Harnessing the Power of Gamma-Delta T Cells

June 2026

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IN8bio - Who We Are

A clinical-stage biotech company developing a new class of treatment for autoimmune diseases and cancer powered by gamma-delta (γδ) T cells, a rare but exceptionally potent white blood cell

Deep Experience Across the Team

William Ho
Co-Founder, Chief Executive Officer

AlephPoint Capital
NEW LEAF VENTURE PARTNERS
Bank of America
PiperJaffray.
COWEN

Lawrence Lamb, PhD
Co-Founder and Chief Scientific Officer

O'NEAL COMPREHENSIVE CANCER CENTER
THE UNIVERSITY OF ALABAMA AT BIRMINGHAM
Palmetto Health USC MEDICAL GROUP
UNIVERSITY OF SOUTH CAROLINA

Patrick McCall, CPA
Chief Financial Officer

Deloitte.
CHUBB
CATALYST BIOSCIENCES
TURNSTONE BIOLOGICS

Kate Rochlin, PhD
President & Chief Operating Officer

CURADIGM
EXPANDING LIFE
BIOTAGENICS
IMMUNOVENT
filament
BIOSOLUTIONS INC.

Lou Vaickus, MD, FACP
Interim Consulting Chief Medical Officer

TOLERX
Immune resolve.
aktaPD
VERTEX

A team built around γδ T cells, cell therapy, strategic finance and execution

- 35+ years of γδ T cell expertise
- Decades of extensive background in oncology discovery, business insights, franchise creation, product development, regulatory affairs, and commercialization
- Clinical development experience across immunology, oncology and cell therapy
- Business development, financing, and commercialization experience

Revolutionizing γδ T cell Therapies

A New Way to Fight Both Autoimmune Disease and Cancer

- **One platform: two major disease areas with massive unmet need**
  - **In autoimmune disease -** reset a misfiring immune system without suppressing it entirely
  - **In cancer -** kill residual tumor cells that surgery and chemo leave behind
- **No serious side effects observed to date in either setting**

IN8bio R&D facility, Birmingham, AL

A Robust Pipeline with Multiple Near-Term Readouts

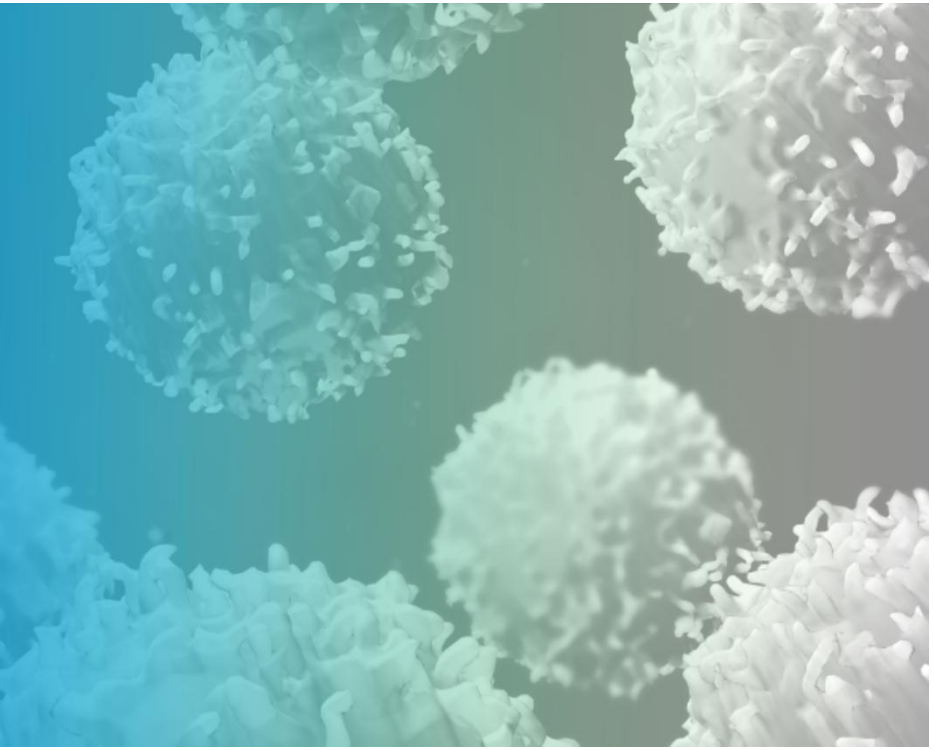
Product Candidate	Approach	Key Indications	Preclinical	Phase 1	Phase 2	Phase 3	Next Anticipated Milestone(s)^
T Cell Engagers (TCEs)							
Preclinical – Autoimmune & Oncology							
INB-619	γδ TCEs	Autoimmune and Oncology					• IND-enabling studies with initial mouse model data in 2026
Cellular Therapies							
Clinical – Leukemias							
INB-100	DeltEx™ Allo γδ T cells	AML					• Complete dosing of additional patients in the DL2 expansion cohort 2026 • Provide clinical updates and follow-up YE 2026
Clinical - Solid Tumors							
INB-200/400#	DeltEx™ DRI*	GBM (1L)**					• Trial completed, pursuing peer-reviewed publication of data • Obtain FDA guidance on potential registrational pathways in 2026 • Present updated mOS data in mid- and late- 2026

* 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-Q, filed with the SEC on May 7, 2026, for additional details about IN8bio's pipeline efforts

Gamma-Delta (γδ) T cells Overview

Our Core Thesis

γδ T cells could be the most effective cells in the immune system to fight disease



- Uniquely positioned to treat both cancer and autoimmune disease, with a single platform
- Play an outsized role by coordinating a broad immune response
- Help drive deeper immune responses
- Higher levels of gamma-delta T cells in tumors are associated with better survival and stronger responses to cancer treatment

γδ T Cells Combine the Best of all Immune Cells

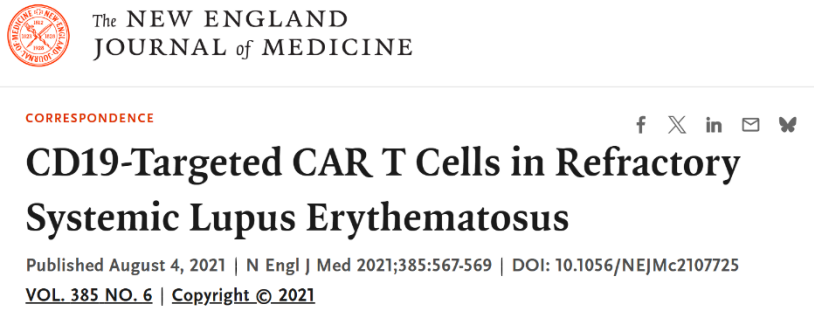
Rare but powerful immune cells that can effectively identify and eradicate target cells

	γδ T cells	CAR-T cells	αβ T cells	CAR NK cells
Activity				
Innate Activity (kills directly)	✓	✗	✗	✓
Adaptive Activity (memory)	✓	✓	✓	✗
Durability & Persistence	✓	✓	✓	✗
No engineering needed	✓	✗	✗	✓
Safety				
Lower risk of side effects (CRS)	✓	✗	✗	✓

The Power of γδ T cells
Next Generation T cell Engagers (TCE's)

Schett's Study Proved CD19 Driven Immune Reset Works

Raises the bar on safety, access, and durability



The NEW ENGLAND JOURNAL of MEDICINE

CORRESPONDENCE

CD19-Targeted CAR T Cells in Refractory Systemic Lupus Erythematosus

Published August 4, 2021 | N Engl J Med 2021;385:567-569 | DOI: 10.1056/NEJMc2107725

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CAR T-cell therapy in autoimmune diseases

Georg Schett, MD [a,b] · Andreas Mackensen, MD [b,c] · Dimitrios Mougiakakos, MD [d,e]

Affiliations & Notes | Article Info | Linked Articles (1)

The NEW ENGLAND JOURNAL of MEDICINE

CORRESPONDENCE

CAR T-Cell Therapy in Autoimmune Disease

Published May 1, 2024 | N Engl J Med 2024;390:1628-1632 | DOI: 10.1056/NEJMc2403705 | VOL. 390 NO. 17

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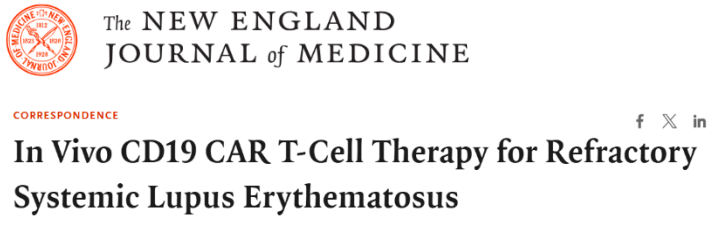

The NEW ENGLAND JOURNAL of MEDICINE

CORRESPONDENCE

In Vivo CD19 CAR T-Cell Therapy for Refractory Systemic Lupus Erythematosus

Published September 17, 2025 | N Engl J Med 2025;393:1542-1544 | DOI: 10.1056/NEJMc2509522

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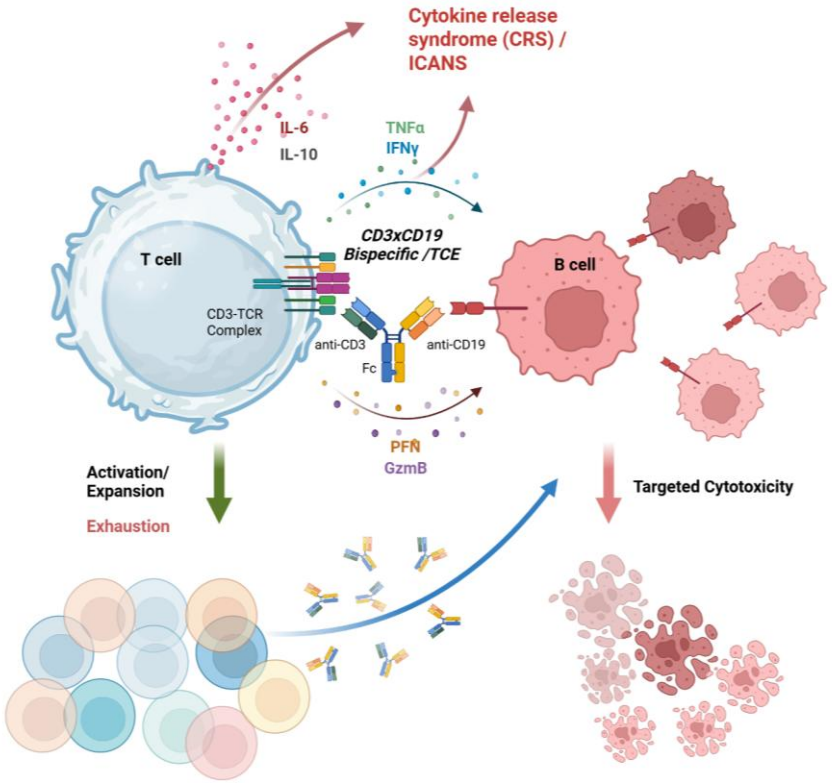
nature medicine

Article | Open access | Published: 07 January 2026

CD19 CAR-T cells for treatment-refractory autoimmune diseases: the phase 1/2 CASTLE basket trial

The CD3 TCE: The Standard Approach Is Broken

CD3-Targeting Creates Dangerous and Unavoidable Side Effects



TCE binds CD3
>90% of TCEs use this target

→

Fundamental Limitations
Broad, non-selective activation

T cell exhaustion
Killing ability lost
Can't dose higher

Toxicity
CRS: 60 – 80% patients
~10% severe (Gr. 3+)

Tissue-resident B cells missed
The cells that drive autoimmune disease

The Result: a narrow therapeutic window — and a tough engineering fix

IN8bio's γδ TCEs Solve All Three Problems

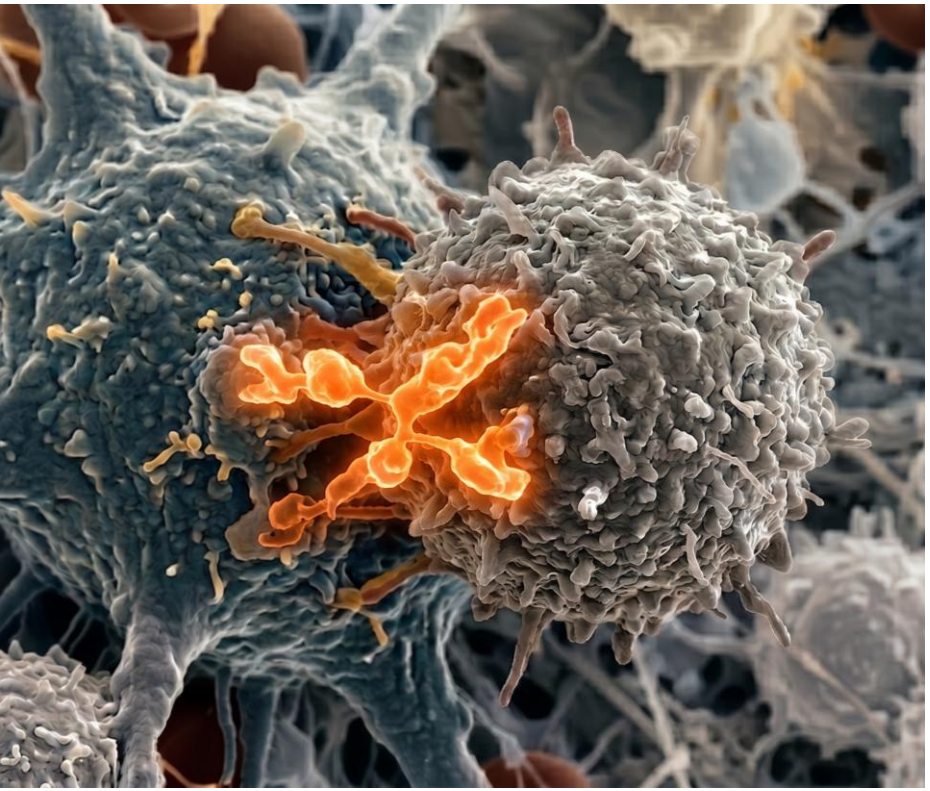
Our approach to TCE development overcomes current TCE limitations to achieving immune reset

CD3 Failure	IN8bio TCE Technology
Immune cell exhaustion	Selective γδ T cell activation - no broad CD3+ T cell activation, MOA resists broad immune exhaustion
Toxicities	Minimal IL-6 and lower TNF-α cytokine release reduces risk of significant CRS and broadens the therapeutic window
Incomplete tissue depletion	Tissue penetration – Dual Vδ1+, Vδ2+ targeting to access tissue, circulating and lymphoid B cell compartments

INB-619: A Pan γδ CD19 TCE

The Power of Cell Therapy Without the Cell Therapy

A TCE that functions like an in vivo CAR-T: Expands **γδ** T cells, eliminates targets, and avoids limiting toxicities



Source: Image generated by Gemini, 2026

- Activates the pan γδ T cell repertoire: including both Vδ1+ and Vδ2+
- Eliminates target cells without CD3-driven toxicity
- Opens TCE-medicated B cell depletion to diseases where current options are too dangerous/ limiting
- No lymphodepletion needed for TCE
- Safer therapeutic approach to treat a broader range of indications

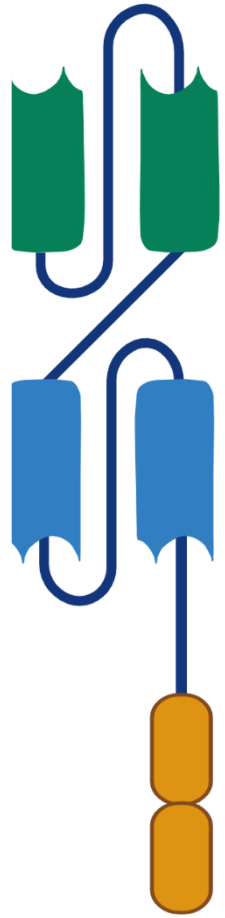
INB-619: The First Pan-γδ T cell Engager to Drive Expansion

