Harnessing the Power of Gamma-Delta T Cells

IN8bio R&D Day 2026

May 21st, 2026

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Agenda

01 Platform & Biology

02 INB-619 in Autoimmune Diseases

03 DeltEx Drug Resistant Immunotherapy (DRI) in GBM

04 Corporate

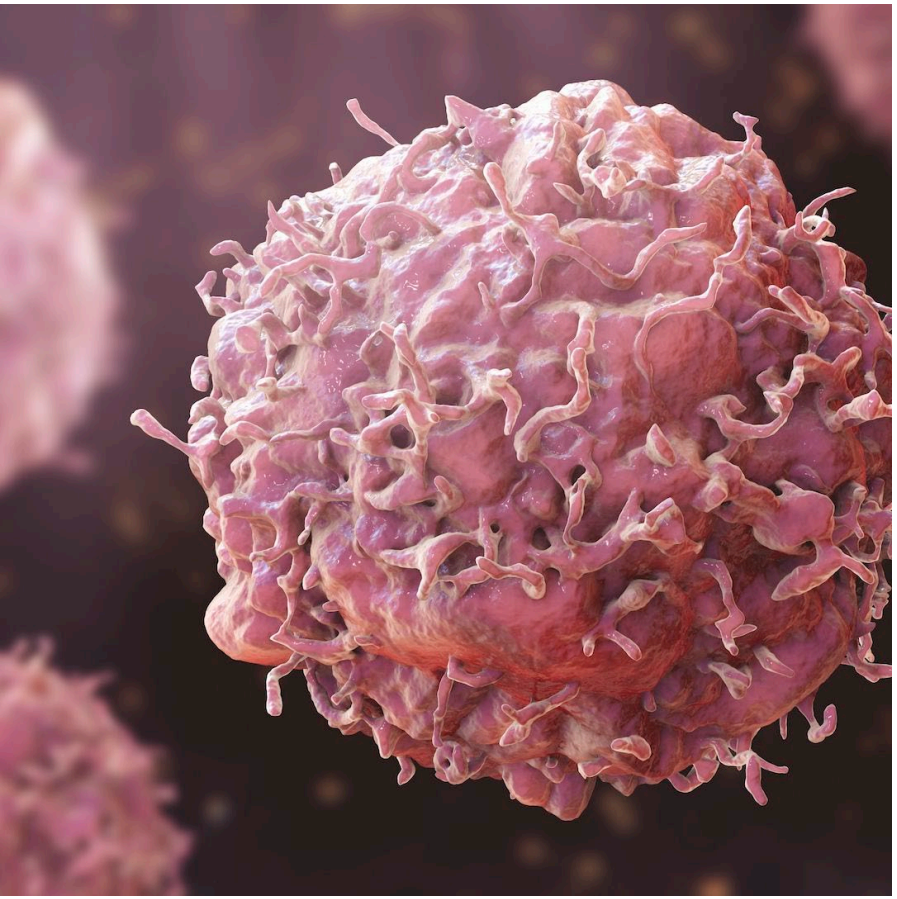
05 Q&A with Reardon

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

Two Sides of the Same Problem

Cancer and Autoimmune Disease



- Millions of patients with cancer or autoimmune disease have no good treatment options
- Current therapies cause dangerous side effects that limit how much doctors can give
- Current autoimmune therapies suppress the entire immune system — leaving patients vulnerable to infections and cancer
- The root cause of both: the immune system is either fighting too little, or fighting the wrong target

Our Goal

Restore Immune Balance

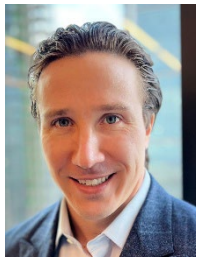
Deep Experience Across the Team

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

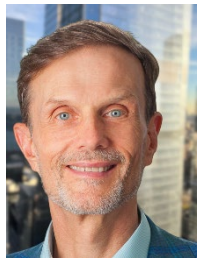


Patrick McCall, CPA
Chief Financial Officer

Deloitte.
CHUBB
CATALYST BIOSCIENCES
TURNSTONE BIOLOGICS

Kate Rochlin, PhD
President & Chief Operating Officer

CURADIGM
EXPANDING LIFE
BIOTAGENICS
IMMUNOVENT
filament
BIOSOLUTIONS INC.



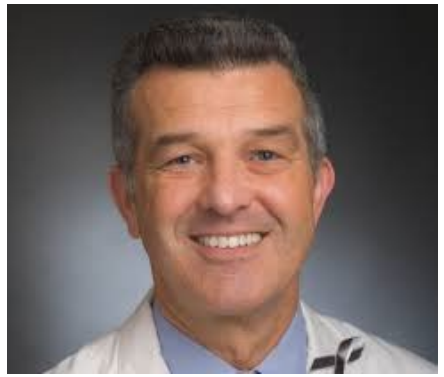
Lou Vaickus, MD, FACP
Interim Consulting Chief Medical Officer

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

Guest Speaker



Dana-Farber Cancer Institute

David Reardon, MD, Dana Farber Cancer Institute

- Dr Reardon, is a Professor of Medicine at Harvard Medical School and currently serves as Clinical Director of the Center for Neuro-Oncology at Dana-Farber Cancer Institute in Boston, MA. He previously served as the Associate Deputy Director of the Preston Robert Tisch Brain Tumor Center at Duke University Medical Center for eleven years. He completed his residency at John Hopkins Hospital in Maryland, USA and was awarded a fellowship at the University of Michigan.

- Dr. Reardon is an active researcher with special interests in the design and implementation of clinical trials for neuro-oncology and the preclinical evaluation of promising therapeutics for central nervous system tumors. His work includes using innovative clinical therapeutic agents to improve outcome for patients with brain and spinal tumors, with particular focus on immunotherapeutics. He has also led investigations of molecular-targeting agents, anti-angiogenic reagents, cytotoxins and other biologically-based therapies such as PD-L1. Dr. Reardon has published over 270 peer-reviewed manuscripts. He received the R. Wayne Rundles Award for Excellence in Cancer Research as well as the Award for Excellence in Adult Clinical Research by the Society for Neuro-Oncology in 2015 and 2016.

Lawrence Lamb, PhD, CSO

Introduction

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**

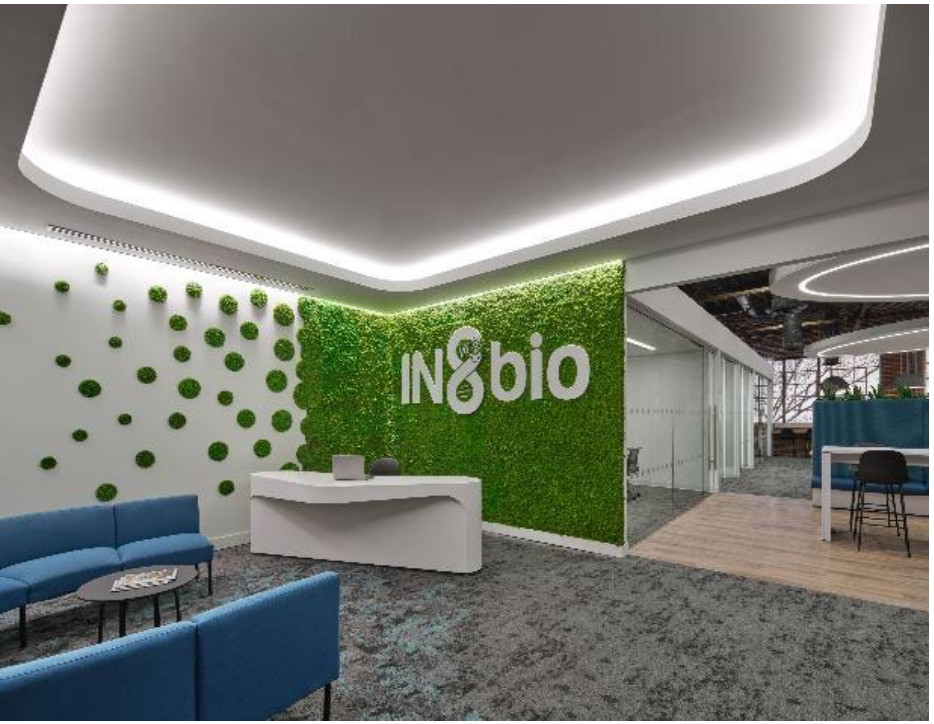
IN8bio R&D facility, Birmingham, AL

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

Today We Will Focus on Two High-Value Opportunities



IN8bio R&D facility, Birmingham, AL

T cell Engager platform (INB-600)

- Off-the-shelf biologic approach; autoimmune + oncology

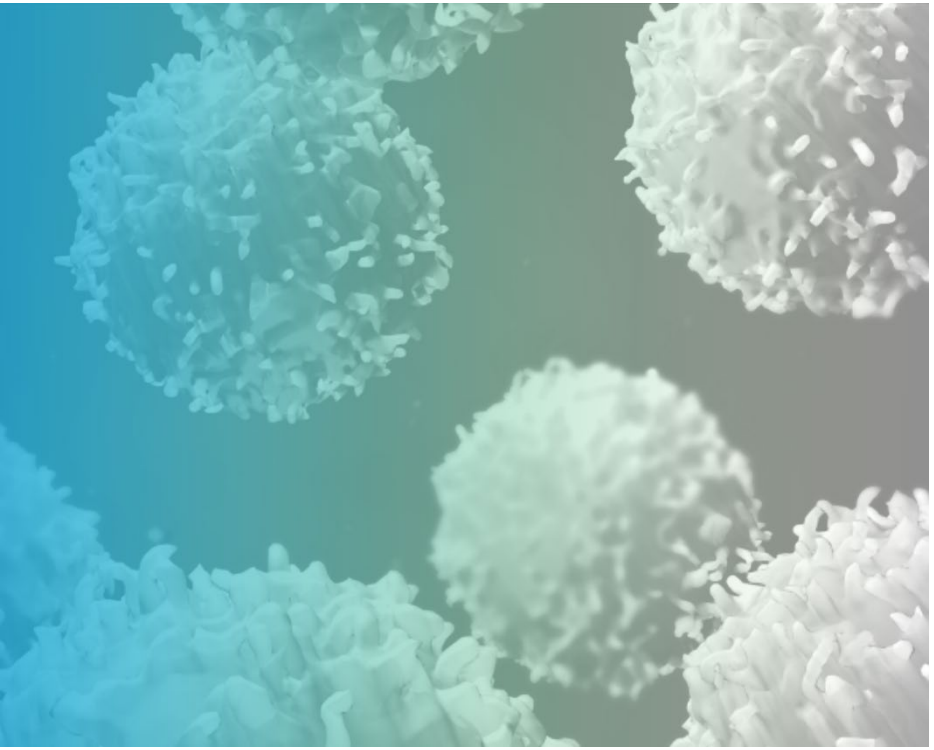
Glioblastoma (INB-200/400)

- Proprietary “DeltEx DRI” cell therapy approach for solid tumors with high unmet need

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

What is a γδ T Cell?

The immune system's battlefield manager

The Problem: There aren't enough of them! IN8bio's team has spent 35 years solving that.

Source: Image generated by Gemini, 2026

Source: Andrew Sewell - https://www.linkedin.com/in/andrew-sewell-26048517/

γδ T Cells Combine the Best of all Immune Cells

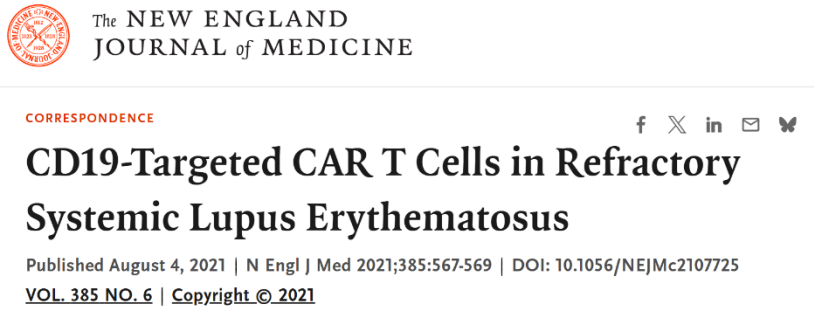
Rare but powerful immune cells that can effectively identify and eradicate target cells

	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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THE LANCET

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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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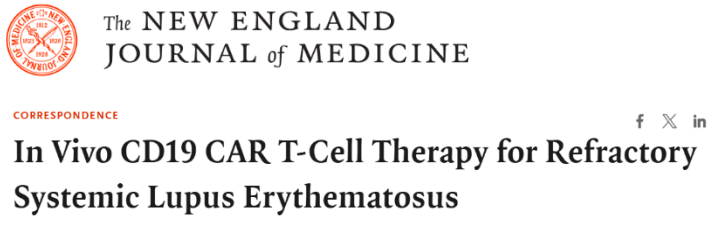

The NEW ENGLAND JOURNAL of MEDICINE

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

Lymphodepletion Induces Long Term Side Effects

ORIGINAL ARTICLE | ARCHIVE

Cyclophosphamide-Induced Ovarian Failure

Authors: G. L. Warne, M.B., B.S., M.R.A.C.P., K. F. Fairley, M.D., F.R.A.C.P., J. B. Hobbs, M.B., B.Sc. Med., Ph.D., F.C.A.P., and F. I. R. Martin, M.D., F.R.A.C.P. Author Info & Affiliations

Published November 29, 1973 | **N Engl J Med 1973;289:1159-1162** | **DOI: 10.1056/NEJM197311292892202**
VOL. 289 NO. 22

Abstract

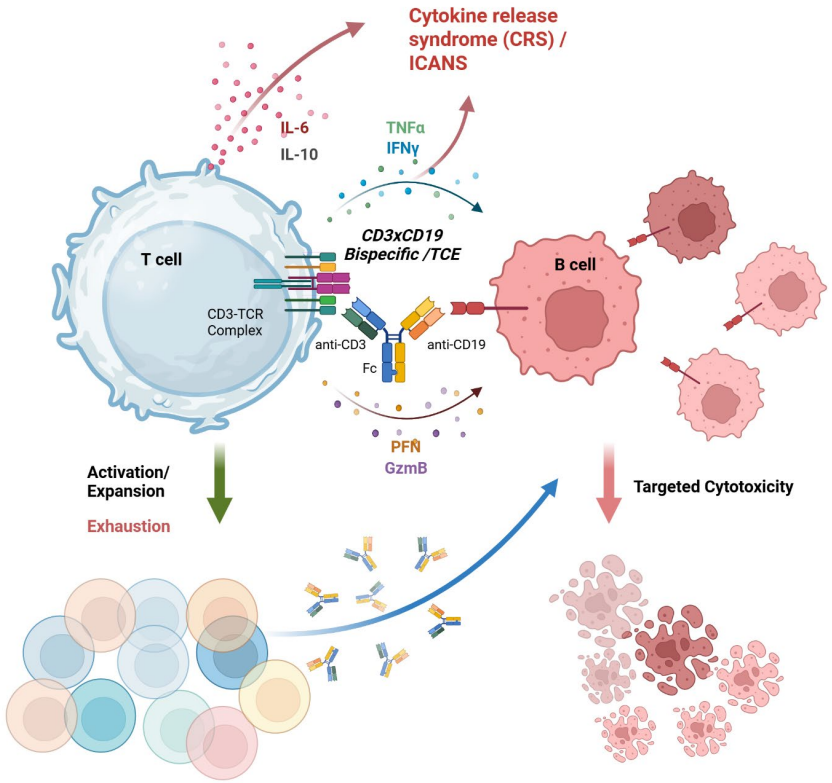
Twenty-two women receiving cyclophosphamide for either progressive glomerulonephritis (20) or rheumatoid arthritis (two) were studied to define more closely the nature of menstrual abnormalities known to occur with this drug. The patients were studied by assay of urinary estrogens and total gonadotrophins and, when possible, by ovarian biopsy. Seventeen patients had definite or probable ovarian failure after cyclophosphamide. No patient on whom ovarian biopsy was performed showed normal follicular maturation, and ova were seen in only two. One patient judged clinically and biochemically to have ovarian failure regained normal ovarian function 10 months after cessation of cyclophosphamide therapy. (N Engl J Med 289:1159–1162, 1973)

Coordinating CAR-T Care in the Hospital is Complex!!!

Source: Image generated by Gemini, 2025

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

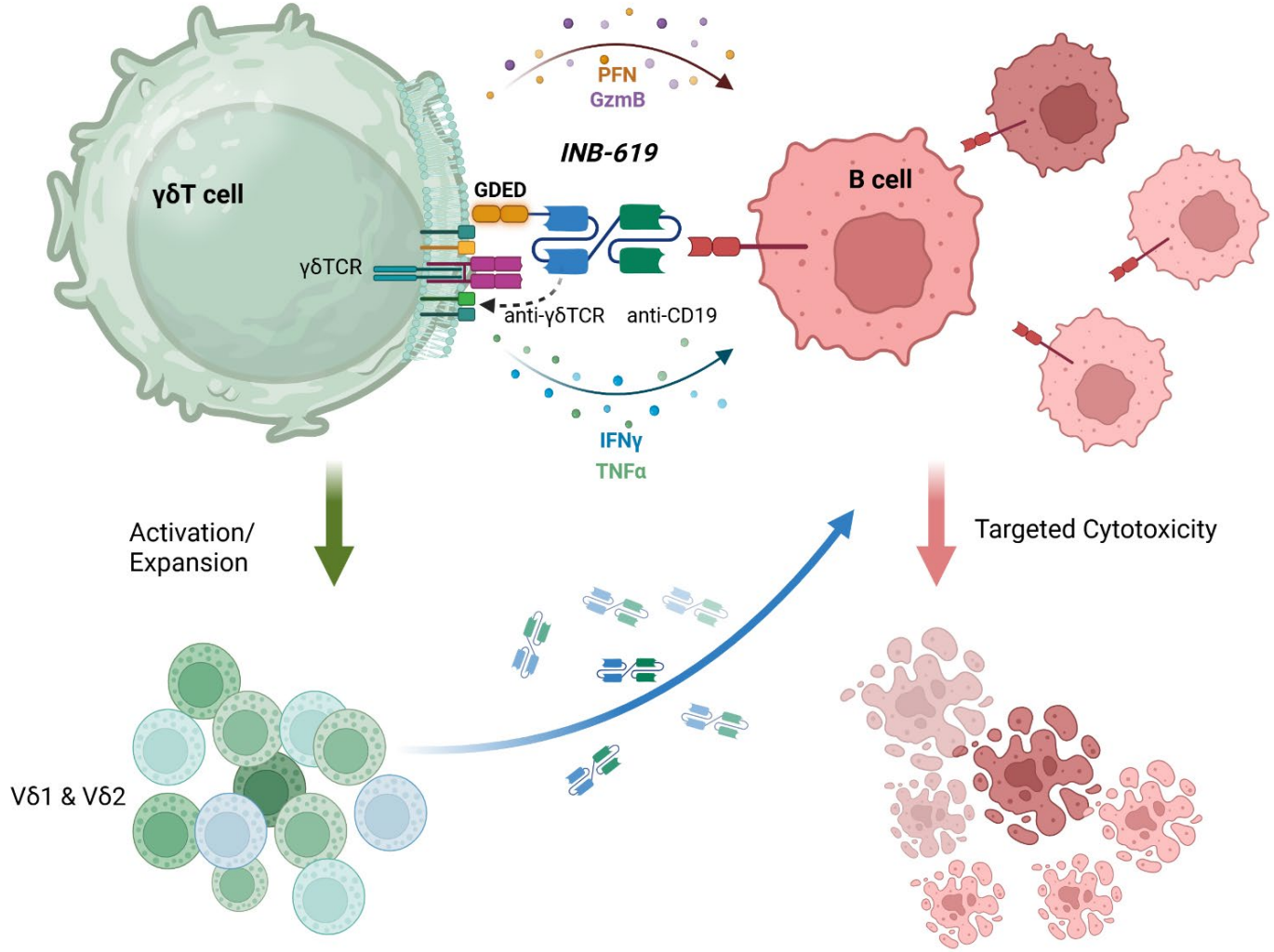
IN8bio's γδ TCEs Solve All Three Problems

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

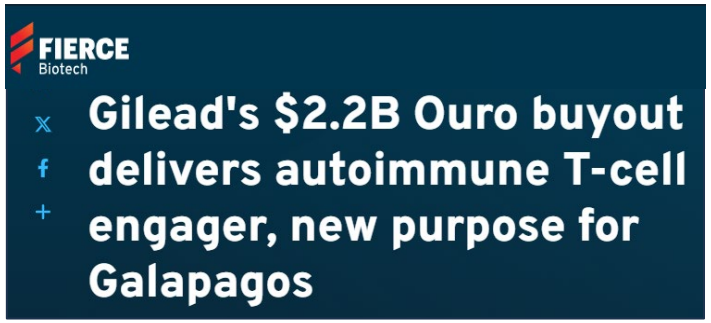
INB-619: Depletes B Cells Without the Toxicity of CD3 TCEs

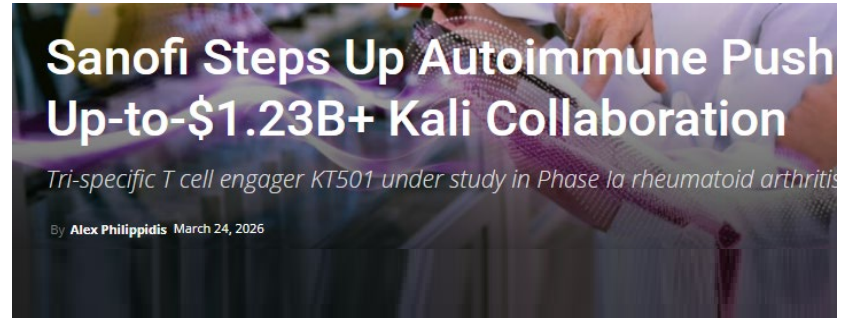
Pan-**γδ** TCE to expand both Vδ1 and Vδ2 cells — reaching B cells CD3 TCEs cannot



The Recent T cell Engager Deal Explosion in 2026

Company	Partner	Upfront Deal Size ($MMs)	Phase	Target
Candid Therapeutics	UCB	$2 Billion	Phase 1	BCMA-CD3, CD20-CD3
Ouro Medicines	Gilead	$1.7 Billion	Phase 1a	BCMA-CD3
Kali Therapeutics	Sanofi	$180 Million	Phase 1	CD19-BCMA-CD3, CD19-CD3





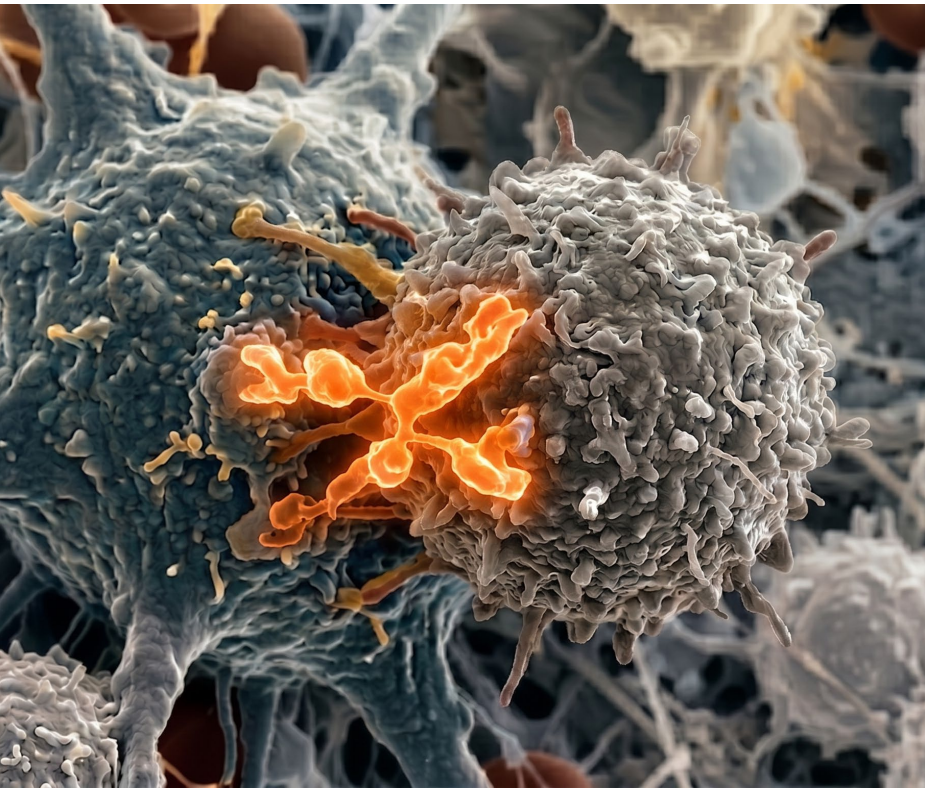
Autoimmune TAM estimated at $135.6B in 2026 and growing at 8.45% CAGR*

Source: *https://www.researchandmarkets.com/report/autoimmune-disease-drug

INB-619: A Pan γδ CD-19 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

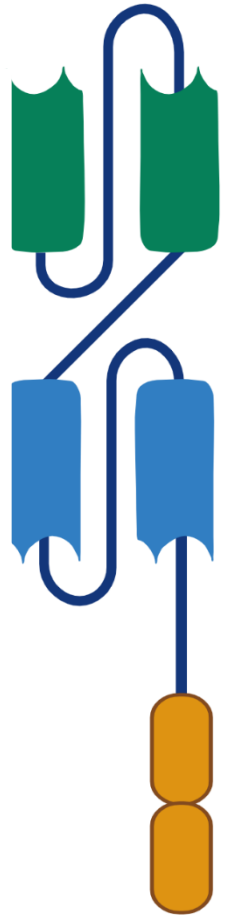
INB-619: The First Pan-γδ T cell Engager to Drive Expansion

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

Cassette-like CD-19 domain targets B cells

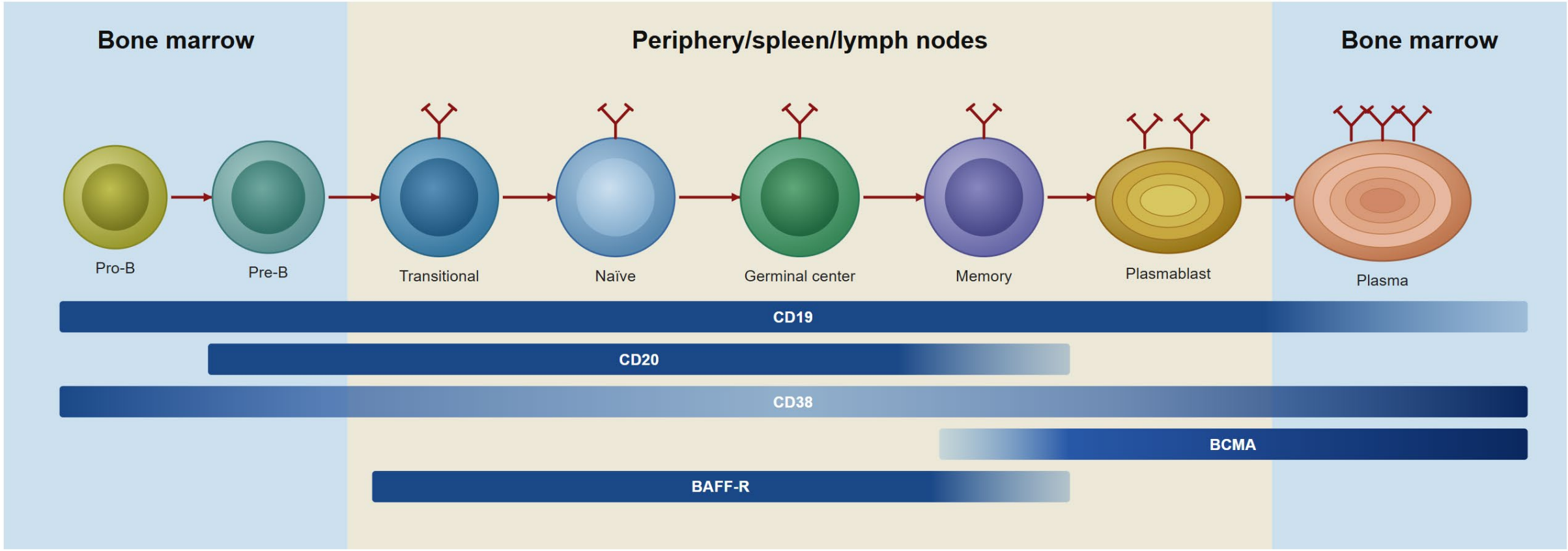
Pan γδ TCR binding activates without CD3

GDED (gamma delta expansion domain) drives in vivo expansion



CD19 Covers Every B cell Stage

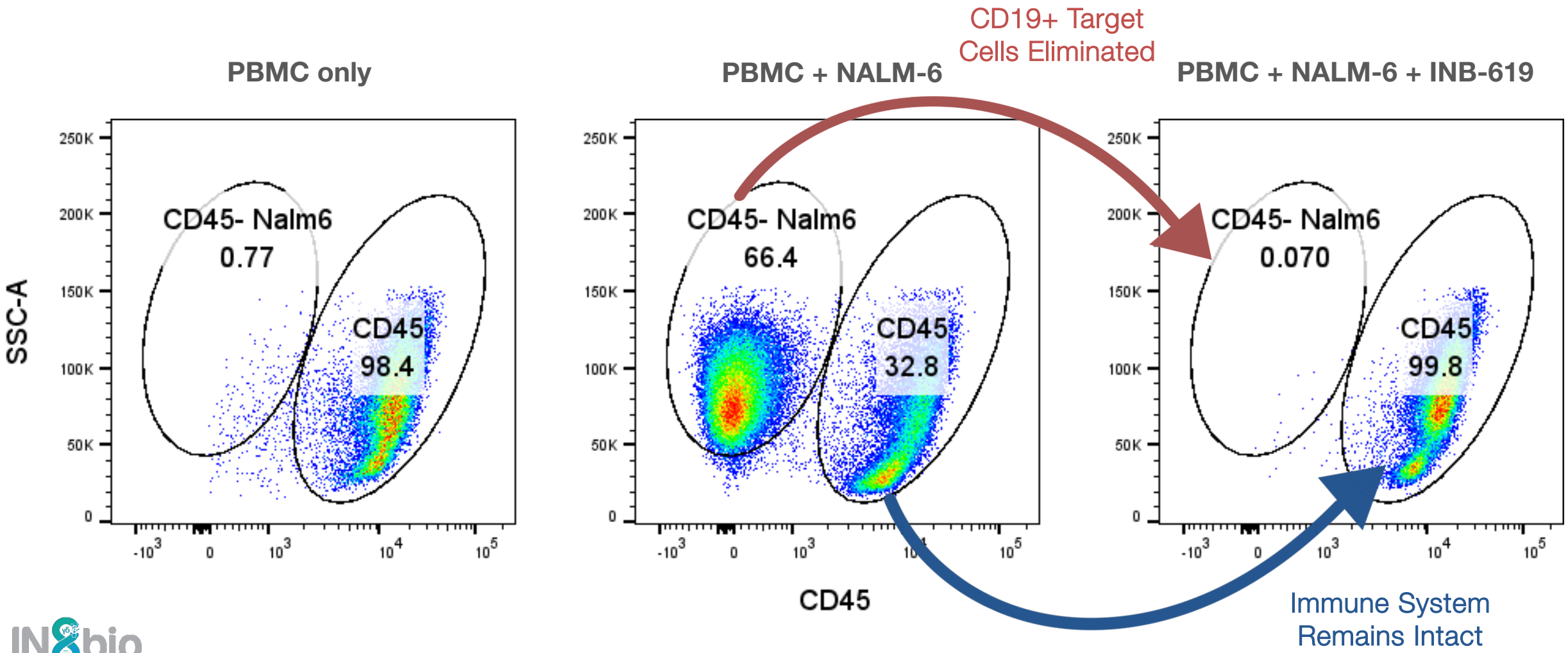
The others miss critical stages — CD19 is validated by Schett's Data across autoimmune indications



