Harnessing the Power of Gamma-Delta T Cells

June 2, 2025

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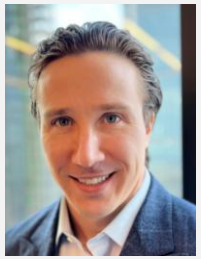
Deep Experience Across Cell Therapy and Oncology

William Ho
Co-Founder, President and Chief Executive Officer

Lawrence Lamb, PhD
Co-Founder and Chief Scientific Officer



Patrick McCall, CPA
Chief Financial Officer

Kate Rochlin, PhD
Chief Operating Officer

Lou Vaickus, MD, FACP
Interim Consulting Chief Medical Officer

IN8bio's team has deep experience in cell therapy & oncology expertise:

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

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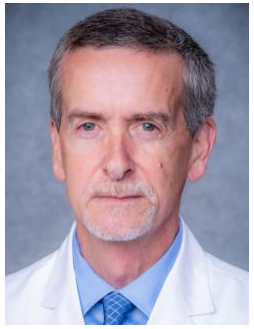
Guest Speakers



L. Burt Nabors, MD - Division Director for Neuro-Oncology at UAB

- L. Burt Nabors, is the M.D., William Austin Brown Endowed Professor, Vice Chair for Research, and Director of the Division of Neuro-Oncology in the UAB Department of Neurology. Dr. Nabors received his M.D. degree from the University of Tennessee in Memphis in 1991 and interned at the National Naval Medical Center. He served on active duty with the United States Navy as a Flight Surgeon from 1992 to 1995. He completed a residency in Neurology at UAB in 1998 and a fellowship in Neuro-oncology in 1999 under the direction of Dr. Steven S. Rosenfeld. He joined the Neuro-Oncology Program in 1999 as an Assistant Professor of Neurology and assumed the position of Program Leader in 2005. He is board-certified in Neurology and holds medical licensure in Alabama. Dr. Nabors maintains active basic, translational, and clinical research efforts. Moreover, Nabors is globally recognized as a leader in neuro-oncology treatment. He has served as an investigator for a large number of clinical trials of novel therapies for brain tumors, leading to significant innovations in the field.

O'NEAL COMPREHENSIVE CANCER CENTER
UAB MEDICINE

Lou Vaickus, MD, FACP – Interim Chief Medical Officer

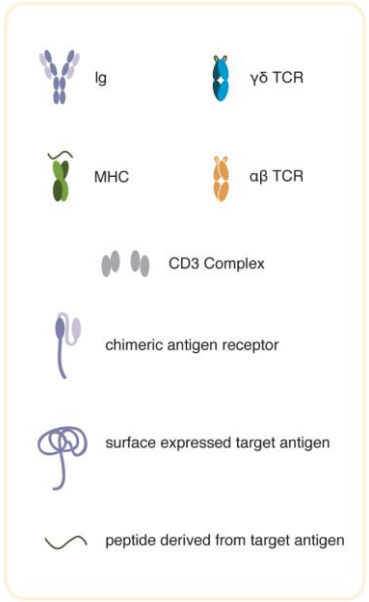
- Lou Vaickus, M.D. FACP, serves as Interim Chief Medical Officer of IN8bio. Dr. Vaickus is Chief Executive Officer and Founder of akta Pharmaceutical Development LLC (aktaPD®) and filled this role for IN8bio through its IPO. His therapeutic areas of specialization include oncology, hematology, pulmonary and other fibrotic disorders, infectious diseases, autoimmunity, internal medicine, immunology and medical devices inclusive of rare diseases. Lou served as Vice President and Head of Clinical Development, Global Medicines Development and Affairs at Vertex Pharmaceuticals. Before Vertex, he was Chief Medical Officer of Tolerx, Inc. Prior to that, he served as Vice President of Clinical Research and later as Senior Vice President of Medical Affairs at Sunovion, and Medical Director of Oncology/Immunology at EMD Serono. Dr. Vaickus has held faculty positions at the U of Iowa (Division of Hematology/Oncology) and at Roswell Park Cancer Institute/SUNY Buffalo (Divisions of Clinical Immunology, Bone Marrow Transplant, Hematologic Malignancies, and Solid Tumor Oncology and Investigational Therapeutics). He is board certified in Internal Medicine, Hematology and Hematologic Malignancies, and Medical Oncology. He completed postdoctoral fellowships in hematology, oncology, and immunology at the Mayo Clinic and Stanford University. He has over 100 peer-reviewed articles, book chapters, and abstracts. Dr. Vaickus received an M.D. from Loyola University's Stritch School of Medicine