Targeting the γδ T cell receptor (TCR) instead of CD3

Cassette-like CD19 domain targets B cells

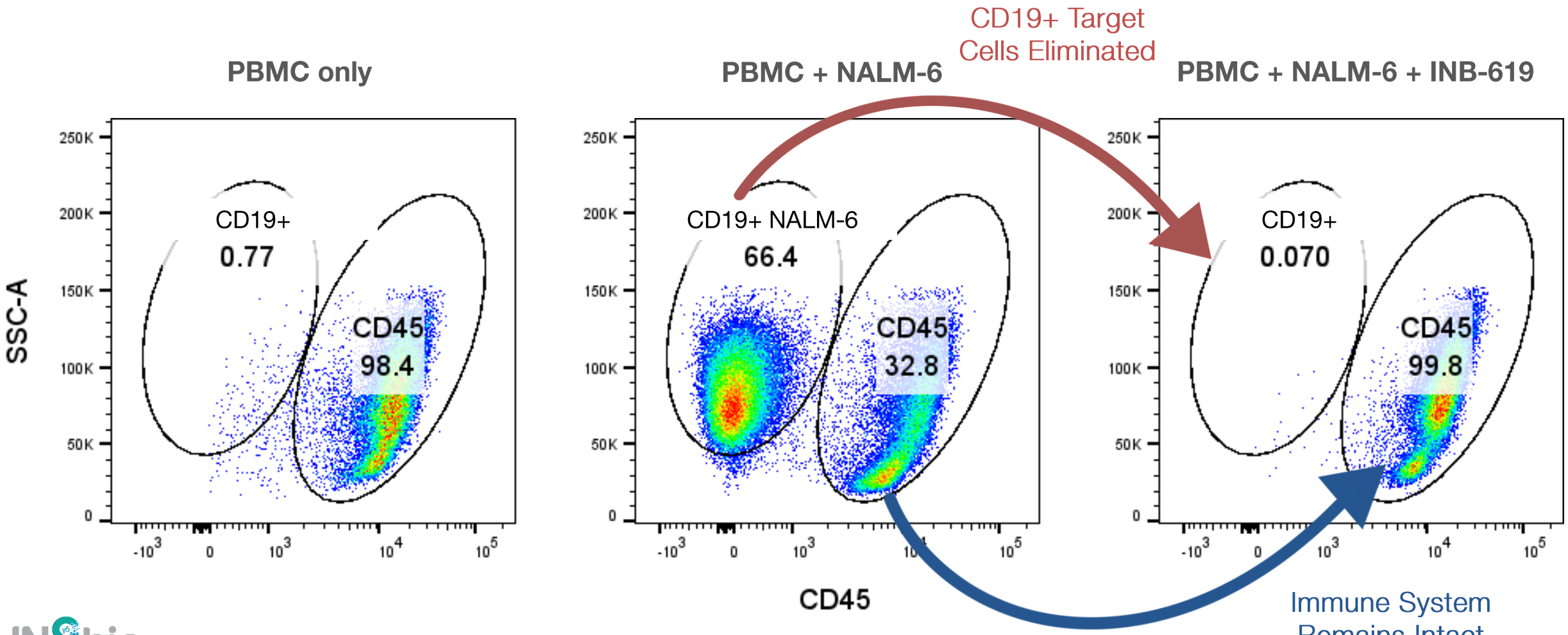
Pan γδ TCR binding activates without CD3

GDED (gamma delta expansion domain) drives in vivo expansion



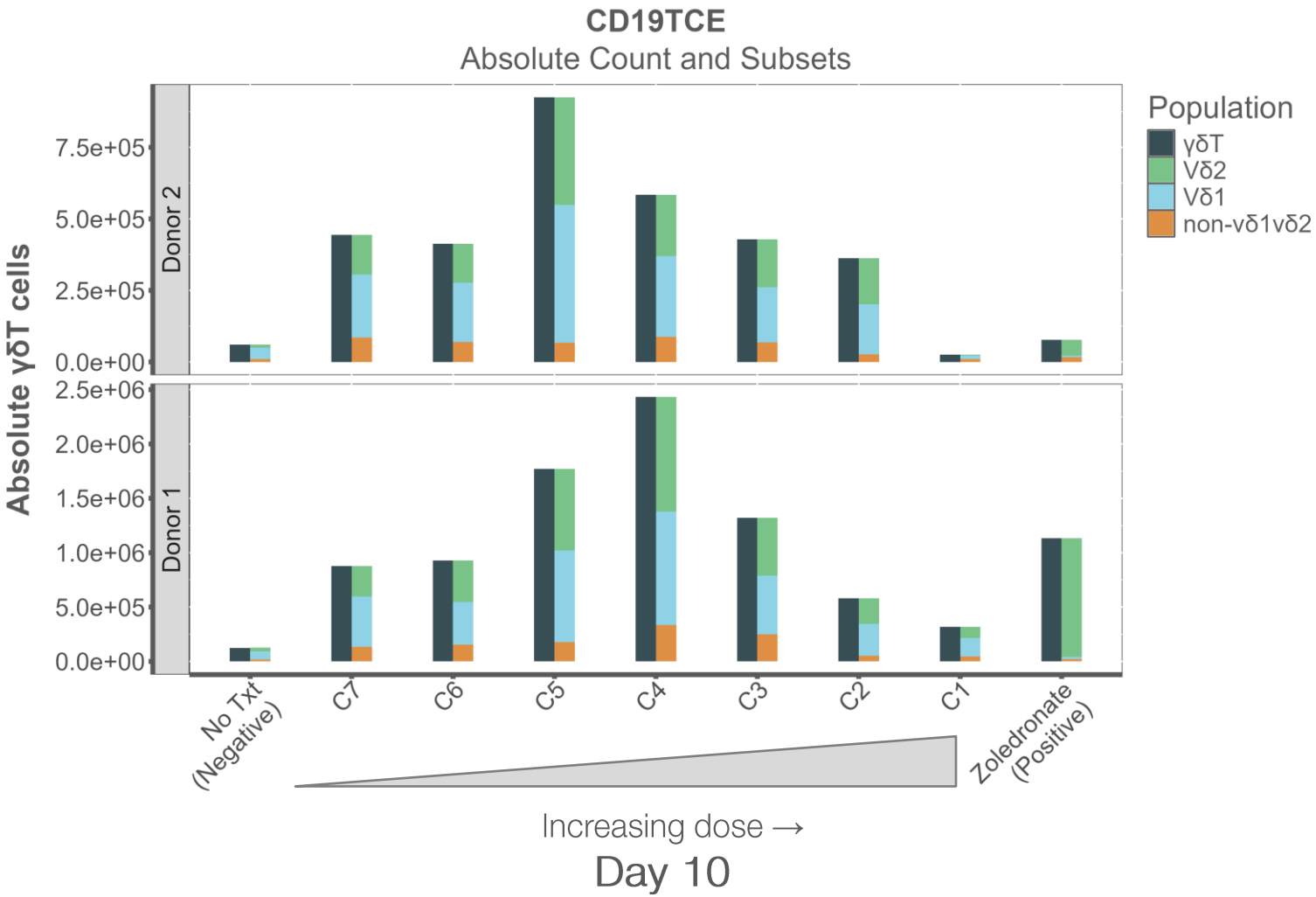
INB-619 Efficiently and Specifically Eliminates Target Cells

Complete CD19+ cell clearance: 66% → 0.07% in PMBC culture



INB-619 is the First TCE driving Pan γδ T Cell Expansion

Both Vδ1+ and Vδ2+ subtypes expand — with the potential to target tissue-resident & circulating B cells

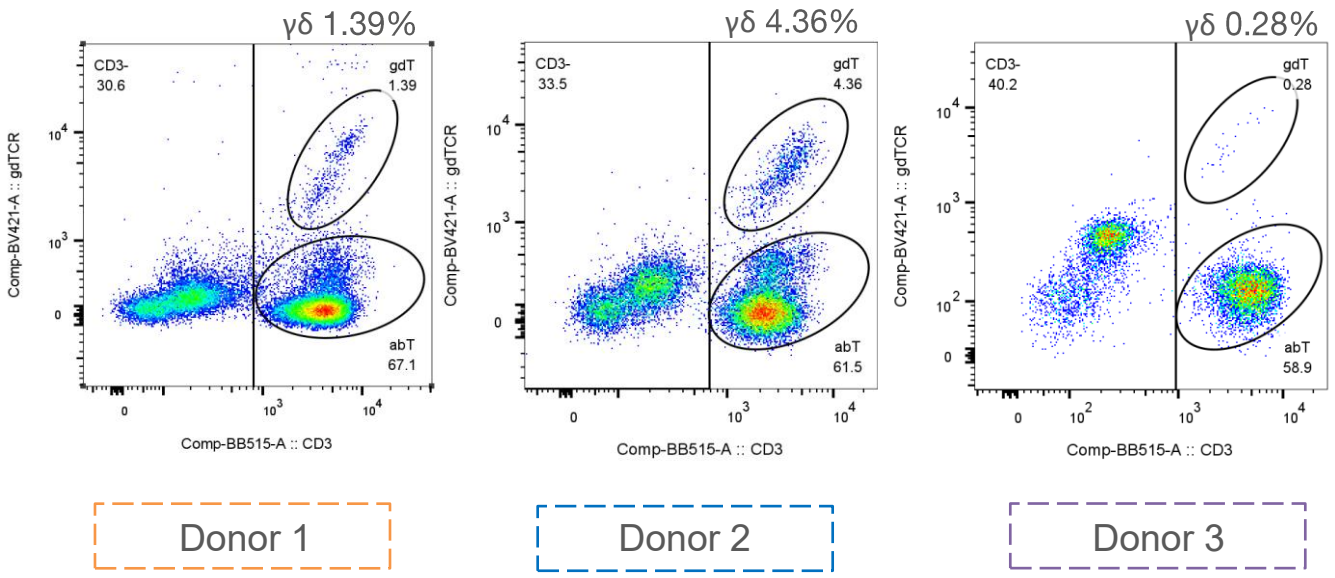


- Expansion is dose-dependent across both γδ subtypes
- Vδ2+ provide surveillance and Vδ1+ tissue residence, enabling deeper B cell depletion
- No expansion without INB-619 (No Txt control)

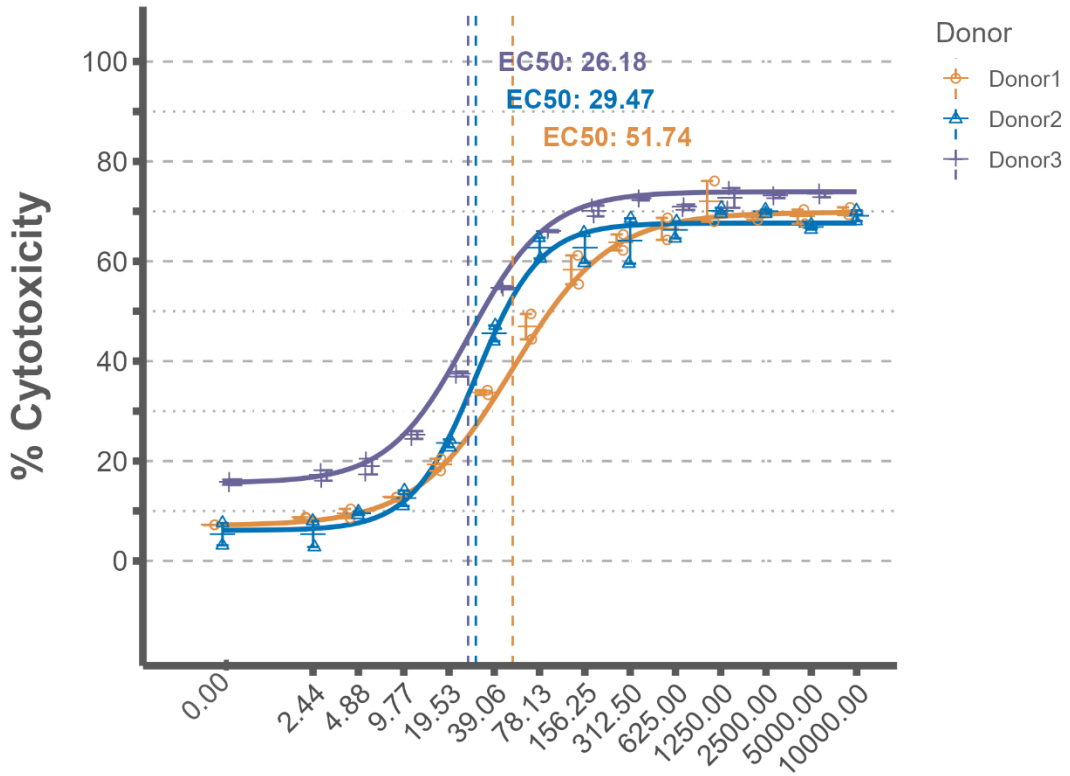
INB-619 Works Even When γδ T cells are Scarce

Potency is consistent regardless of starting γδ T cell levels — critical for rare cell types like γδ T cells

- Donors ranged from 0.28% to 4.36% γδ T cells at baseline
- All three showed overlapping potency (EC50: 26–52 pM)