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

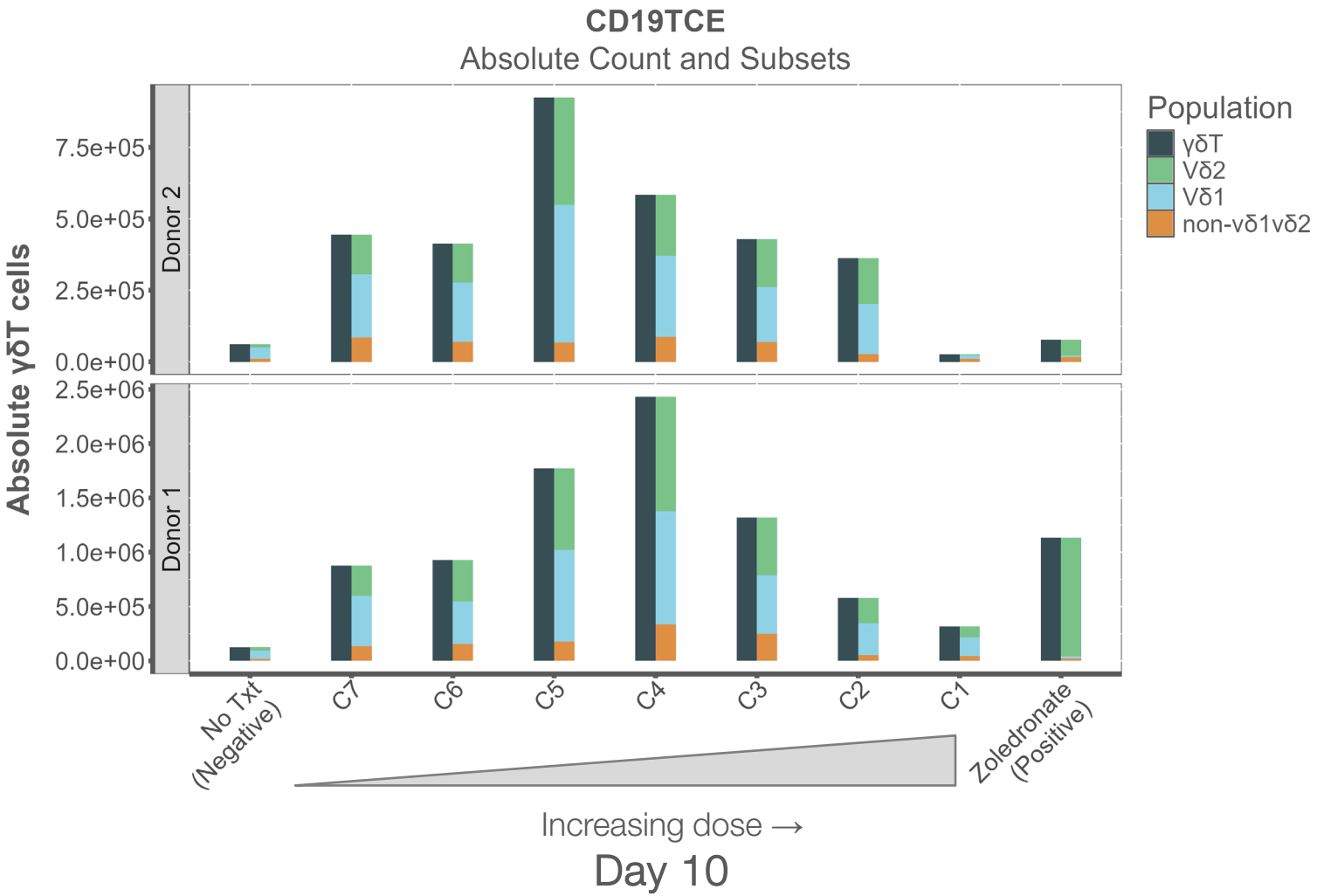
INB-619 Efficiently and Specifically Eliminates Target Cells

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

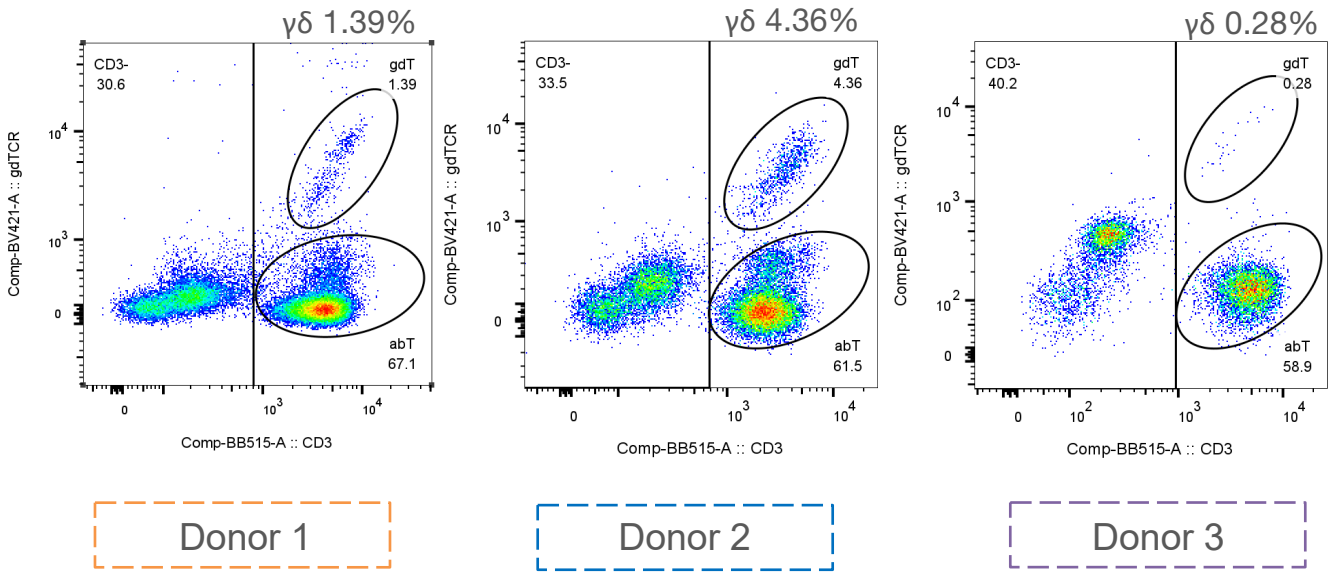


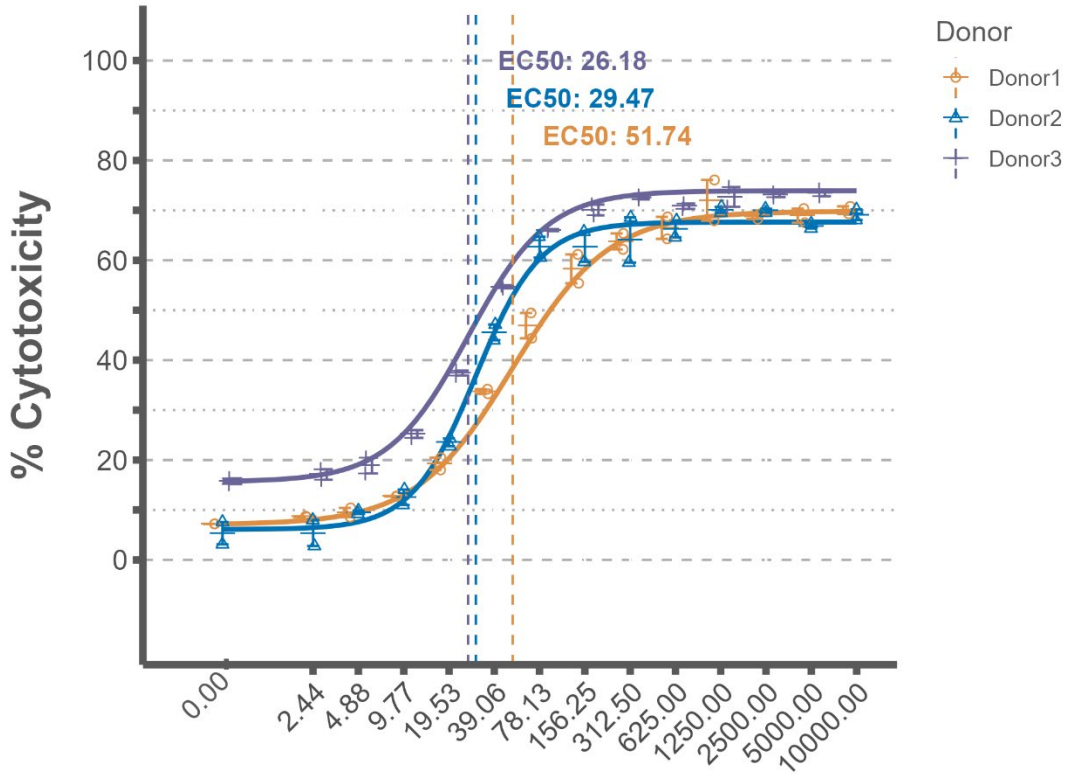
- Expansion is dose-dependent across both γδ subtypes
- Vδ2+ provide surveillance and Vδ1+ tissue residence, enabling deeper B cell depletion
- No expansion without INB-619 (No Txt control)

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

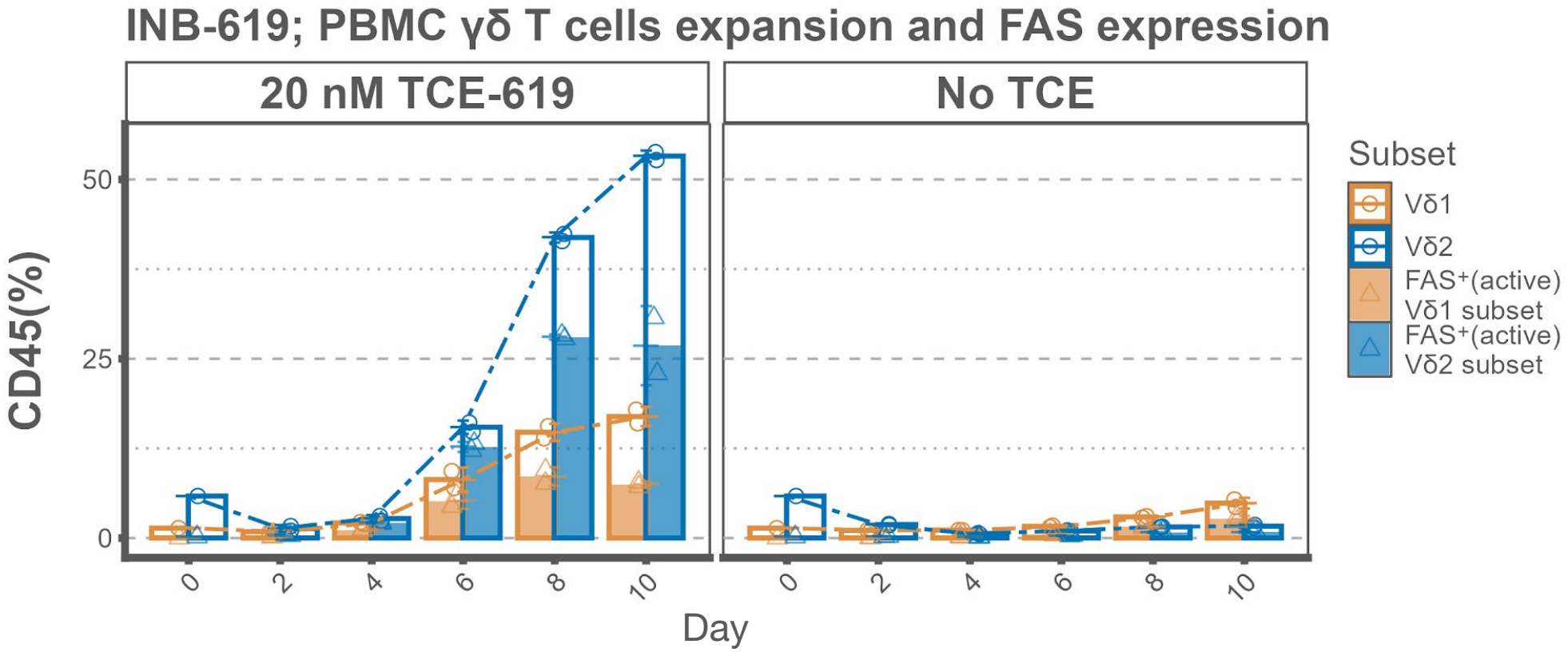
- Donors ranged from 0.28% to 4.36% γδ T cells at baseline
- All three showed overlapping potency (EC50: 26–52 pM)





INB-619 Induces Controlled Activation of Both Vδ1+ and Vδ2+

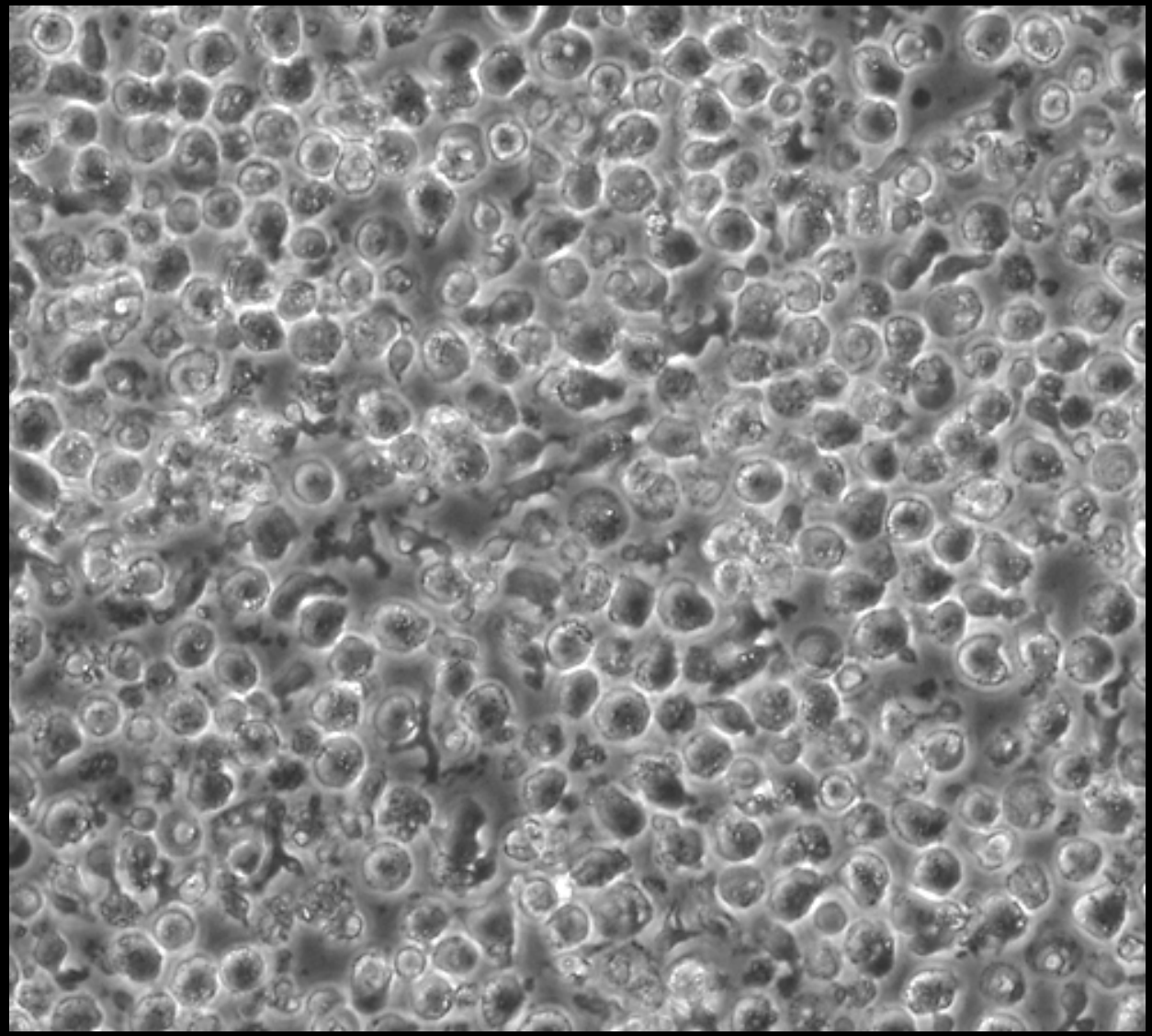
No expansion activation without INB-619 — full expansion of both subtypes with it



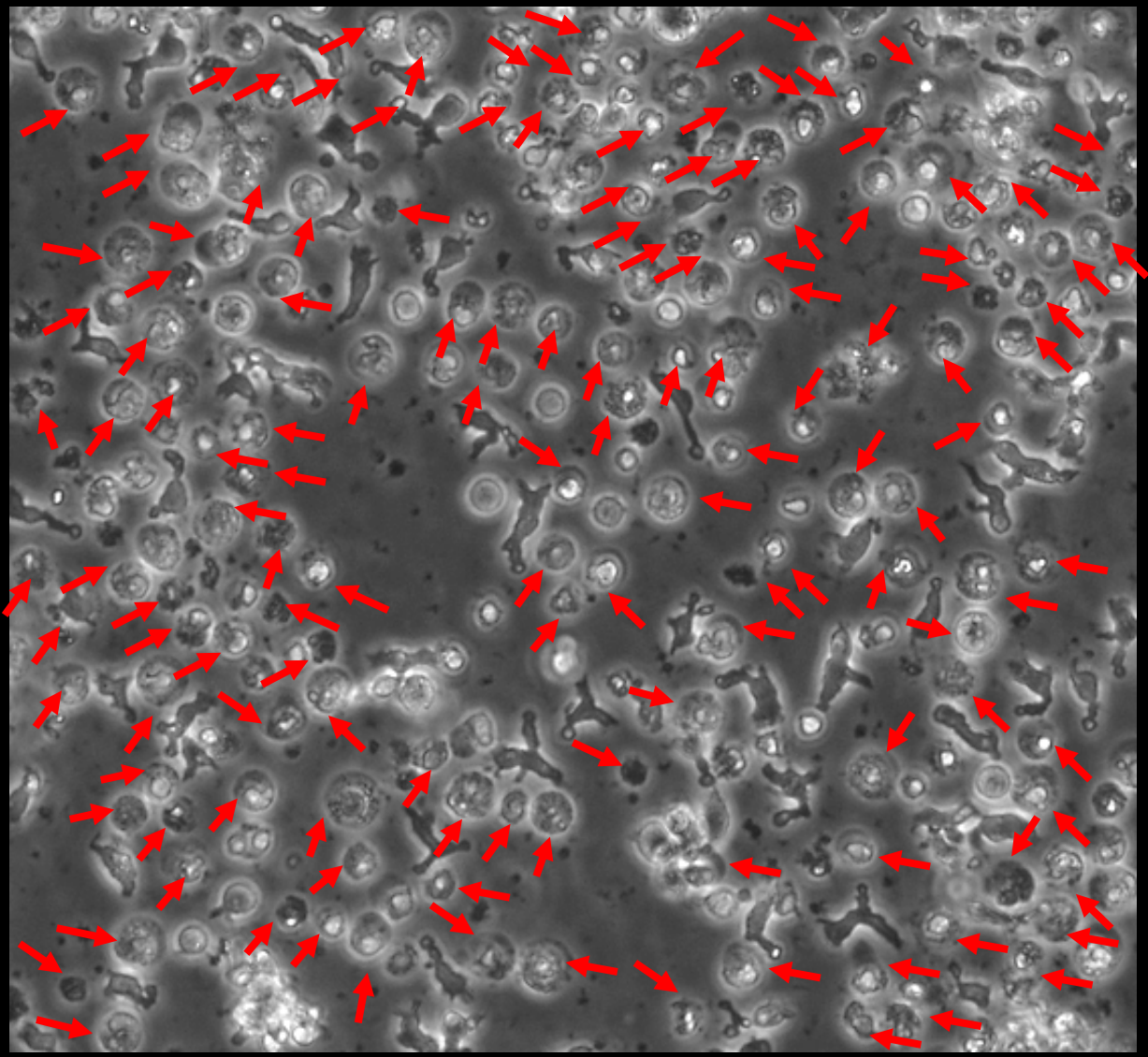
INB-619 causes expansion and controlled activation of both Vδ1+ and Vδ2+

No significant expansion without INB-619

CD19-γδ TCE Induced Killing is Clearly Visible



γδ T vs. Nalm6 without 19xTCE

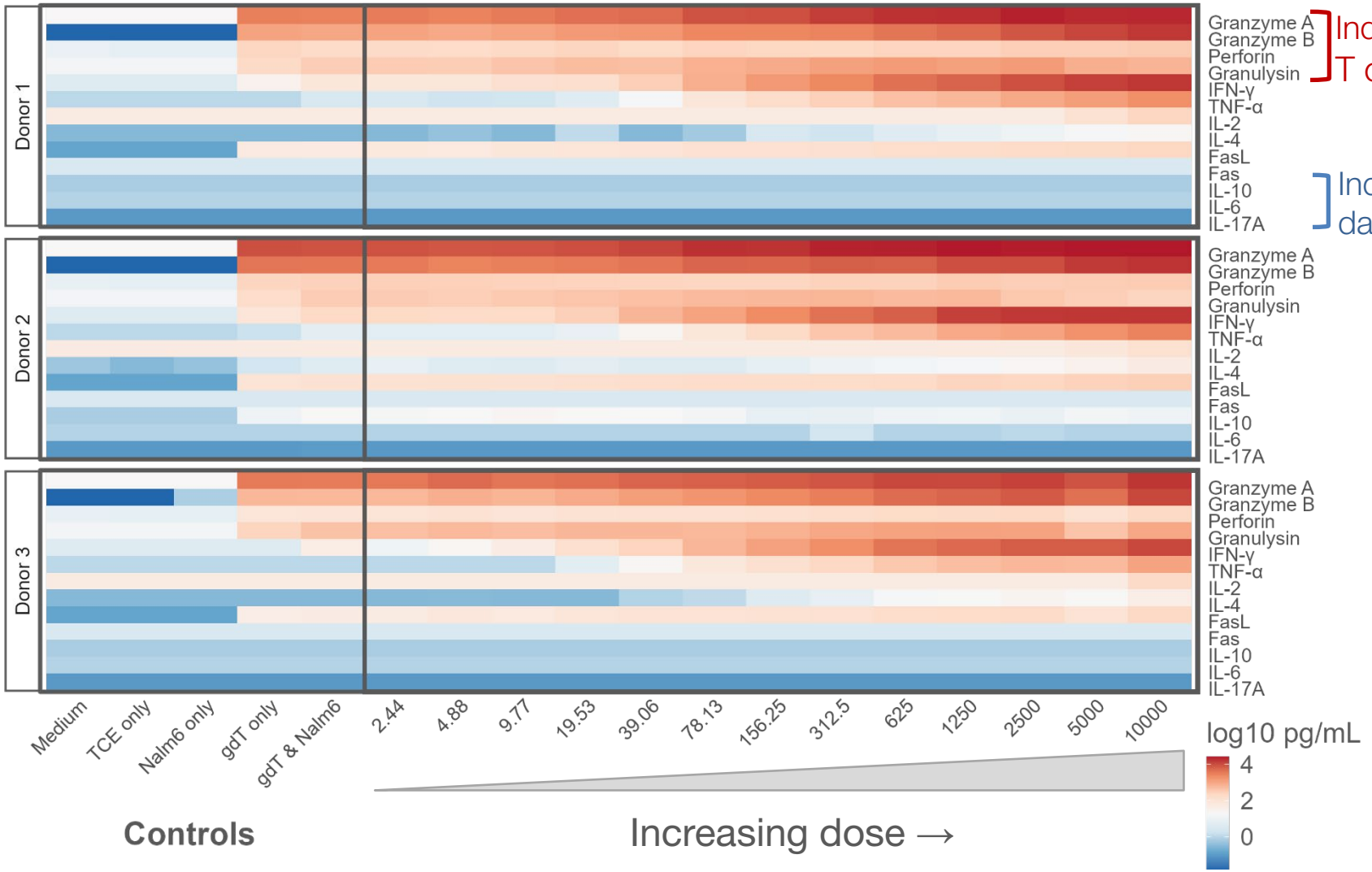


γδ T vs. Nalm6 with 19xTCE

E:T = 1:1 @ 24 hours

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

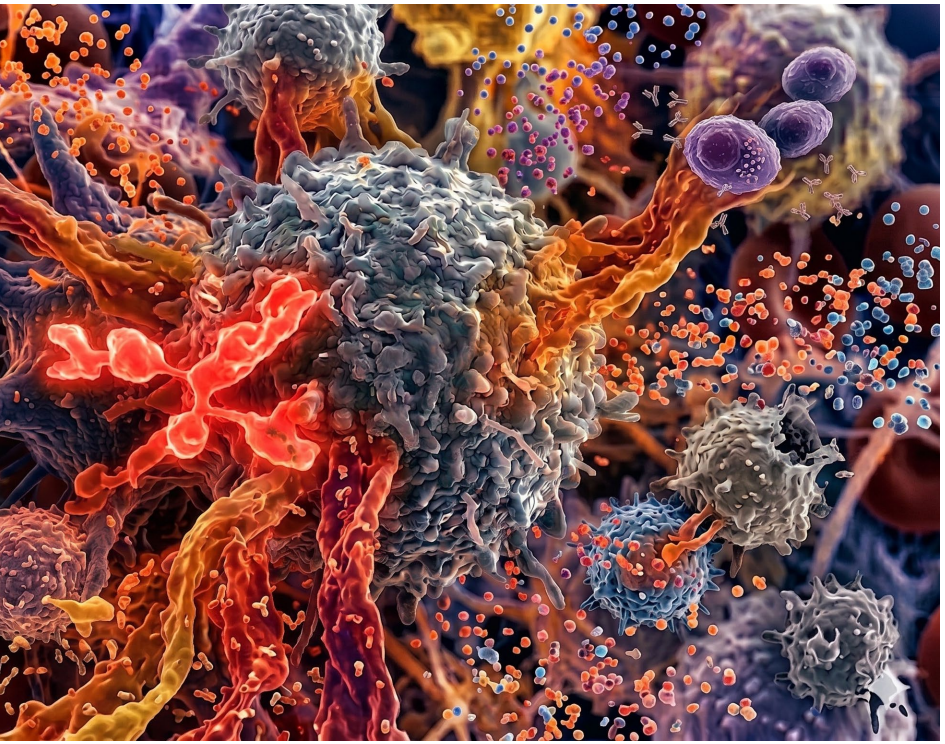


Indicates Activation of γδ T cell Killing Pathways

Indicates no increase in dangerous cytokines

INB-619 for Autoimmune Disease

Autoimmune Patients Can’t Tolerate Broad CD3 Toxicity



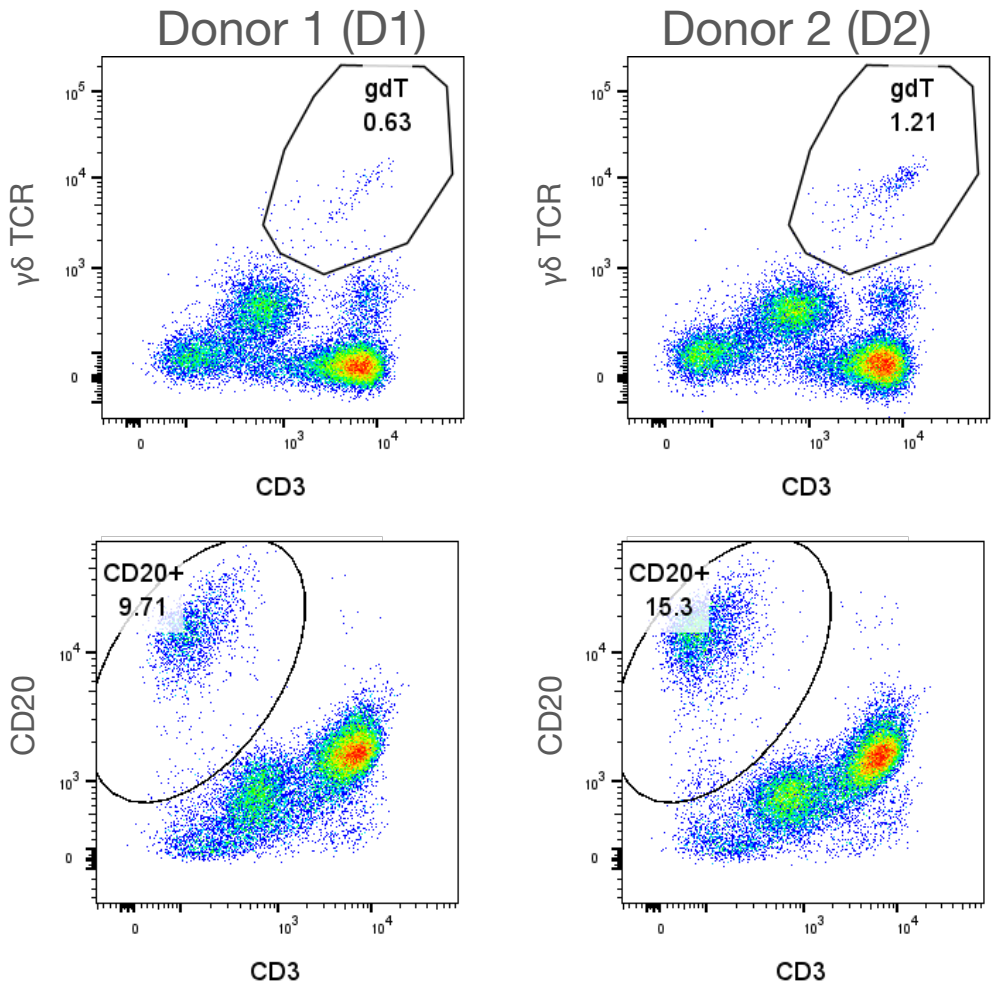
- Cancer patients can tolerate severe side effects because the alternative is often death, autoimmune patients cannot
- Existing CD3 TCEs trigger system-wide immune activation and often lead to excess cytokine release, exactly what autoimmune patients already suffer from
- γδ TCE offers precise immune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

