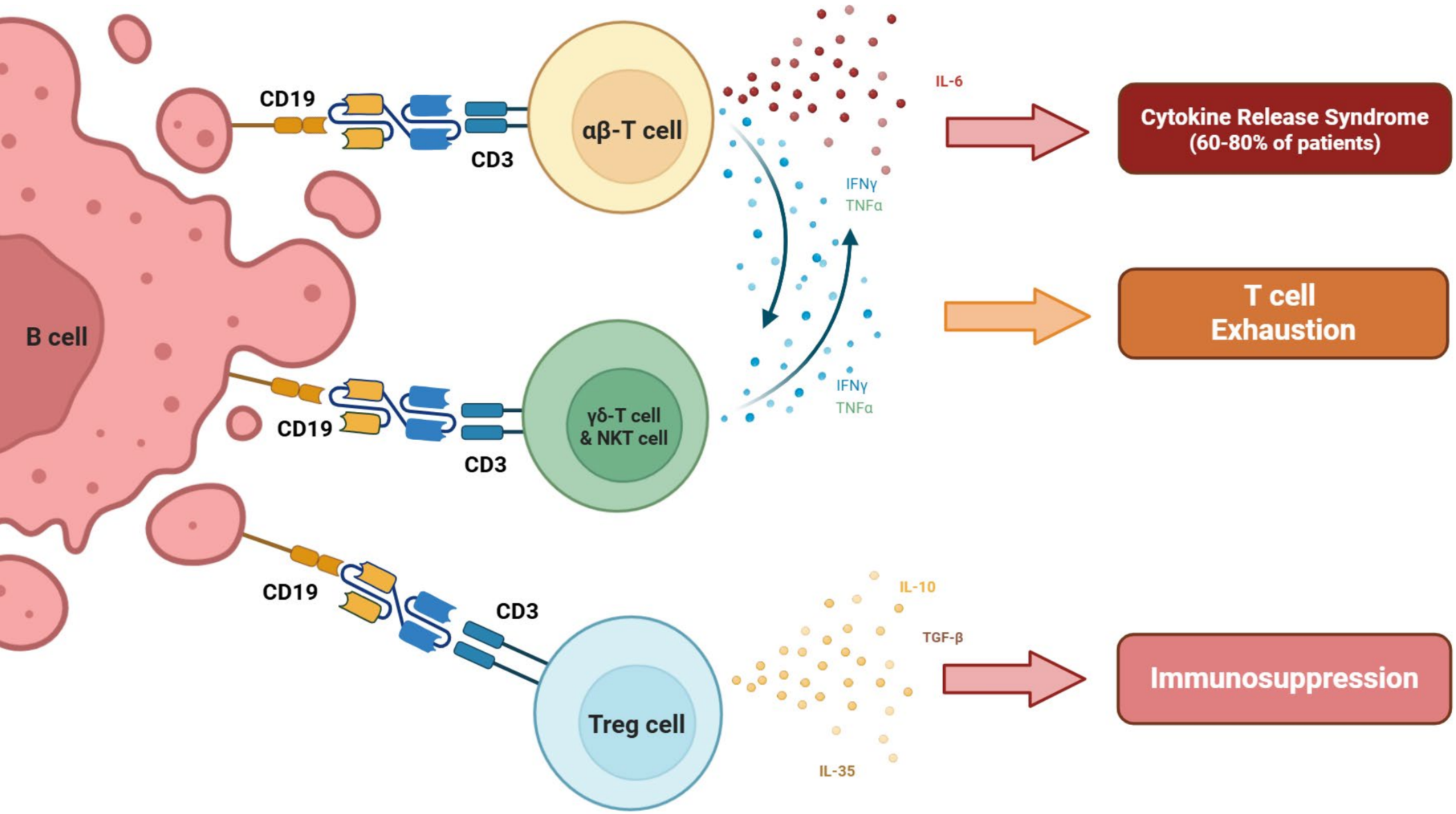
- Relapse in leukemia patients who have undergone haplo-HSCT are significant and often leads to death
- INB-100 patients have an **increased rate of Progression Free Survival (PFS) and Overall Survival (OS) at 1-year** compared to retrospective control data sets
- **100% AML patients remaining in remission to-date**, with median follow-up of 20.1 months and 23.3 months excluding patients in the expansion cohort

γδ T Cell Engagers (TCE)

Enhancing the cancer killing function of γδ T cells

IN8bio's TCE Platform Leverages the Power of γδ T cells for Autoimmune Diseases

Conventional CD3 TCEs Have a Narrow Therapeutic Window



… Resulting in Failure to Achieve Immune Reset to Date

- X Many TCE's in development do not offer the broad B cell coverage of CD19
- X 60-80% of patients experience CRS and ~10% >Gr. 3 CRS
- X Broadly targeting CD3 pushes T cells to exhaustion limiting their killing abilities
- X This narrow therapeutic window prevents higher dosing with current TCEs
- X Affinity de-tuning of the binding domains also causes the TCE to fall off the T cell
- X As a result, CD3 based TCE's may be ineffective at targeting tissue resident B cells

These factors drive the low depth of B cell depletion from protein engagers in Schett's data

IN8bio Has a Strategy to Overcome These TCE Limitations

Our approach to TCE development:

Targeting and Tissue Penetration

Deep B cell depletion can be achieved through specific activation of immune subsets that can drive circulatory, lymphoid and tissue-specific targeting

T cell Expansion

Meaningful responses require sufficient numbers of effector T cells, which can be achieved by targeted cell expansion through both the TCR and an expansion domain

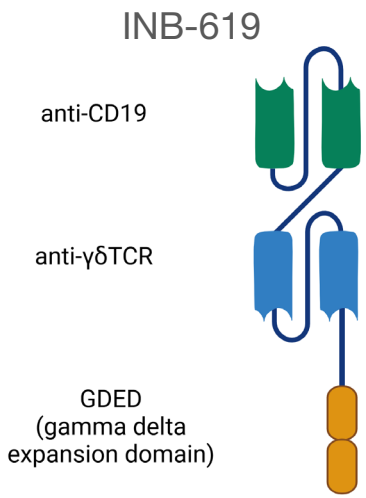
Higher Tolerability

Most TCEs are limited by toxicity, such as CRS and ICANs that prevent higher dosing and deeper target elimination

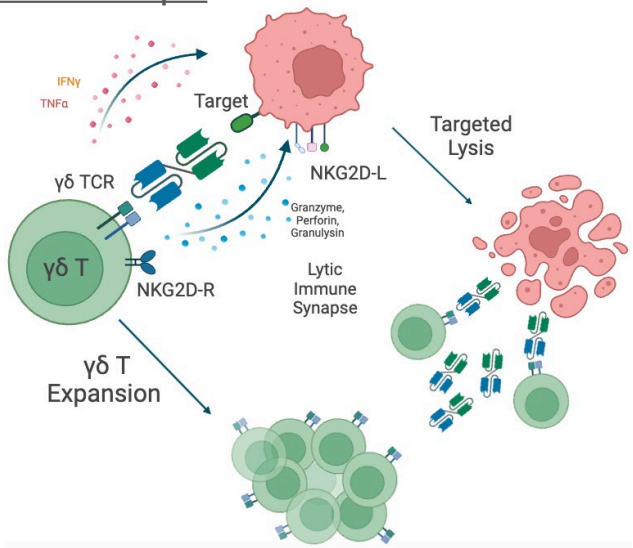
IN8bio's γδ TCE Platform: Differentiated and Broadly Applicable

- The first γδ TCE to show pan-γδ T cell expansion and activation
- Engages γδ T cells through a unique mechanism, binding of the γδ TCR
- Precision activation and expansion of γδ T cells drives efficient target cell elimination without the cytokine activation associated with CD3-directed TCEs
- The platform's design is versatile, allowing for the development of multiple products targeting different antigens, providing broad potential for treatment
- The ability to expand powerful γδ effector cells, together with the potentially enhanced safety profile, positions this approach for broad applicability across the autoimmune and oncology landscape