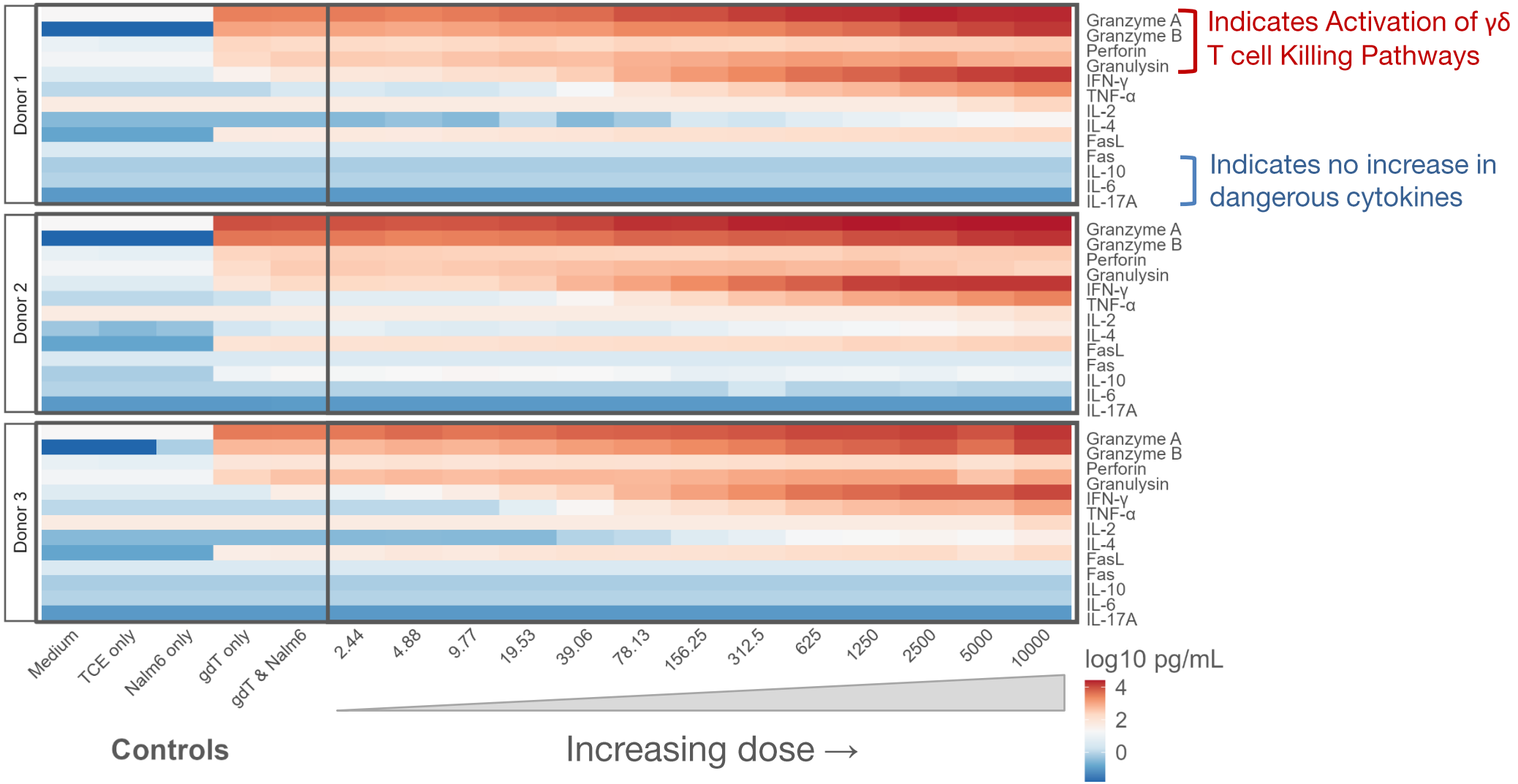


Donor 1 | Donor 2 | Donor 3



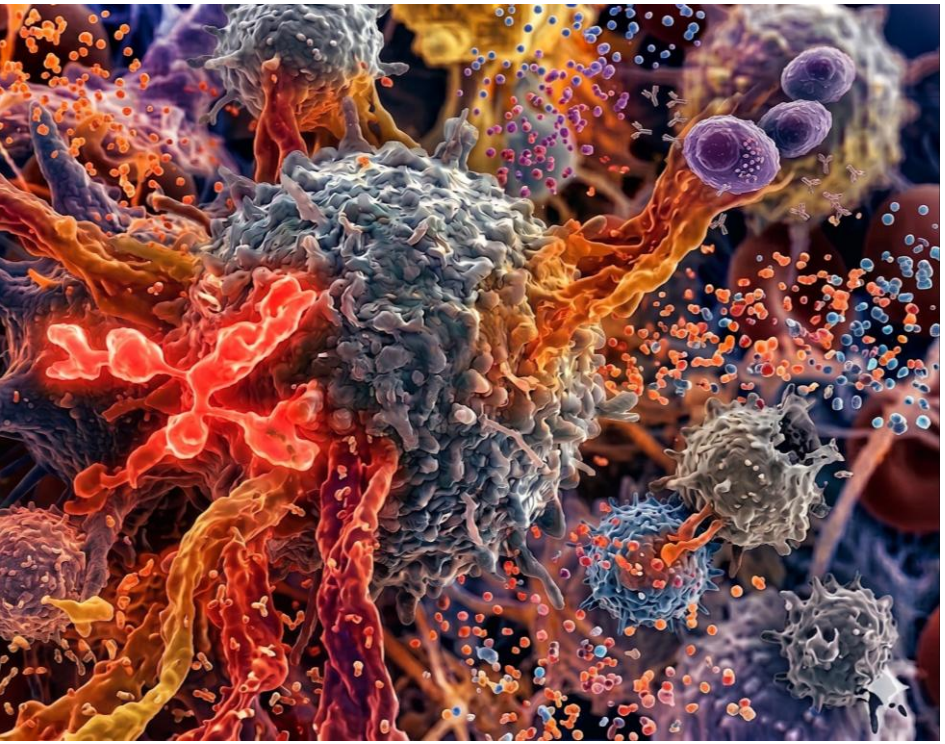
Inflammatory Cytokines are not Induced by INB-619

Killing markers (Granzyme, Perforin) rise with dose — CRS cytokines (IL-6, IL-10, IL-17) stay flat



INB-619 for Autoimmune Disease

Autoimmune Patients Can’t Tolerate Broad CD3 Toxicity



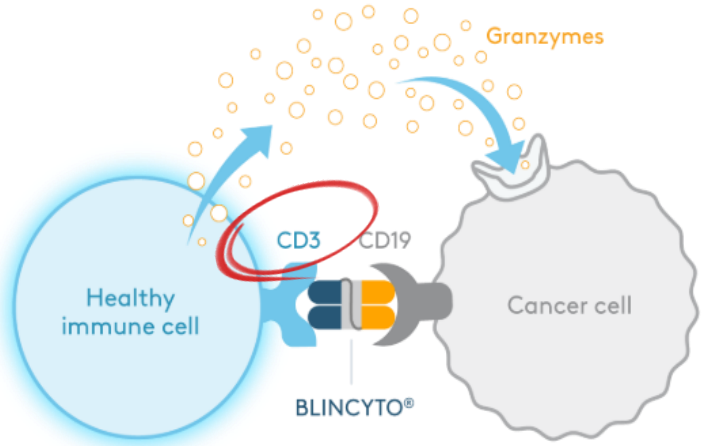
- Cancer patients can tolerate severe side effects because the alternative is often death, autoimmune patients cannot
- Existing CD3 TCEs trigger system-wide immune activation and often lead to excess cytokine release, exactly what autoimmune patients already suffer from
- γδ TCE offers precise immune system activation and target cell elimination without igniting the whole immune system

Source: Image generated by Gemini, 2026

INB-619 Depletes B Cells Without the Toxicity of CD3 TCEs

Blinatumomab and Mosunetuzumab both use CD3, triggering the toxicities INB-619 avoids

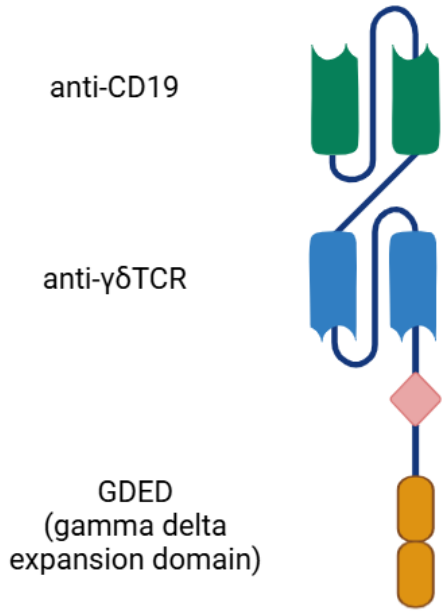
Blinatumomab (BLI)
CD19 TCE



2025 sales: $1.6B

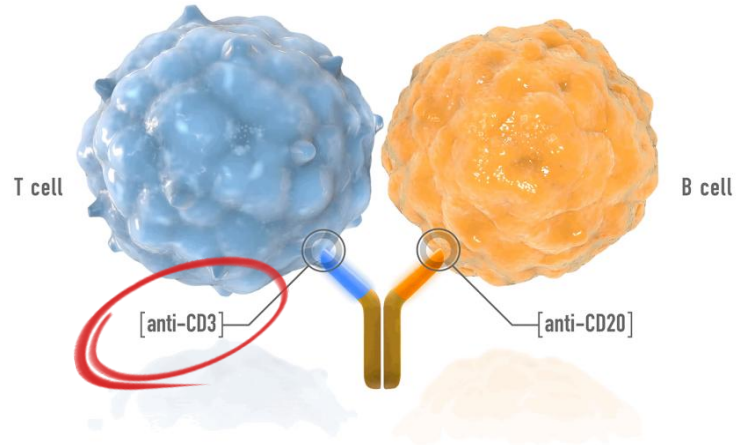
~54 kDa

INB-619
CD19 TCE



~75 kDa

Mosunetuzumab (MOS)
CD20 TCE



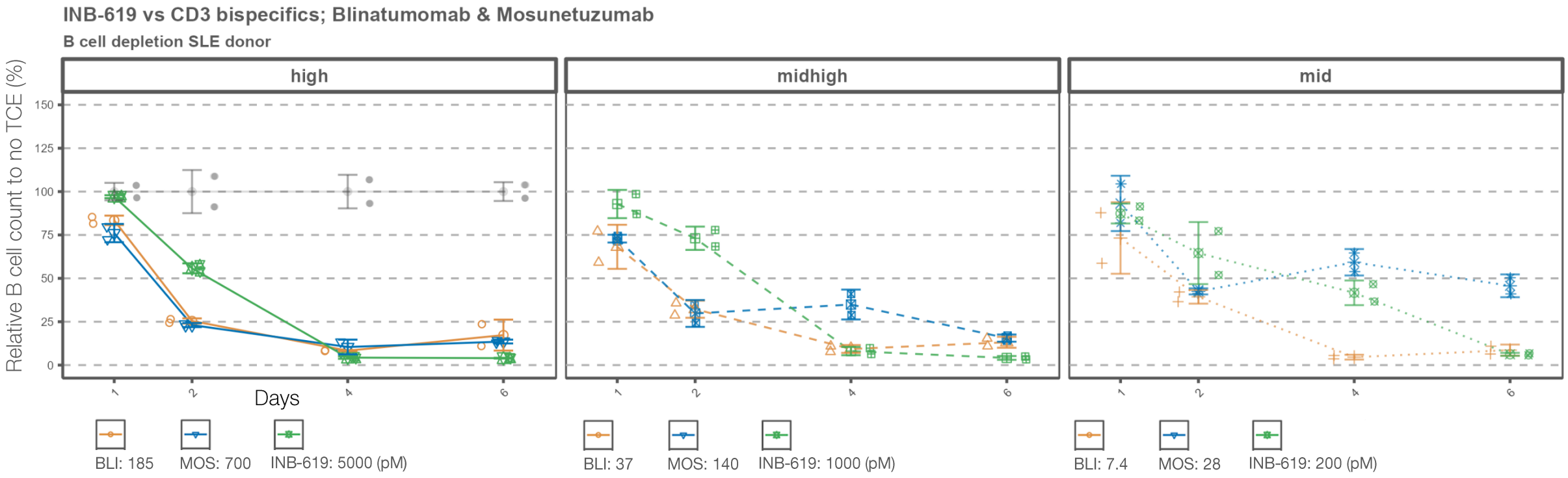
Est. 2025 sales: $120M+

~146 kDa

*https://pharmaphorum.com/news/approvals-extend-use-roche-abbvie-drugs-lymphoma?

INB-619 Depletes SLE B cells Across a Range of Concentrations

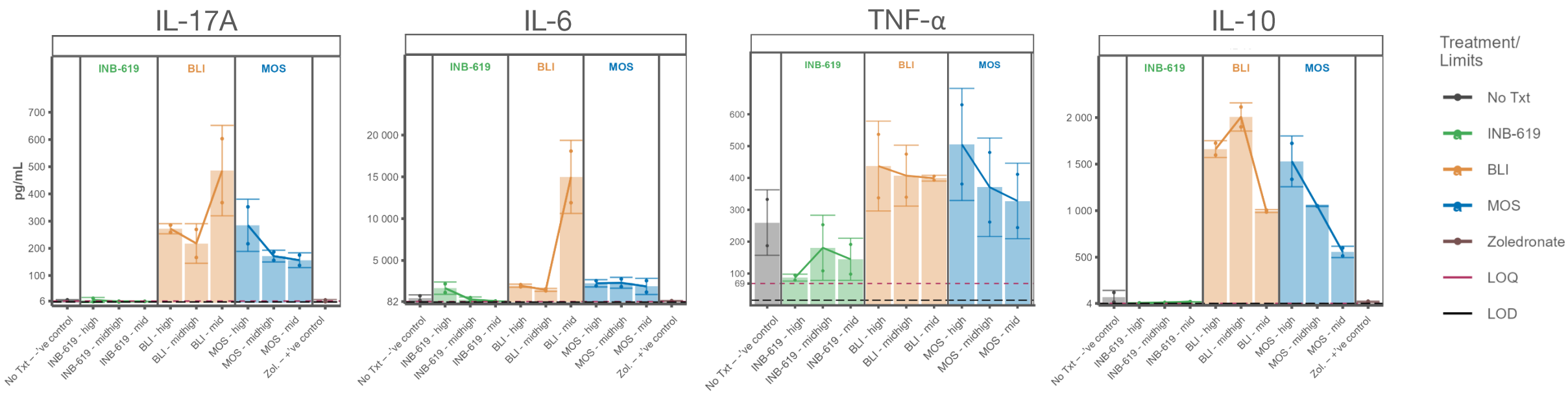
Most CD3 TCEs dose-reduced in autoimmune disease due to potential toxicity