Source: Image generated by Gemini, 2026

Lupus (SLE) Patients Have Low & Variable Levels of γδ T cells

γδ T cells are low & variable

B cells are expanded & high

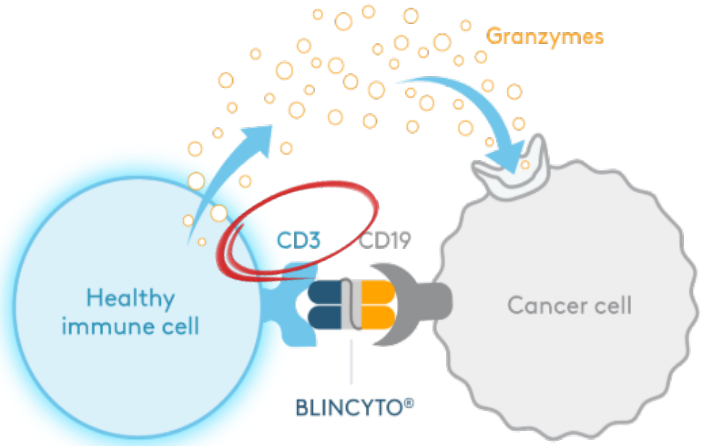


Donors with active SLE disease were selected for this study

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

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

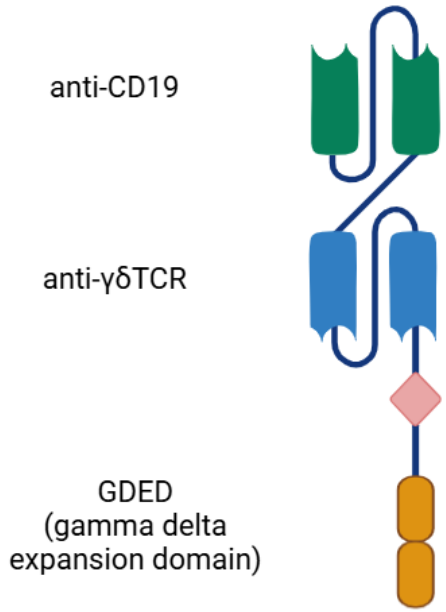
Blinatumomab (BLI)
CD-19 TCE



2025 sales: $1.6B

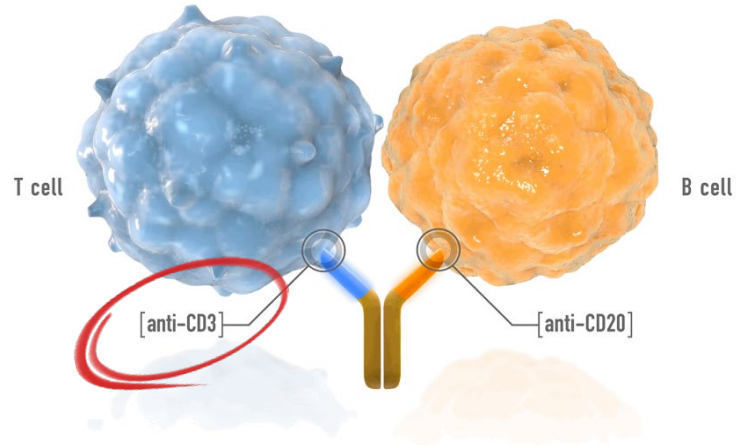
~54 kDa

INB-619
CD-19 TCE



~75 kDa

Mosunetuzumab (MOS)
CD-20 TCE



Est. 2025 sales: $120M+

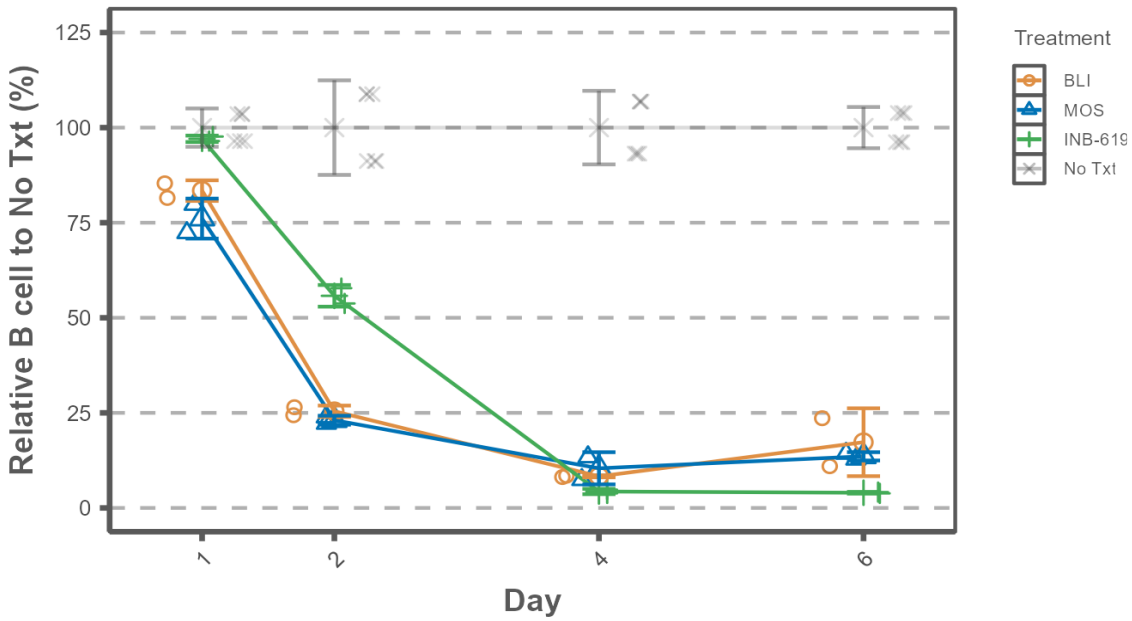
~146 kDa

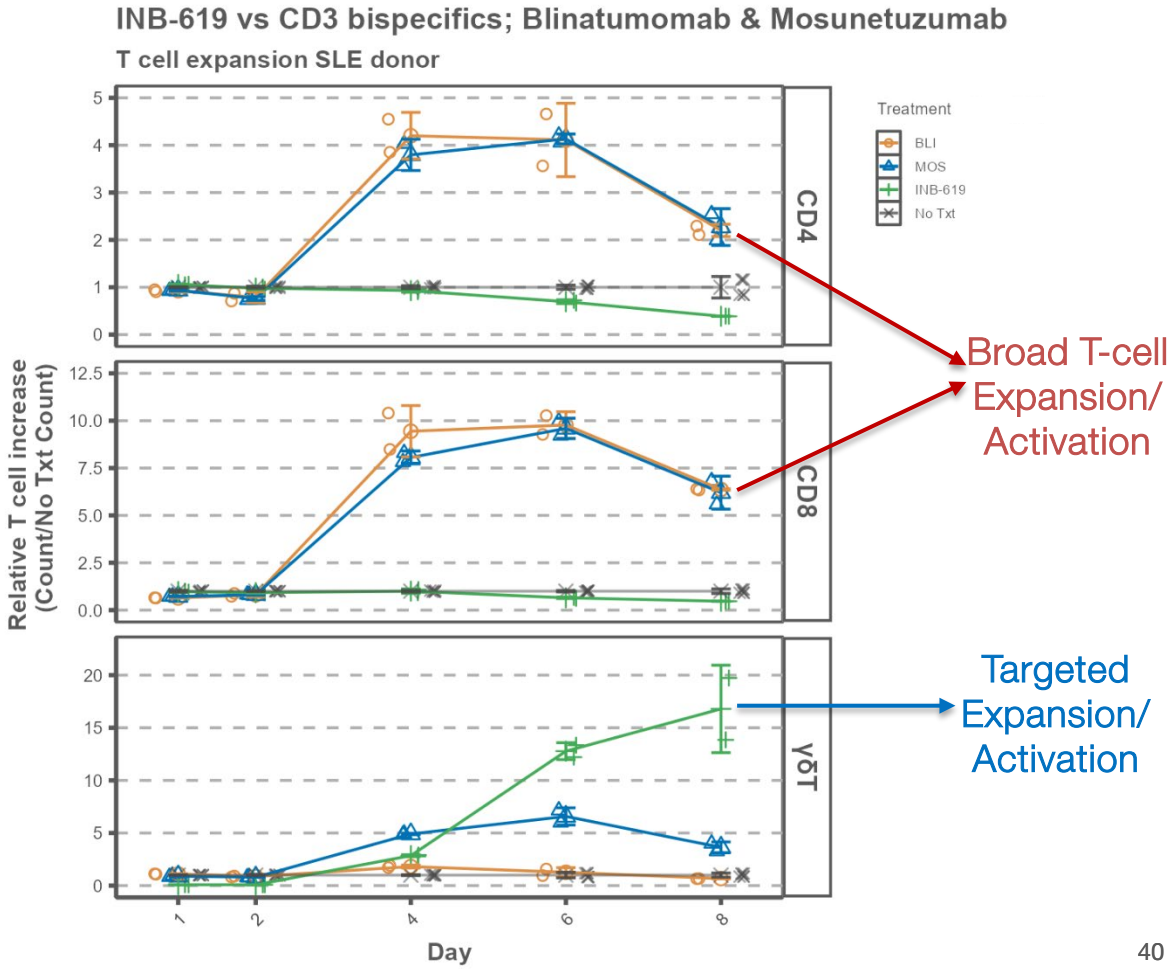
*https://pharmaphorum.com/news/approvals-extend-use-roche-abbvie-drugs-lymphoma?

INB-619 Targeted Activation and Complete Target Elimination

BLI and MOS activate broadly across CD4/CD8 cells, triggering toxicity. INB-619 expands only γδ T cells.

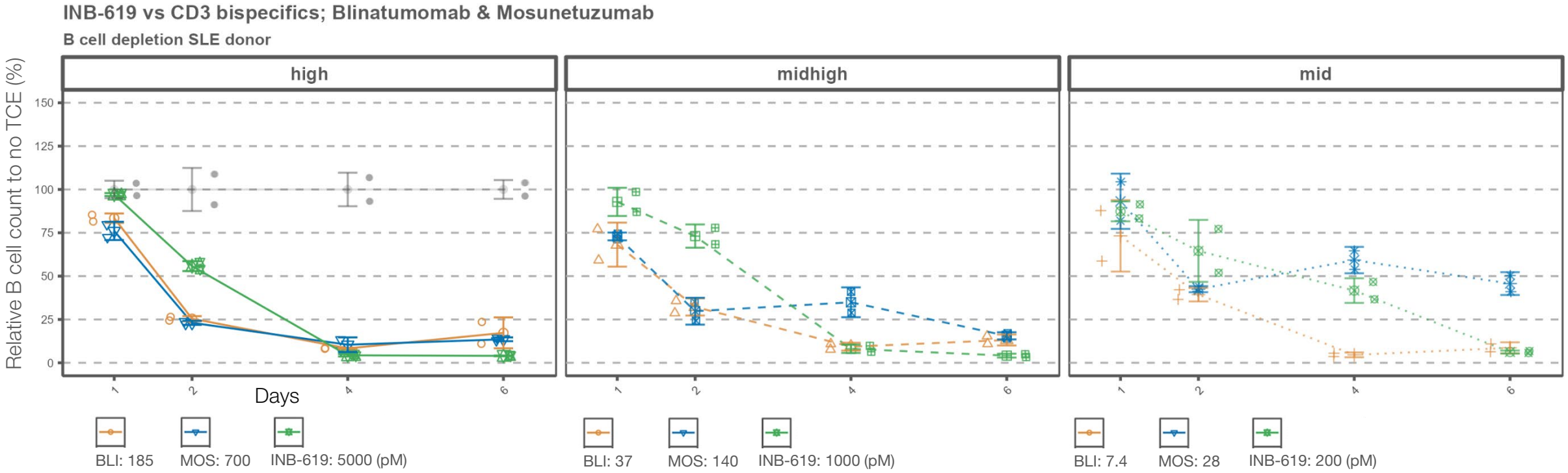
SLE donor B cell depletion





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

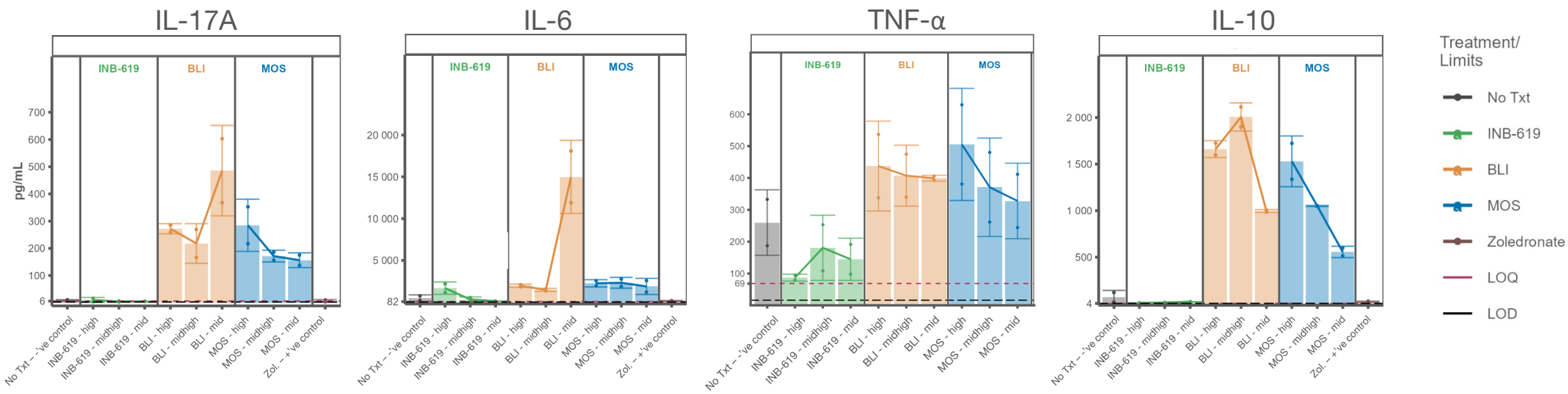
Most CD3 TCEs dose-reduced in autoimmune disease due to potential toxicity



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

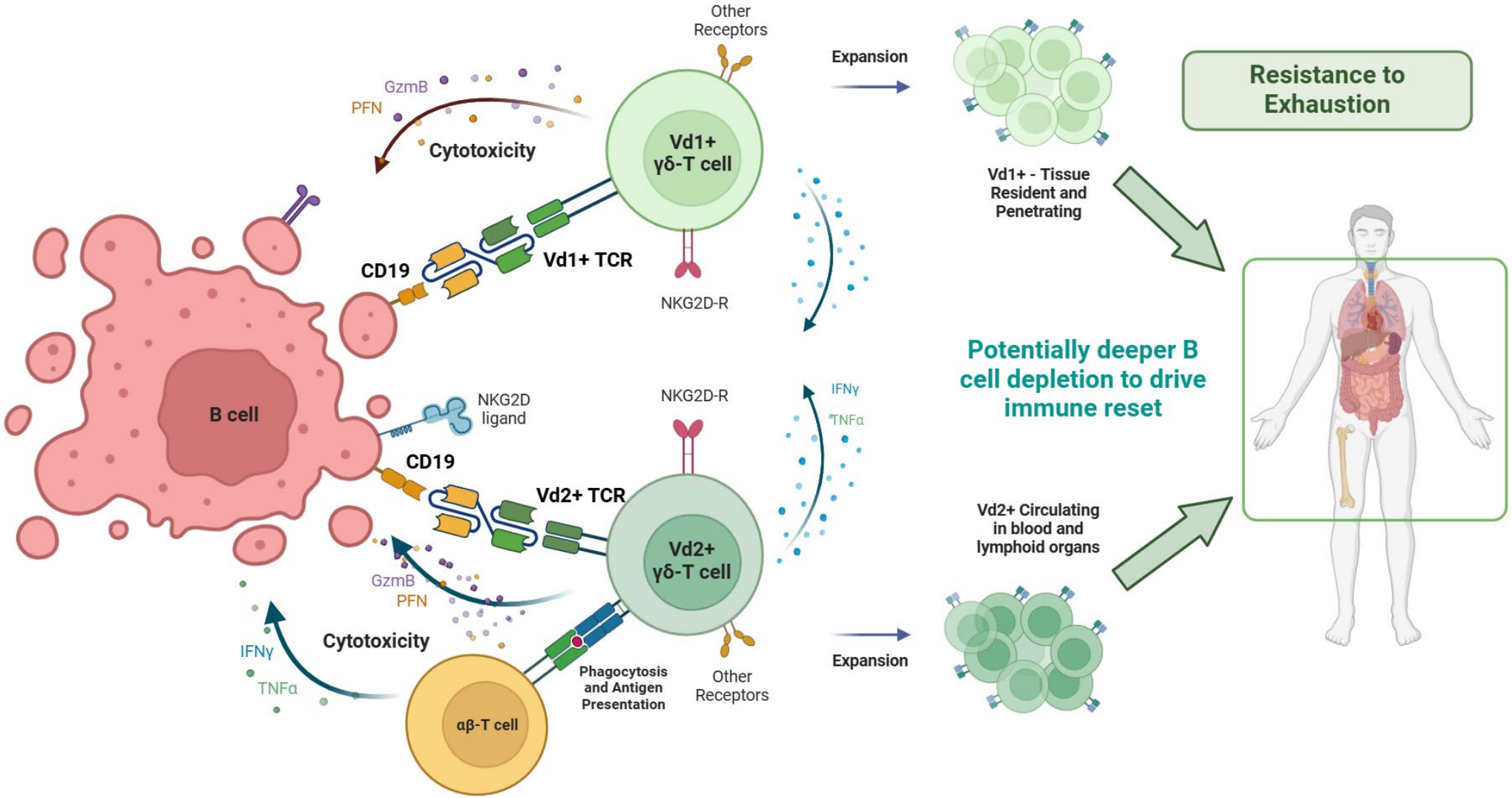
INB-619 Designed to Overcome Autoimmune Challenges

Single-subtype γδ TCEs fail if patients respond differently; INB-619 binds pan-γδ TCR and activates both major subtypes

- Autoimmune patients have dysregulated, shifting and variable immune profiles
- Vδ1+ or Vδ2+ compartments may be more responsive; INB-619 activates whichever responds
- First γδ TCE to demonstrate expansion and activation across all γδ T cell subclones

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

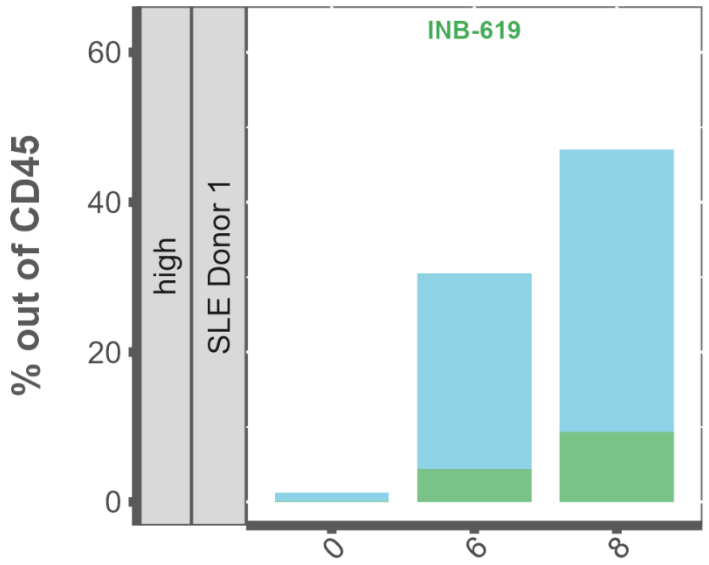
Pan γδ TCR targeting is more powerful to drive B cell elimination



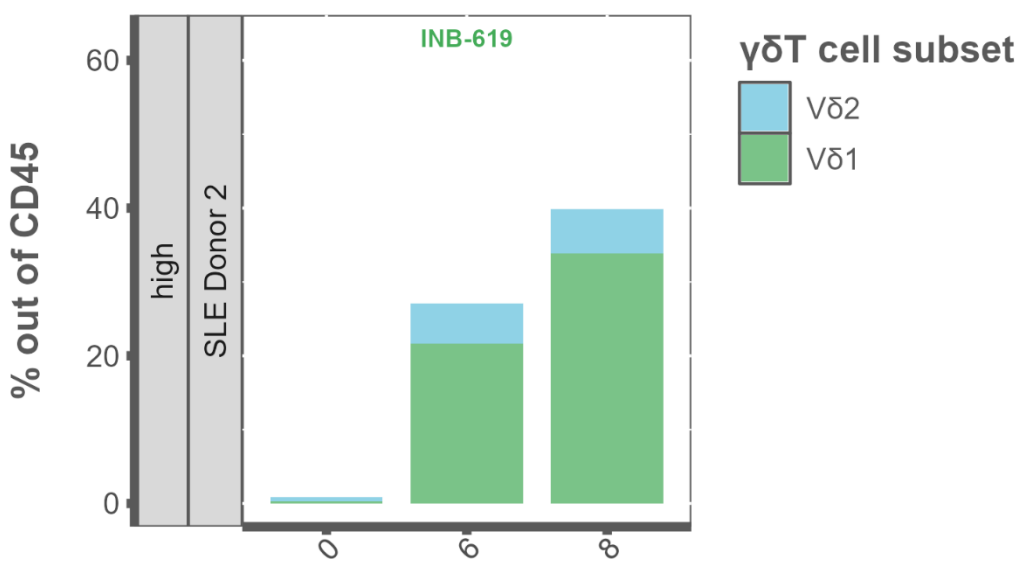
Different Patients, Different γδ Response; Same Outcome

D1 expanded Vδ2+, D2 expanded Vδ1+ → INB-619 achieved B cell elimination in both

SLE Donor 1 (D1)



SLE Donor 2 (D2)

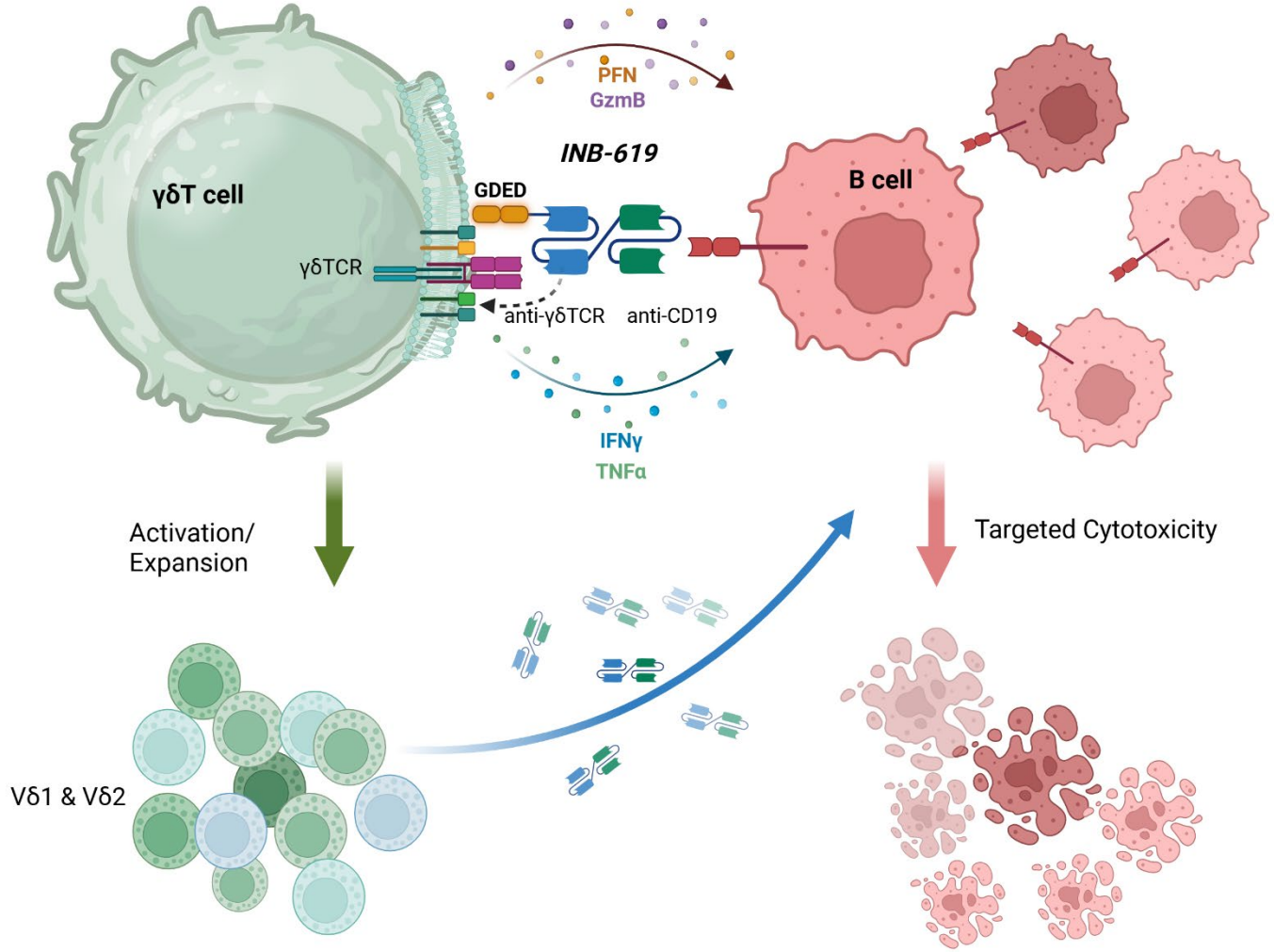


- Both patients started with low but indistinguishable γδ T cell levels
- Complete B cell elimination by Day 4
- This could only be accomplished by a pan- γδ TCE

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



David Reardon, MD

Dana Farber Cancer Institute

Dana-Farber Cancer Institute

Glioblastoma: ***The*** Major Unmet Need in Current Medical Oncology

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Disclosures

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Consulting/Advisory Boards (paid to DR): AnHeart Pharmaceuticals; BlueRock Therapeutics LP; CeCaVaGmbH & Co.KG; Chimeric Therapeutics; Enterome; Genenta Sciences; Jupiter Life Sciences Consulting, LLC; Kintara; Miltenyi Biomedicine GmbH; Nuvation Bio; Neuvogen; Servier; Paradigm Medical Communications; Putnam Inizii Associates, LLC