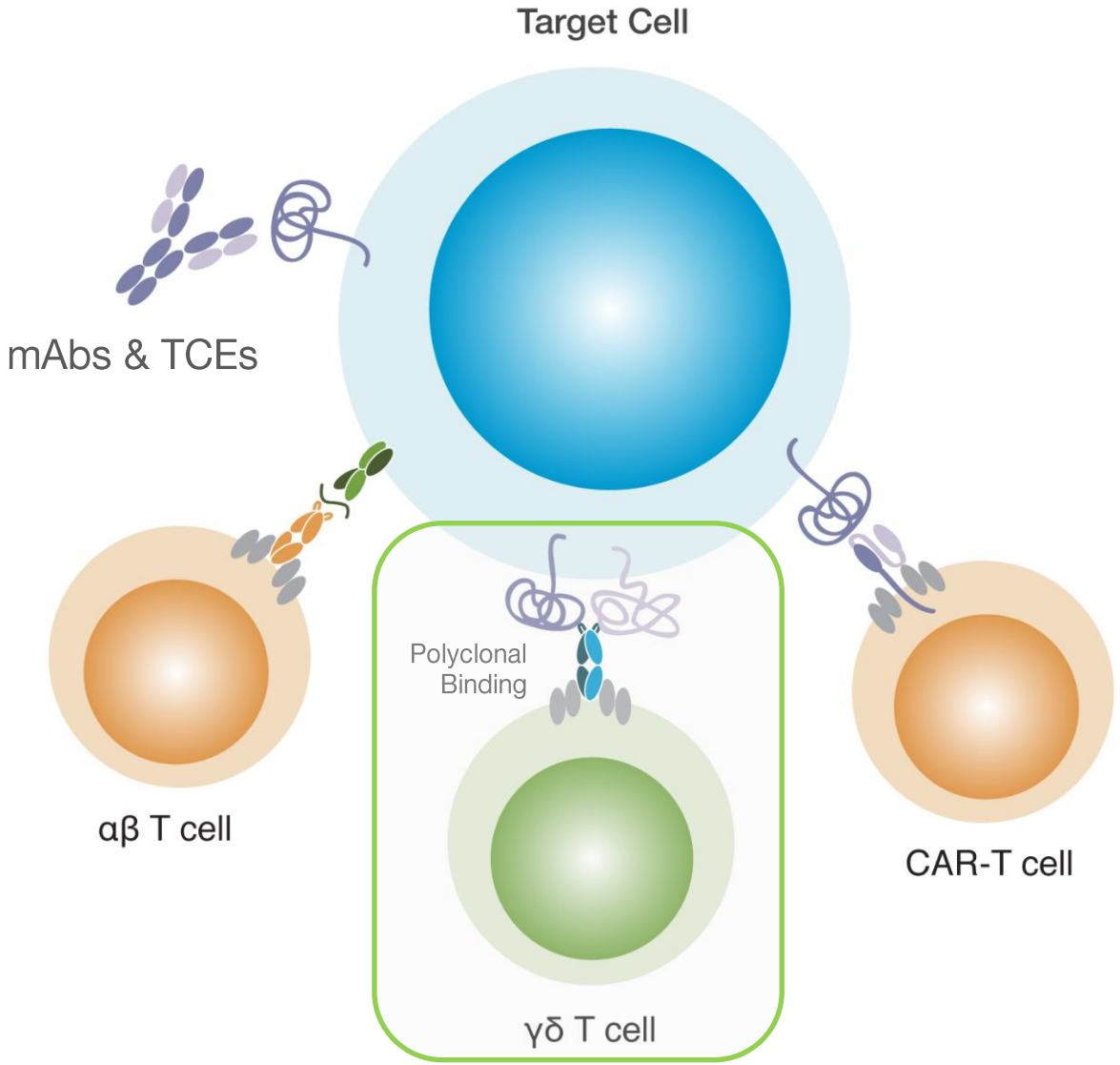
Revolutionizing γδ T cell Therapies

- **IN8bio is a leader in the development of gamma-delta (γδ) T cell therapies and T cell engagers (TCEs) in oncology and autoimmune diseases**
  - **Harnessing the Power of Immune Cells:** γδ T cells are “Nature’s CAR-T” cells that act as direct killers while orchestrating a comprehensive immune response to eliminate cancers and other dysfunctional cells
  - **Durable Cancer Remissions:** IN8bio is targeting significant unmet needs by pioneering novel approaches to keep patients' progression-free longer with multiple remissions exceeding 3+ years in patients with difficult to treat cancers
  - **Precision and Safety:** γδ T cells have demonstrated in patients a better safety profile to date, with lower rates of adverse events and toxicities including cytokine release syndrome (CRS) and neurotoxicity (ICANs)
  - **Strong Capabilities:** We are translating over 30 years of expertise in γδ T cell research, our DeltEx™ platform has solved certain key biological, clinical and manufacturing issues that historically plagued the field across cell therapy and TCEs
  - **Powerful Platform Approaches**: We have developed a γδ TCE that can efficiently drive depletion of target cells, while inducing expansion of γδ T cells. This construct has unique differentiated properties to drive deeper cell depletion in cancer and autoimmune indications
- Our Mission: **Cancer Zero**™ - Driven by our goal to safely eradicate residual cancer cells. Join us in transforming cancer care