Novel Expansion Domain Technology



Novel pan-γδ TCR binding & activation for powerful effector function



IN8bio Leverages Biology & Engineering for a Powerful γδ TCE

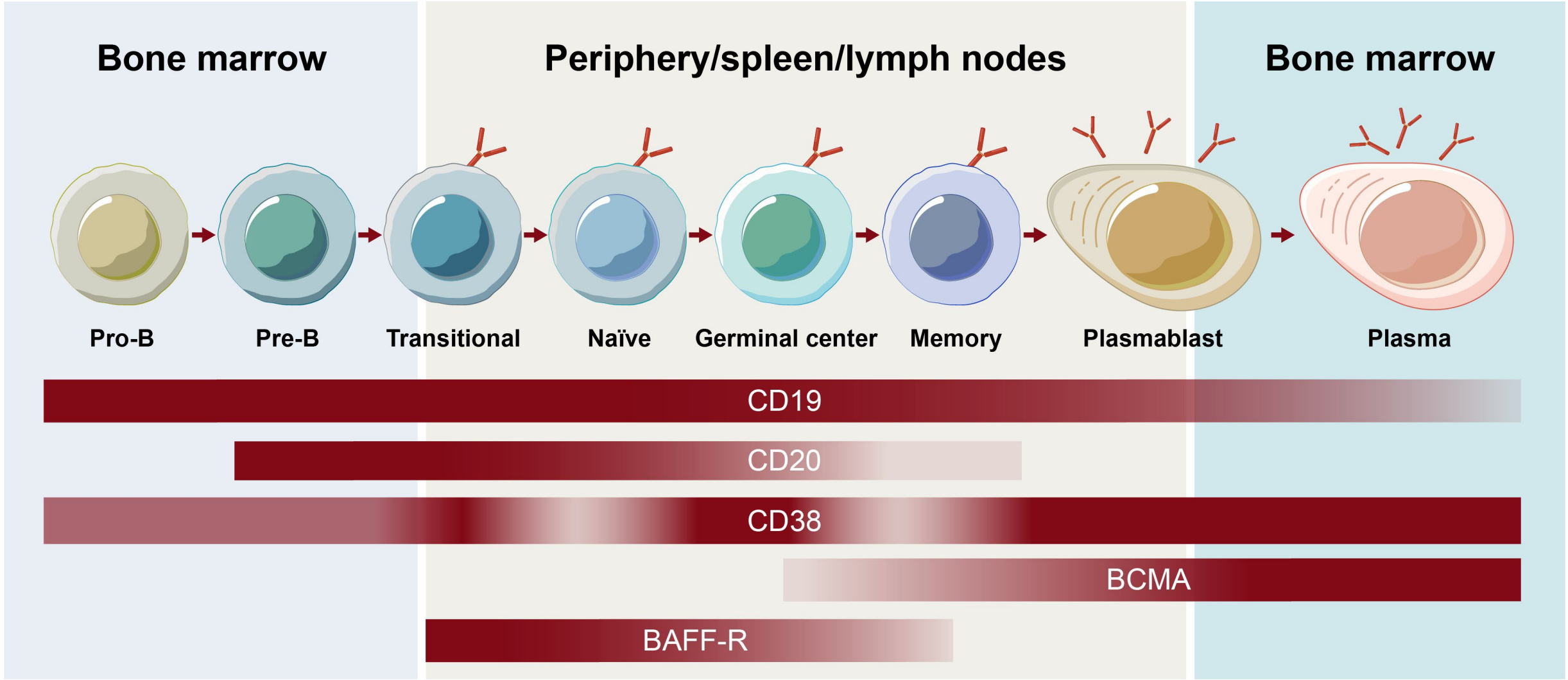
The Key Features and Advantage of IN8bio's γδ TCE Platform

- Increasing γδ T cell levels can drive effective and deep target elimination
- Reduced cytokine levels are needed for higher TCE doses and broad application in autoimmune diseases
- Pan-γδ T cell expansion and activation of both Vd1+ and Vd2+ drives complete target cell elimination
- Pan-γδ TCE activity directs complete target elimination in healthy & dysregulated immune background
- This novel TCE approach is designed to work across healthy & disease associated immune variation

This approach was developed based on IN8bio's deep understanding of γδ T cell biology & effector function

INB-619: A Unique B cell Depleting γδ TCE

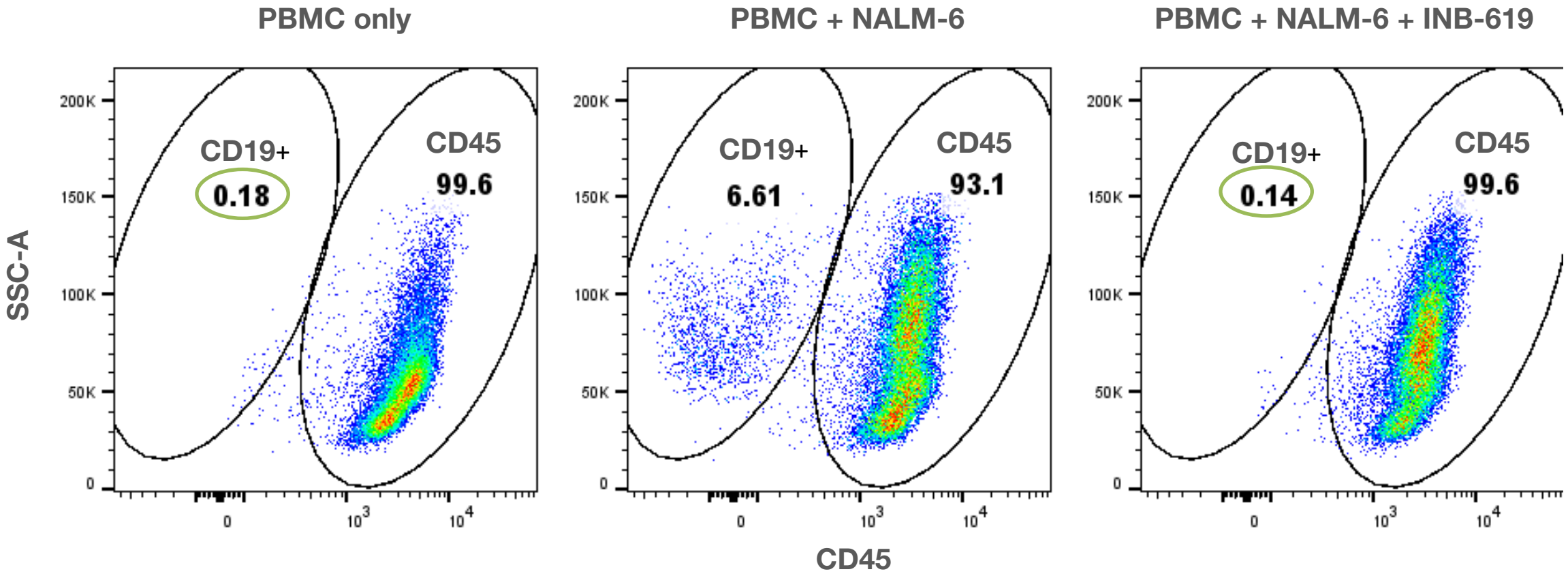
Targeting CD19 for the Broadest B cell Coverage



Source: Robinson et al., Front. Immunol. 15:1454747. doi: 10.3389/fimmu.2024.1454747