Data Safety Monitoring Board: ImVax; CeCaVaGmbH

Stock: AnHeart Therapeutics; Bionaut Labs

CNS Cancers: Disproportionate Mortality Relative to Incidence

WHO International Agency for Research on Cancer: Global Cancer Observatory, 2022

Incidence

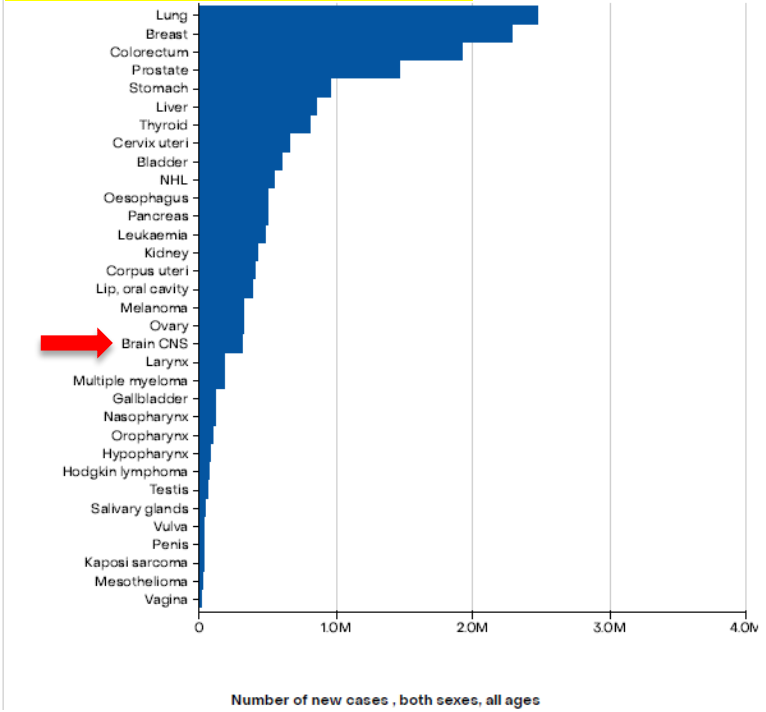
Cases	ASR (World)
19 976 499	196.9

Mortality

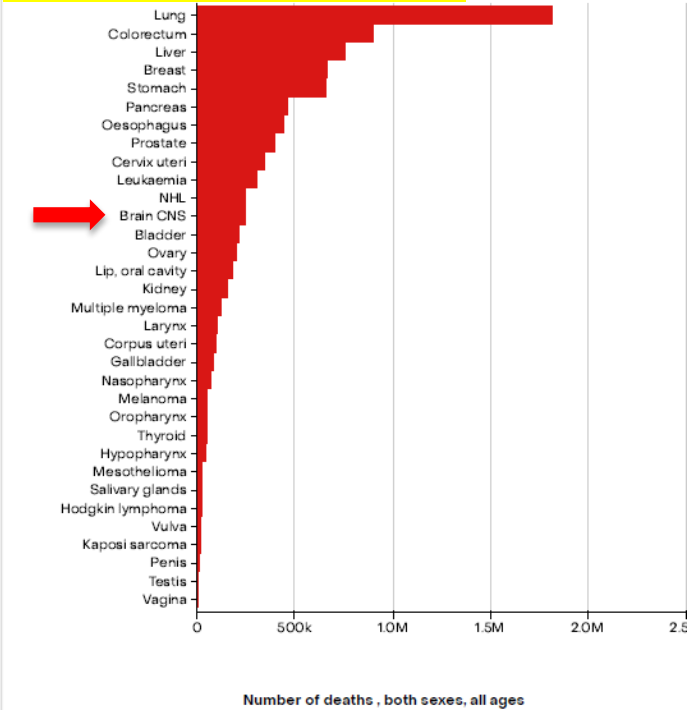
Deaths	ASR (World)
9 743 832	91.7

Cancer site ranking

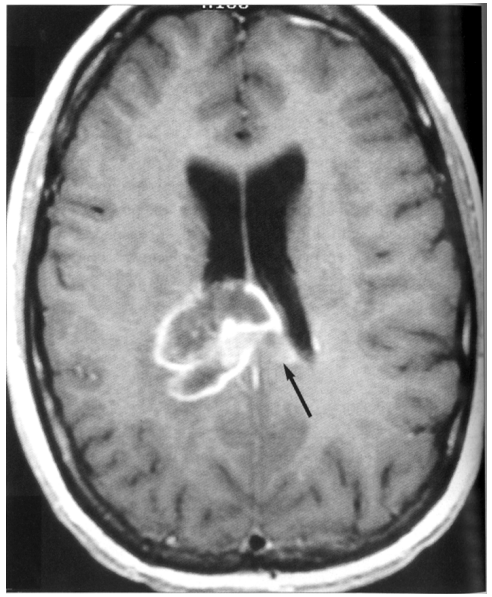
Global Incidence: CNS #19

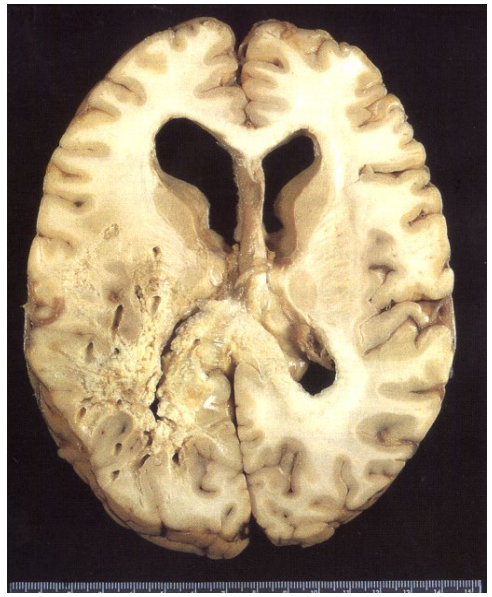


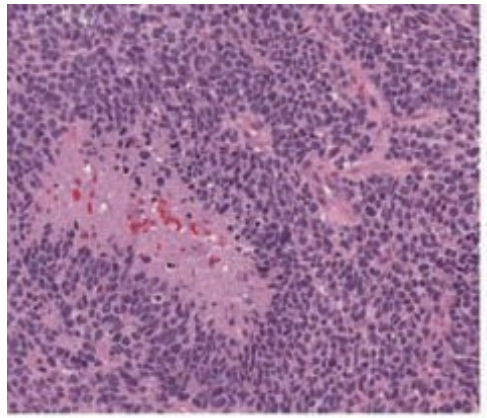
Global Mortality: CNS #12



Glioblastoma







13,000 cases in US/year

Male:female (1.5:1); occurs at all ages with peak 6^{th}-7^{th} decade

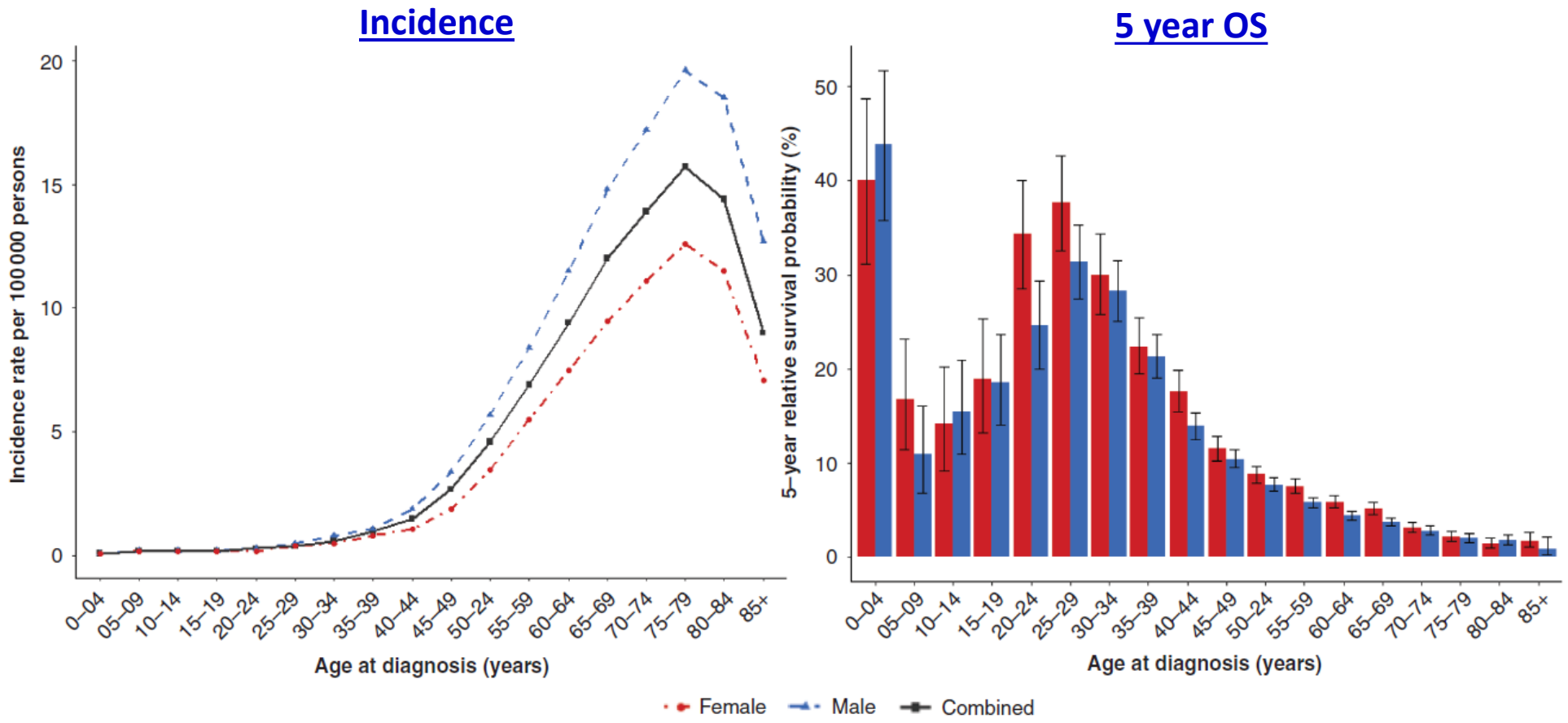
Lack of systemic metastases (local infiltrative/destructive growth)

Incurable and universally fatal

Current standard therapy (defined 2005): surgery, radiation, temodar
- median PFS: 6.9 months
- median OS: 14.6 months
- MGMT unmethylated (55%) vs MGMT methylated (35%)

Survival poorer if:
- MGMT unmethylated
- older
- poor performance status
- unresectable

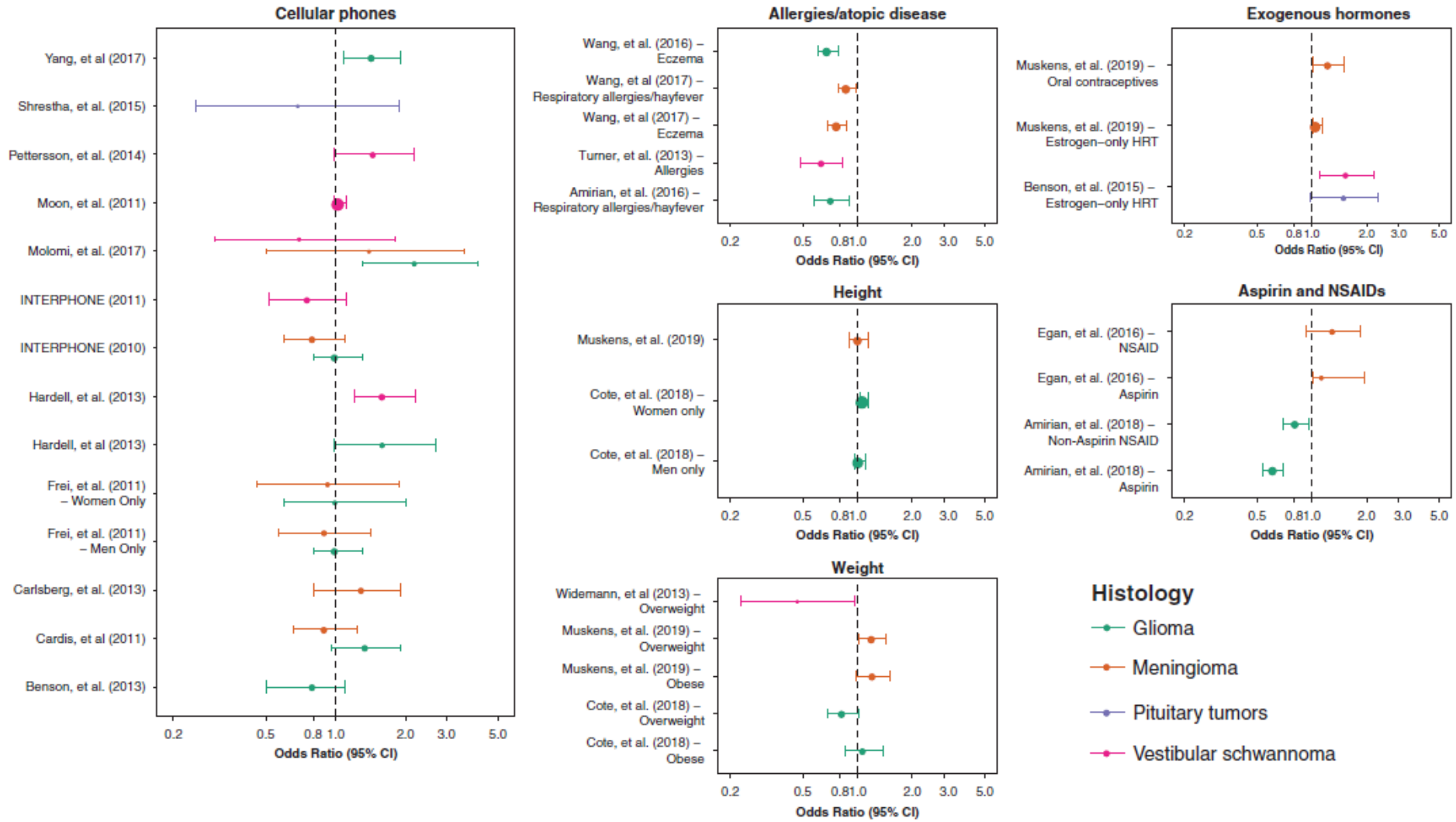
Glioblastoma: Epidemiology



Wen/Weller Neuro-Oncol 22(8):1073-1113, 2020

Glioblastoma: Environmental Risk Factors

Environmental: prior CNS irradiation is the only established risk factor; lower risk with atopy



Ostrom QT Neuro-Oncol 21(11) 1357-1375, 2019

Glioblastoma: Genetic Risk Factors (< 5%)

Gene Symbol (Chromosome Location)	Disorder/Syndrome (OMIM ID)	Mode of Inheritance	Phenotypic Features	Associated Brain Tumors
APC, MMR (5q21)	Familial adenomatous polyposis (FAP, 175100), Turcots syndrome type 2	Dominant	Development of multiple adenomatous colon polyps (>100), predisposition to colorectal cancer, and brain tumors	Medulloblastoma, glioma
ATM (11q22.3)	Ataxia- telangiectasia (208900)	Autosomal recessive trait	Progressive cerebellar ataxia, susceptibility to infections, predisposition to lymphoma and lymphocytic leukemia.	Astrocytoma and medulloblastoma
CDKN2A (9p21.3)	Melanoma-neural system tumor syndrome (155755)	Dominant	Predisposition to malignant melanoma and malignant brain tumors	Glioma
IDH1/IDH2 (2q33.3/15q26.1)	Ollier disease	Acquired post-zygotic mosaicism, dominant with reduced penetrance	Development of intraosseous benign cartilaginous tumors, cancer predisposition	Glioma
MLH1, PMS2	Turcots syndrome type 1	Autosomal recessive trait	Development of multiple adenomatous colon polyps (<100), predisposition to colorectal cancer, and brain tumors	Medulloblastoma, glioma,
MSH2,MLH1,MSH6,PMS2	Lynch syndrome (120435), biallelic mismatch repair deficiency, constitutional MMR *deficiency*	Dominant	Predisposition to gastrointestinal, endometrial and other cancers	Glioblastoma, other gliomas
MSH2,MLH1,MSH6,PMS2	Mismatch repair deficiency syndrome (276300)	Recessive	Pediatric cancer predisposition; café-au-lait spots; colon polyps	Glioma
NF1 (17q11.2)	Neurofibromatosis 1 (NF1) (162200)	Dominant	Neurofibromas, schwannomas, café-au-lait macules	Astrocytoma, schwannomas, optic nerve glioma
RB1 (13q14)	Retinoblastoma	Dominant	Development of multiple tumors of the eye, increased risk of some brain tumors	Retinoblastoma, pineoblastoma, malignant glioma
TP53 (17p13.1)	Li–Fraumeni syndrome (151623)	Dominant	Predisposition to numerous cancers, especially breast, brain, and soft-tissue sarcoma	Glioblastoma, other gliomas
TSC1,TSC2 (9q34.14,16p13.3)	Tuberous sclerosis (TSC) (191100, 613254)	Dominant	Development of multisystem nonmalignant tumors	Giant cell astrocytoma

Abbreviations used: ATM, ataxia telangiectasia; APC, adenomatous polyposis coli; CDKN2A, cyclin-dependent kinase inhibitor 2A; MLH1, MutL homolog 1, colon cancer, nonpolyposis type 2; MSH2, MutS protein homolog 2; MSH6, MutS protein homolog 6; OMIM, Online Mendelian Inheritance in Man; PMS2, postmeiotic segregation increased homolog 2; RB1, retinoblastoma transcriptional corepressor 1; TP53, tumor protein p53.

Wen/Weller Neuro-Oncol 22(8):1073-1113, 2020

Presentation and Diagnosis

Presentation: Usually acute progressive neurologic symptoms that evolve over days to a few weeks

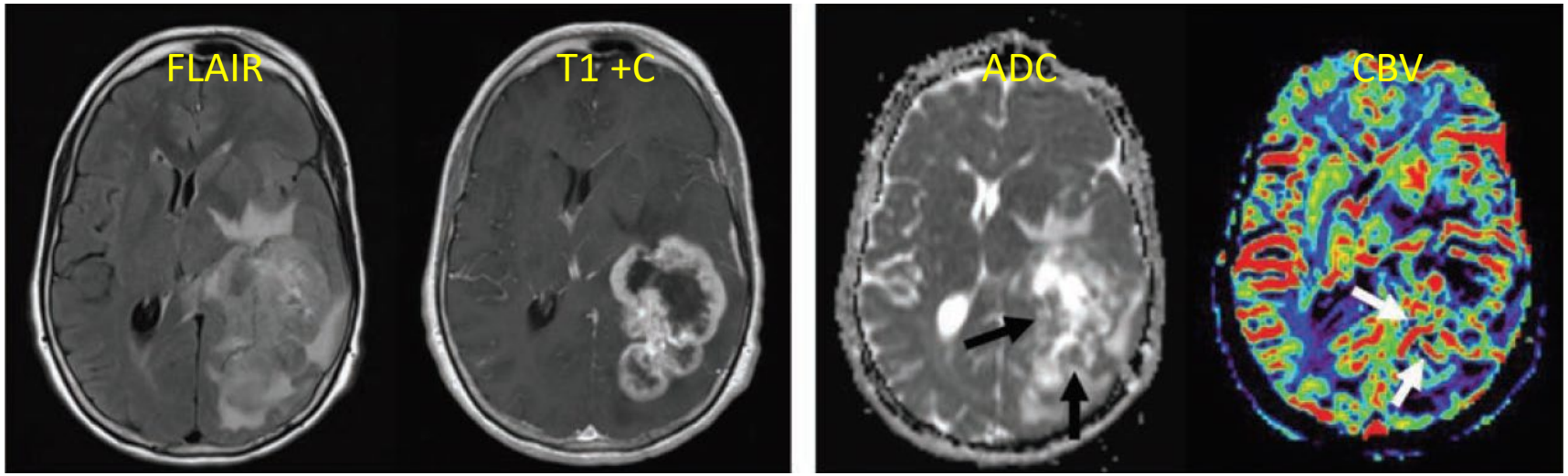
- Focal neurologic deficits: weakness, balance, speech, or vision changes
- Cognitive, memory, personality/behavioral changes
- Headaches, seizures

Work-up

- Magnetic resonance imaging (MRI) with contrast

Diagnosis

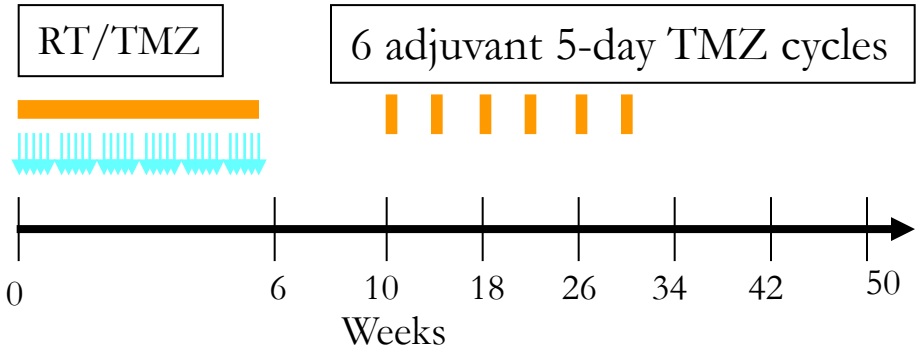
- Tumor sampling and study (biopsy or resection)
- *Detailed analysis of the blueprint of the genetic features of the tumor cells*

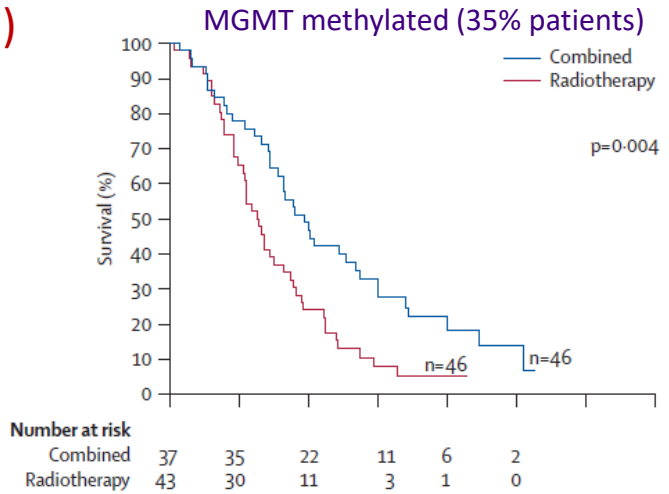


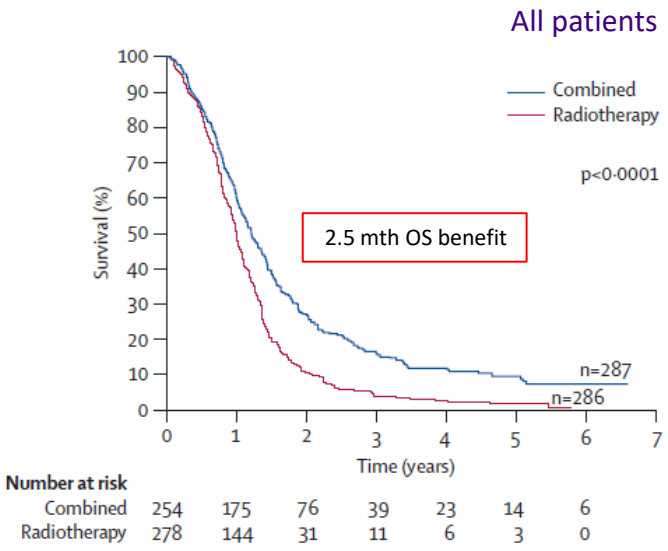
Glioblastoma Therapy: The Standard of Care was Defined in 2005 (21 years ago)

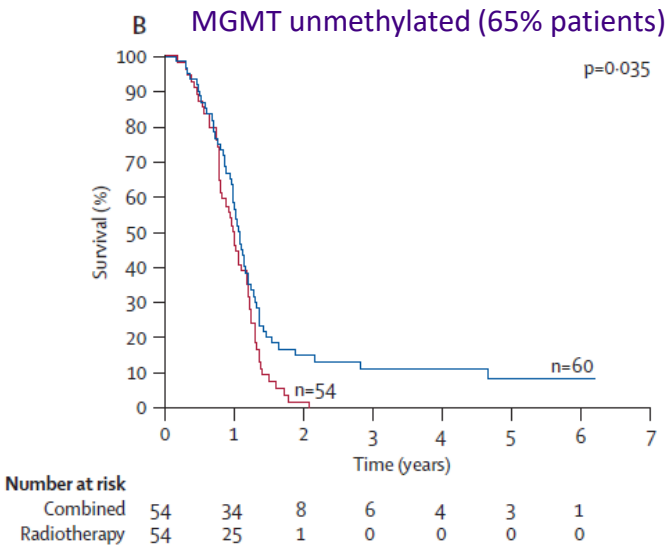
Step 1. Maximum safe resection (helpful but never curative)

Step 2. Radiation and temozolomide chemotherapy









Stupp et al. NEJM 352:987, 2005

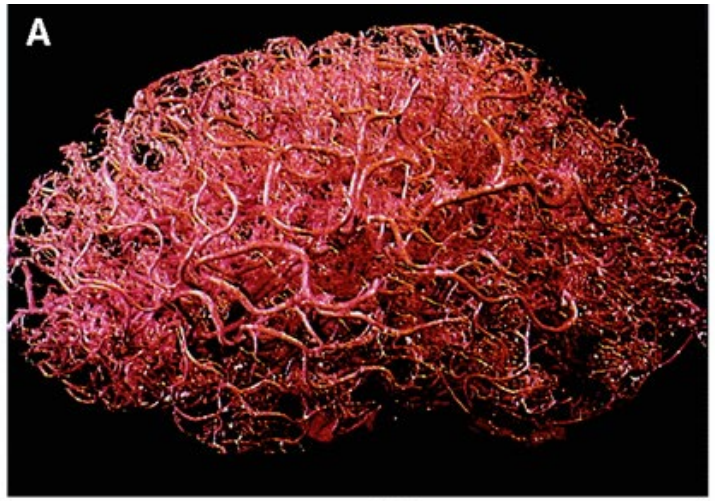
GBM: Negative Phase 3 Trials in Past 10 years

Agent	n	Population	Primary endpoint	Result
Dose dense TMZ (RTOG)	833	Newly dx	OS	Negative
Avastin (RTOG)	621	Newly dx	OS	Negative
Avastin (AvaGlio)	921	Newly dx	OS	Negative
Cilengitide (Centric)	545	Newly dx	OS	Negative
Tocagen	403	Recurrent	OS	Negative
ABT414 (Intellance)	640	Newly dx	OS	Negative
Nivolumab (MGMT unmth; CM498)	550	Newly dx	OS; PFS	Negative
Nivolumab (MGMT Meth; CM548)	693	Newly dx	OS; PFS	Negative
Nivolumab (CM143)	363	Recurrent	OS	Negative
ICT 107	414	Newly dx	OS	Suspended
Marizomib (EORTC)	750	Newly dx	OS	Negative
11 Trials	**6,733 patients**			

Why Do Drugs Fail in GBM?

1. Delivery
2. Delivery
3. Delivery

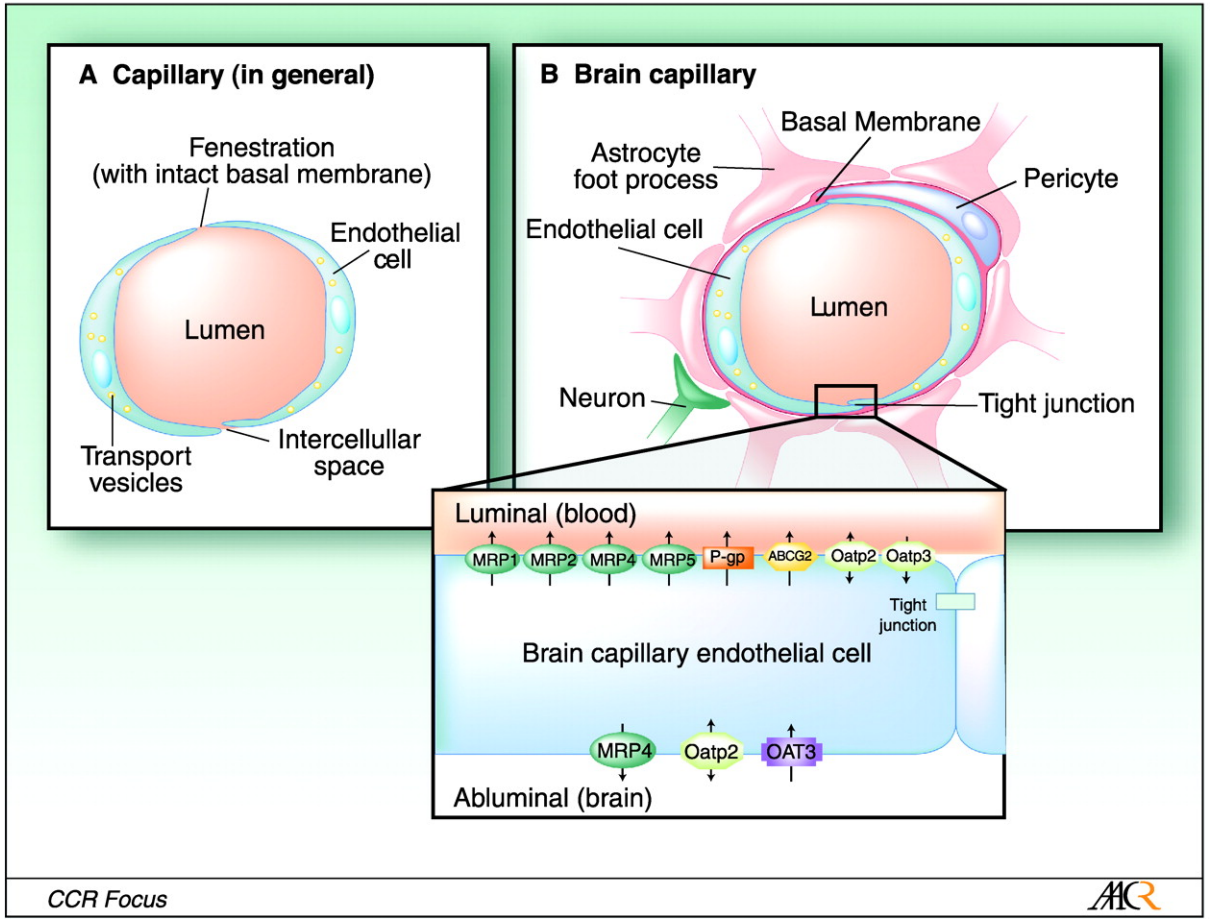
4. Redundancy
5. Heterogeneity
6. Resistance – intrinsic and acquired



Delivery: Blood Brain Barrier

Adult brain
- 400 miles of capillaries
- 12 m^2 surface area

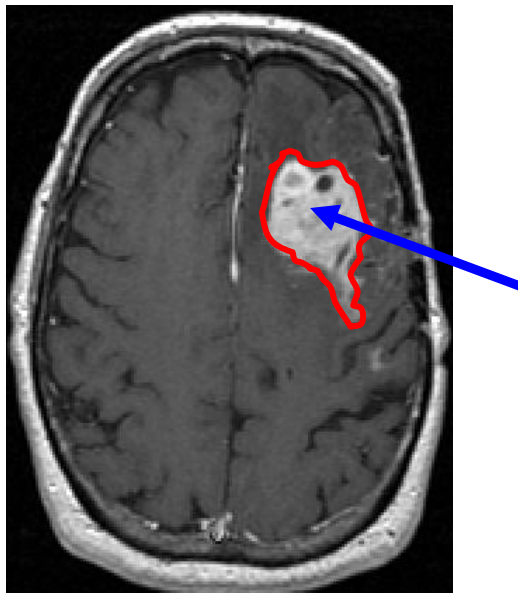
Disrupted in areas of contrast enhancement

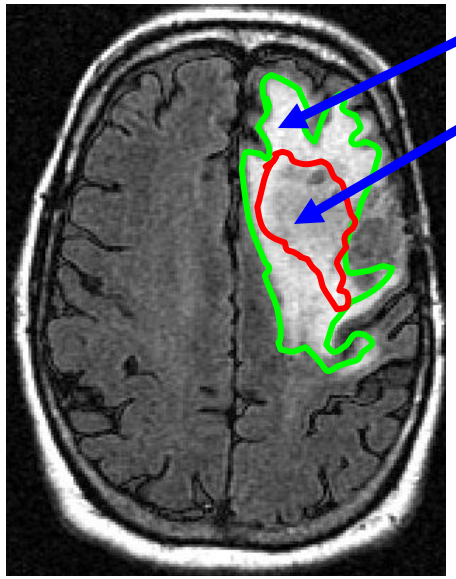


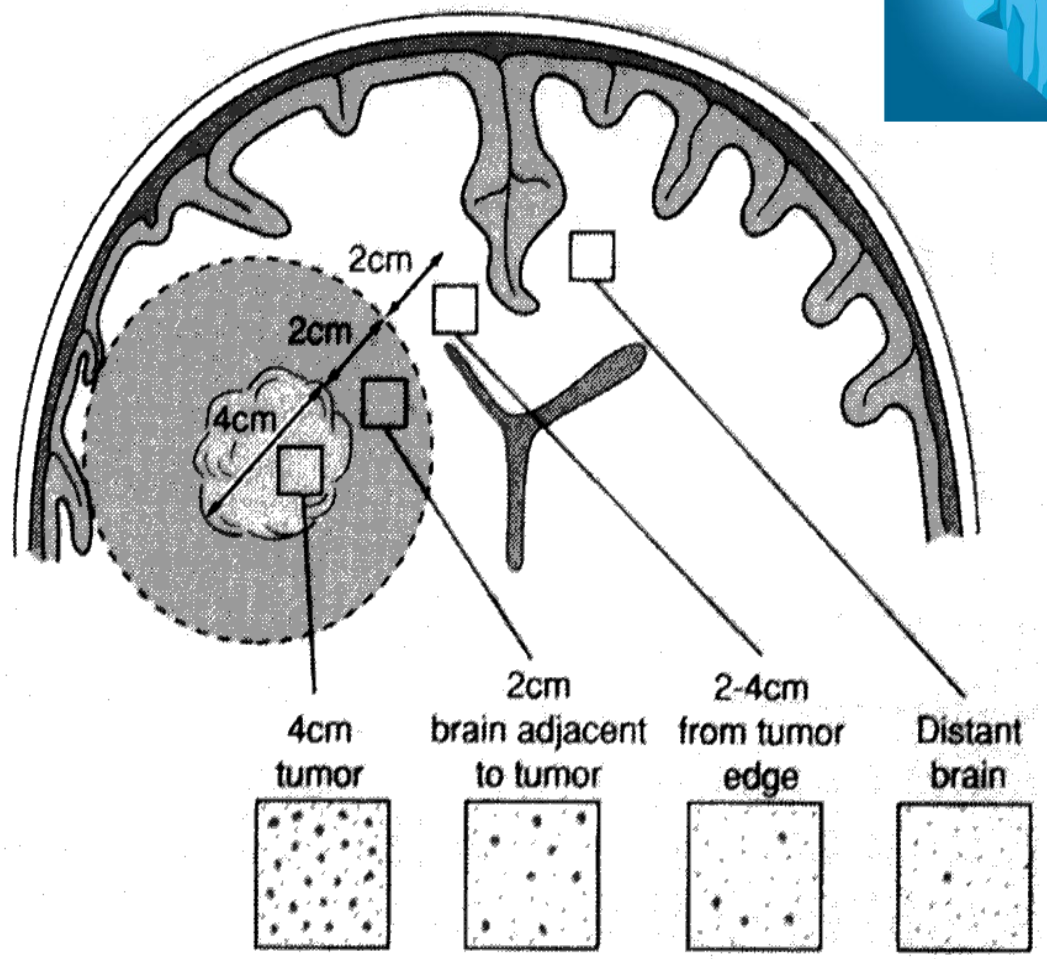
Excludes
- size, charge, lipophilicity
- efflux transporters

Ca Res 13:1663,2007

GBM: Infiltrates Extensively (does not metastasize!)