We Need Continued Immune Surveillance to Eliminate Recurrence and Progression

γδ T Cells: Nature's CAR-T Cell





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

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

L. Burt Nabors, MD

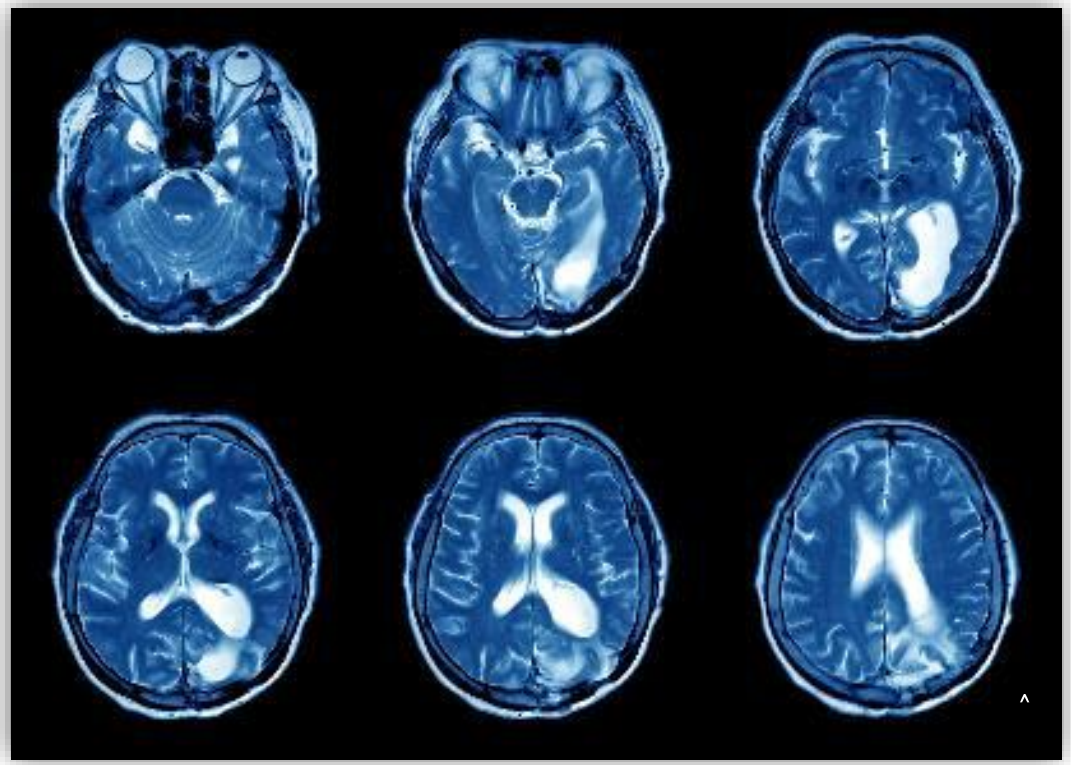
William Austin Brown Endowed Professor, Vice Chair for Research, and Director of the Division of Neuro-Oncology in the UAB Department of Neurology

Glioblastoma Multiforme (GBM)

GBM is the most aggressive form of brain cancer, where the Standard-of-Care was established in 2005

New cases of GBM diagnosed each year in the U.S.:	New cases GBM diagnosed each year globally:
~14,500	~300,000

For over 20 years, the existing standard of care has resulted in median overall survival of	The existing standard of care has resulted in median progression-free survival of
~14 to 16 months	~7 months