INB-619 Drives Target Elimination with γδ T cells from PBMC

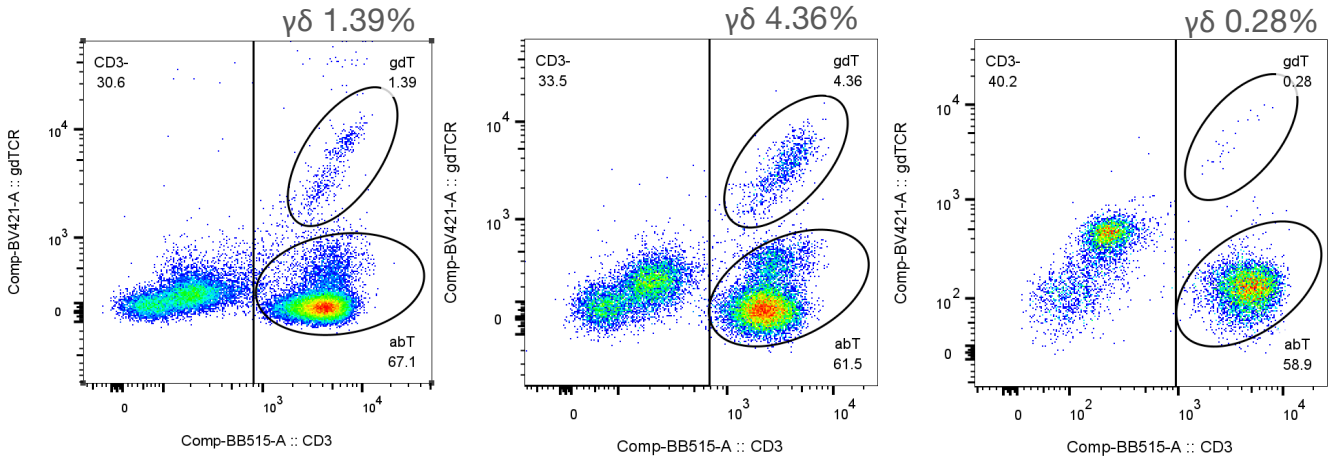
NALM-6 (CD19+) ALL cells were spiked into PBMC and cultured +/- CD19TCE over 6 days - Note complete eradication of CD19+ cells after 6 days culture with the CD19 TCE (INB-619)



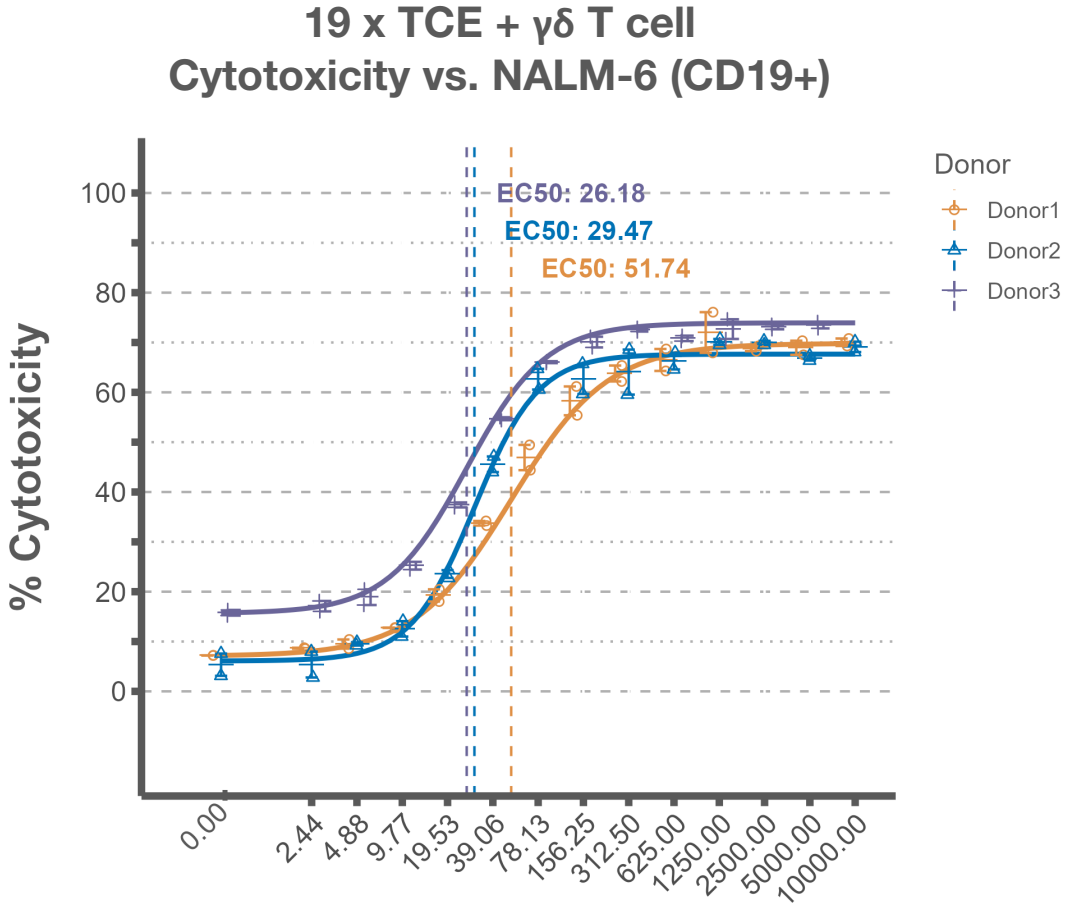
INB-619 Demonstrates Clustered EC50 across Donors

γδ T cell levels range from 1-5% in healthy donors and can be as low as 0.2% in cancer & autoimmune patients

- INB-619 shows tight clustering of EC50 in donors ranging from 0.2-5% initial γδ T cell levels
- This suggests that target cell elimination and tight EC50 clustering is due to the ability to induce γδ expansion and activation and not initial γδ levels