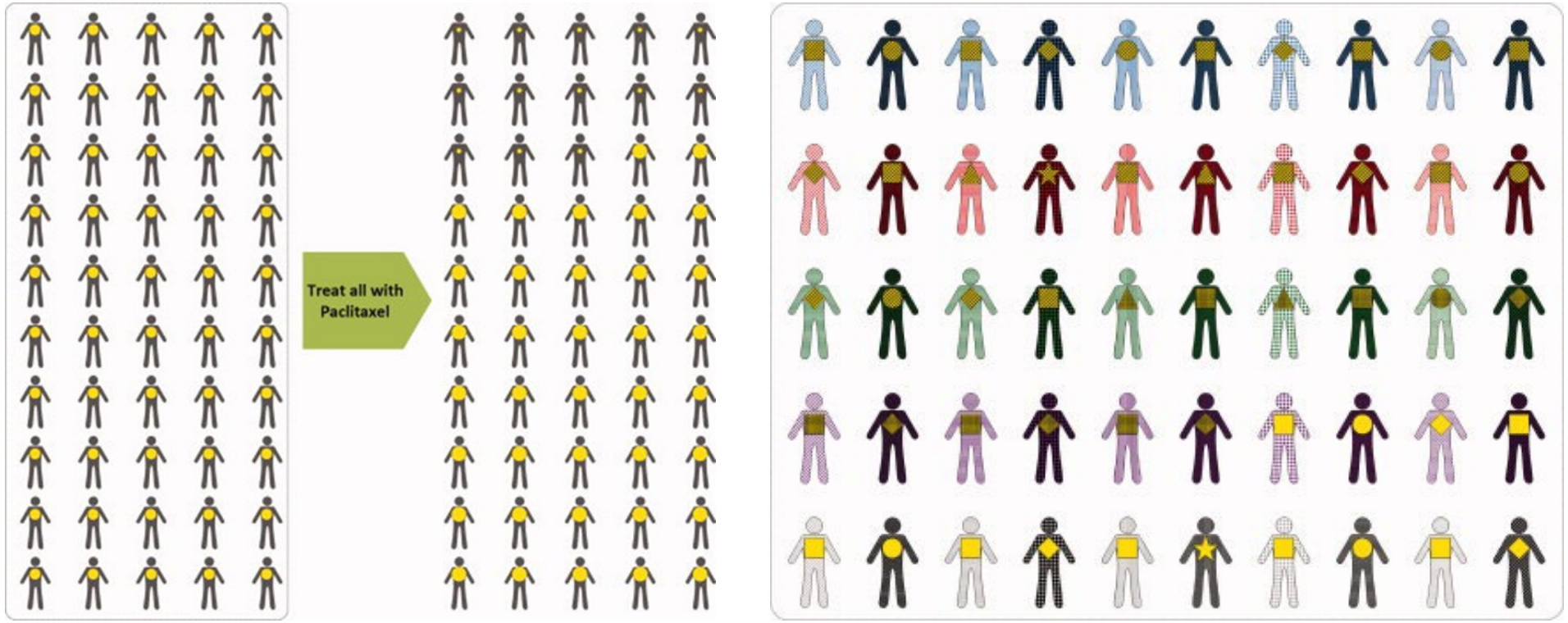
	4cm tumor	2cm brain adjacent to tumor	2-4cm from tumor edge	Distant brain
Ratio of tumor cells to total cells	1:1	1:10	1:100	1:1000
Percentage of tumor cell population	92%	6%	1.8%	0.2%

Why Do Drugs Fail in GBM?

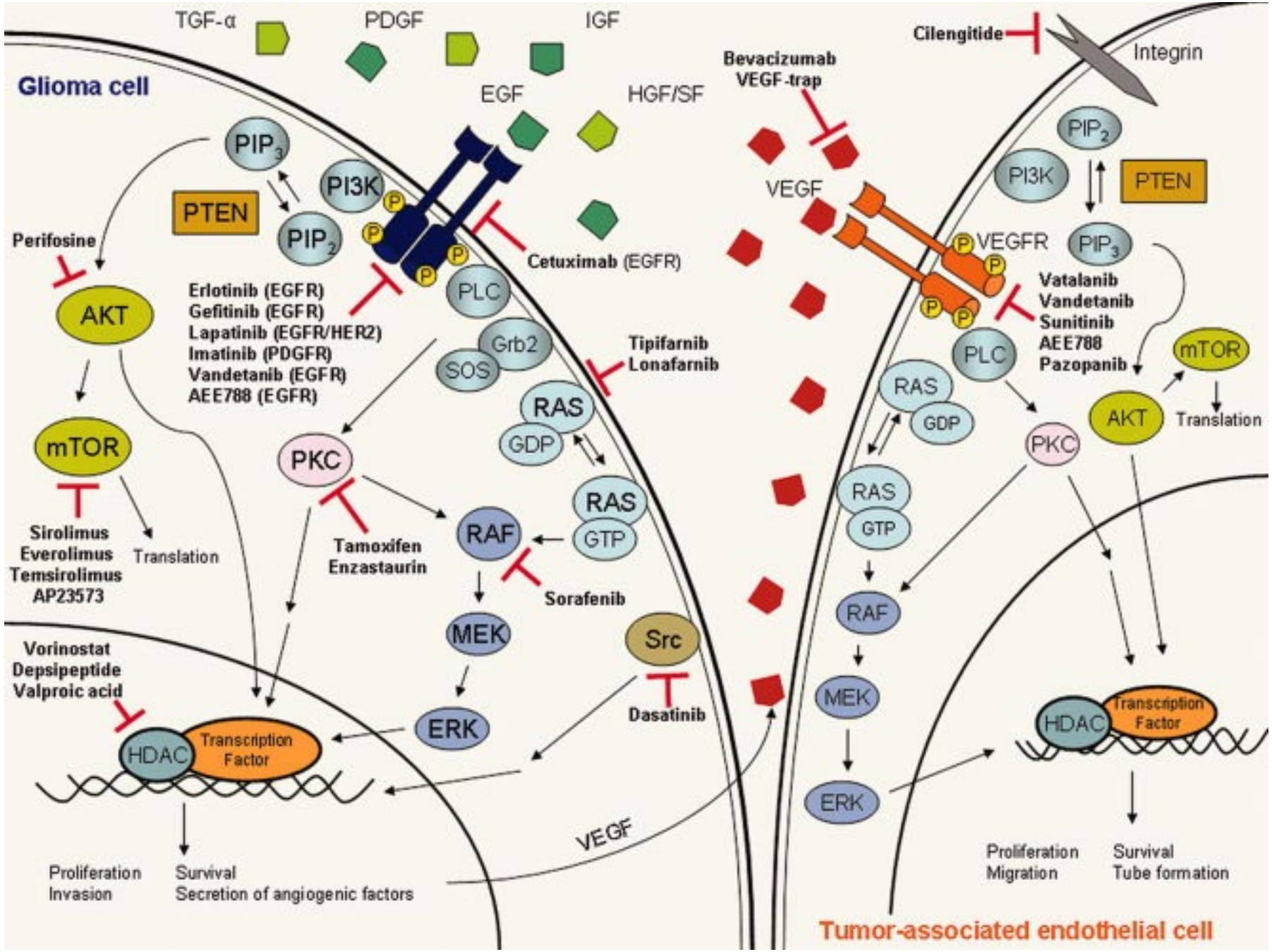
1. Delivery
2. Delivery
3. Delivery

4. Redundancy
5. Heterogeneity
6. Resistance – intrinsic and acquired

The Era of Personalized Oncology

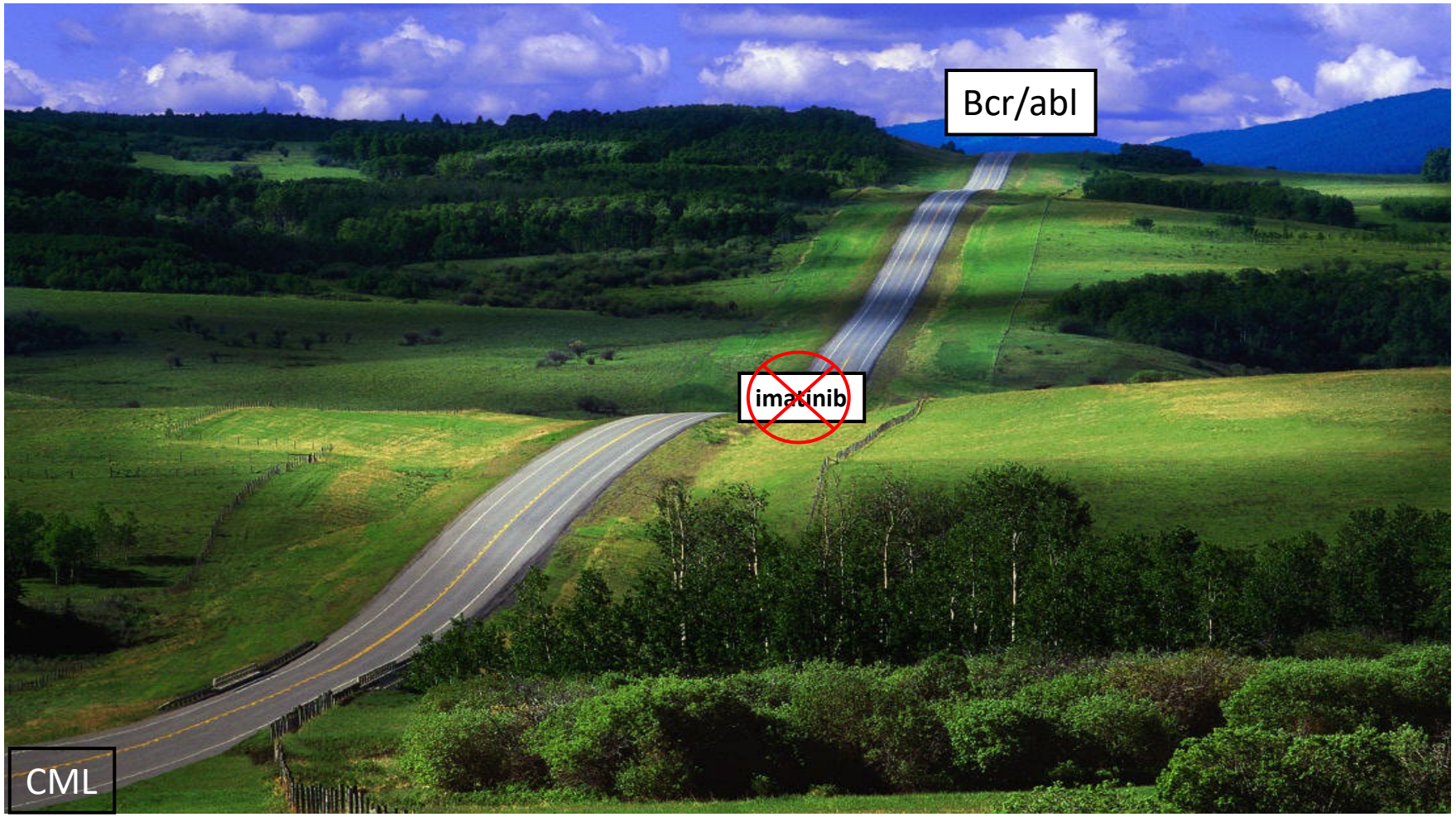


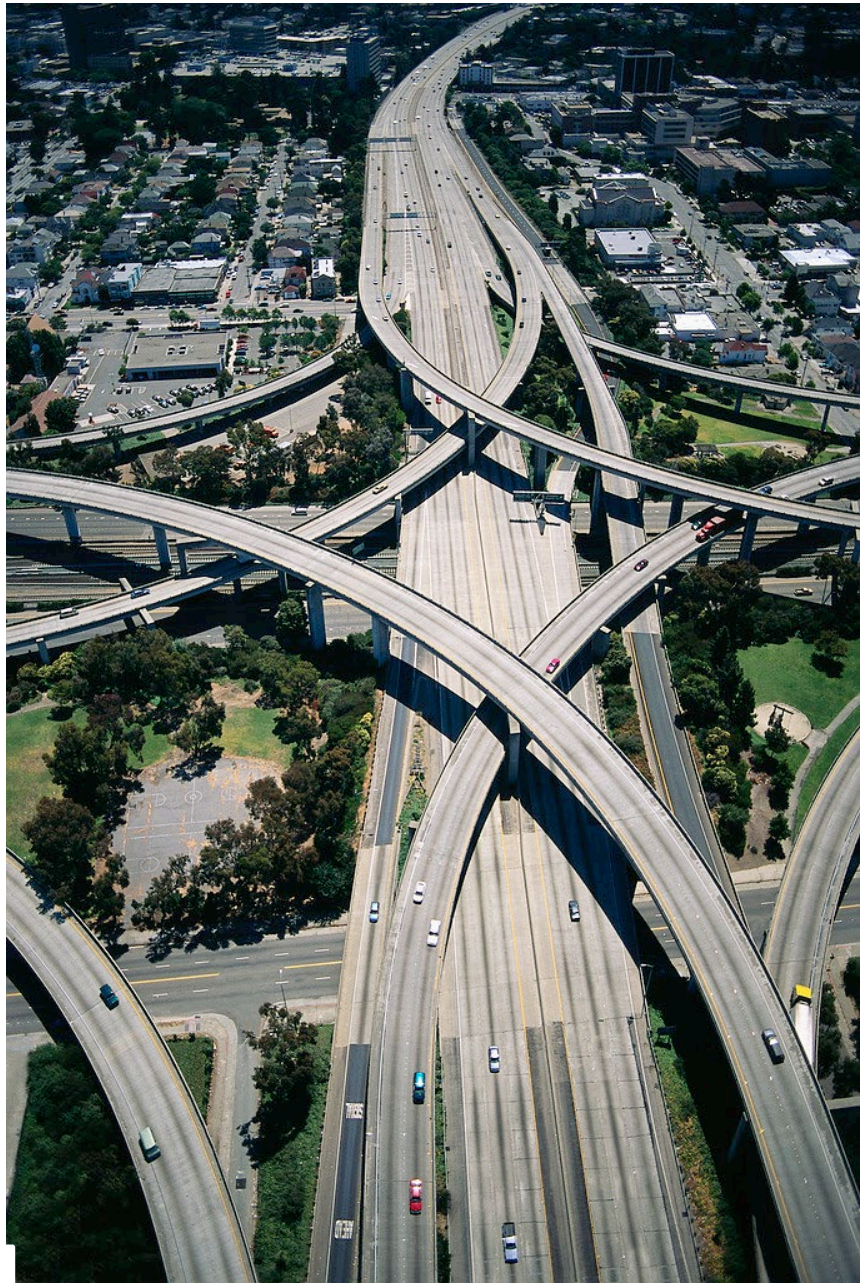
CA: A Cancer Journal for Clinicians
pages n/a-n/a, 27 OCT 2011 DOI: 10.3322/caac.20135



Sathornsumetee S, et al. *Cancer.* 2007;110:13-24

Chronic Myelogenous Leukemia (CML)





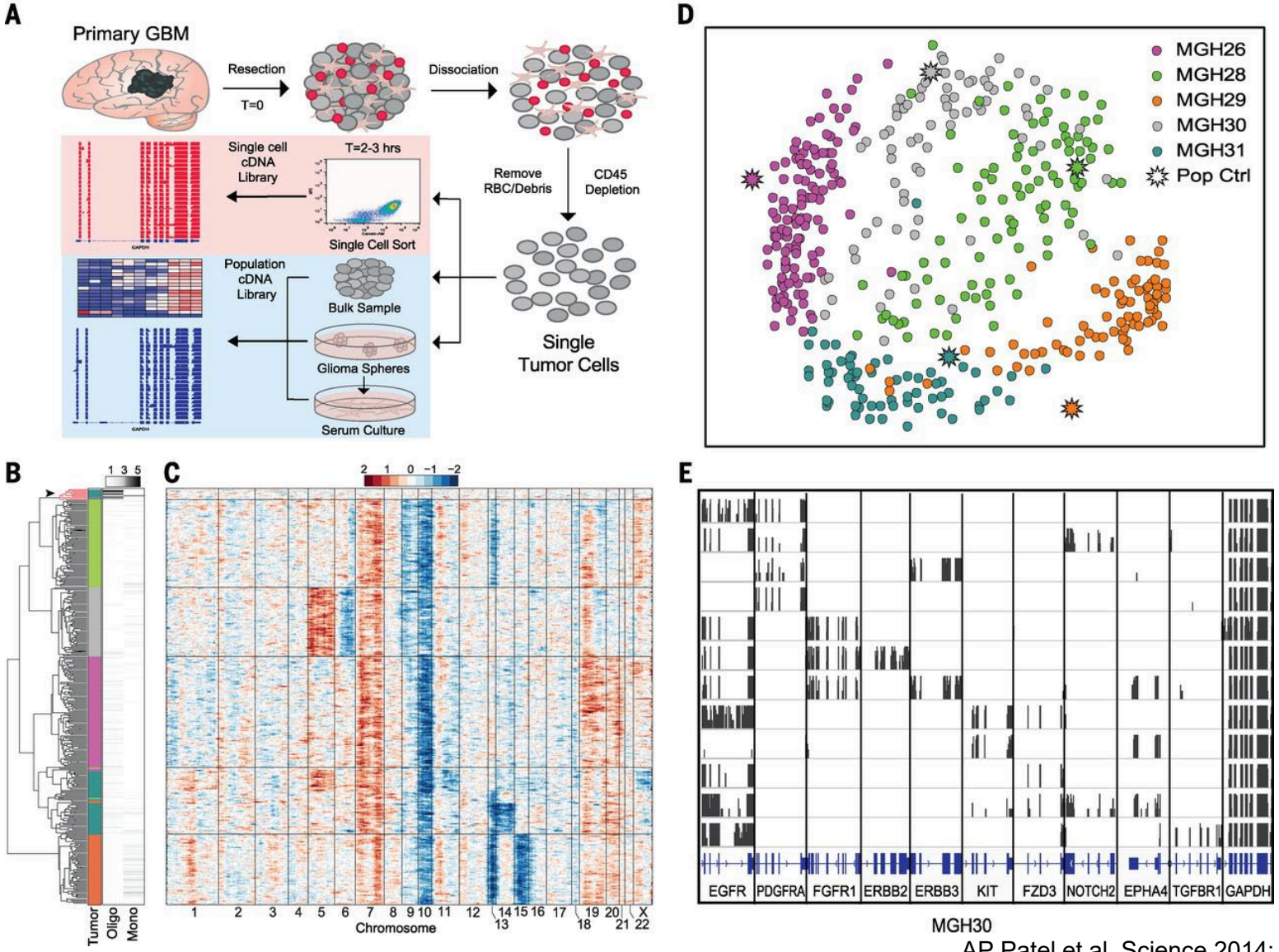
GBM: complexity of pathway signaling

Targeted Therapy 1st Generation Trials: Malignant Glioma

EGFR	Gefitinib	II	Rec GBM	53	9 SD >6 mo	Rich. *J Clin Oncol.* 2004
EGFR	Erlotinib	II	Rec MG	45	1 PR/6 SD; PFS: 8–12 wk	Raizer. ASCO, 2004. 1502
EGFR	Erlotinib	II	Rec GBM	24	5 PR/5 SD >6 mo	Vogelbaum. ASCO, 2004. 1558
EGFR	Erlotinib	II	Rec GBM	31	6 PR/5 SD	Prados. ASCO, 2003
EGFR	Erlotinib	II	Rec GBM	48	1 CR/1 PR/11 SD	Yung. ASCO, 2004. 1555
PDGF	Imatinib	II	Rec GBM	51	3 PR/5 SD >6 mo	Van den Bent. ASCO, 2004. 1501
PDGF	Imatinib	I/II	Rec GBM	95	2 PR/14 SD	Wen. SNO, 2004. TA 63
mTOR	CCI 779	I	Rec MG	12	4 SD	Chang. *Invest New Drugs. 2004*
mTOR	CCI 779	II	Rec GBM	31	No rad response	Galanis. ASCO, 2004. 1503
VEGF	PTK787	I	Rec GBM	47	2 PR/31 SD; PFS:12.1 wk	Conrad. ASCO, 2004. 1512
PKC-β2	LY317615	I	Rec GBM	28	5 PR	Fine. ASCO, 2004. 1511
αVβ3	Cilengitide	I	Rec MG	51	2 CR/3 PR/13 SD	Nabors. SNO, 2004. TA 39

MG=malignant glioma; SD=stable disease; PR=partial response; CR=complete response.

Glioblastoma: Intra-Tumoral Heterogeneity Fosters Tumor Evolution and Clonal Resistance



AP Patel et al. Science 2014; 344:1396-401

Tumor Evolution: A Central Obstacle to Curative Cancer Therapy

INTRA-TUMORAL HETEROGENEITY

+

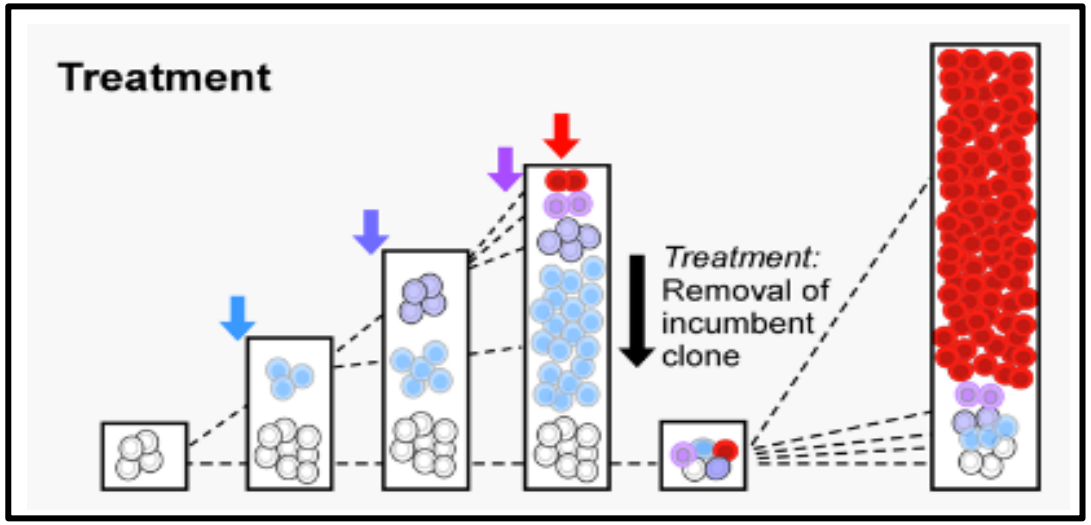
EFFECTIVE TREATMENT

▼

Cytotoxic agents

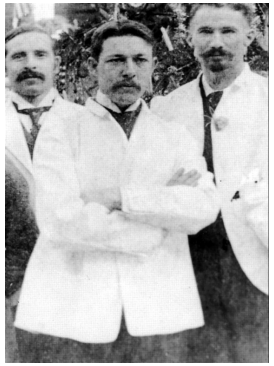
Novel targeted agents

TUMOR EVOLUTION = RESISTANCE



Wang NEJM 2011
Landau Cell 2013
Landau Cancer Cell 2014

The Odyssey of Immunotherapy's Transformation of Medical Oncology



William B. Coley, MD – 1893 "Coley's Toxins"

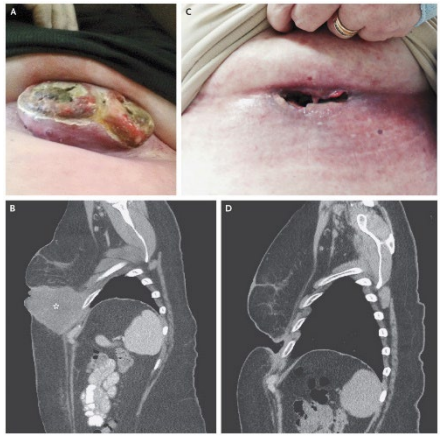
*"The Treatment of Malignant Tumors by Repeated Innoculations of Erysipelas: With a Report of Ten Original Cases." American J Medical Sciences **10**: 487–511.*

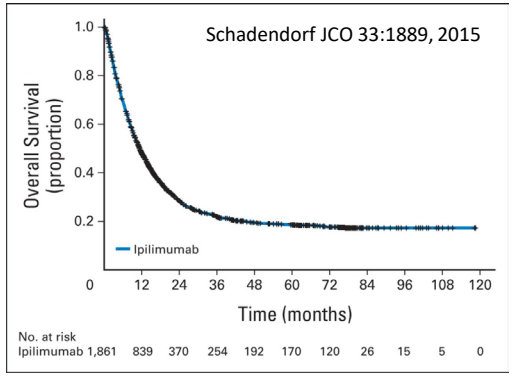
+125 years
(lack of clinical success)

Past 10 years

- **Vaccines**
- **Cellular Therapies (CAR T cells, TILs)**
- **Checkpoint inhibitors (CTLA-4, PD-1)**

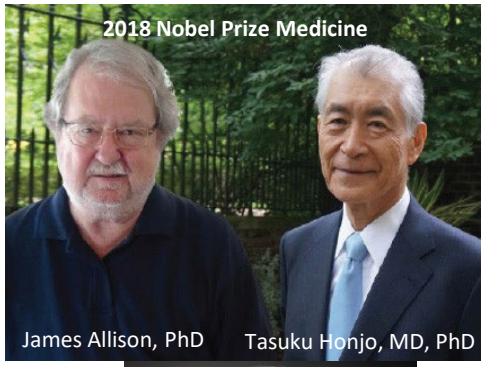
1 dose nivo/ipi
NEJM 2015





2026:

Approx 100 U.S. FDA approved cancer indications for immunotherapy drugs



Gordon J. Freeman, Ph.D.
2024 AACR/CRI Lloyd J. Old Award

Immunotherapy Transforming Medical Oncology but *NOT* Primary Brain Cancer

Generic Name	Brand Name	Agent Class	# FDA Approved Cancers	FDA Approved Cancers
Pembrolizumab	Keytruda	Checkpoint inhibitor	14	Melanoma, NSCLC, head and neck CA, Hodgkin's lymphoma, B cell lymphoma, urothelial CA, bladder CA, gastric CA, cervical CA, hepatocellular CA, merkel cell CA, renal cell CA, SCLC, esophageal CA, endometrial CA, squamous cell CA, , triple neg breast CA, MSI high CA, TMB high CA
Nivolumab	Opdivo	Checkpoint inhibitor	10	Melanoma, NSCLC, mesothelioma, renal cell CA, Hodgkin's lymphoma, head and neck CA, urothelial CA, colorectal CA, hepatocellular CA, SCLC, esophageal CA
Atezolizumab	Tecentriq	Checkpoint inhibitor	6	Bladder Ca, NSCLC, breast CA, SCLC, hepatocellular CA, melanoma
Nivolumab + ipilimumab	Opdivo + Yervoy	Checkpoint inhibitor	6	Mesothelioma; NSCLC; RCC; HCC; melanoma; CRC (MSI-H/dMMR)
Durvalumab	Imfinzi	Checkpoint inhibitor	3	Urothelial CA, NSCLC, SCLC, hepatocellular, biliary tract CA
Avelumab	Bavencio	Checkpoint inhibitor	3	Merkel cell CA, urothelial CA, RCC
Ipilimumab	Yervoy	Checkpoint inhibitor	3	Melanoma, renal cell CA, colorectal CA
Cemiplimab	Libtayo	Checkpoint inhibitor	3	Squamous cell CA; Basal cell CA; NSCLC
Axicabtagene ciloleucel	Yescarta	CAR T (CD19)	2	Large B cell lymphoma Follicular lymphoma
Tisagenlecleucel	Kymriah	CAR T (CD19)	2	Large C cell lymphoma Pediatric and adult B-ALL
Brexucabtagene autoleucel	Tecartus	CAR T (CD 19)	2	Mantle cell lymphoma Adult B-ALL
Lisocabtagene maraleucel	Breyanzi	CAR T (CD 19)	1	Large B cell lymphoma
Idecabtagene vicleucel	Abecma	CAR T (BCMA)	1	Multiple myeloma
Ciltaceabtagene autoleucel	Carvykti	CAR T (BCMA)	1	Multiple myeloma
Sipuleucel-T	Provenge	Vaccine	1	Prostate CA
Bacille-Calmette-Guerin	BCG	Vaccine	1	Bladder CA
Imlygic	T-VEC	Oncolytic virus	1	Melanoma

Approximately 100 US FDA Approved Oncology Indications for Immunotherapy Agents

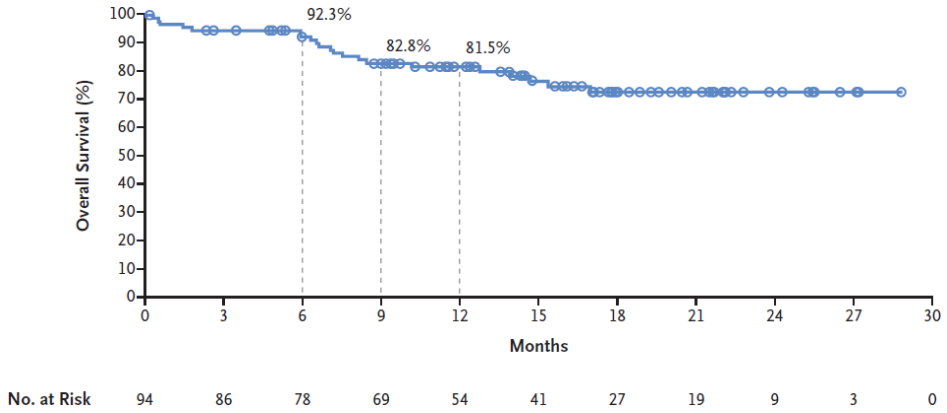
Negative Phase 3 Trials: Immunotherapy for Glioblastoma

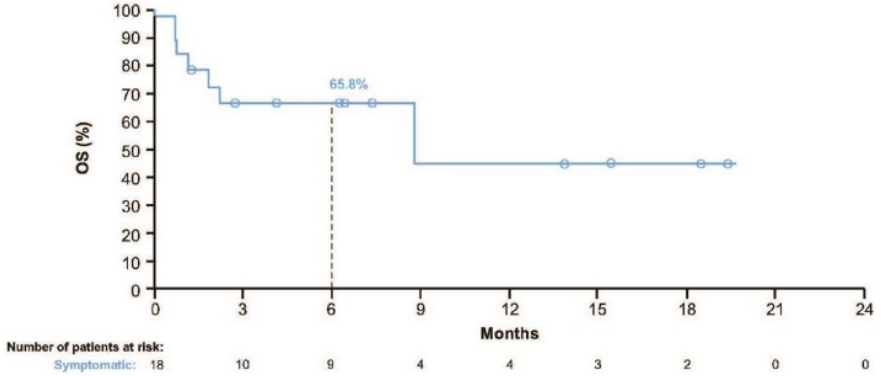
Agent	n	Study Population	Primary endpoint	Result
Nivolumab (CM 143)	369	Recurrent	OS	Negative
Nivolumab (CM 498)	550	Newly diagnosed; MGMT unmethylated	OS; PFS	Negative
Nivolumab (CM 548)	693	Newly diagnosed; MGMT methylated	OS; PFS	Negative
Rindopepimut (ACT IV)	745	Newly diagnosed; EGFRvIII+	OS	Negative
ICT 107	414	Newly diagnosed	OS	Suspended
Tocagen	403	Recurrent	OS	Negative
TOTAL	**3,174**			

Immune Checkpoint Therapy: Activity in CNS Metastases

CM 204: Nivolumab + Ipilimumab for CNS Melanoma Metastases

	Asymptomatic Patients	Symptomatic Patients
N	101	18
ORR	58%	22% (4/18)
OS-6	92.3%	65.8%
Median OS	Not reached	8.7 months
Citation	Tawbi NEJM 2018	Tawbi Neuro-Oncol *2021*





Why Does Immunotherapy Fail in GBM?