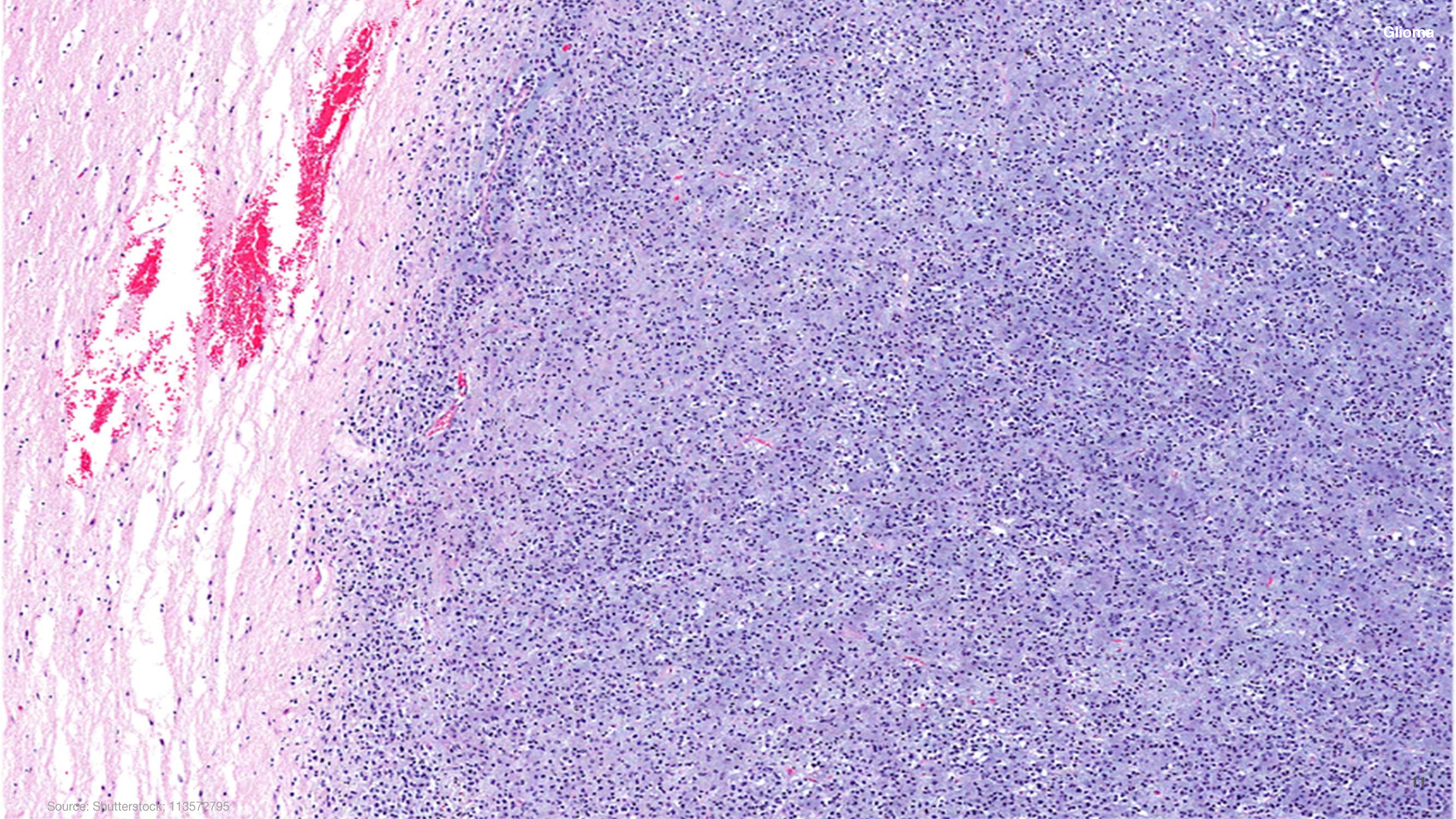


A novel gamma-delta T cell treatment used in front-line disease to address the challenges in GBM

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

William Ho

CEO and Co-founder IN8bio



Source: Shutterstock; 113572795

Shortfalls of Conventional Cell Therapies in Solid Tumors

CAR-Ts have demonstrated efficacy in blood cancers but have not had similar results in solid tumors

Tumor heterogeneity

Tumor cells harbour distinct molecular signatures with varying treatment sensitivity

T Cells unable to penetrate tumor

Unrecognizable and impenetrable; malignant cells turn off antigens and receptors for immune evasion

Few targets that can be ablated

Unfavorable therapeutic profile of conventional immunotherapy with off-target recognition

Immune-suppression

T_{Regs} of the tumor microenvironment (TME) suppress $T_{Effector}$ function and anti-tumor response

Trafficking of T cells into Tumors

Effective immunotherapy requires concerted and abundant T cell migration to the disease site

Chemotherapy kills immune cells

Non-selective cytotoxicity kills immune cells required for tumor surveillance and targeting

Pursuing Treatment in GBM: Following the Biology

The biology shows us the multiple advantages of γδ T cells in the solid tumor setting, particularly in glioblastoma, where patients have **very limited available treatment options.**

The brain offers a separate compartment that allows direct delivery of cells through a catheter directly to the site of the tumor, increasing E:T ratio and reducing the variable of cell trafficking.