INB-619 Target B cell eradication achieved across multiple doses in SLE donor

INB-619 Demonstrates Lower Secretion of CRS Cytokines

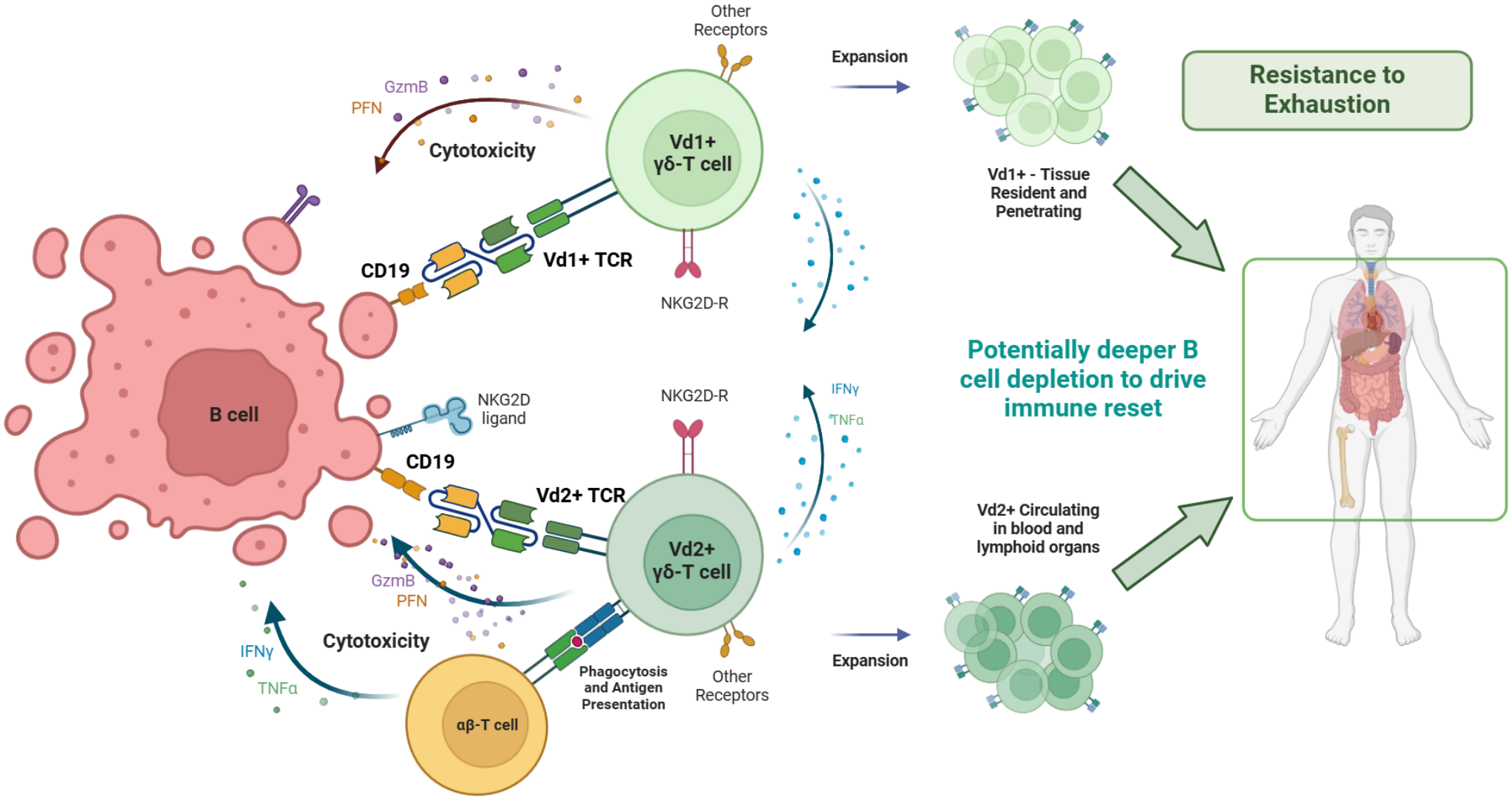
SLE donor cytokine secretion at Day 4



INB-619 has 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

Vδ1+ and Vδ2+ Attack from Two Directions — Tissue & Blood

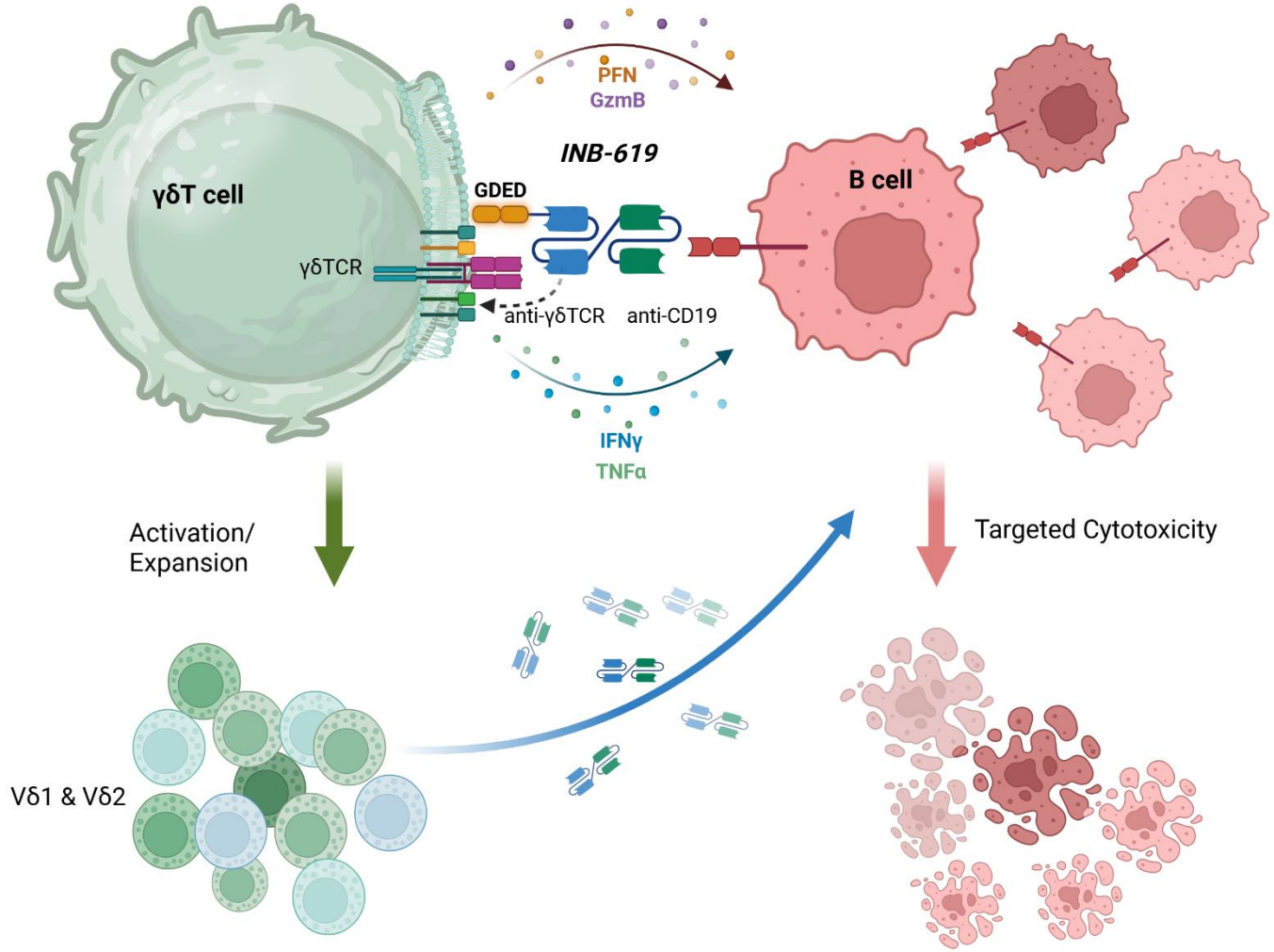
Pan γδ TCR targeting is more powerful to drive B cell elimination



INB-619 - CD19-γδ TCE Provides Unique Advantages

A pan-γδ-TCE with built-in co-stimulation that secretes few CRS inducing cytokines

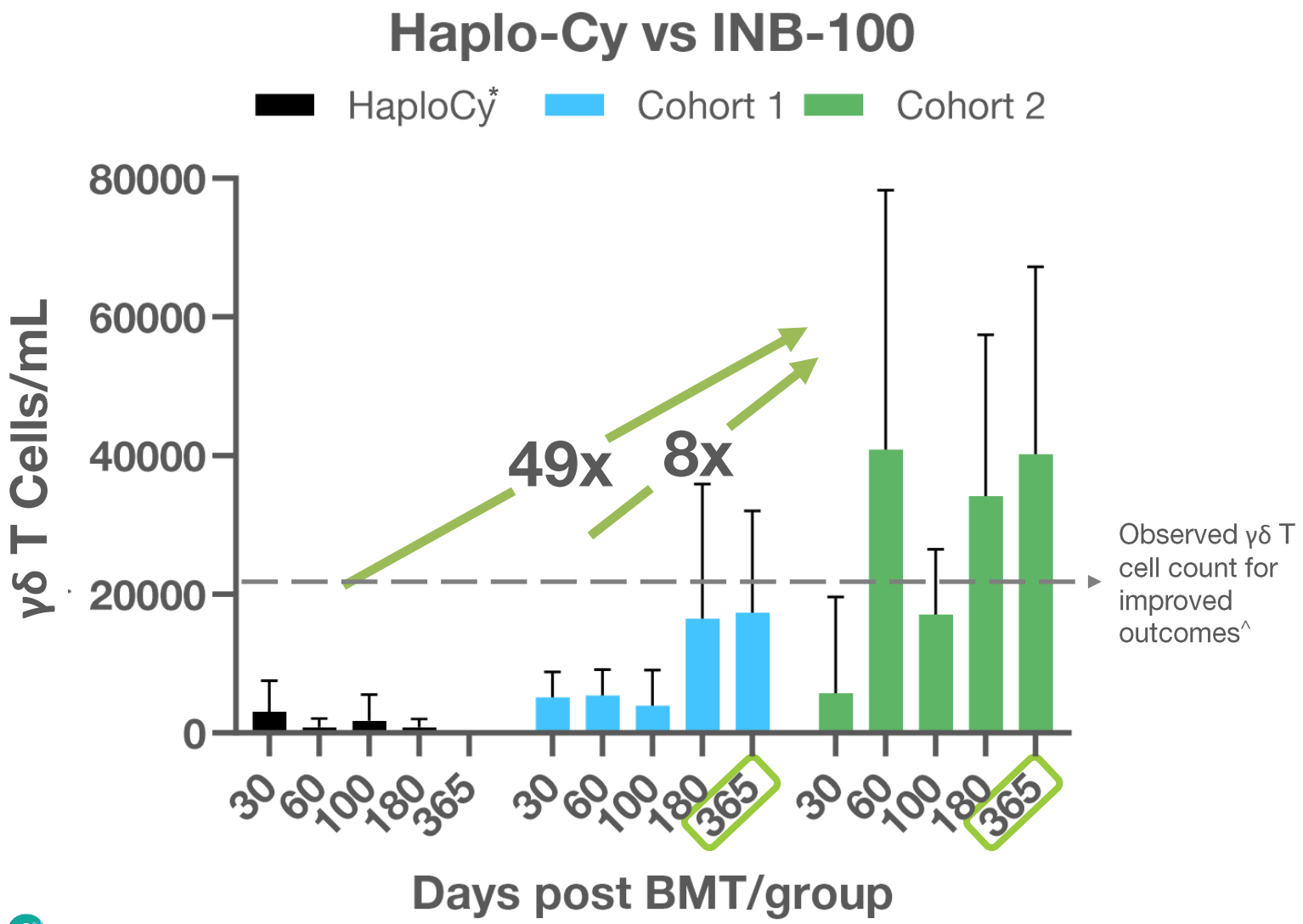
- Expands γδ T cells to eliminate target cells in a dose-dependent manner
- Vδ1+ cells target tissue resident B cells
- Vδ2+ cells are phagocytes that help drive deeper B cell depletion
- γδ T cells don't secrete IL-6 to reduce CRS toxicities
- Mimicking CAR-T with simpler manufacturing, lower costs, avoids lymphodepletion and repeat dosing with TCE's



INB-100

DeltEx™ Allo for Leukemias

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



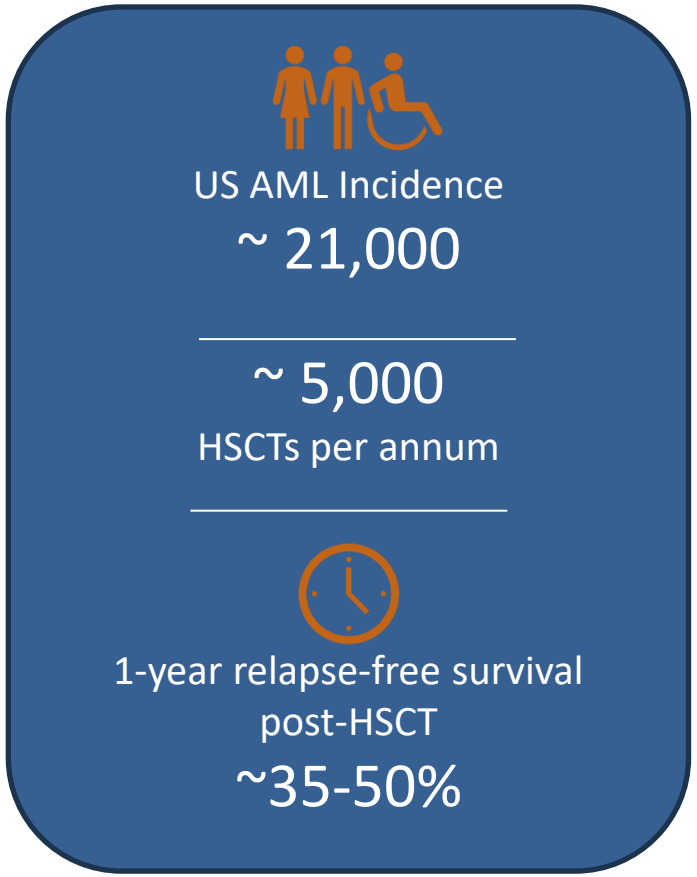
- Higher γδ T cell levels at 365 days suggests ***in vivo*** **expansion** and **persistence** enabling continued surveillance against residual cancers
- IN8bio's INB-100 results in a **dose-dependent** rise in circulating γδ T cells above the threshold associated with improved progression-free survival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



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

The Ohio State University Comprehensive Cancer Center

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**

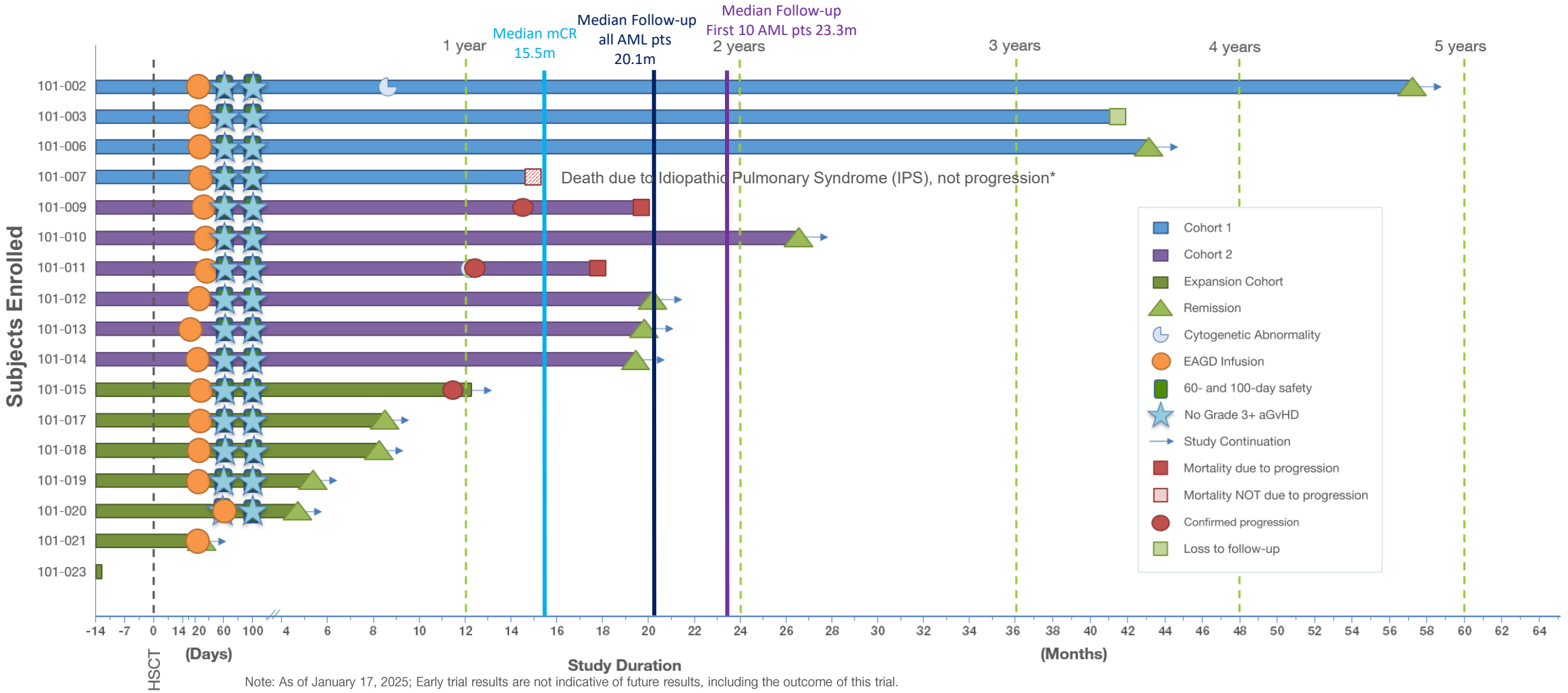
All patients with intermediate or high-risk disease

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)