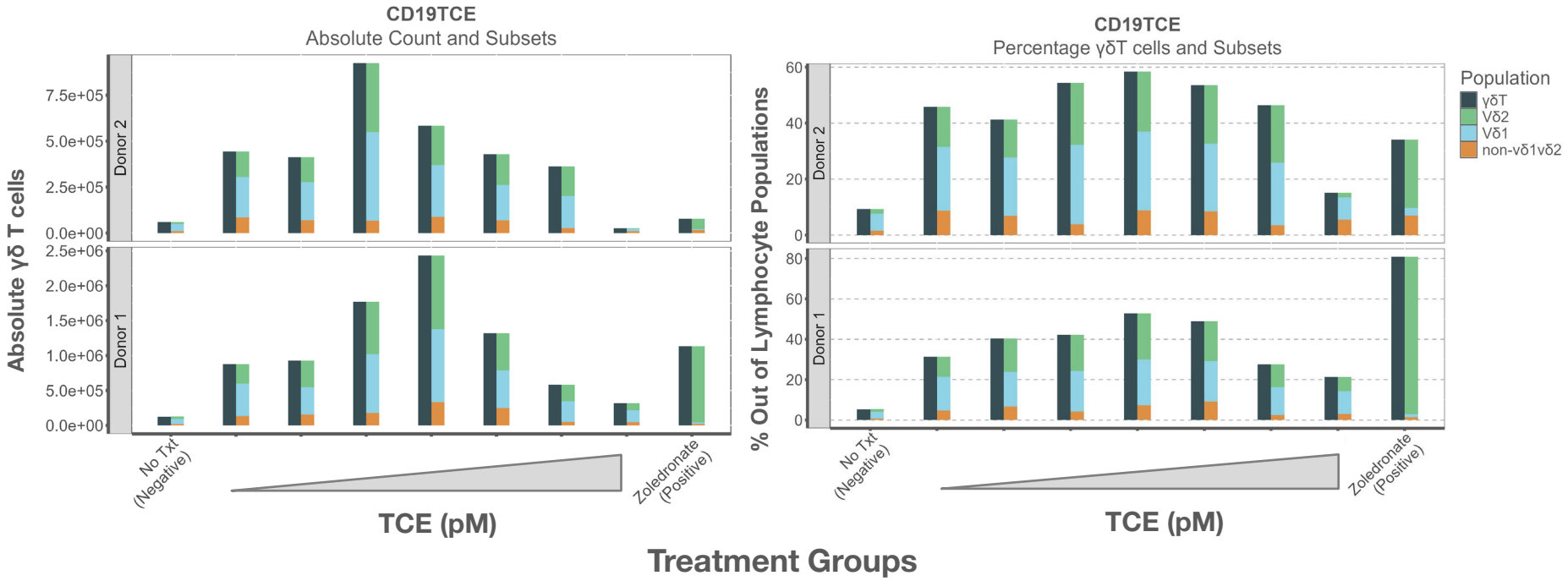


Donor 1, Day 0 | Donor 2, Day 0 | Donor 3, Day 0



IN8bio's γδ TCE's function as a Pan-γδ T cell Expander

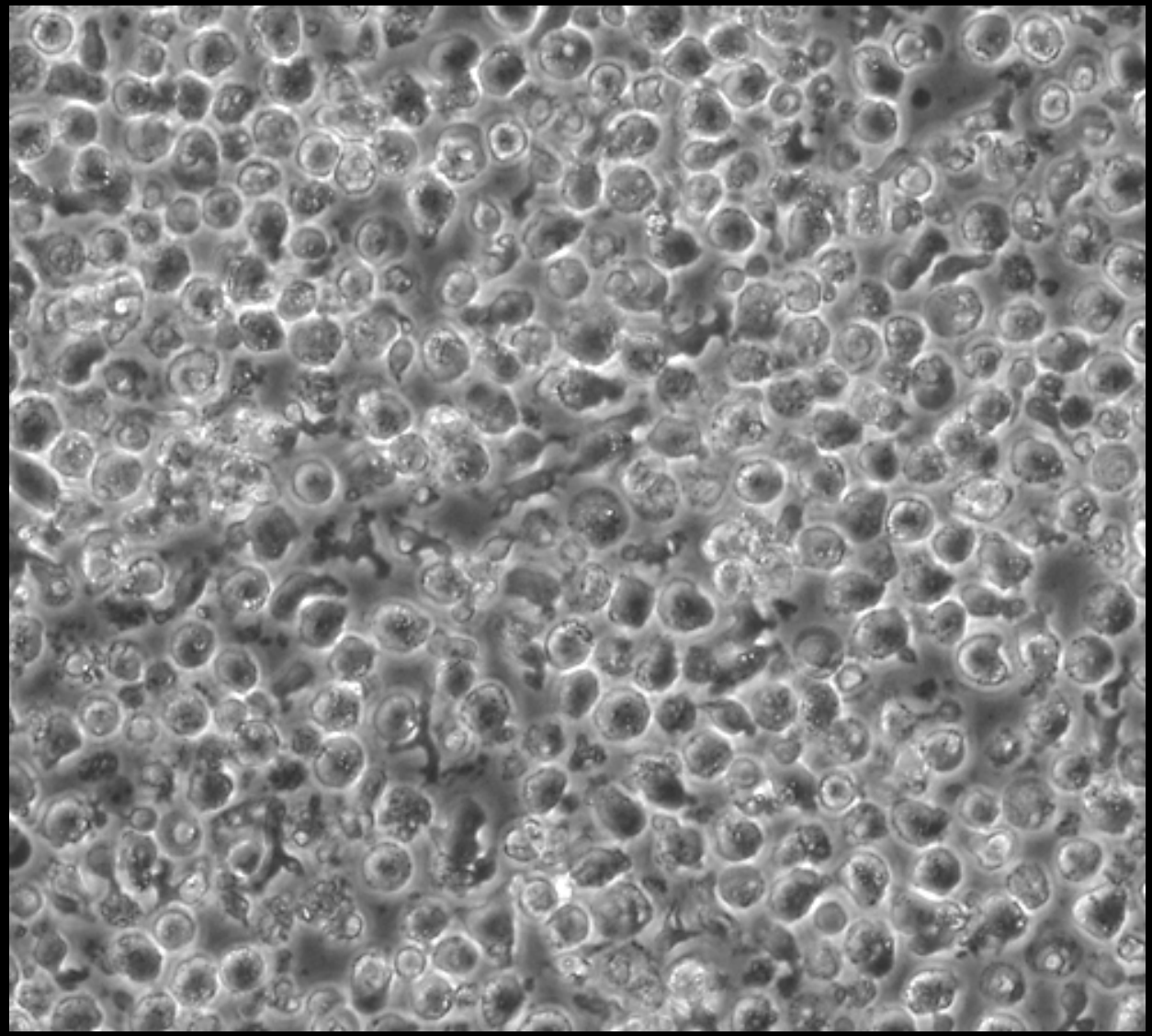
CD19-γδ TCE significantly expanded both Vδ1+ and Vδ2+ T Cells



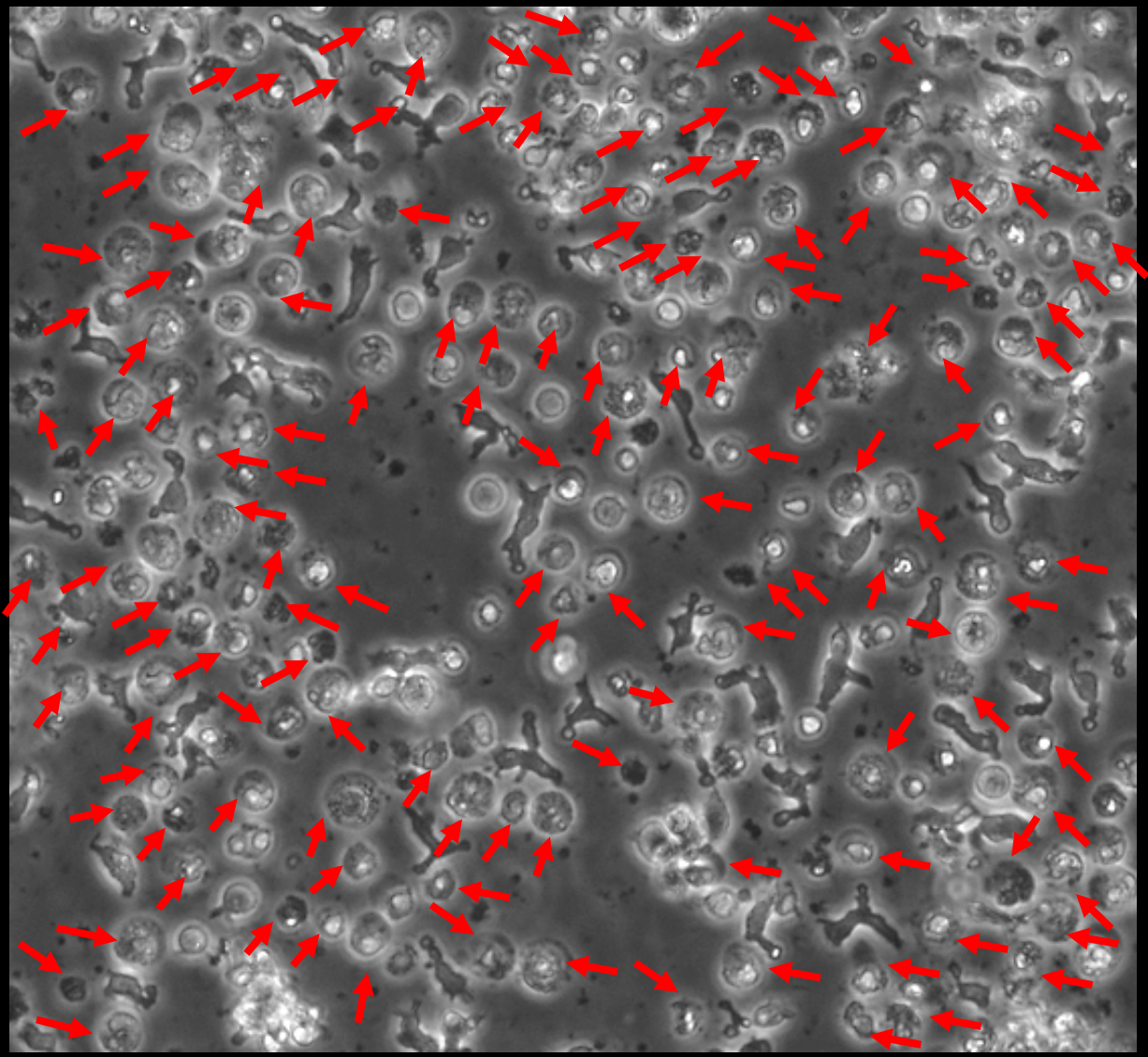
Frequency of Expanded γδ T cell Numbers (Day 10)