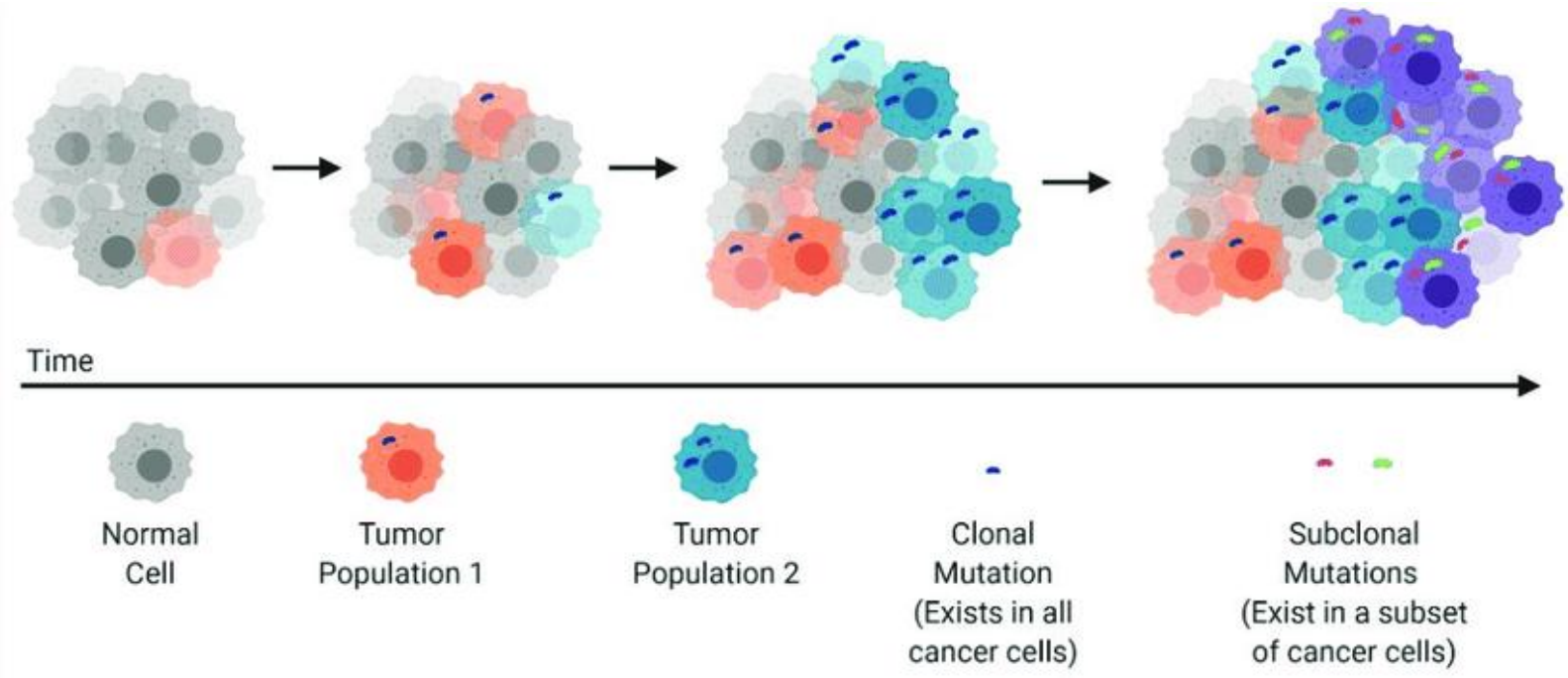
As we move towards allogeneic cell therapy in the solid tumor setting it simplifies the challenges around dealing with host-versus-graft (HvG) effect and the persistence of the delivered cells.

The advantage of going into the brain is that it is one of three organ centers in the body historically considered immune-privileged.

In neuro oncology, the standard of care, Temodar, is lymphodepleting in itself. A separate lymphodepleting protocol such as Flu/Cy is not necessary.