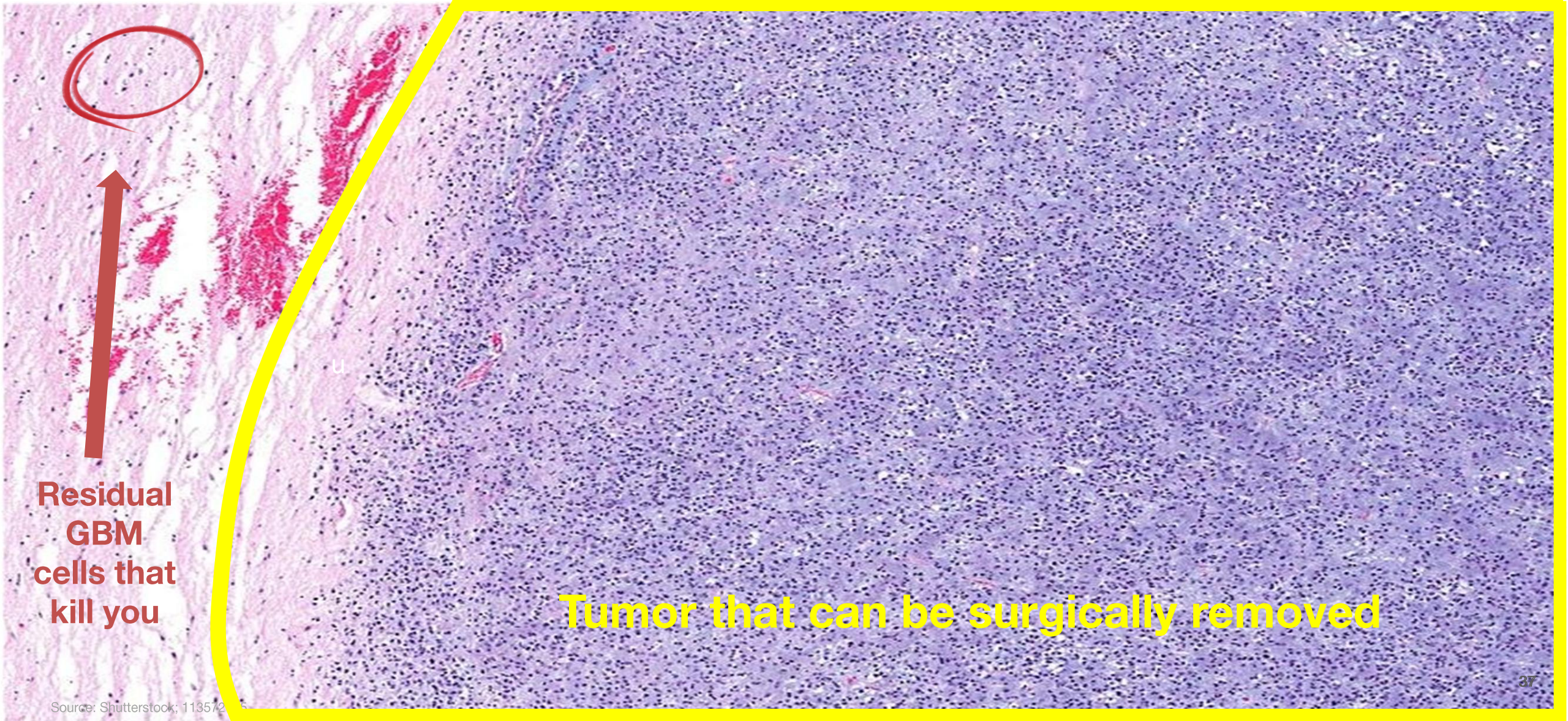
^ CIBMTR CM24-35, "Retrospective analysis of RFS and OS in AML patients undergoing haploidentical transplant with 62% (N=424) receiving reduced intensity conditioning (RIC) regimen."
* Patient and transplant-related characteristics after FluCyTBI non-myeloablative RIC haploidentical HCT with PT-Cy-based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

INB-200 & 400

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

The Problem



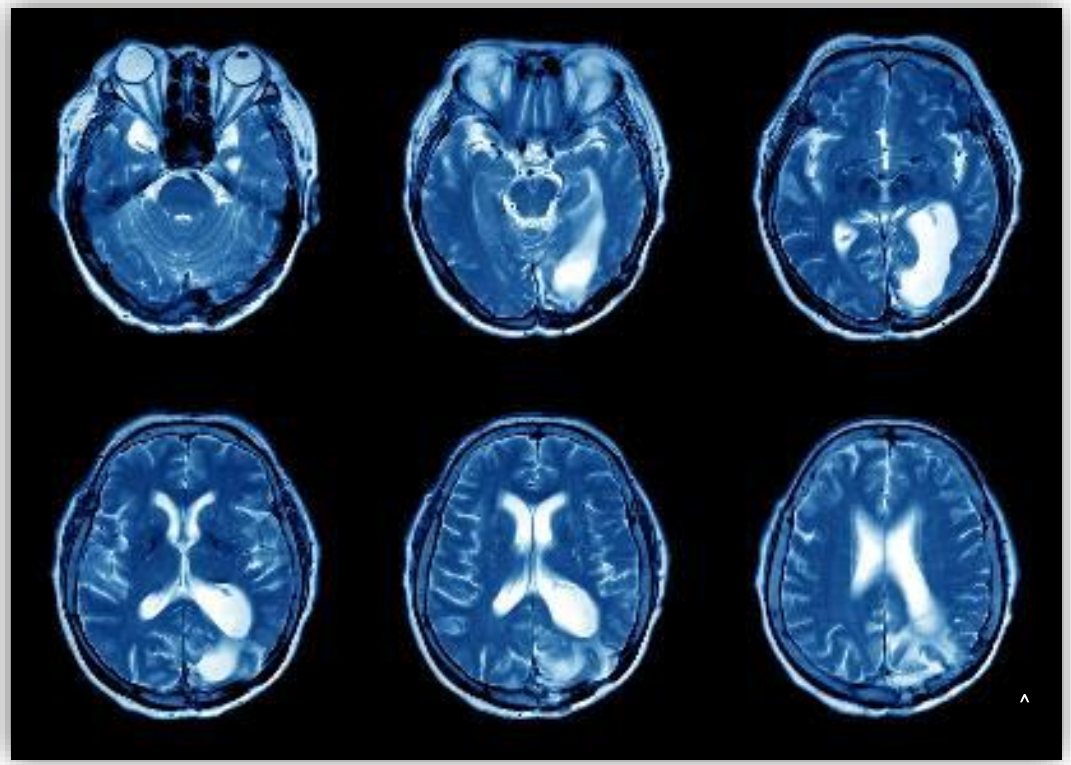
Source: Shutterstock; 113572

Little Has Changed in Over 20 Years

GBM is the most aggressive form of brain cancer, where the SOC (Temozolomide) was established in 2005

Patients diagnosed annually (in each of US & EU)	Est. WAC of CAR-T therapy in 2026
~14,500	~$516,000

Median survival on standard-of-care	Median progression-free survival
~14 to 16 months	~7 months



A novel **γδ** T cell treatment used in front-line disease to address the challenges in GBM

Source: EMA, ABTA, NCI, Stupp et al., NEJM 2005; 352: 987-96, Drug Development & Delivery - https://bit.ly/35KLLJ5, ^Brain scan of general GBM patient ^ estimate by Gemini (from sales of FDA-approved CAR-T in 2025

GBM Biology Makes an Ideal Setting for γδ T cell Therapy

Four structural advantages no other solid tumor offered as first proof-of-concept

Direct delivery

Catheter to tumor site eliminates cell trafficking and the **delivery** problem of the blood brain barrier

Targets Heterogeneity

Forced expression of NKG2D-ligands allows γδ T cells to addresses heterogeneity

Simpler with no additional lymphodepletion

Standard-of-care (Temodar) already does it

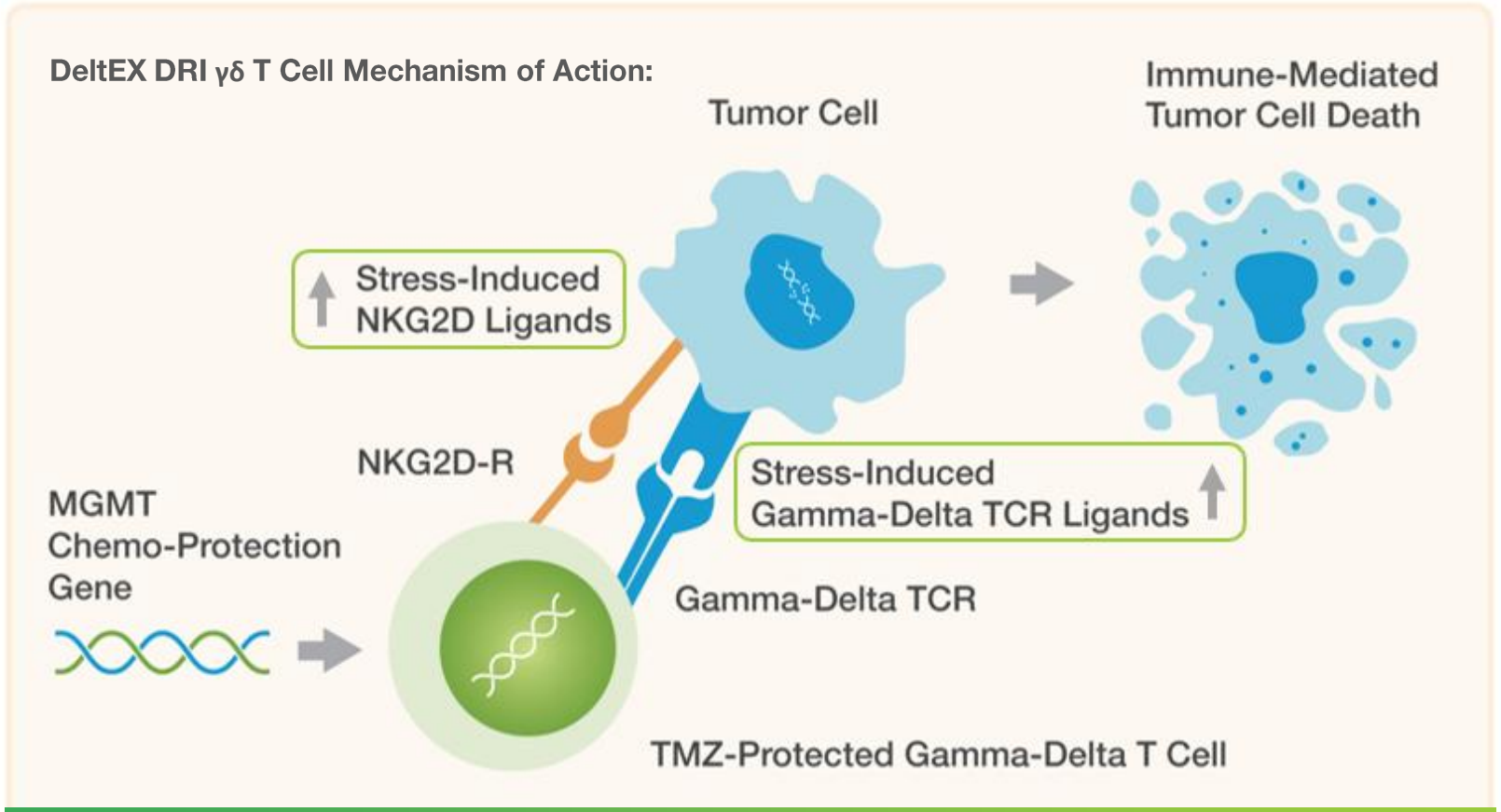
Immune-privileged

Allogeneic γδ cells persist without host-vs-graft immune attack

γδ T cells are Powerful Killing Cells for Immunotherapy

γδ T Cells Find and Kill Tumors That Hide from Everything Else

Chemotherapy stresses tumor cells; γδ T cells recognize that stress signal and attack

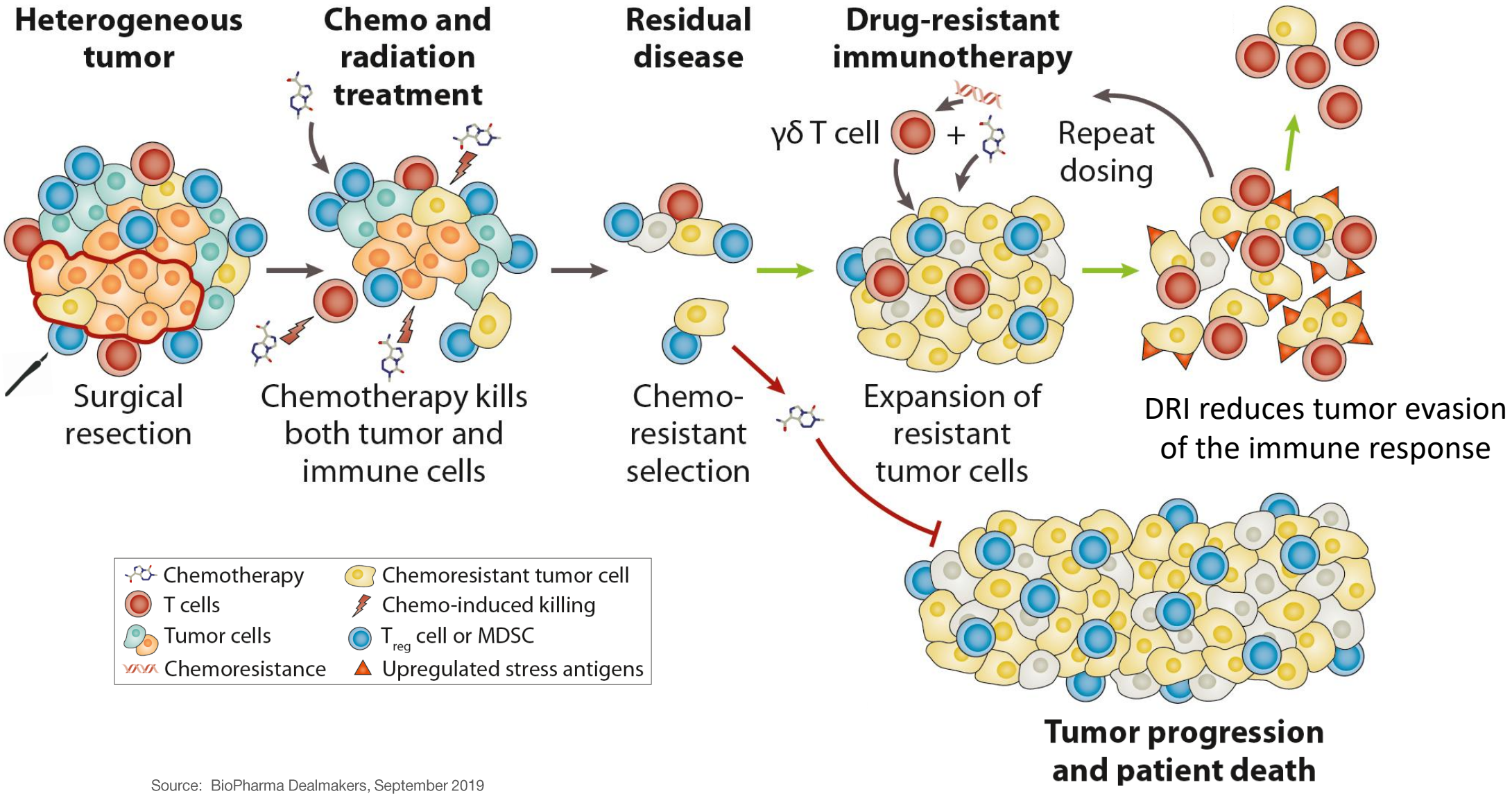


Chemo damages tumor DNA → Tumor displays stress signals → γδ T cell recognizes and kills it

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

Effective therapy



Source: BioPharma Dealmakers, September 2019

INB-200/400 Data

Phase 1/2 Trial: Do Repeat Doses Drive Deeper Response?