- CD19-γδ TCE expanded γδ T cells from PBMC during the cytotoxic lysis of normal B cells or malignant B cell lines
- Zoledronate (positive control) expanded Vδ2+ cells from PBMC as expected
- γδ T cells + PBMC without added CD19-γδ TCE (NoTxt) did not expand any γδ T cells (negative control)
- Both Vδ1+ and Vδ2+ T cells are activated and proliferate
- Vδ1+ γδ T cells are known to be **tissue resident** potentially allowing for **deeper B cell depletion**
- **To our knowledge, no other TCE has been shown to drive expansion and proliferation at this magnitude**

CD19-γδ TCE Induced Killing is Clearly Visible



γδ T vs. Nalm6 without 19xTCE

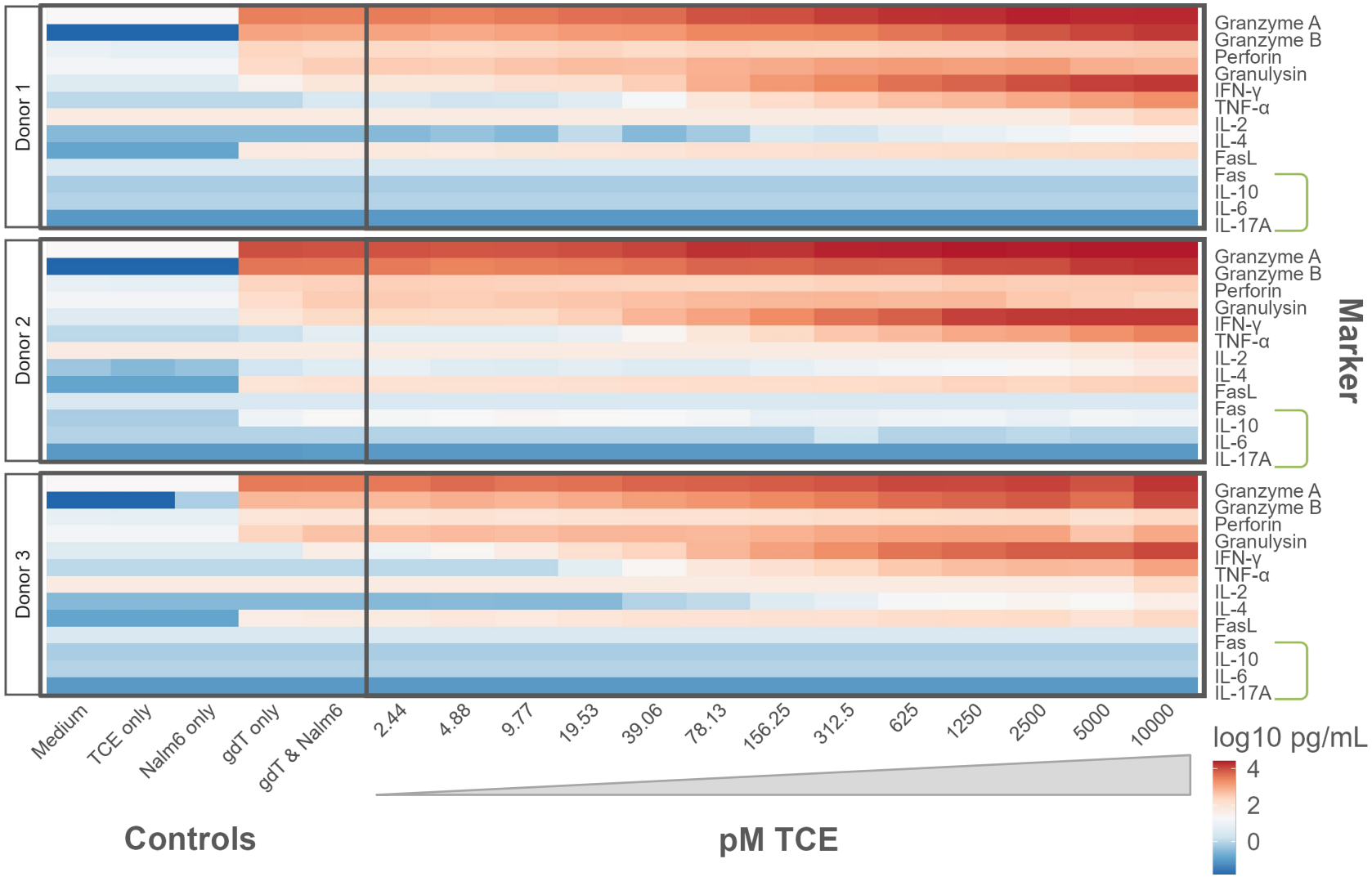


γδ T vs. Nalm6 with 19xTCE

E:T = 1:1 @ 24 hours

Adverse Cytokines Not Observed with IN8bio's CD19-γδ TCE

IL-6, IL10 or IL-17 cytokine secretion remain flat across the dose curve, in-line with controls



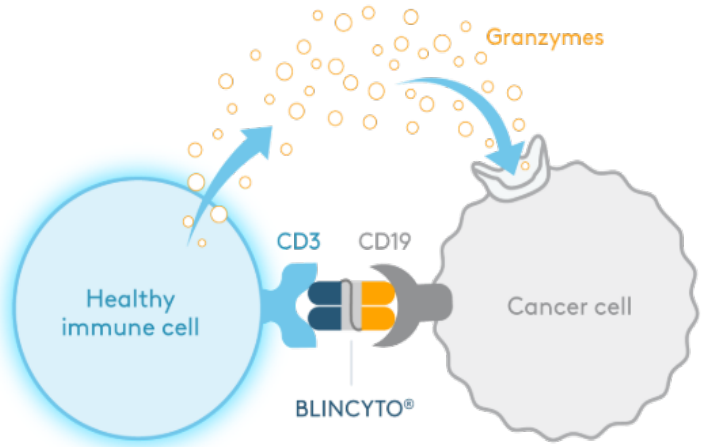
INB-619 for Autoimmune Disease

INB-619 vs Blinatumomab and Mosunetuzumab: A Comparative Analysis of B cell Killing