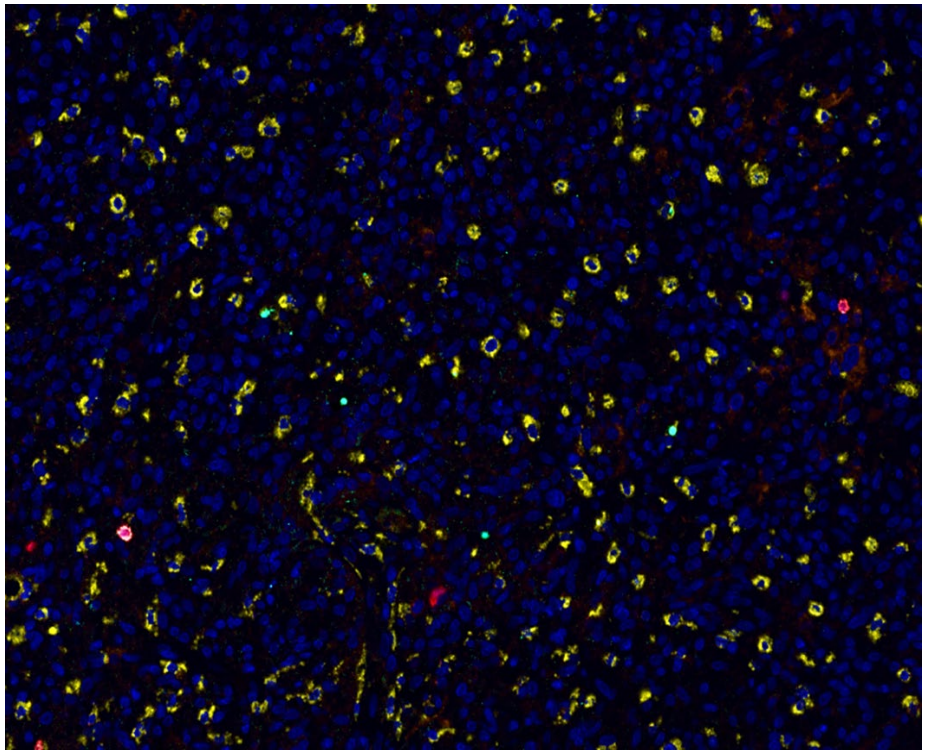
The Tumor Microenvironment is Programmed to Block the Immune System

GBM tumor microenvironment: A “perfect storm” of multiple complementary mechanisms that promote tumor growth and block effector immune cell infiltration and activation.

- Multiple suppressive molecules/cytokines actively secreted by GBM tumors

- Overwhelming dominance of suppressive myeloid cells

- Hijacks normal astrocyte and neuron function



Courtesy of A. Heimberger, MD, PhD

Sebastian Junger

How Do We Raise the Bar for GBM Outcomes?

1. Need for innovative drugs/therapeutic approaches
   A. Need to move beyond traditional cytotoxic therapies

2. Does the drug get into the brain and exert a meaningful biologic effect?
   A. Early investment in small studies but detailed analyses
   B. Integrated biocorrelative analyses

3. Combination approaches are key: Complementary mechanisms of action
   A. Integrate with standard of care radiation therapy and temozolomide chemotherapy for additive/synergistic benefit
   B. Harder to develop acquired resistance

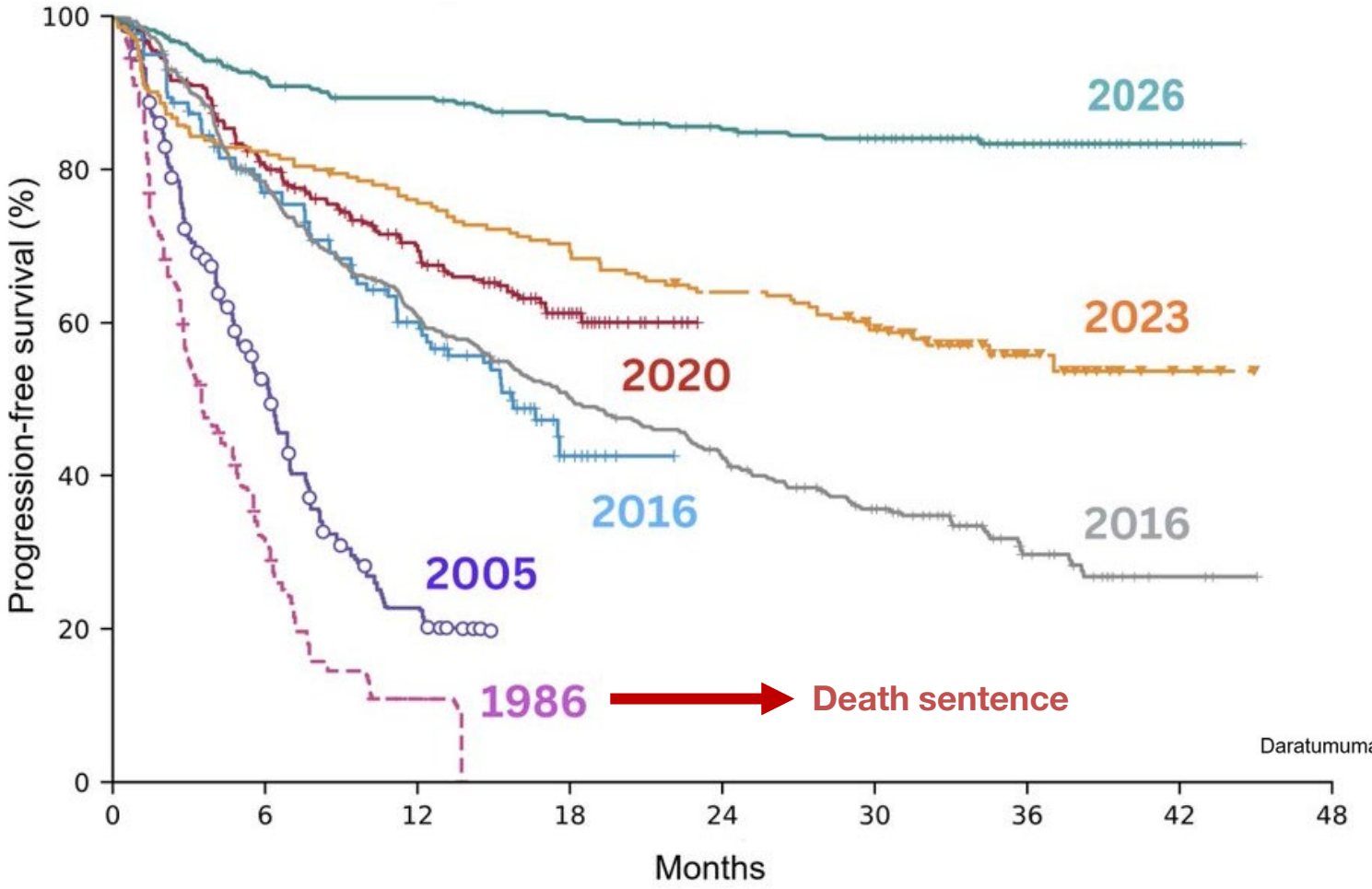
4. Evaluate the drug's effect on the GBM tumor microenvironment.

INB-200 & 400

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

Myeloma Was Once a Death Sentence. Now It's Manageable.

GBM is where myeloma was in 2005; We are working to “crack” the biology



- Myeloma now an estimated ~$30B annual market

Teclistamab-daratumumab
Cilta-Cel
Carfilzomib + dexamethasone + daratumumab
Daratumumab-dexamethasone + pomalidomide/bortezomib
Carfilzomib + dexamethasone
Bortezomib
High-dose dexamethasone

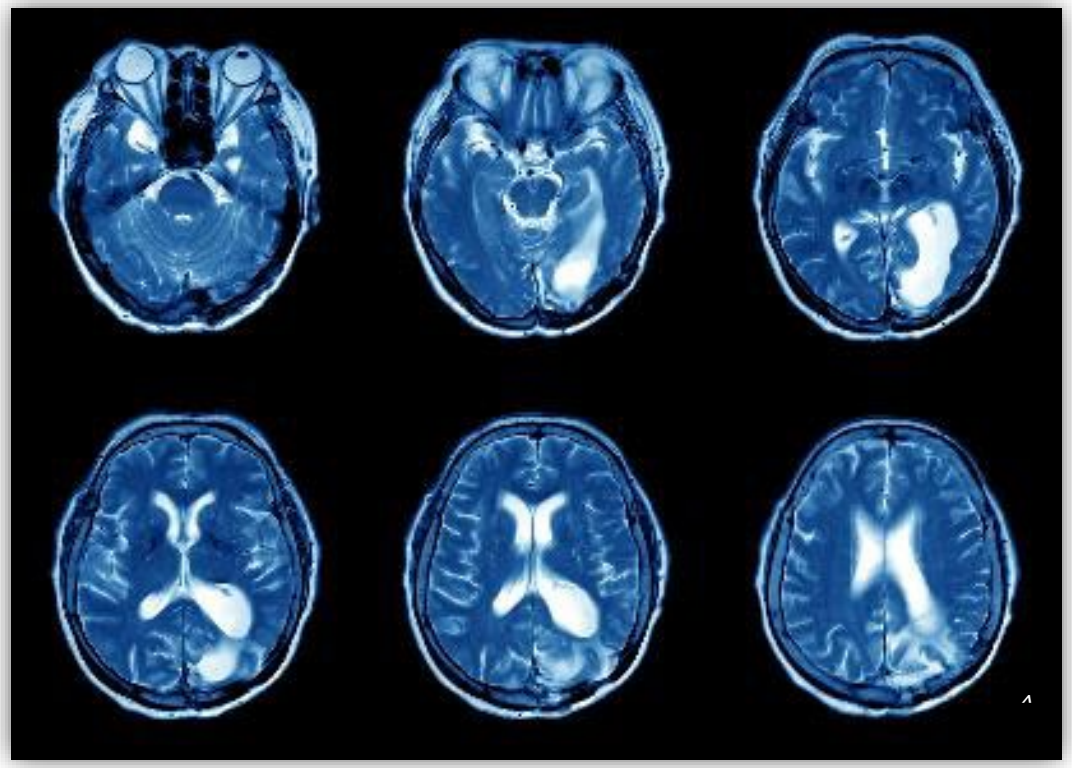
Sources: https://x.com/DrSamuelBHume/status/2040100943023227188?s=20 Multiple Myeloma Market Climbs to USD 49.89 Billion by 2034

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

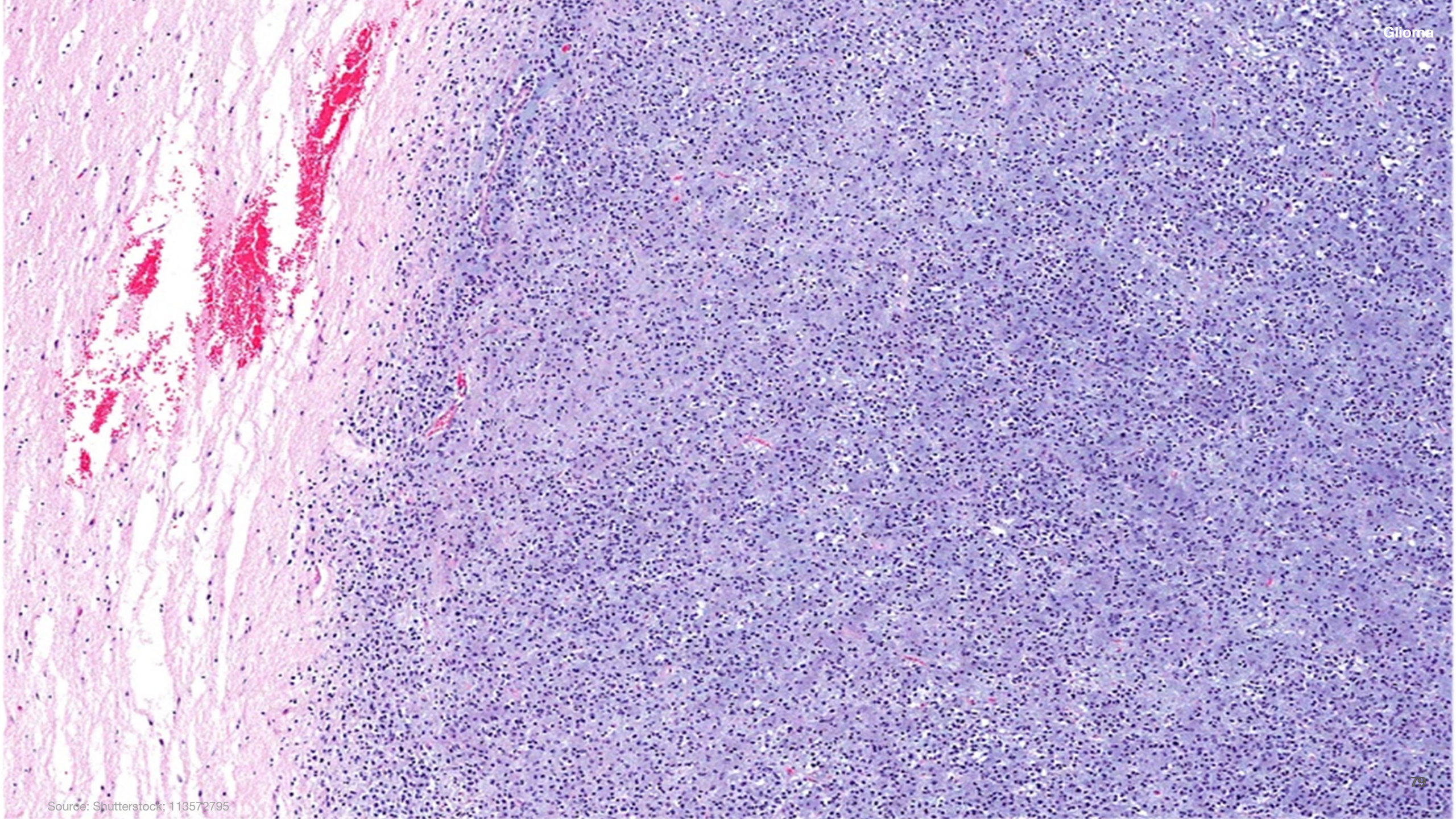
Forced expression of NKG2D-ligands allows γδ T cells to addresses heterogeneity

Simpler with no additional lymphodepletion

Standard-of-care (Temodar) already does it

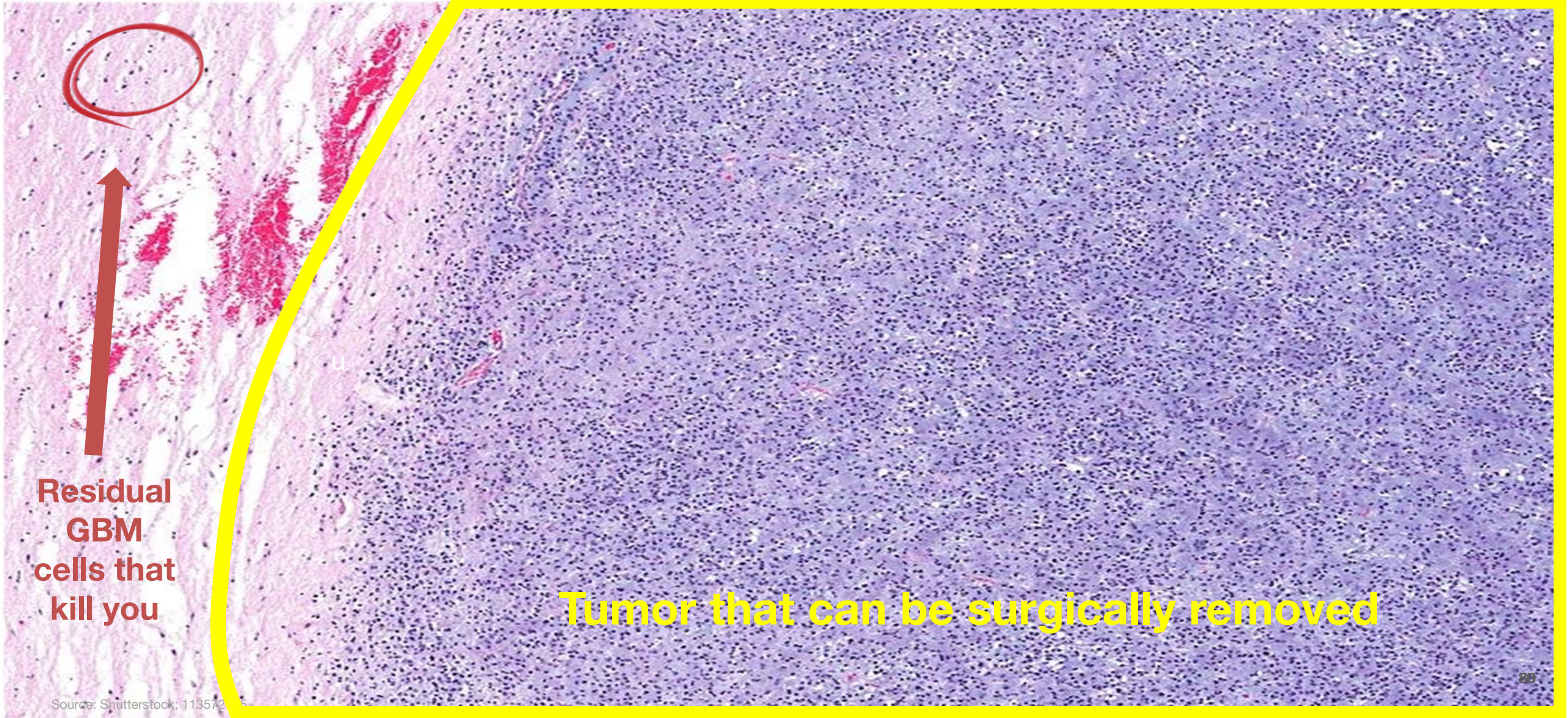
Immune-privileged

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



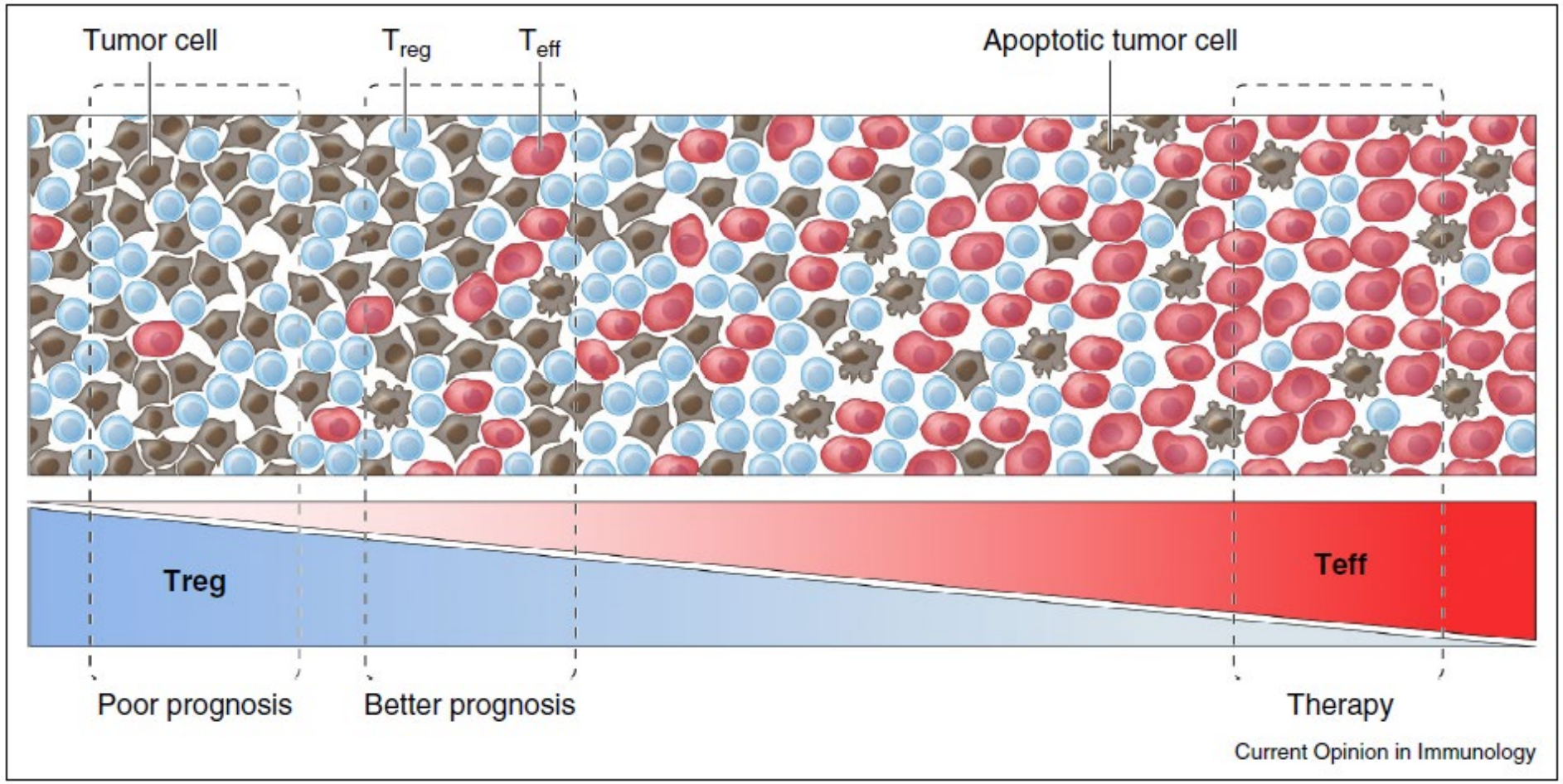
Source: Shutterstock; 113572795

The Problem



Source: Shutterstock; 113572

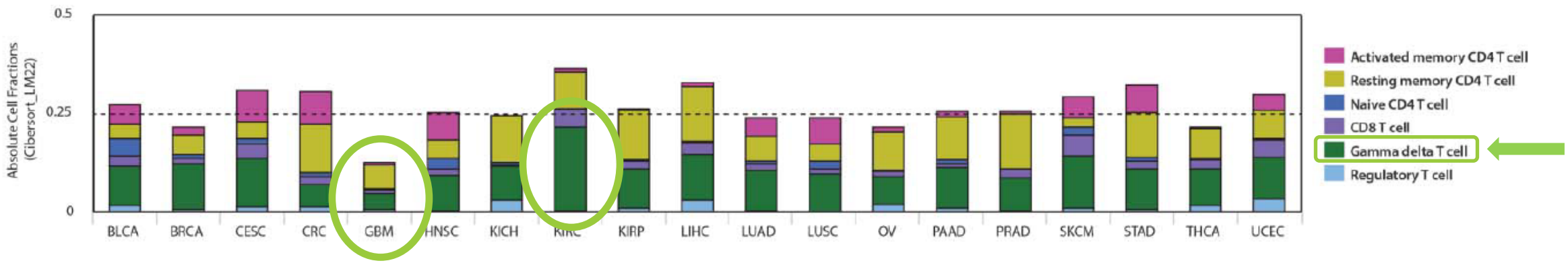
Shifting the Effector-to-Target Ratio is Key to Improving Outcomes



Source: Roychoudhuri et al. Curr Opin Imm, 2015, 33:101–111

Glioblastoma Demonstrates Low Immune Infiltration

High levels of TILs including γδ T cells predict positive responses to immune checkpoint inhibitors

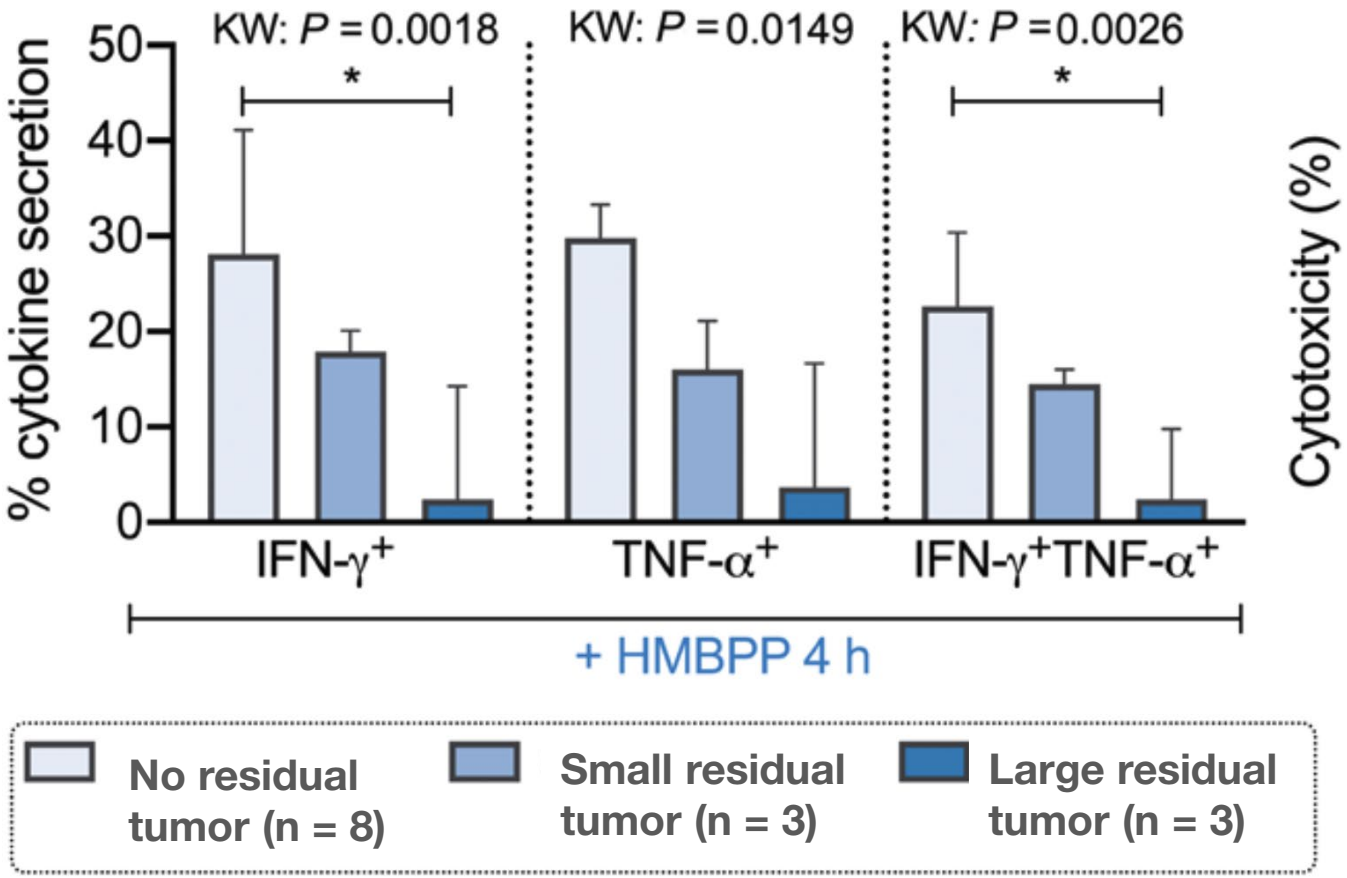


Increasing the number of killer T cells into Tumor is critical

Source: Garg et al. Oncoimmunology, Vol 6, No. 4, 2017

Less Tumor = Stronger Immune Response

Contrary to conventional dogma - Immune cells fight hardest when tumor burden is lowest, exactly when IN8bio delivers therapy

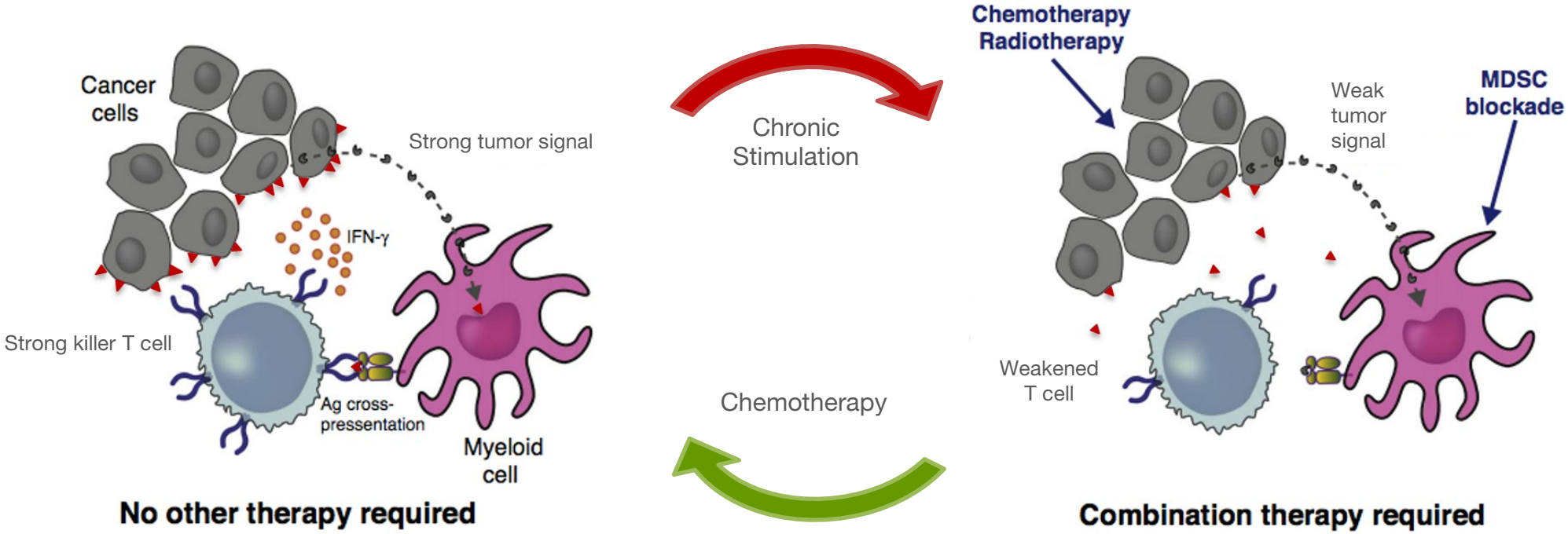


→ taller bars = stronger immune response

Source: Foord et al., Sci. Transl. Med. 13, eabb0192 (2021) - * = statistically significant difference

Tumors Evolve to Escape Your Immune System

Single therapies train tumors to resist elimination; attacking from multiple angles prevents escape