Treatment Arms

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

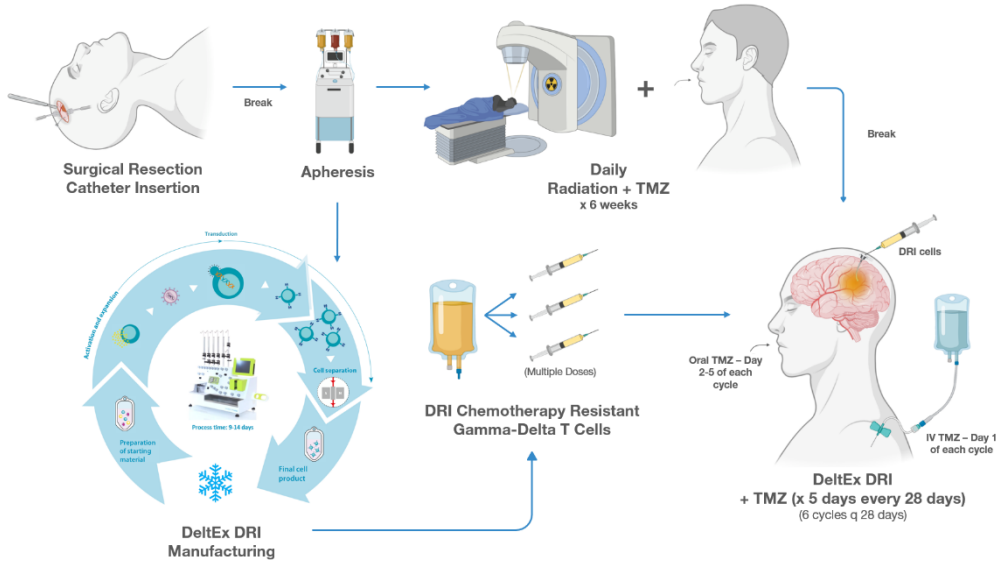
DL1: N = 3 (up to 6) patients, single dose of 1 x 10^7 cells on C1D1
DL2: N = 3 (up to 6) patients, three doses of 1 x 10^7 cells, one dose every 28 D1 of C1-C3
DL3: N = 3 (up to 6) patients, six doses of 1 x 10^7 cells, one dose every 28 days on D1 of C1-C6

Treatment Regimen & Timing

Surgical resection followed by apheresis

6 weeks induction TMZ + radiation

6 cycles maintenance TMZ + DRI



Primary Endpoints

- Safety + Maximum Tolerated Dose

Secondary Endpoints

- Time to progression
- Overall survival
- Biologic response

Site

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

UAB HEERSINK SCHOOL OF MEDICINE

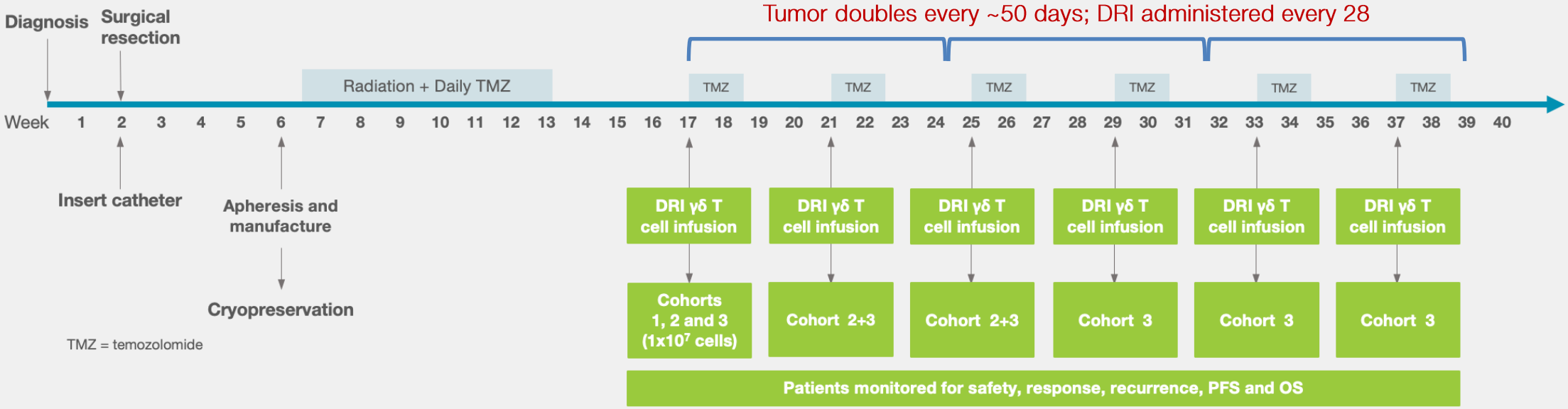
Source: IN8bio, image created with biorender.com

Repeated Doses to Intercept Residual Cells Every 28 Days

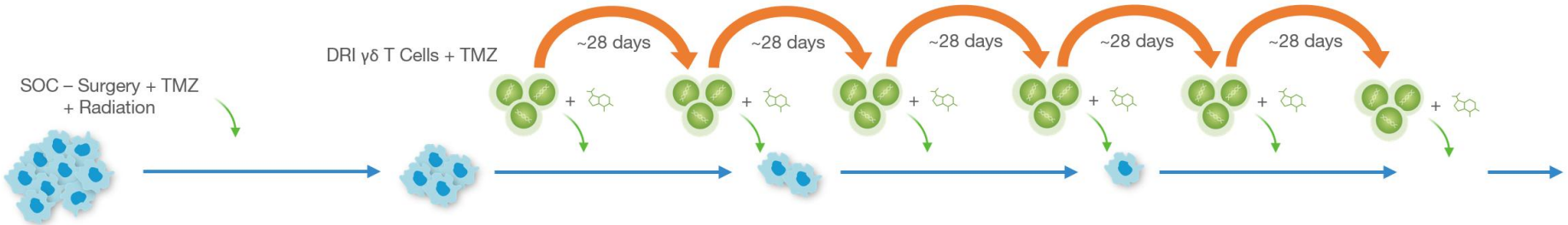
Timed to outpace tumor regrowth, each infusion catches residual cells as they multiply to maintain remission

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

Four Prestigious Cancer Centers…One Consistent Finding

Consistent treatment activity and no major toxicity signals across all sites and treatment arms

- INB-200 (Phase 1, UAB) and INB-400 (Phase 2, three sites) treated 17 patients combined
- No major toxicity or significant adverse events across any site or treatment arm
- Enrollment suspended in 2024 — no safety or efficacy concerns; data collection on enrolled patients continues

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 May 15, 2026; 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

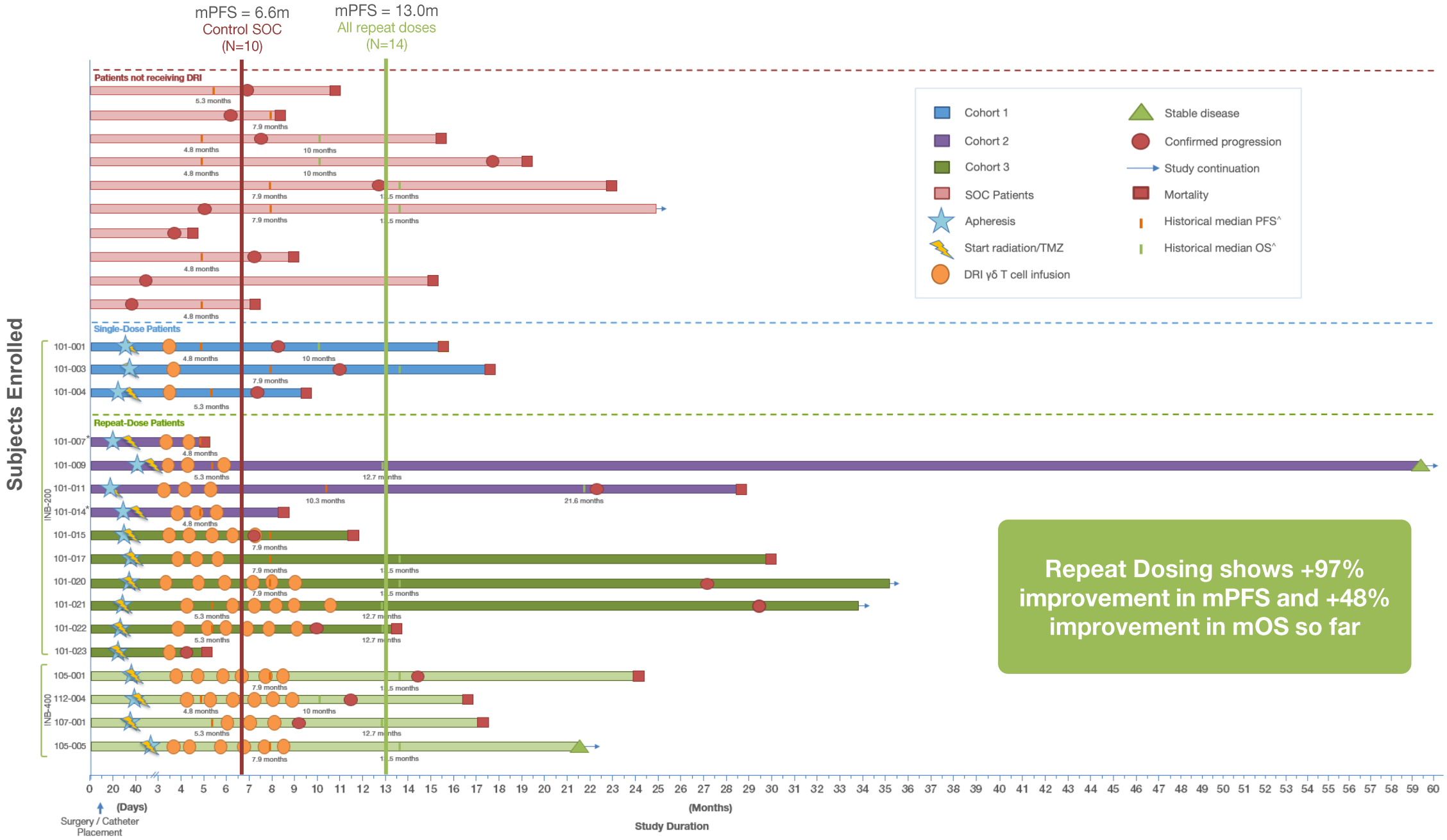
- ^Pt 004 received a single dose of bevacizumab during induction for edema, Control patients unknown outside of SOC

*As of May 15, 2026; Early trial results are not indicative of future results, including the outcome of this trial.

Patient Demographics Comparable Across Cohorts

SOC control group had 80% total resections vs. 43% for DRI patients, a disadvantage for the treatment arm

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%	69	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



Repeat Dosing shows +97% improvement in mPFS and +48% improvement in mOS so far

Note: As of May 15, 2026; *Pts 007 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	NR (19.5+)	(3/4) 75%
All Repeat Dose Patients	13.0	NR (19.5+)	(8/14) 57%

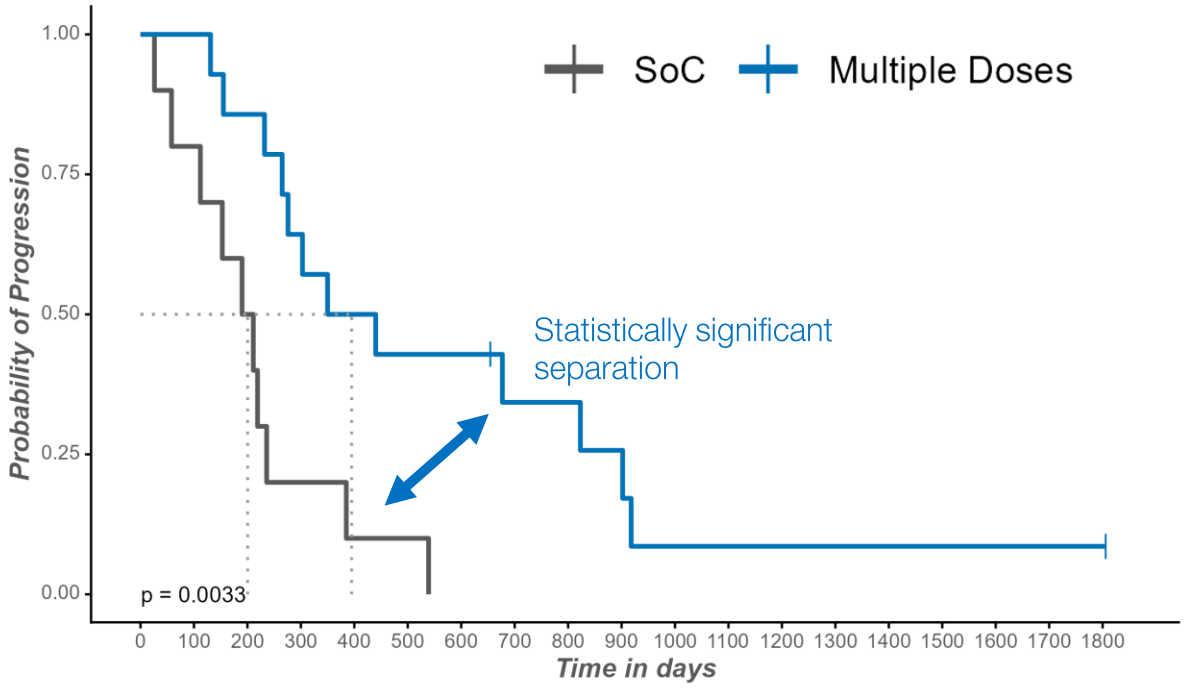
+ = median OS not yet reached

Note: As of May 15, 2026; *Pts 007 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

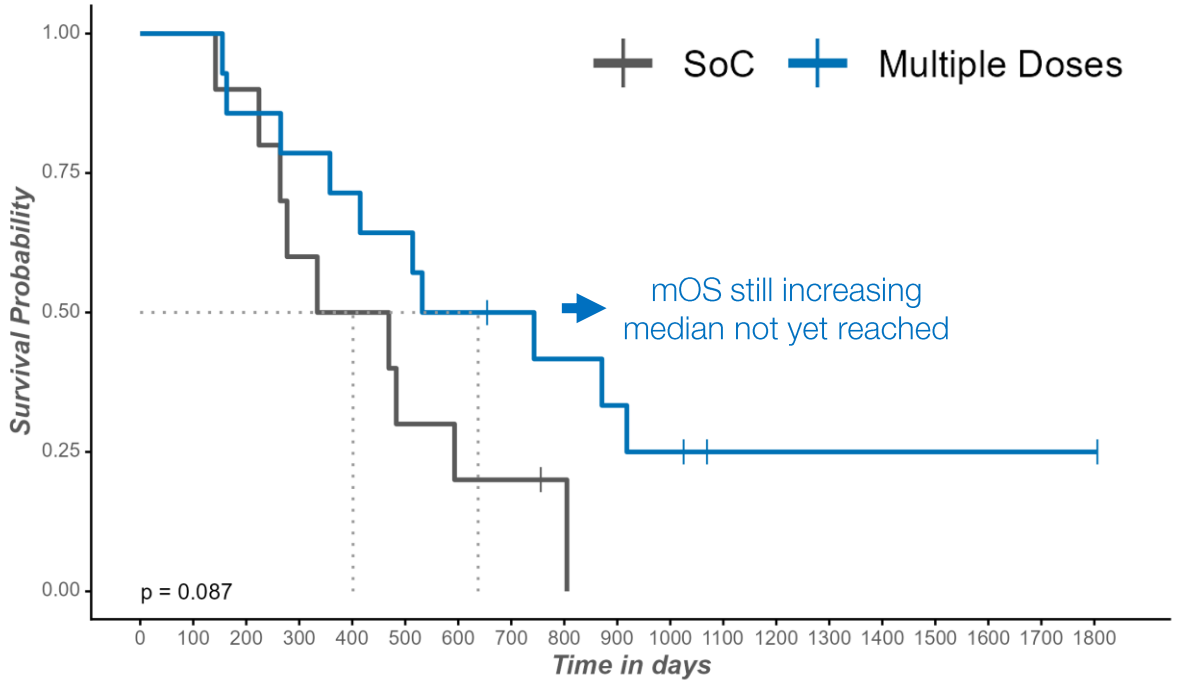
DRI Patients Live Significantly Longer Without Progression

PFS separation is statistically significant; OS median not yet reached in DRI arm