Commercial B cell Targeting Agents vs. IN8bio's CD19-TCE

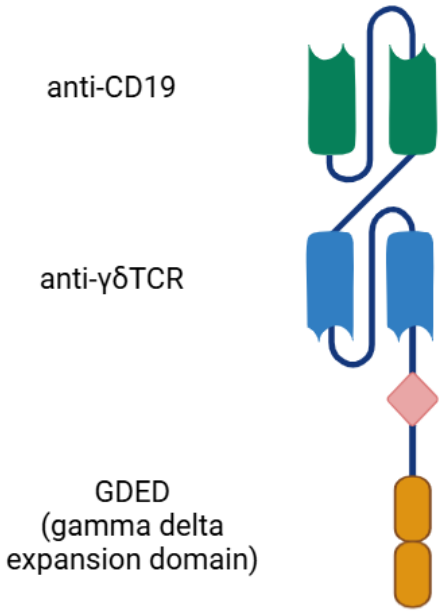
Structure of IN8bio's γδ TCE compared to commercially available CD19 or CD20 B cell targeting therapies

Blinatumomab
CD-19 TCE



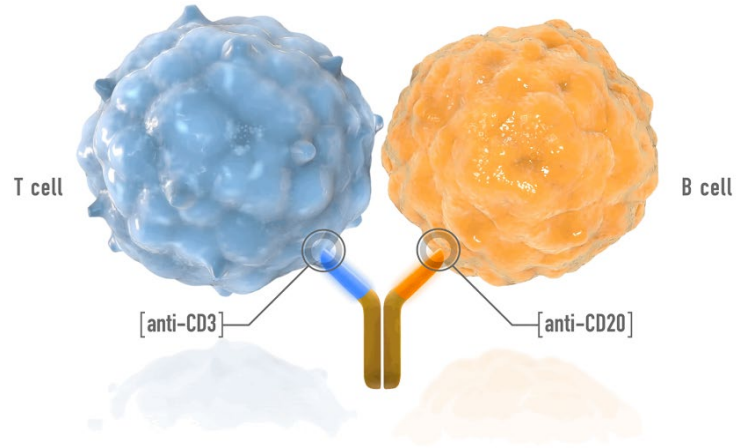
~54 kDa

INB-619
CD-19 TCE



~100 kDa

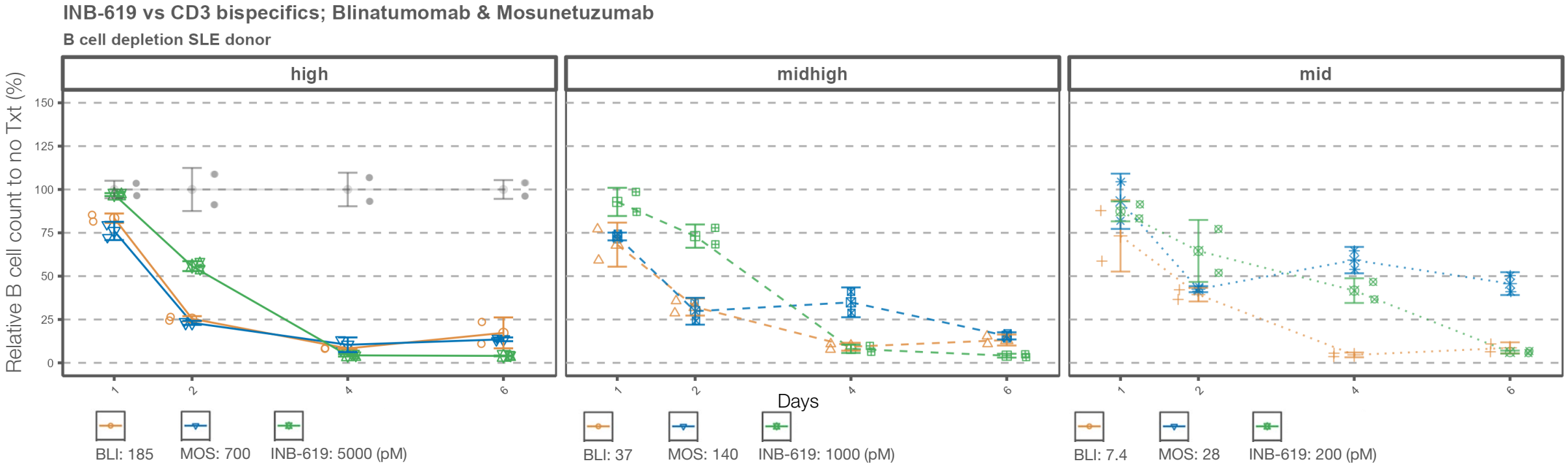
Mosunetuzumab
CD-20 TCE



~146 kDa

INB-619 Depletes B cells Across a Range of Concentrations

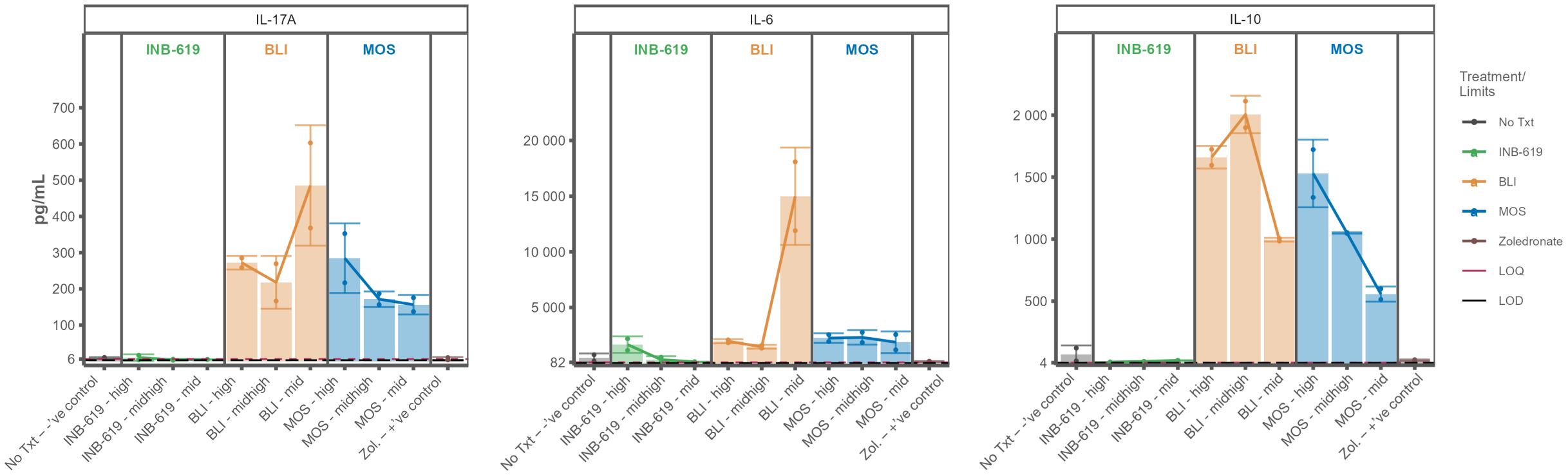
SLE donor B cell depletion comparing INB-619, BLI and MOS



INB-619 can eradicate target B-cells as efficiently or more efficiently than commercial BLI and MOS therapies at multiple concentrations, all compounds are less effective at low concentration overtime likely due to degradation

INB-619 Demonstrates Lower Secretion of CRS Cytokines

SLE donor cytokine secretion at Day 4



INB-619 demonstrated significantly lower secretion of cytokines associated with CRS at doses that completely deplete B cells. This widens the therapeutic index related to commercial BLI and MOS therapies at multiple concentrations