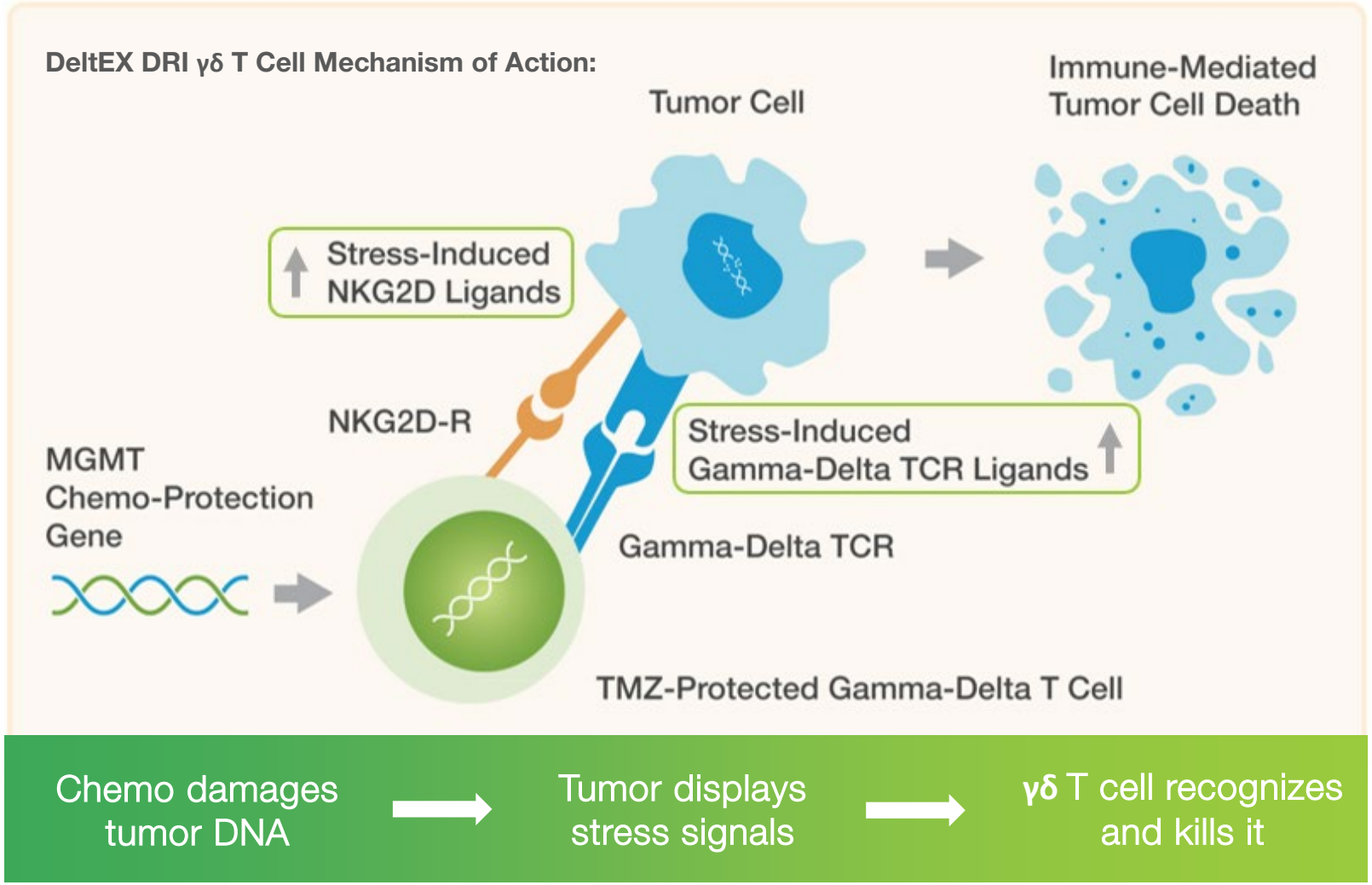
Chemotherapy resensitizes tumors to the immune response

Source: Adapted from Arina et al. Curr Opin Imm, 33 2015

γδ T cells are Powerful Killing Cells for Immunotherapy

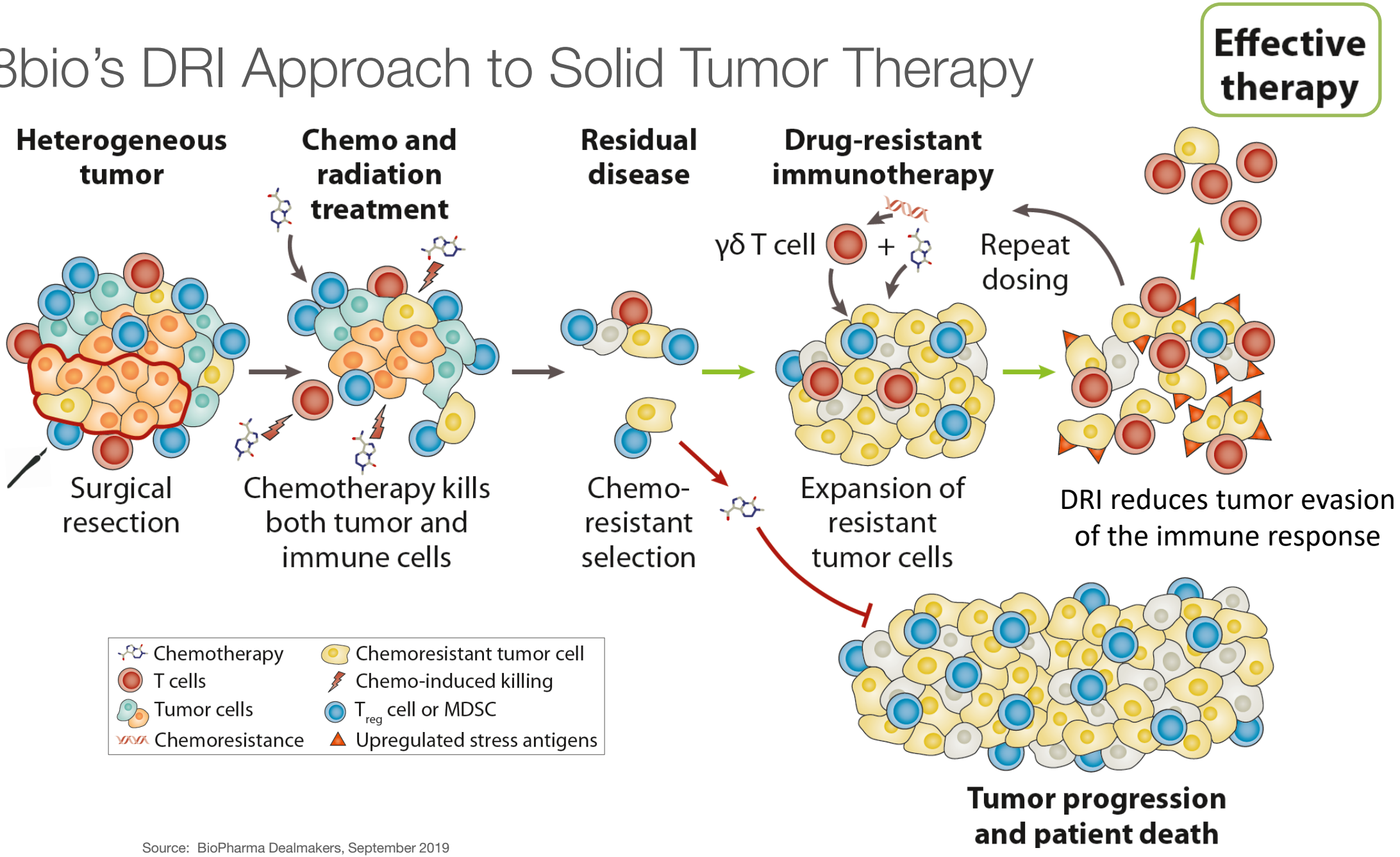
γδ T Cells Find and Kill Tumors That Hide from Everything Else

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



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

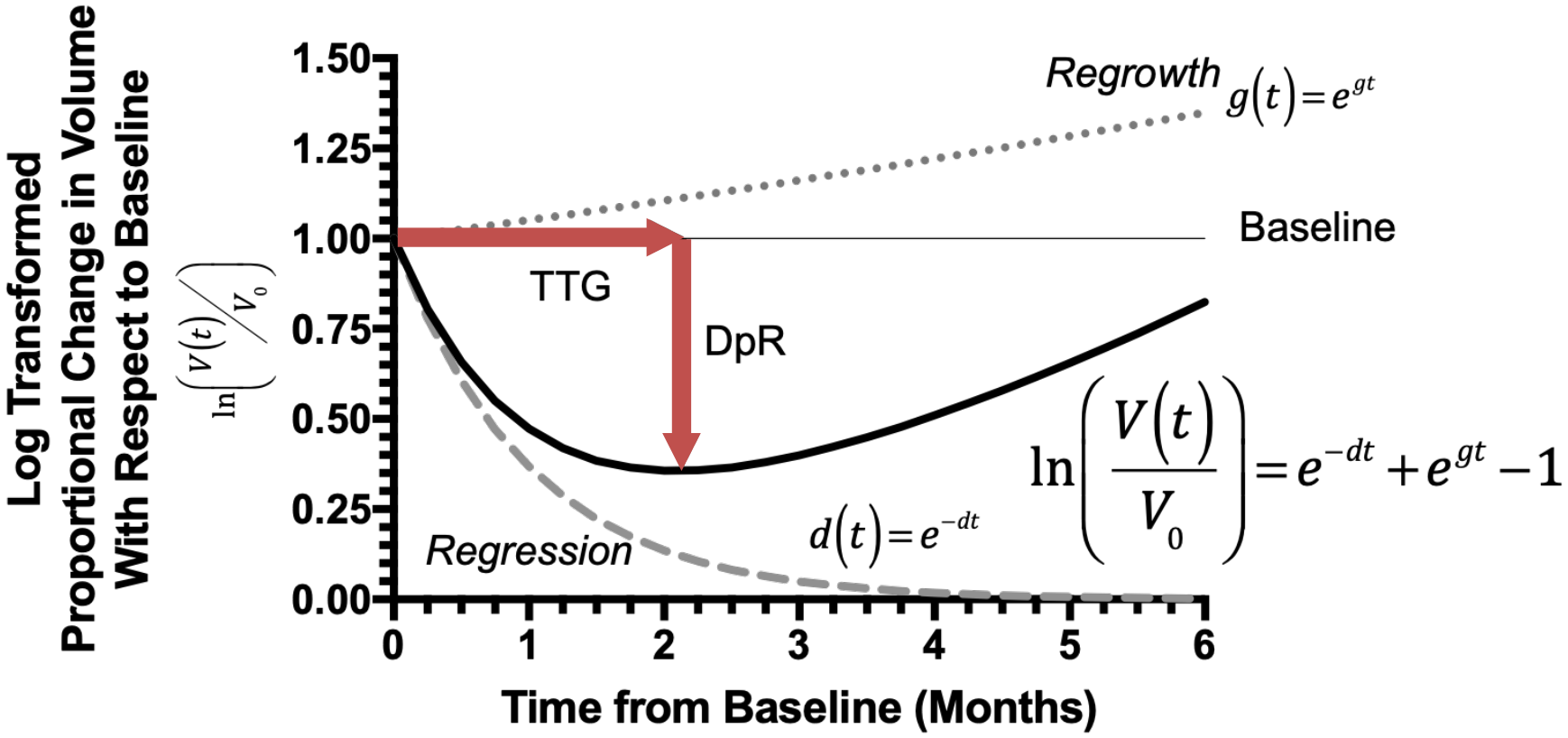
IN8bio's DRI Approach to Solid Tumor Therapy



Source: BioPharma Dealmakers, September 2019

Two Numbers Predict GBM Survival Time

Depth of Response and Time to Regrowth – IN8bio's approach specifically targets both



"d" = Tumor Regression Rate
"g" = Tumor Regrowth Rate
"TTG" = Time to Tumor Regrowth
"DpR" = Depth of Response


Depth of Response (DpR) and Time to Tumor Regrowth (TTG) Predicts Survival in Recurrent Glioblastoma Treated with Anti-VEGF Therapy

Benjamin M. Ellingson, Ph.D.
Professor and Director of MRI Research
Director, UCLA Brain Tumor Imaging Laboratory (BTIL)
Depts. of Radiological Sciences, Psychiatry, and Neurosurgery
David Geffen School of Medicine at UCLA

Akifumi Hagiwara, MD, PhD[1,2], Connor Morris, BS[1,2], Nicholas S. Cho, BS[1,2], Sonoko Oshima, MD, PhD[1,2], Francesco Sanvito, MD[1,2], Talia C. Oughourlian, BS[1,2], Catalina Raymond, MS[1,2], Lauren I. Abrey, MD[3], Jaeap Garcia, PhD[3], Dana T. Aftab, PhD[3], Colin Hessel, MS[3], Tamar Rachmilewitz Minei, MD[3], Shifra Fain Shmueli, MBA[3], David A. Nathanson, Ph.D.[3,4], Patrick Y. Wen, MD[3,5], and Timothy F. Cloughesy, MD[3,5]

ASCO 2023
Abstract #420

UCLA Health Radiology
UCLA Brain Tumor Imaging Laboratory

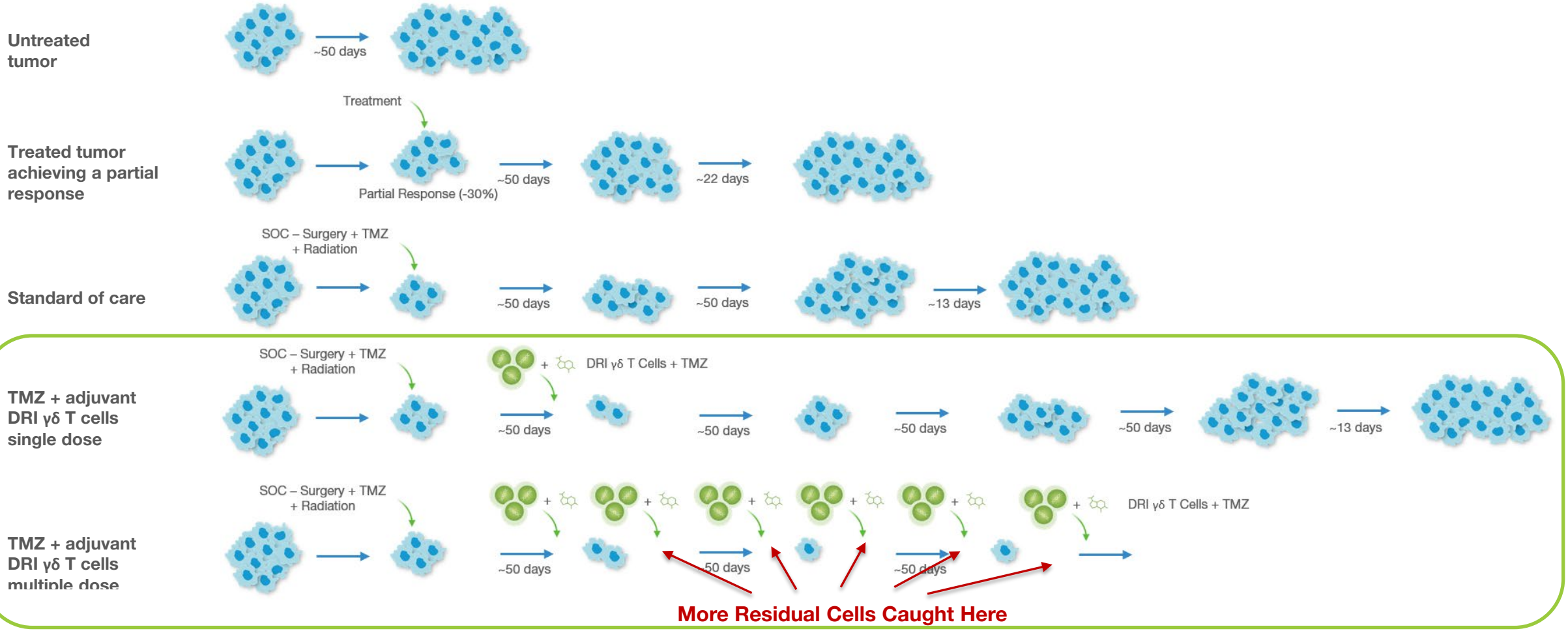
David Geffen School of Medicine

Wilkerson et al., Lancet Oncol 2017;18(1):143-54.
Stein et al., Clin Cancer Res 2012;18(8):2374-81.
Maitland, Clin Cancer Res 2020;26(24):6464-74

Multiple Opportunities to Eliminate More GBM Cells

A single dose buys a little time; multiple doses keep catching resistant cells before they take over

Hypothetical tumor cell dynamics to various treatment regimens and combinations



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

Data to be Updated at ASCO
June 1, 2026

Poster 442 (Abstract 552638)

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):

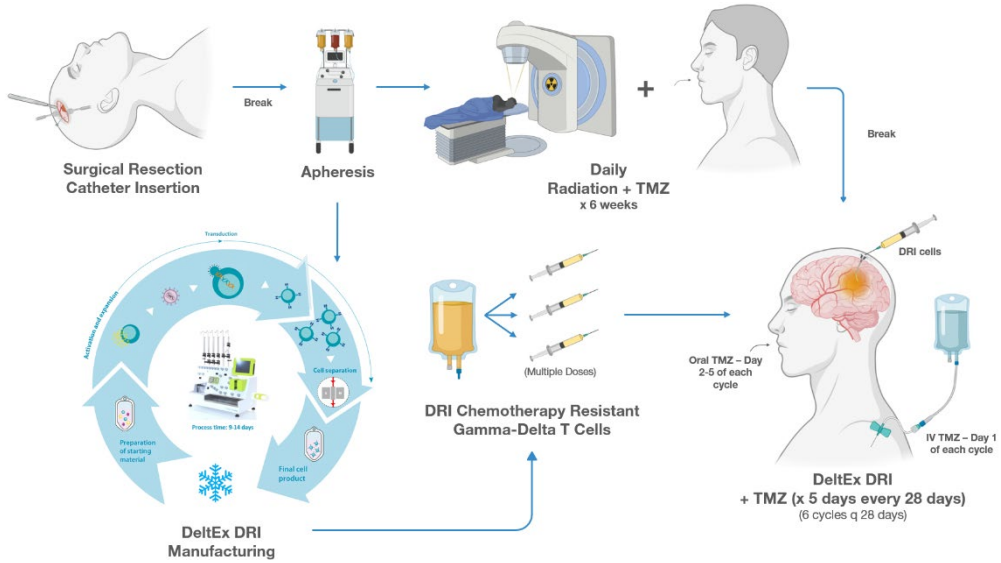
DL1: N = 3 (up to 6) patients, single dose of 1 x 10^7 cells on C1D1
DL2: N = 3 (up to 6) patients, three doses of 1 x 10^7 cells, one dose every 28 D1 of C1-C3
DL3: N = 3 (up to 6) patients, six doses of 1 x 10^7 cells, one dose every 28 days on D1 of C1-C6

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

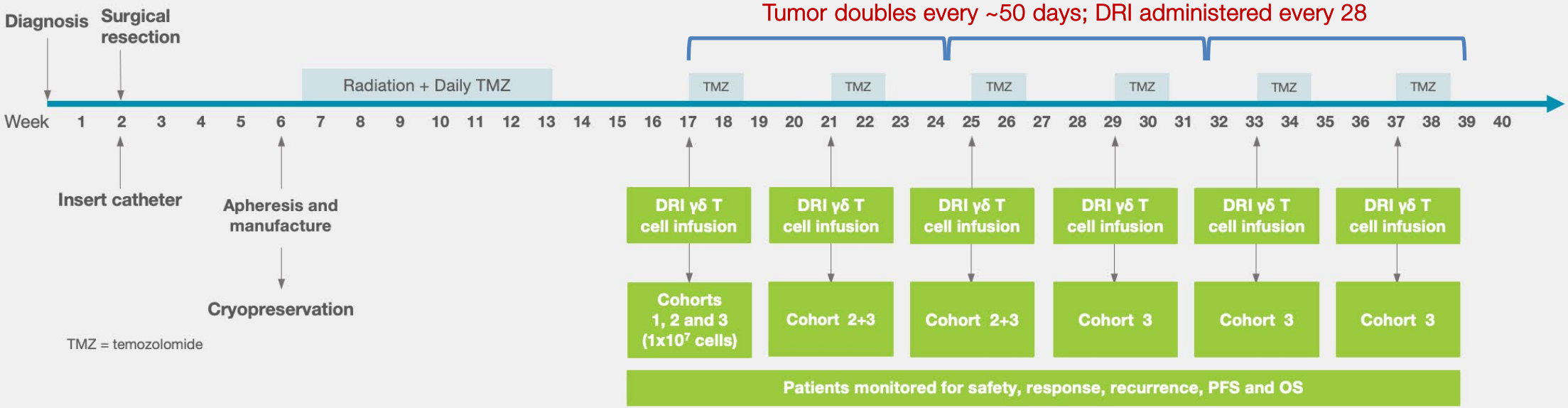
Source: IN8bio, image created with biorender.com

Repeated Doses to Intercept Residual Cells Every 28 Days

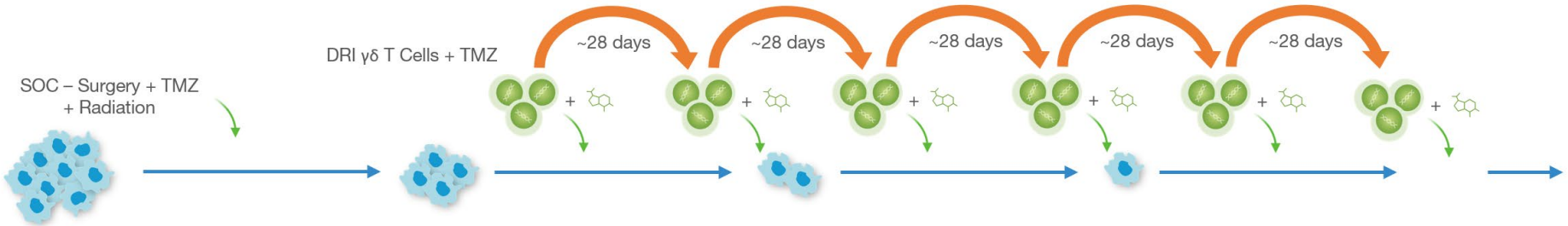
Timed to outpace tumor regrowth, each infusion catches residual cells as they multiply to maintain remission

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

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%	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

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%

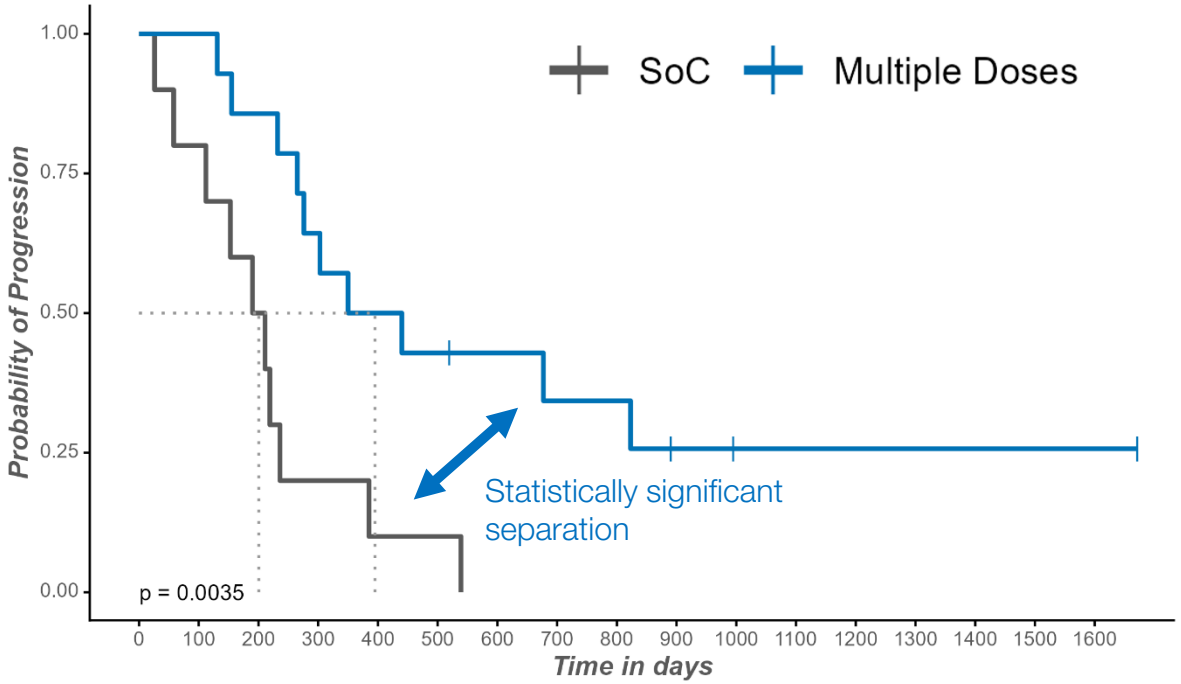
+ = median OS not yet reached

Note: As of December 31, 2025; *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

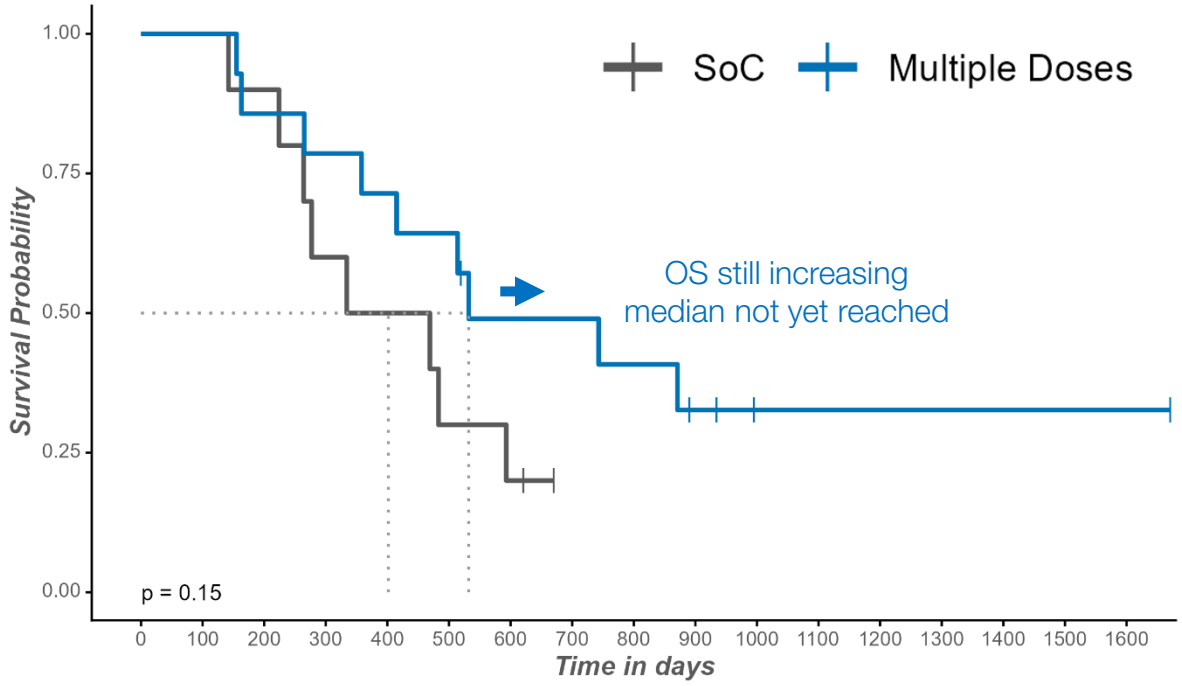
DRI Patients Live Significantly Longer Without Progression

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

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



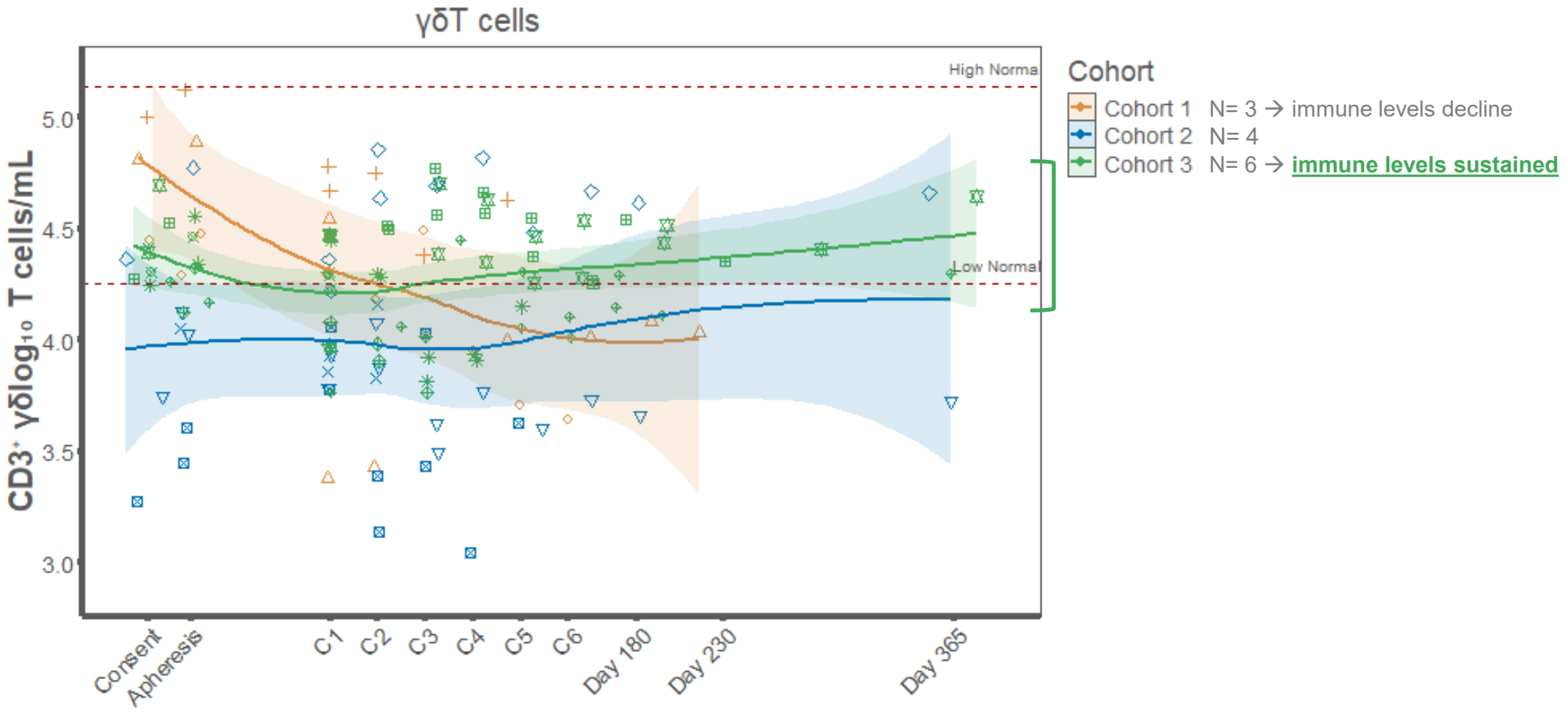
GBM Survival Analysis



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.