DeltEx DRI intervention; GBM Progression-free analysis
Kaplan-Meier Estimates



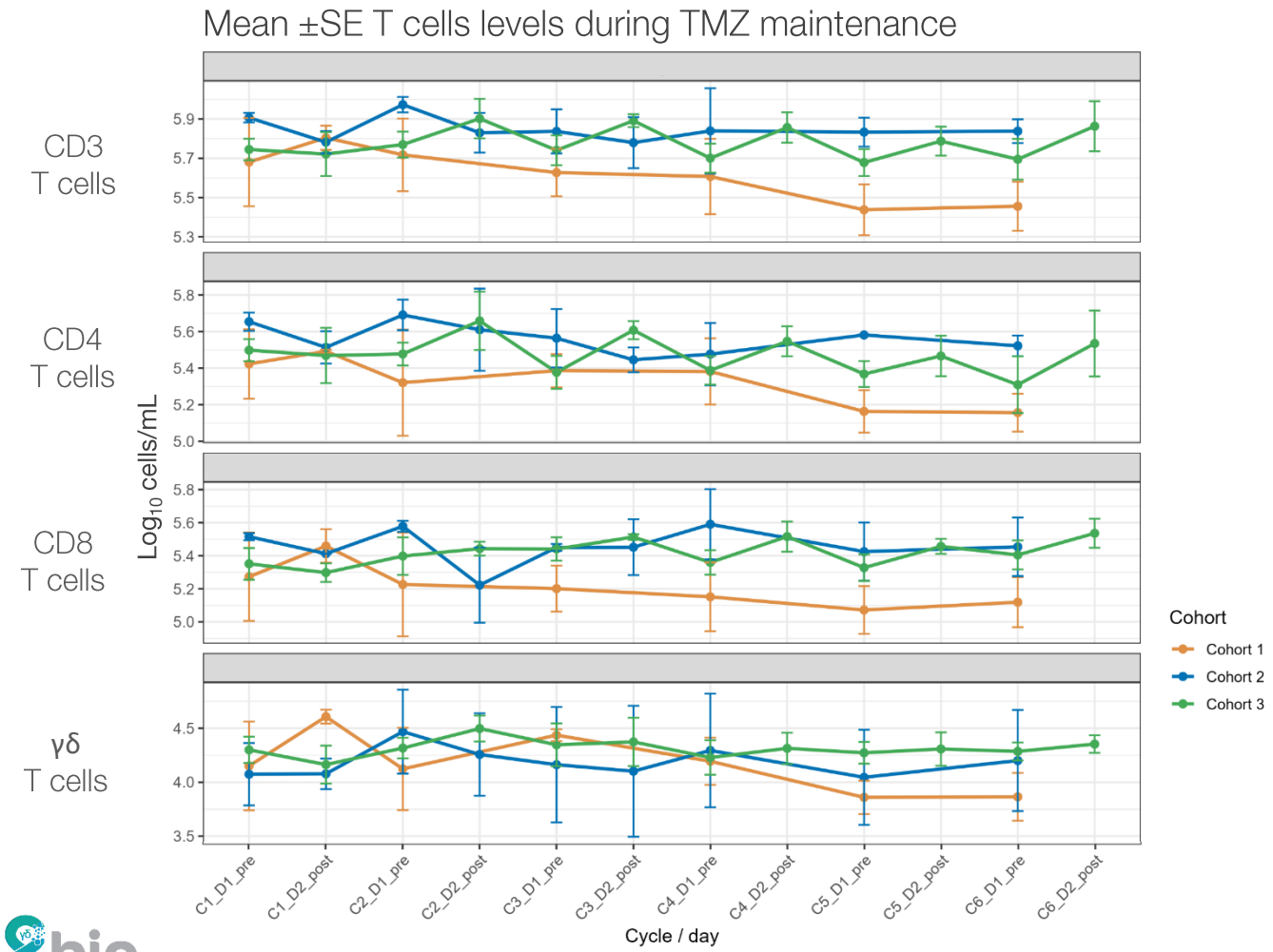
DeltEx DRI Intervention: GBM Survival Analysis
Kaplan–Meier Estimates



Note: As of May 15, 2026; 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.

Repeat Dosing Preserves the Immune System

Preserved immune cell levels mean less TMZ induced lymphopenia and potentially fewer treatment delays



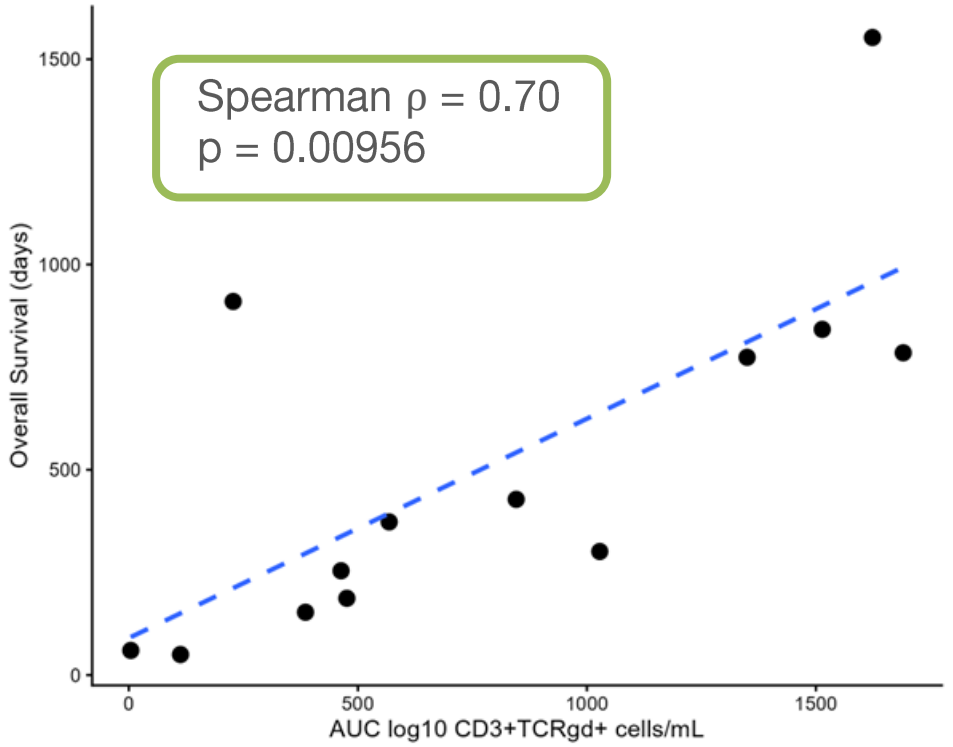
- Cohort 1 (single dose) shows steady decline — the dose frequency difference is decisive
- Cohorts 2 and 3 (repeat dose) maintain T cell levels across all immune subsets during TMZ
- Higher peripheral immune reconstitution is known to be correlated with improved survival outcomes

CRT → immune disruption → γδ dosing → CD4 preservation → cytotoxic state → improved outcomes

Patient Survival and Correlation to Peripheral γδ T cell Levels

Strong, statistically significant correlation: Higher sustained γδ levels predict better outcomes

Cumulative $\log_{10}$ γδ T cells/mL vs survival



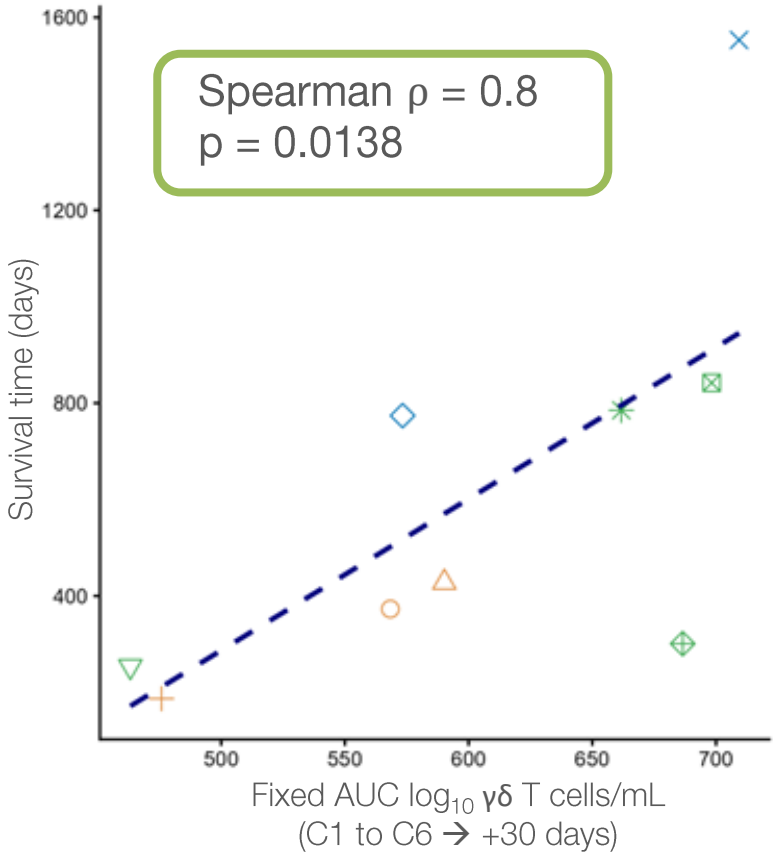
(Sustained γδ T cell levels after treatment →)

Post-infusion peripheral γδ T cell levels were summarized as area under the curve (AUC) to capture the systemic response over time and showed a correlation to overall survival by Spearman's correlation (ρ and p-value).

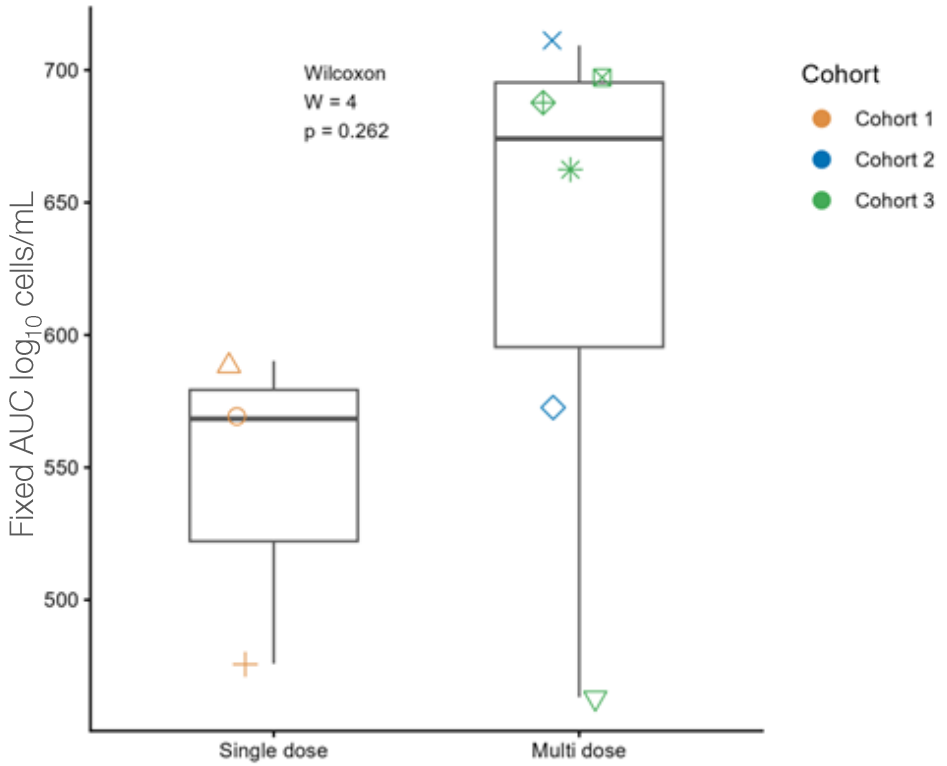
Repeat Dose Cohorts Trend to Higher γδ T cell Levels

Correlation between sustained γδ levels (AUC) and improved outcomes is strong

Fixed AUC log_{10} γδ T cells/mL exposure vs OS (post-infusion)



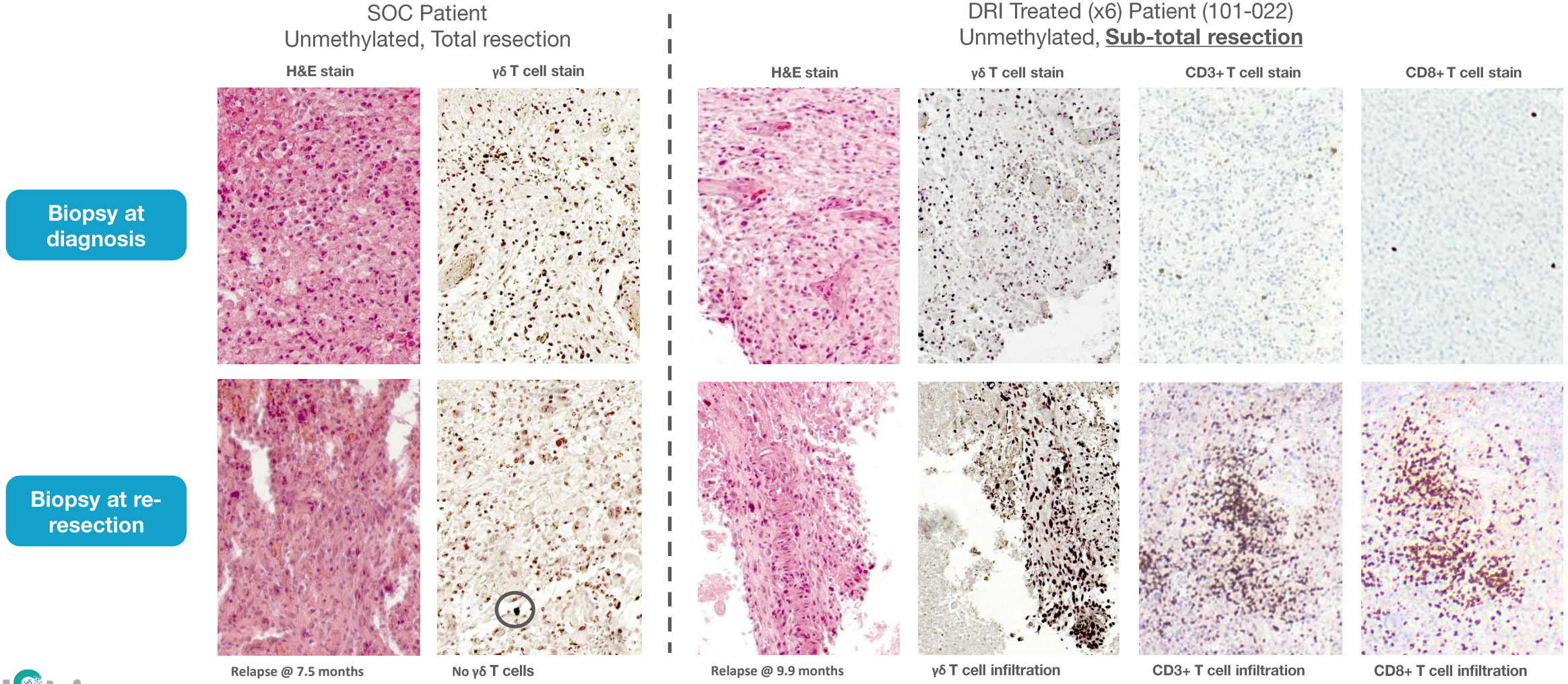
Single vs multi-dose γδ T cells (post-infusion)