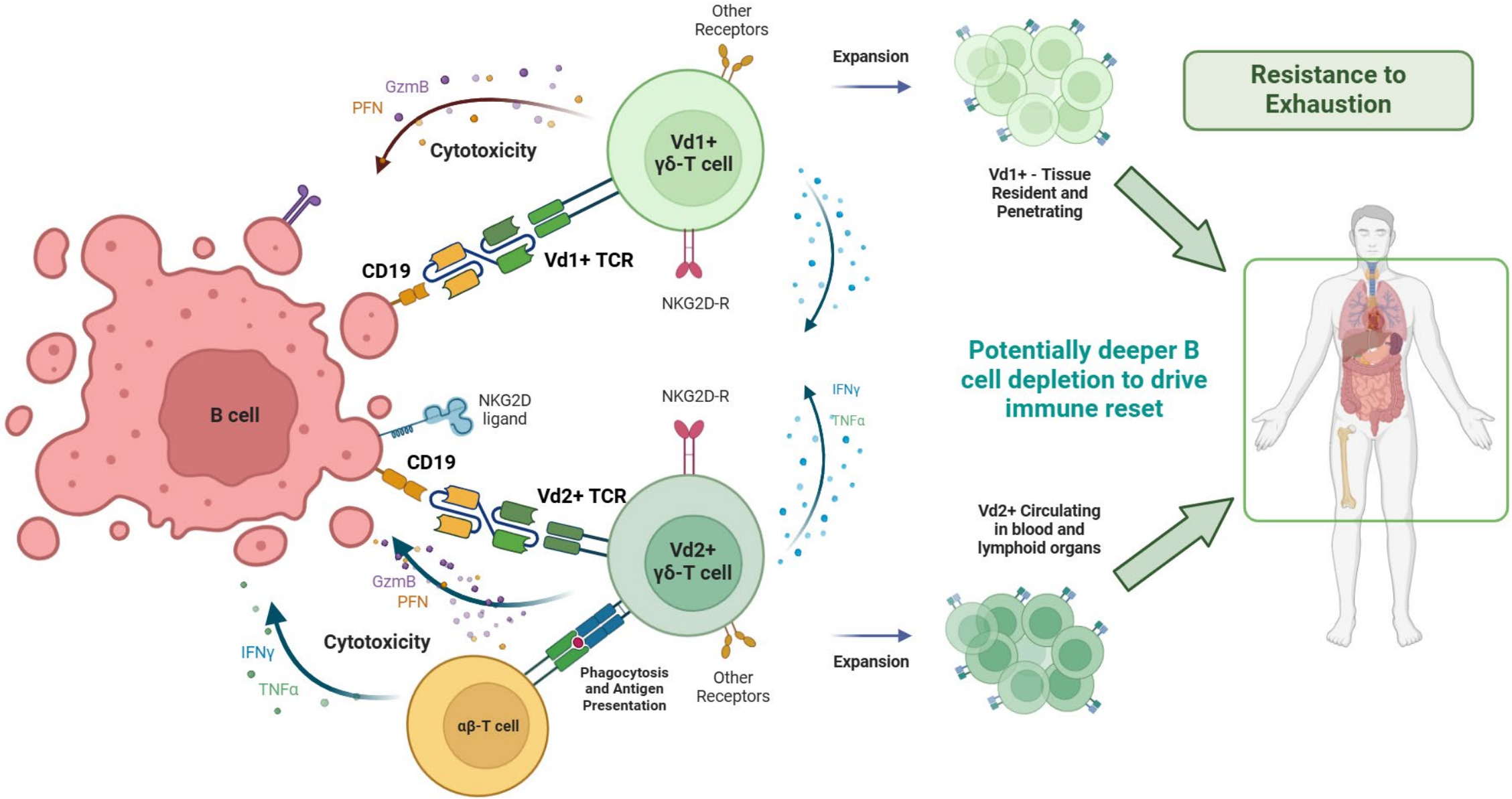
INB-619: Designed to Overcome Autoimmune Challenges

IN8bio's γδ TCE significantly expands both Vδ1+ and Vδ2+ T cells

- Autoimmune disease often exhibits immune dysregulation with both overactive and exhausted immune cell subsets, which can change overtime
- Different immune subsets may respond differently in different patients, which can be difficult to predict at the time of treatment
- To effectively drive B cell elimination, the IN8bio TCE was designed to function as a pan-γδ T cell engager, expanding and activating both primary γδ T cell subtypes, the Vd1+ and Vd2+
- This can induce B cell depletion through activation of either or both γδ T cell subtypes, driving target elimination in patients and tissues with variable immune response to stimulation
- This cannot be achieved by the other γδ TCE's that only target a single γδ T cell subtype, which leads to inefficient target elimination in some patients

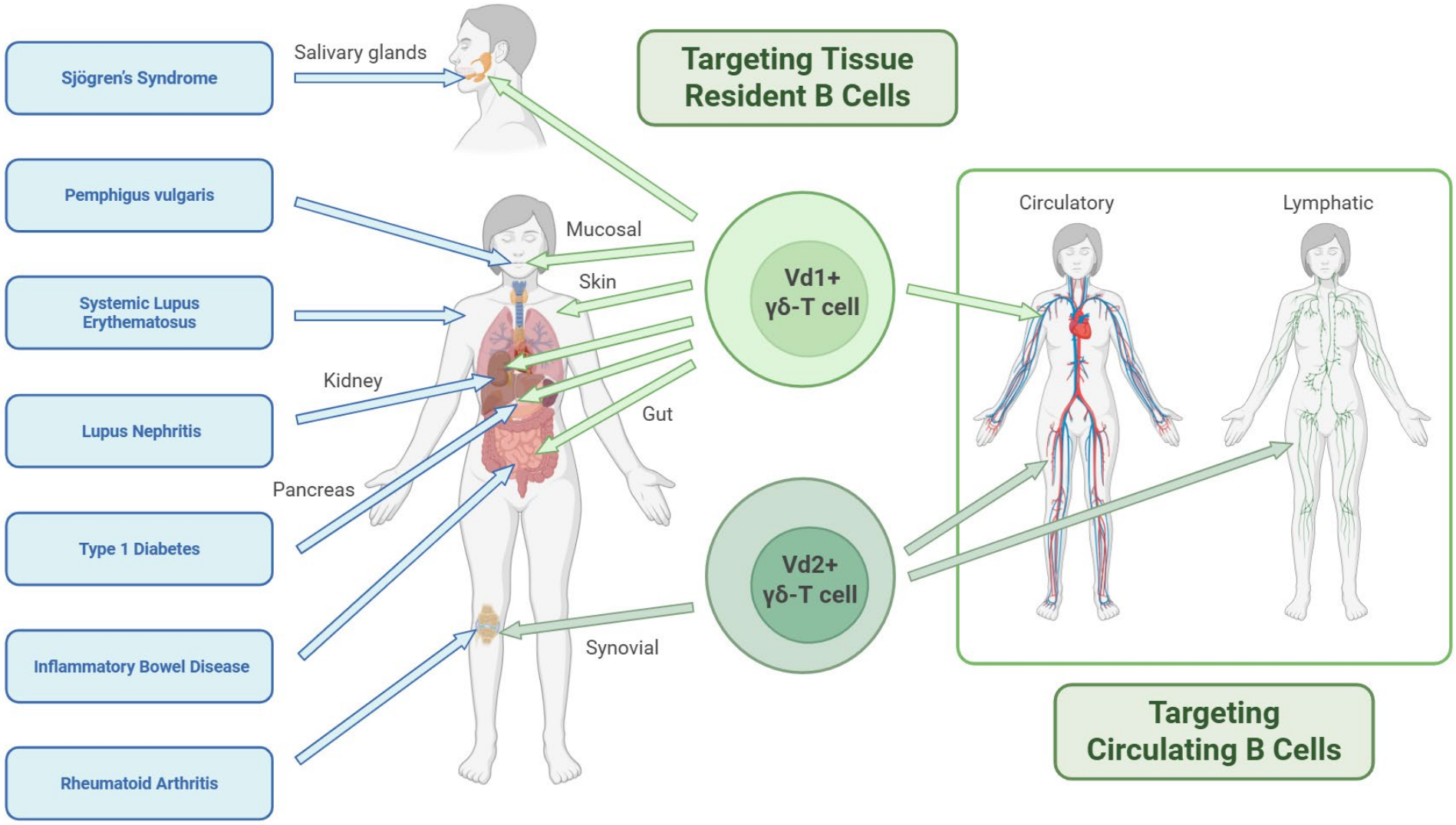
Our γδ TCE has Therapeutic Advantages

Driving to Deeper B cell Depletion with a Pan-γδ TCE



γδ T cell Residence in Tissues of Autoimmune Diseases

Tissue, circulatory and lymphoid residence of γδ T cells may result in deeper B cell depletion