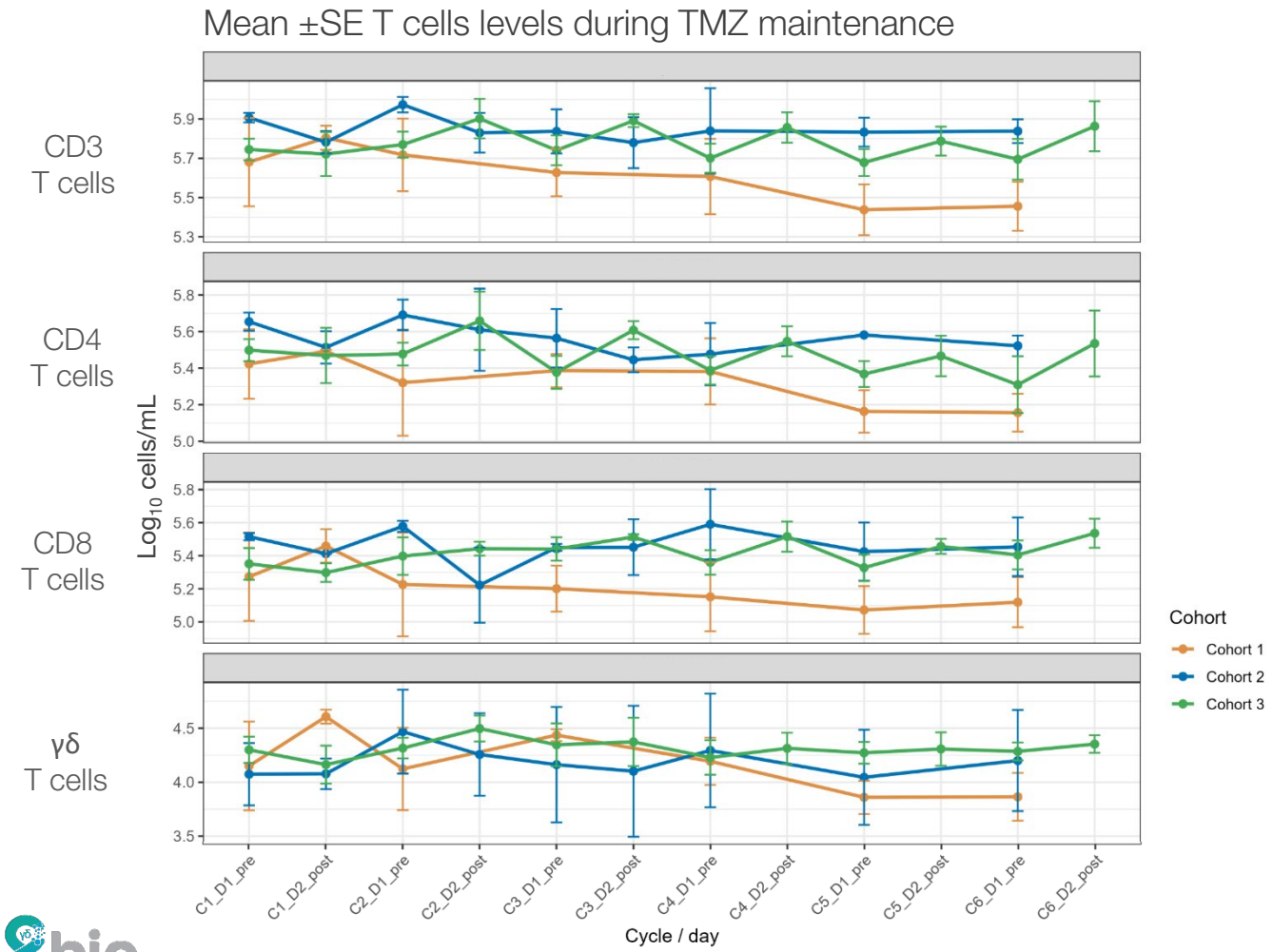
Repeat Dosing Supports Peripheral Immune Strength

Chemotherapy depletes the immune system, DeltEx DRI patients have better peripheral immunity



Repeat Dosing Preserves the Immune System

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



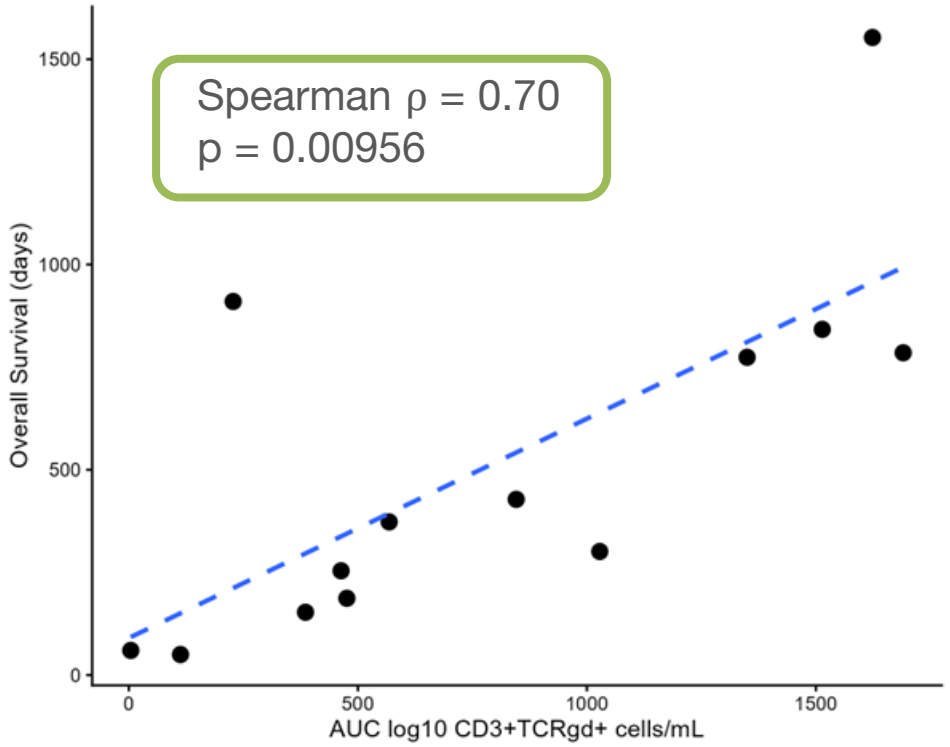
- Cohort 1 (single dose) shows steady decline — the dose frequency difference is decisive
- Cohorts 2 and 3 (repeat dose) maintain T cell levels across all immune subsets during TMZ
- Higher peripheral immune reconstitution is known to be correlated with improved survival outcomes

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



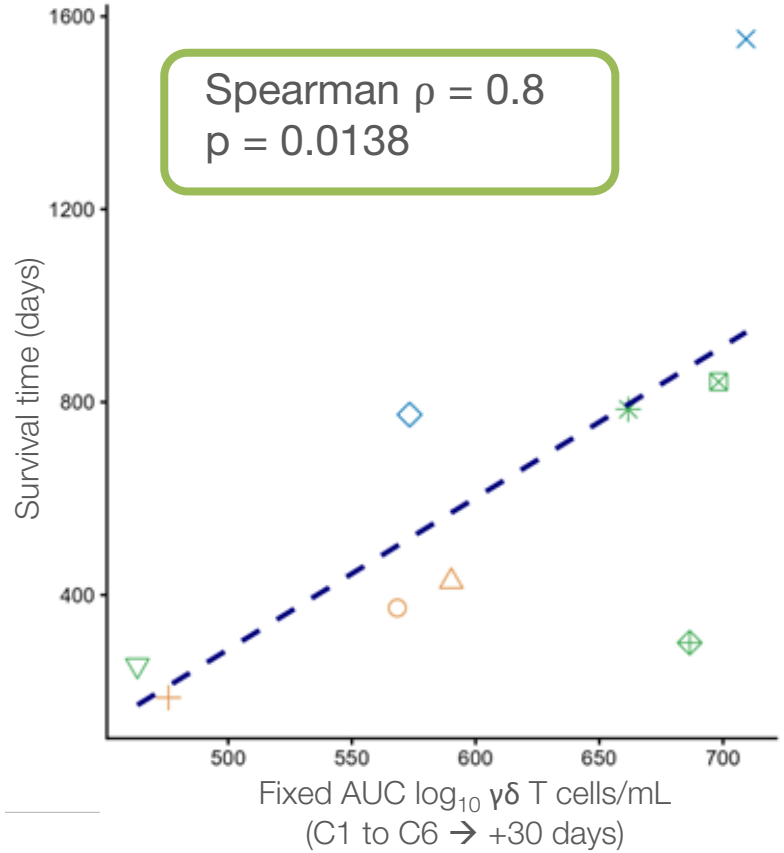
(Sustained γδ T cell levels after treatment →)

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).

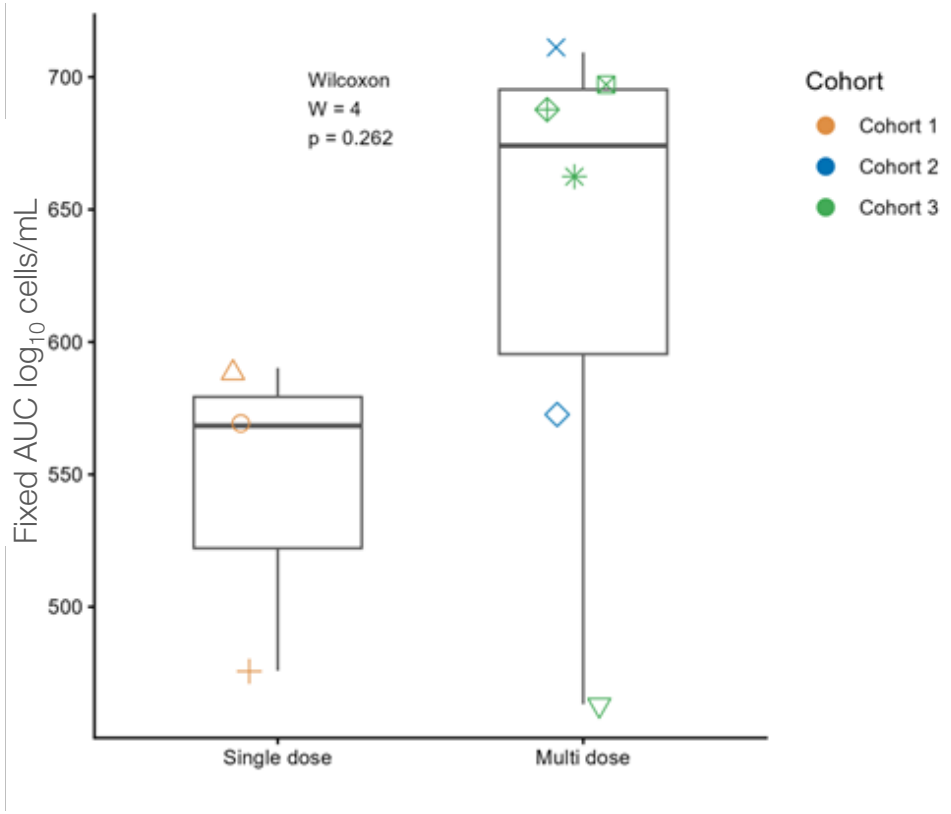
Repeat Dose Cohorts Trend to Higher γδ T cell Levels

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

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



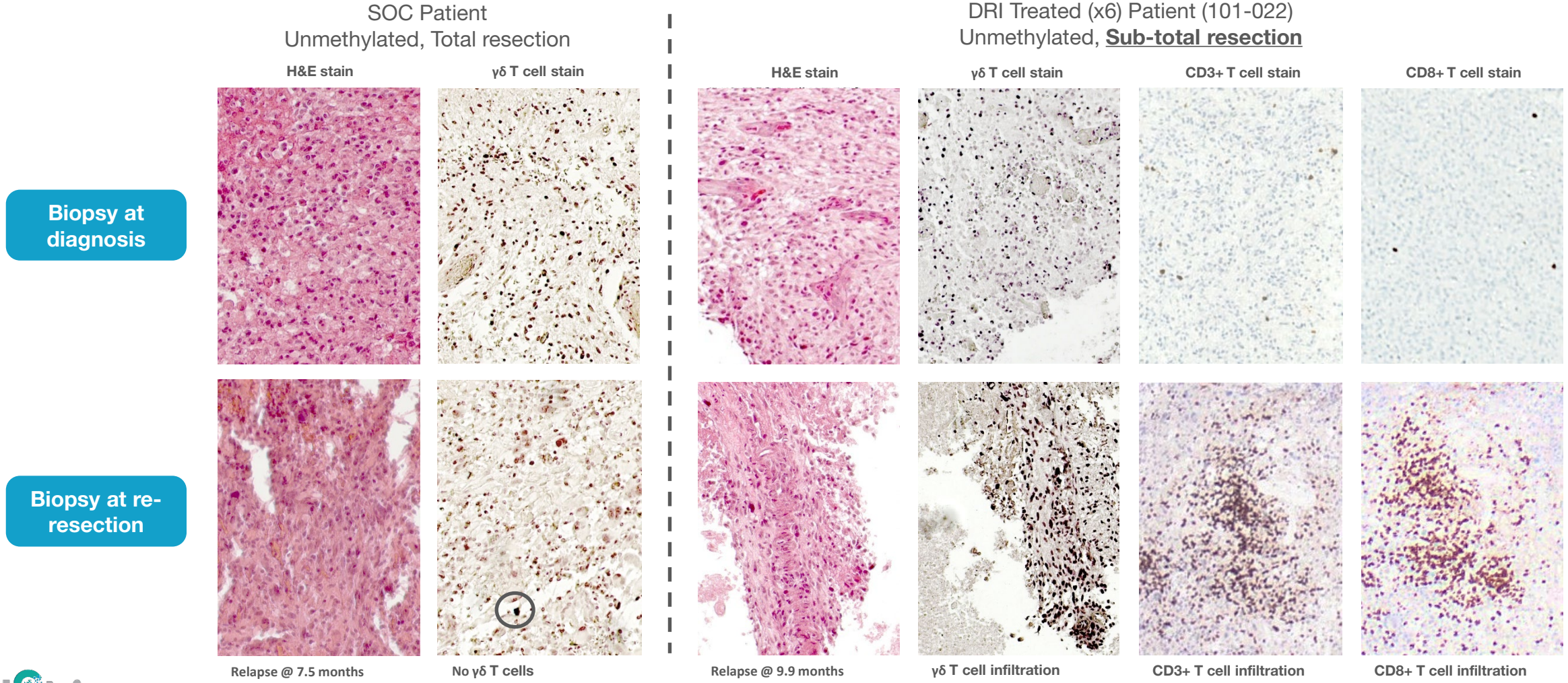
Single vs multi-dose γδ T cells (post-infusion)



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

Histopathology of the TME Shows γδ T cell Infiltration

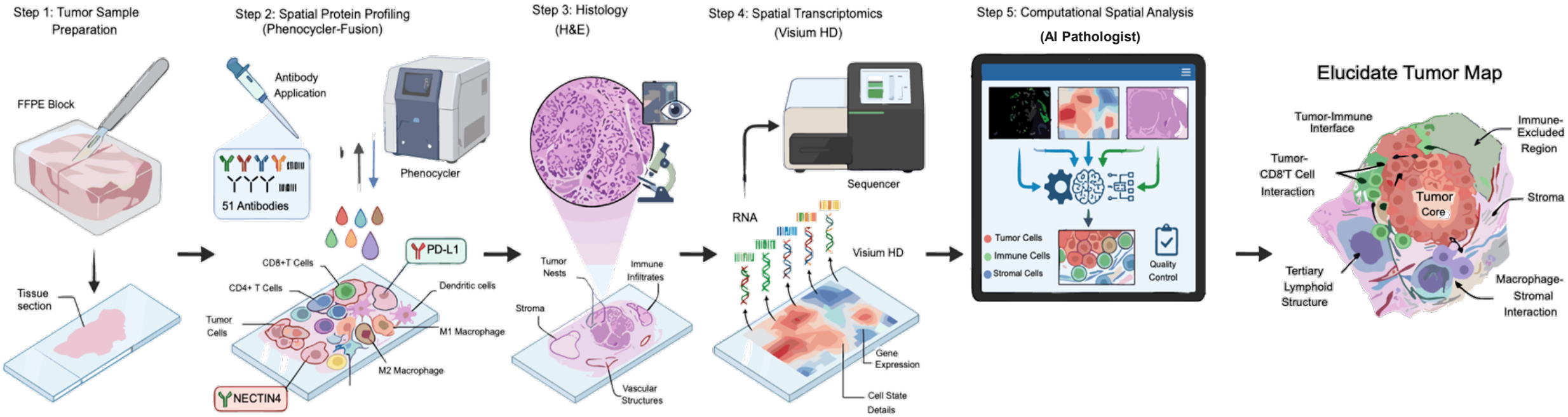
Broad immune cell infiltration demonstrate biological mechanism for activity and responses



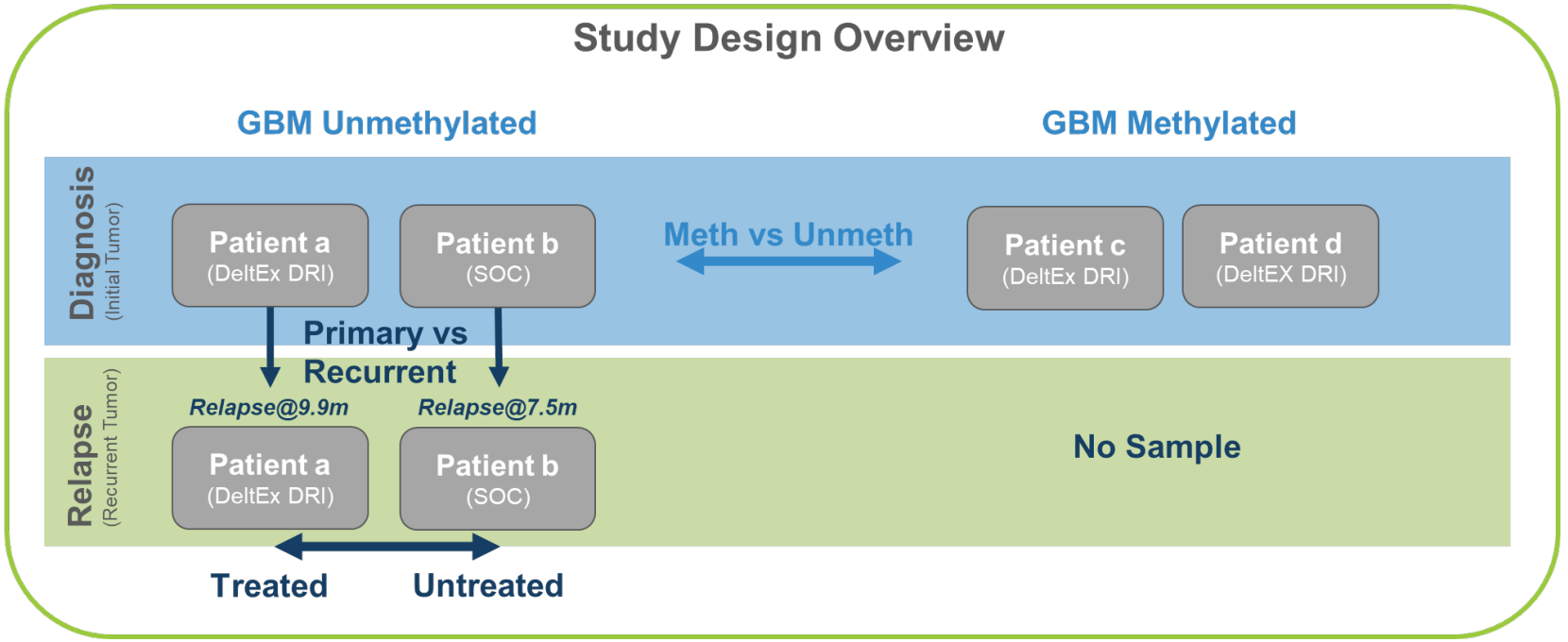
Source: IN8bio and UAB - Early trial results are not indicative of future results, including the outcome of this trial.

AI-Powered Tumor Mapping Gives IN8bio Deeper Insights

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



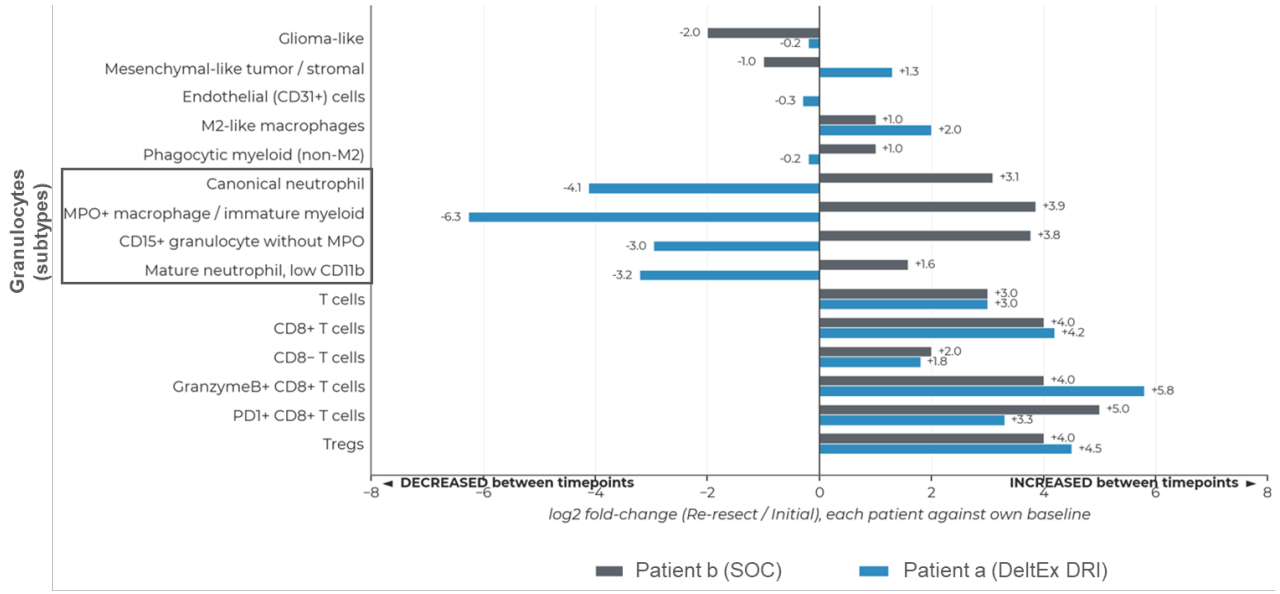
Single-Cell Analysis: What's Happening Inside GBM Tumors

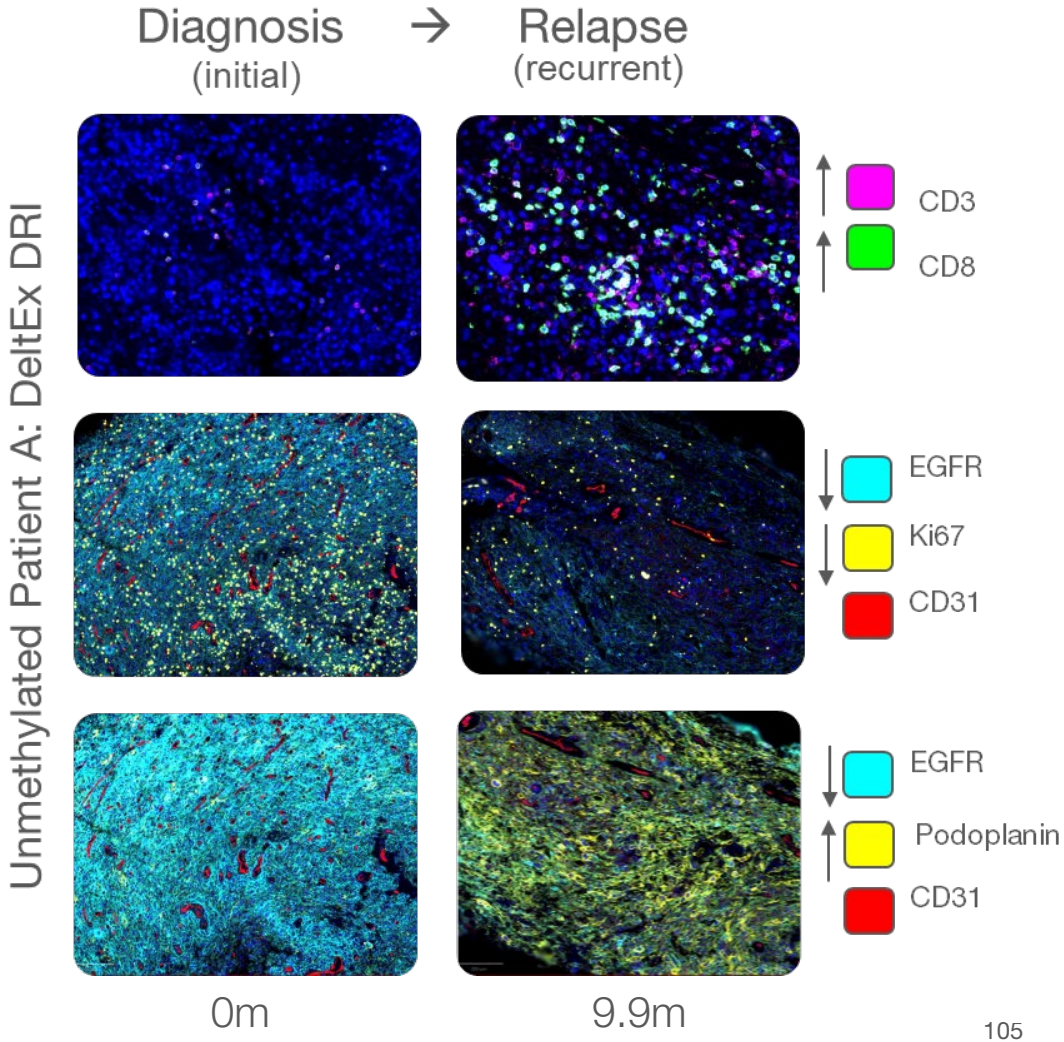


- Single-cell spatial mapping of tumor samples at diagnosis and relapse
- Direct comparison of DRI-treated vs. SOC patient tumors
- Precise visualization of how DRI reshapes the tumor immune environment

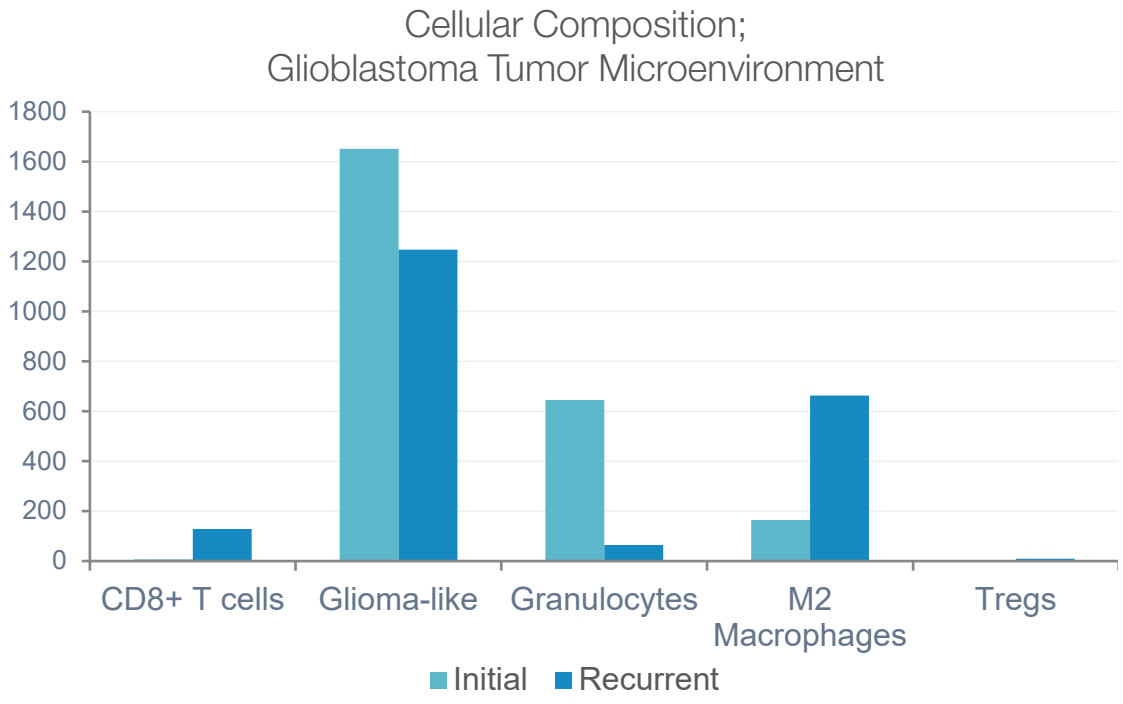
DeltEx DRI Shifts TME from Cold to Hot

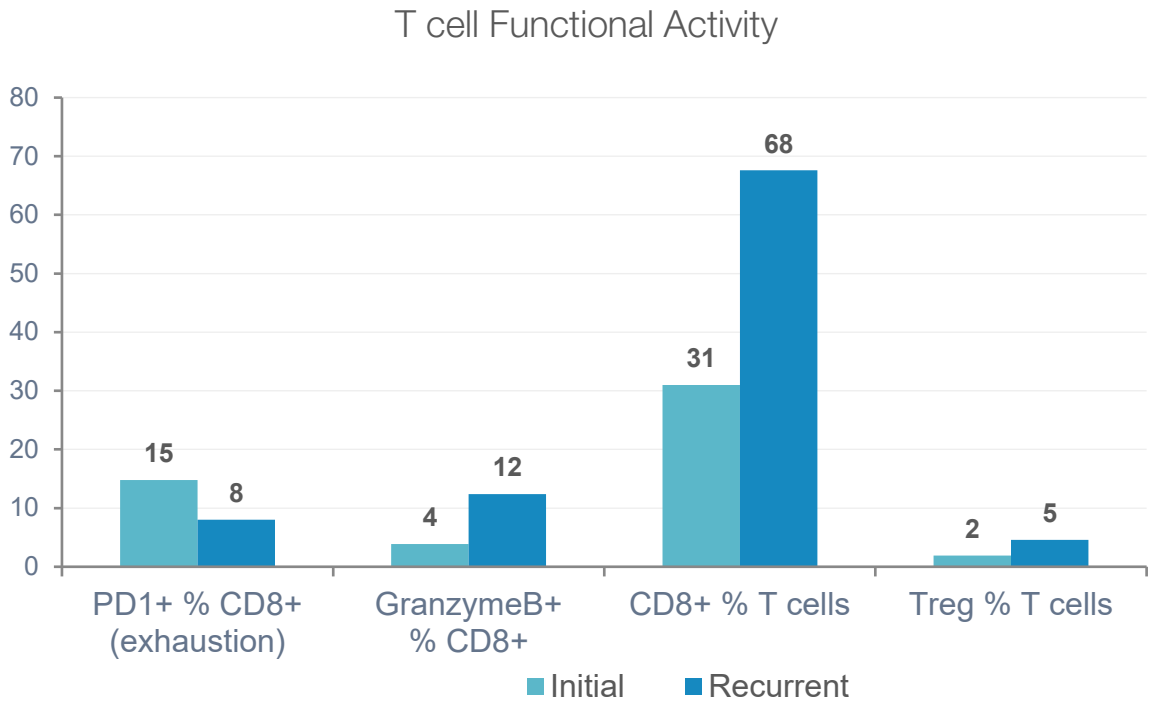
Increased T cell infiltration, reduced tumor proliferation and granulocyte clearance in DeltEx DRI treated patient





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 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

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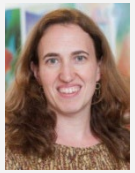

Bianca Santomasso, MD, PhD
MSKCC

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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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