Heterogeneity is a Challenge in Glioblastoma

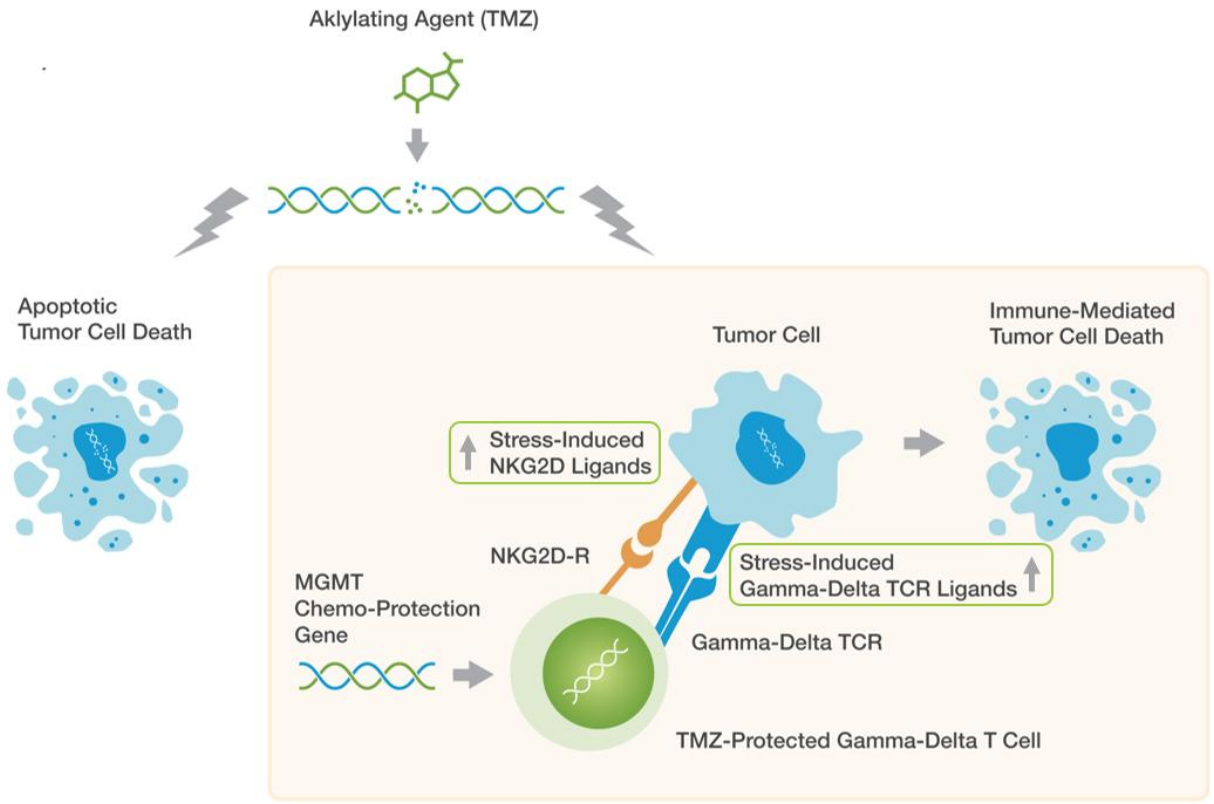
Clonal and subclonal mutations and the pace of tumor growth disrupts the immune response



- Rapid neoantigen generation, tumor microenvironment, and doubling time outrun the effectiveness of the adaptive immune response
- Innate recognition and killing by γδ T cells covers the heterogeneity of gliomas and remains effective but these “first responders” are down-regulated and/or consumed by antigen induced cell death (AICD).

Targeting the DNA Damage Response (DDR) to Kill Tumors

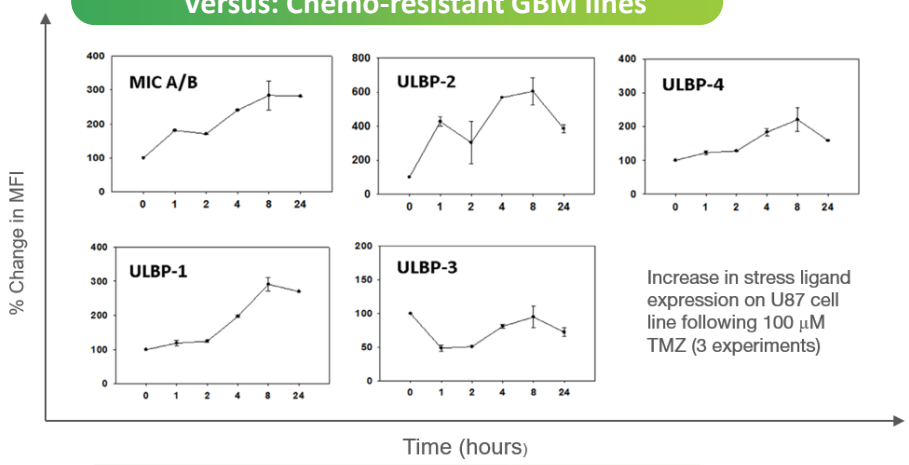
DDR is a biological process that can detect and eliminate cells with DNA damage through increased avidity