INB-619 - CD19-γδ TCE Provides Unique Advantages

- ✓ Pan γδ TCE demonstrates ability to eliminate specific target cells in a dose-dependent manner
- ✓ Significantly expands both Vδ1+ and Vδ2+ T cells
- ✓ CD19 broadly targets the B cell compartment
- ✓ Vδ1+ cells resist exhaustion and can target tissue resident B cells for deeper B cell depletion
- ✓ Vδ2+ cells are phagocytes that drive deeper B cell depletion as seen with Dren Bio's myeloid cells
- ✓ γδ T cells secrete less IL-6 and may reduce CRS and ICANs as is common with CAR-T & CD3 TCE's
- ✓ TCEs allow simpler manufacturing, lower costs, repeat dosing and avoids lymphodepletion

Corporate

Anticipated Milestones Across Pipeline^

- Ticker: **INAB**
- $27.1M Cash on hand at Dec. 31, 2025
  - Cash runway into 2Q27
  - Potential 2nd close for additional $20.1M on TCE data in 2026
- Potential for up to ~$8.9M in additional capital available
- $0 debt
- 9.8 million common shares outstanding as of March 9, 2026

2026

INB-100	Complete treatment of expansion cohort patients at multiple centers 1H26
	Report long-term follow-up of Phase 1 results at medical meeting in late 2026
INB-200 / INB-400	Present long-term data with final median OS from treated patients at medical meetings in mid- and late-2026
	Peer-reviewed publication of Phase 1 data
	Complete discussions with the FDA on potential clinical pathways, including any potential for accelerated approval
INB-619	Present initial animal data on CD-19 targeting γδ T cell engager (TCE)
	Report additional autoimmune data at medical meetings in 2026

^Timing of next anticipated milestones are estimates based on the successful raise of additional capital to fund our programs and subject to change

IN8bio Board of Directors & Key Advisors

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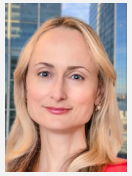

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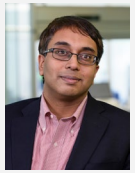
Scientific Advisory Board



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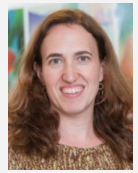

Bianca Santomasso, MD, PhD
MSKCC

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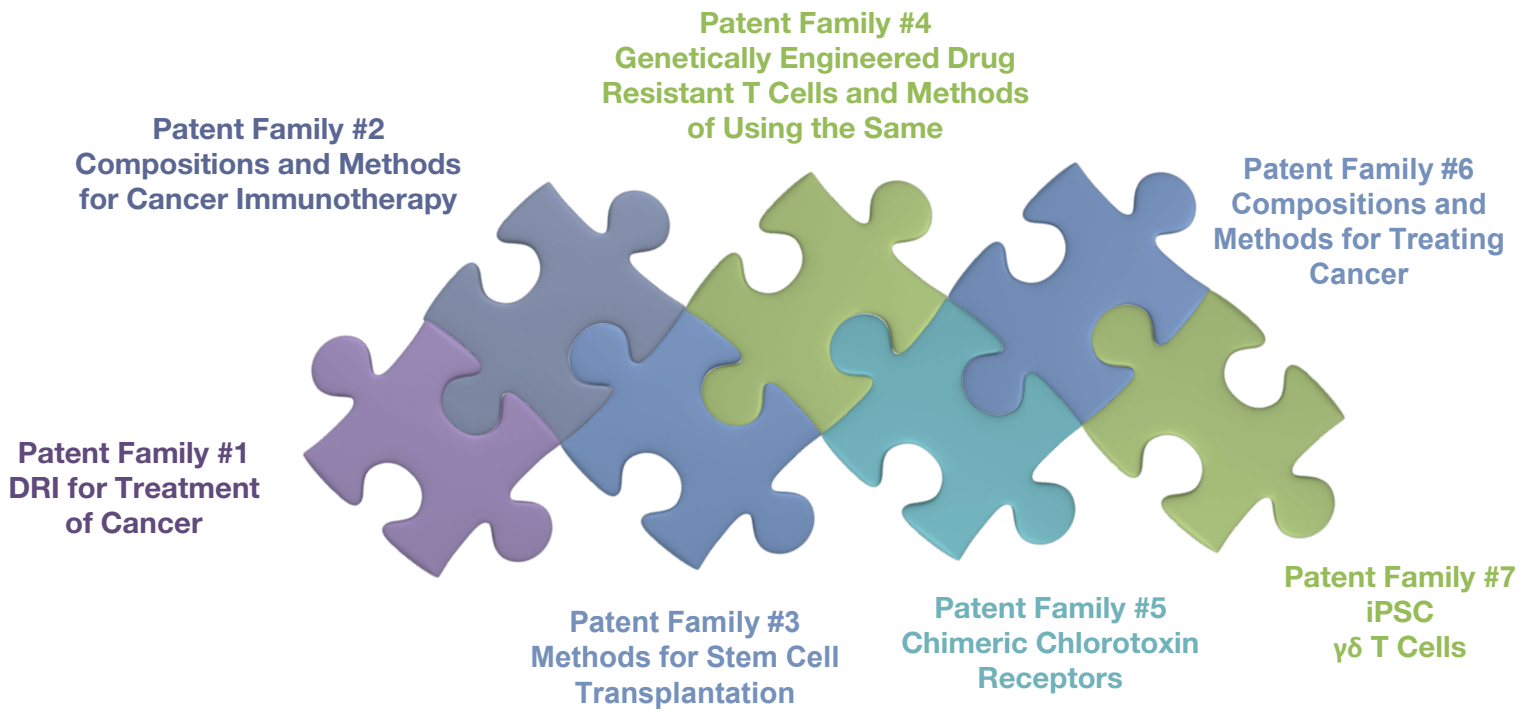
IN8bio

A Robust Intellectual Property Portfolio

Coverage inclusive of both issued and allowed (US, EU and worldwide) methods-of-use and composition-of-matter patents

- Data and “Know-How” exclusively licensed from the University of Alabama at Birmingham (UAB), Emory University (Emory) and Children’s Healthcare of Atlanta (CHOA)
  - Includes all in-vivo and in-vitro data and patient data from any clinical trials
  - Manufacturing expertise including GMP expansion and transduction of γδ T cells
- Broad strategy for coverage across multiple disease states



IN8bio Harnessing the Power of γδ T cells

- **Differentiated Mechanism of Action –** The unique properties of IN8bio's TCE and cellular therapies set it apart from other approaches by providing dual mechanisms to targeting both residual cancer cells and dysfunctional immune cells and achieving deeper target depletion
- **Long-Term Remissions in Oncology –** Sustained, durable remissions exceeding 4+ years observed in difficult indications including AML and GBM
- **Superior Safety Profile –** Our clinical data demonstrates no CRS nor ICANs to date. Current approaches are often limited by a narrow therapeutic window, preventing higher doses to achieve complete target depletion
- **Unique Value in a Large Market –** Our programs are addressing key unmet needs for autoimmune diseases and oncology
- **Proven Execution –** An experienced team with a track record of achieving milestones and delivering strong clinical data to advance a differentiated pipeline
- **Multiple Near-Term Value Catalysts –** Creating clear opportunities for additional funding and stock price appreciation

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