Single cell correlative analysis of INB-200/400 tumor presentation & progression

Histopathology of the TME Shows γδ T cell Infiltration

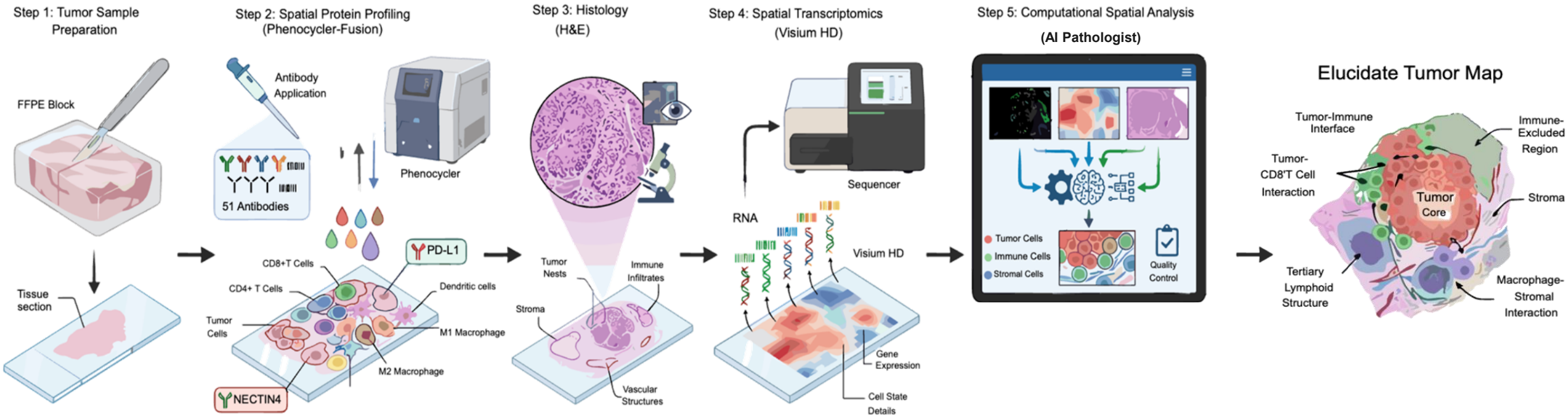
Broad immune cell infiltration demonstrate biological mechanism for activity and responses



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

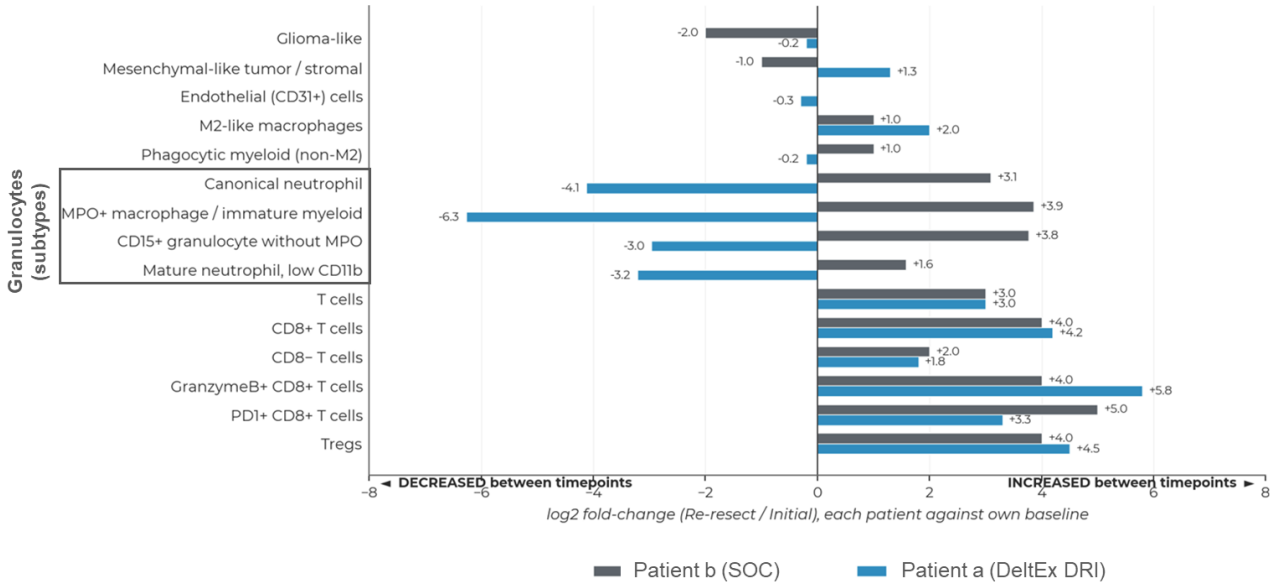
AI-Powered Tumor Mapping Gives IN8bio Deeper Insights

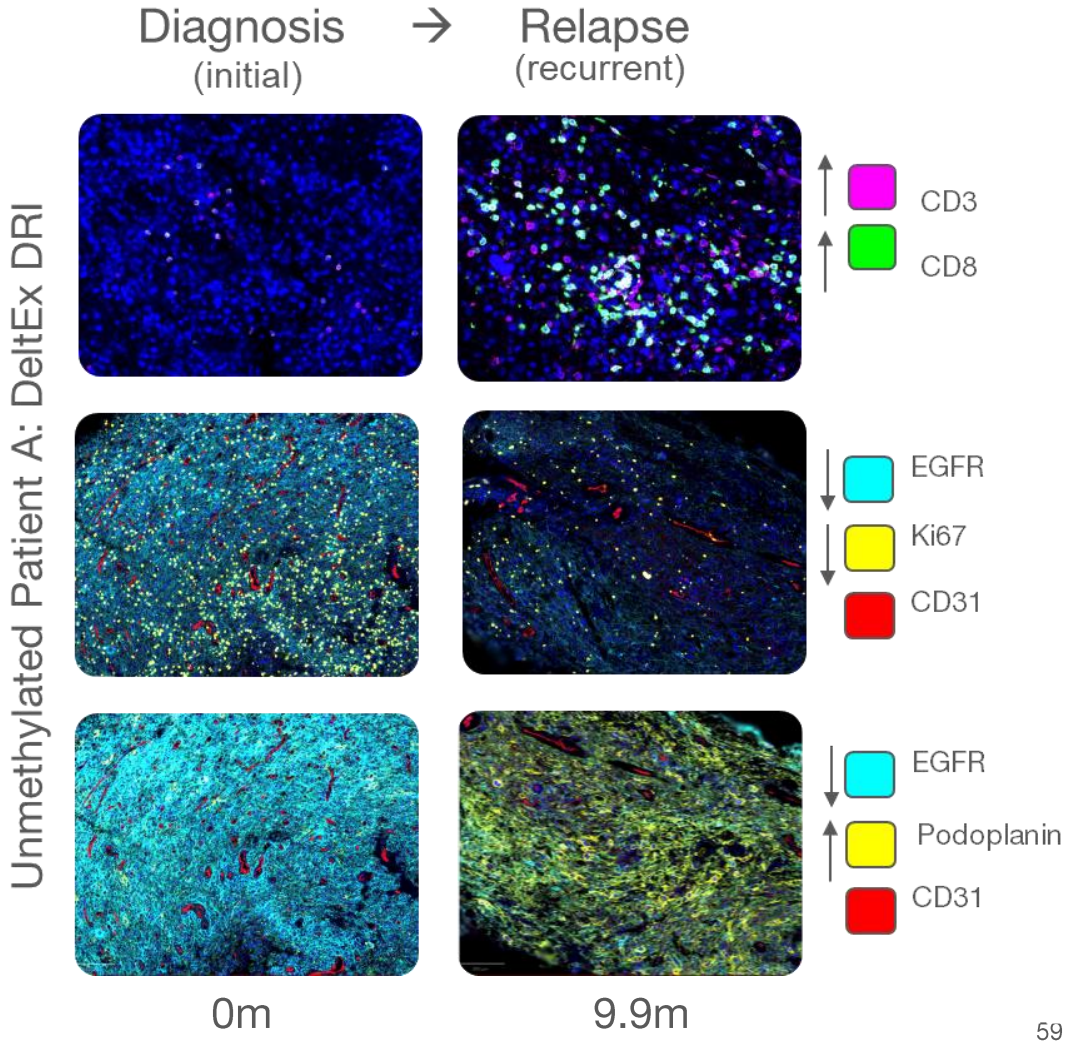
Partnership with Elucidate Bio enables single-cell spatial mapping of the tumor immune environment



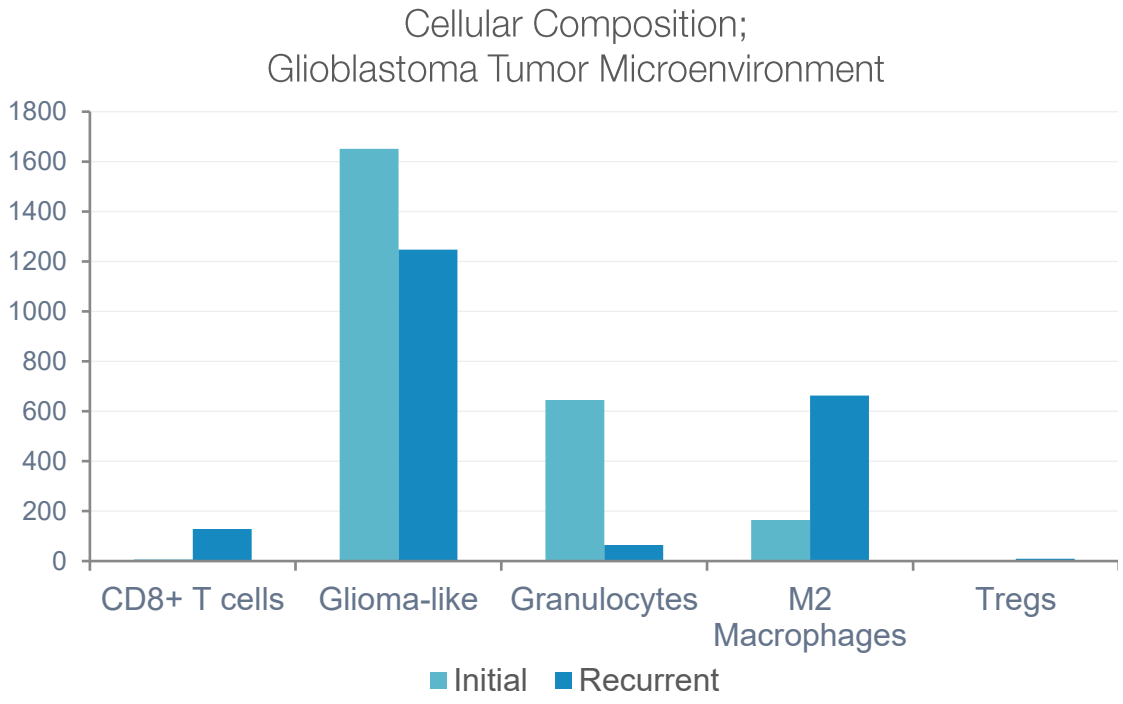
DeltEx DRI Shifts TME from Cold to Hot

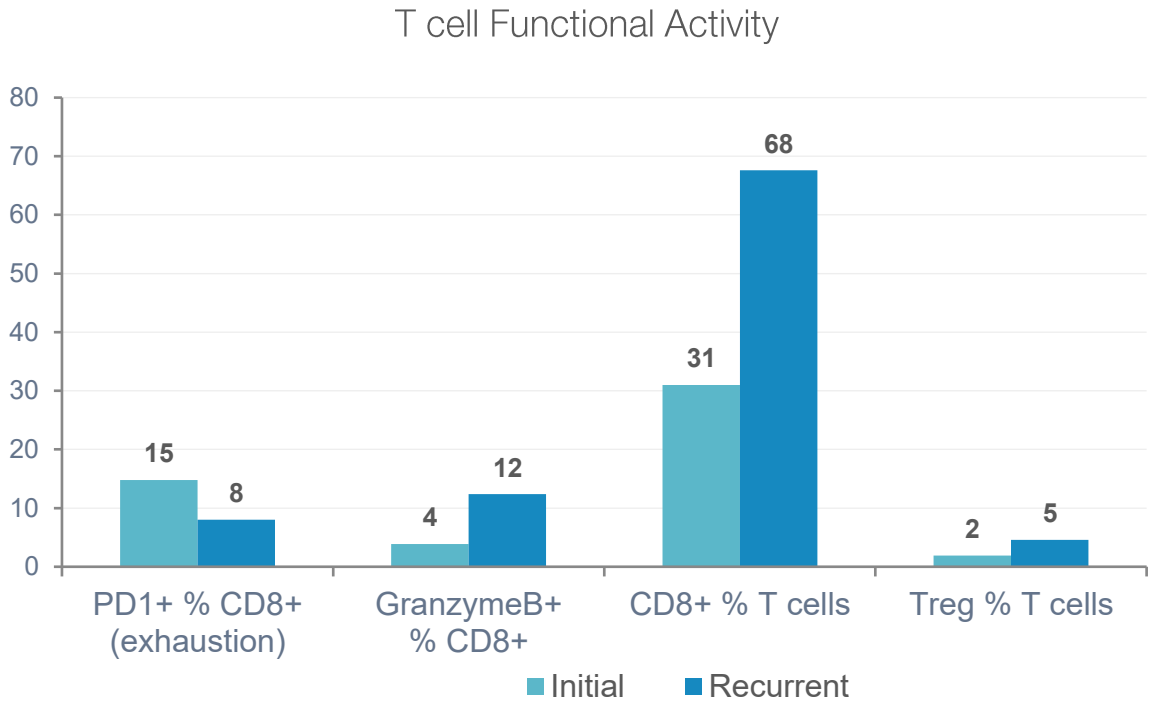
Increased T cell infiltration, reduced tumor proliferation and granulocyte clearance in DeltEx DRI treated patient





DeltEx DRI and GBM Tumor Remodeling





CD8+ T cells
18×
7.2 → 128.6 /mm²

Tumor burden
−24%
Glioma density

Ki-67 (tumor)
−82%
9.4% → 1.7%

Granulocytes
−90%
645 → 65 /mm²

M2 Macrophages
+4×
164 → 663 /mm²

Tregs % T cells
+2.4×
1.9% → 4.6%

PD1+ = exhaustion marker · GranzymeB+ = cytotoxic activity ·

The Data Show Benefits Across Every Dimension

- ✓ Repeat dosing is decisive — more doses, stronger immune response, longer survival
- ✓ ~6x more DRI patients remained progression-free relative to expected survival
- ✓ γδ T cell levels directly predict survival (ρ=0.8, p=0.01)
- ✓ DRI turns immune-desert tumors into immune-active (18x more killer T cells)
- ✓ Escape mechanisms identified; intensification and combination therapy are a logical next step

IN8bio

Corporate Updates

2026 Catalysts Across Pipeline^

$21.9M cash on hand, $0 debt; runway into 2Q27 with potential additional capital of ~$29M

- Ticker: **INAB**
- $21.9M Cash on hand at March 31, 2026
  - 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 May 4, 2026

INB-100	Complete treatment of expansion cohort patients at multiple centers in 2026
	Report long-term follow-up of Phase 1 results at a medical meeting in late 2026
INB-200 / INB-400	Present long-term data, including final median OS from treated patients at medical meetings, including ASCO 2026 and conferences in late 2026
	Peer-reviewed publication of Phase 1 data
	Complete discussions with the FDA on clinical pathways forward, including any potential path for accelerated approval
INB-619	Present initial animal data on CD19 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

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IN8bio

Why IN8bio?

- The only company with 35 years of γδ T cell biology and clinical expertise across both TCE and cell therapy modalities
- Biology informed design of next generation technologies
  - Our TCE activates and expands both γδ subtypes to achieve deeper target depletion with potentially fewer toxicities
  - Strong clinical data including 4+ year remissions in difficult indications with no observed CRS or ICANs
- Experienced team with a track record of attaining milestones and delivering strong clinical data
- Multiple near-term and high-value data catalysts in 2026

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