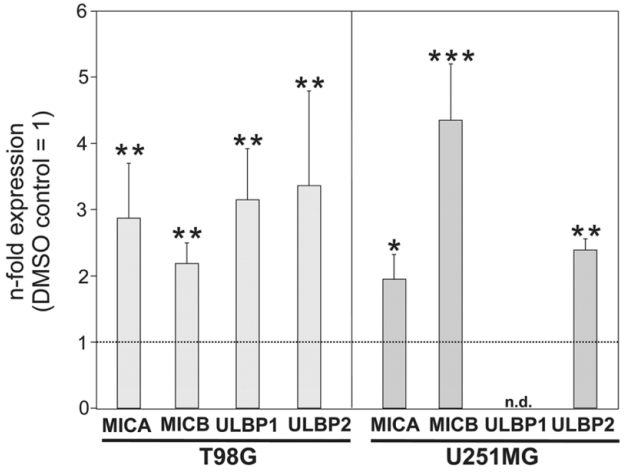
DRI gamma-delta T cell mechanism overview

TMZ Increases NKG2D-L Expression:

Versus: Chemo-resistant GBM lines

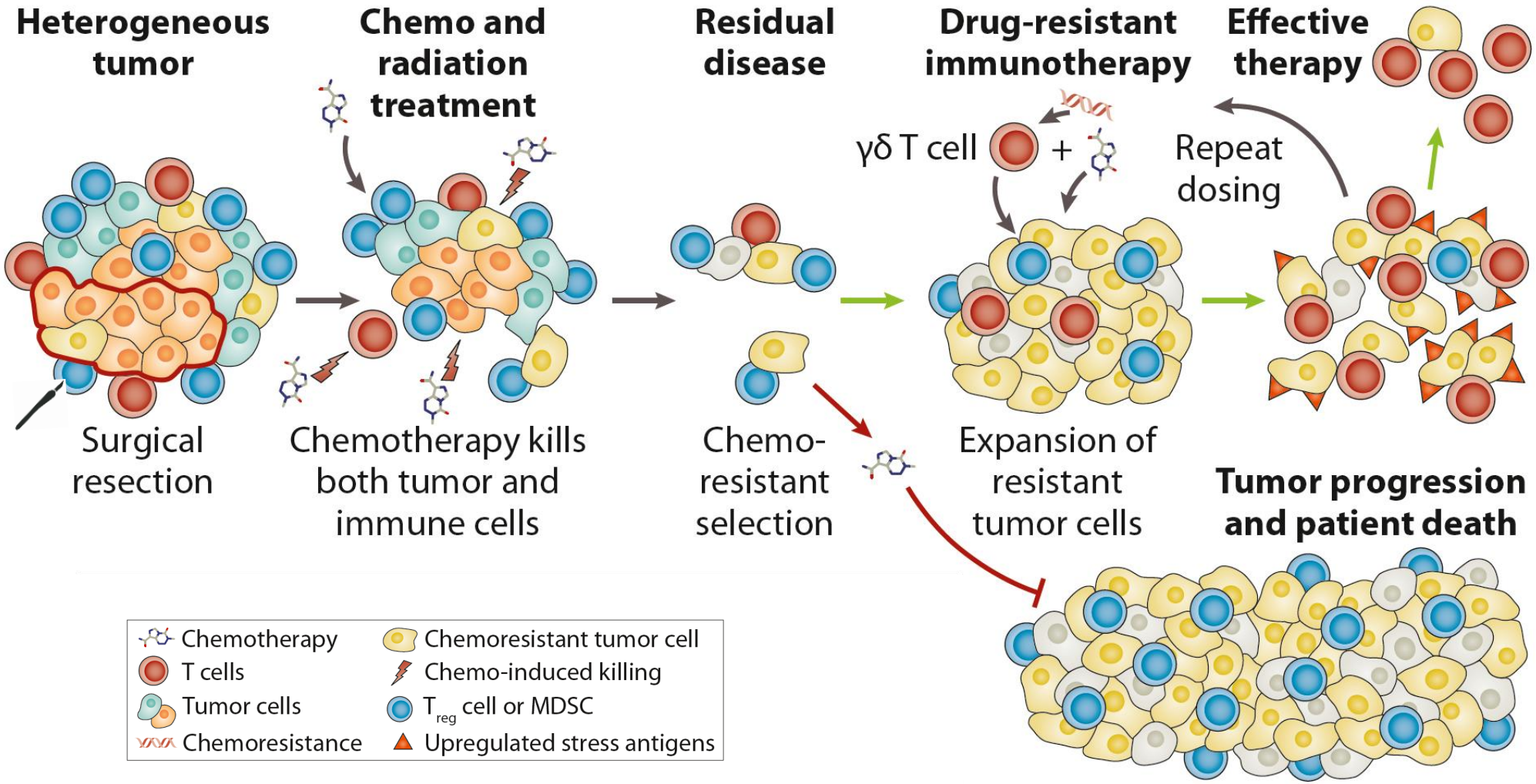


Versus: Glioma stem-like cells



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

IN8bio's DRI Approach to Solid Tumor Therapy



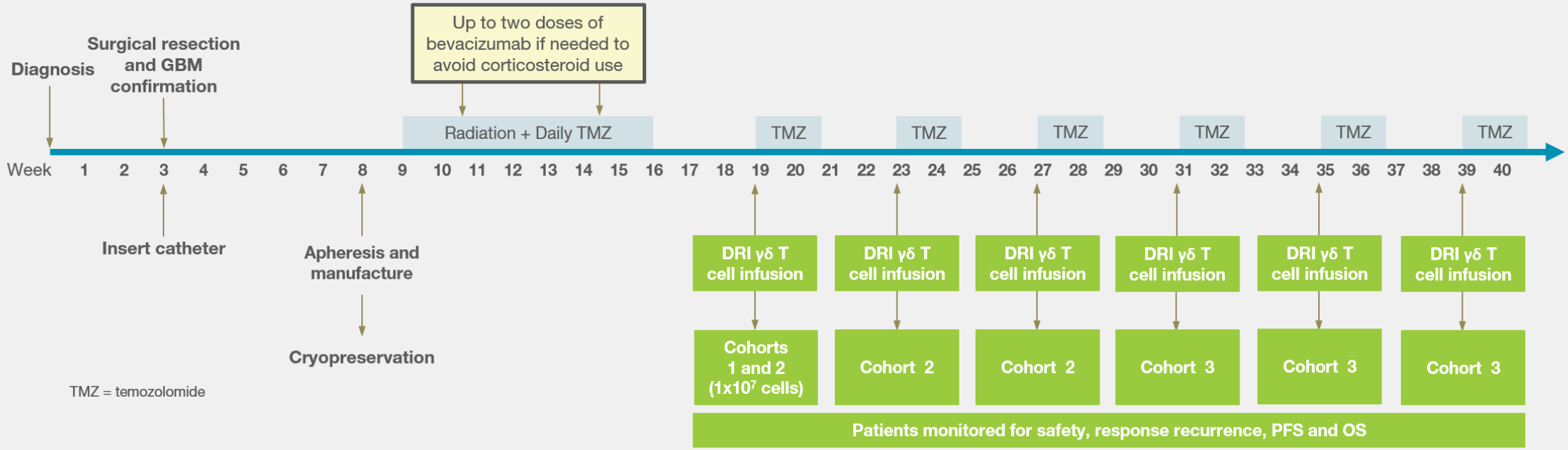
Source: BioPharma Dealmakers, September 2019

Repeated Dosing to Eliminate Residual Cells

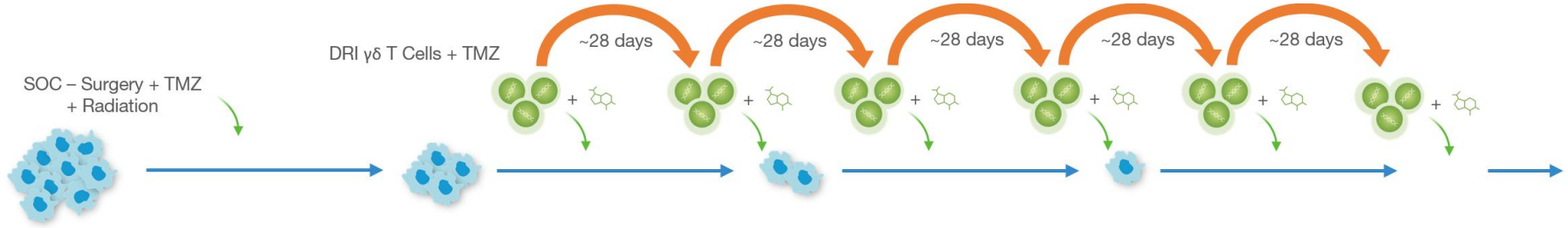
Targeting residual cells at a rate faster than the glioma doubling time to maintain remission

SOC: Stupp Regimen

IN8bio: DeltEx DRI Therapy



TMZ + adjuvant DRI γδ T cells multiple repeat doses



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

Lou Vaickus, MD, FACP

Interim Consulting Chief Medical Officer

INB-200

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

2025 ASCO ANNUAL MEETING

INB-200: Phase 1 Study of Gene-Modified Autologous Gamma-Delta (γδ) T Cells in Newly Diagnosed Glioblastoma Multiforme (GBM) Patients Receiving Maintenance Temozolomide (TMZ)

M Lobbous[1,2], LS Lamb[3], K Rochlin[3], T Pillay[1], M ter Haak[3], LB Nabors[1]

Department of Neurology, University of Alabama at Birmingham, Birmingham, AL, USA[1]; Brain Tumor and Neuro-Oncology Center, Cleveland Clinic Foundation, Cleveland, OH[2;]; IN8bio, Inc. New York, New York[3]

INB-200: Phase 1 Study Design and Treatment Regimen

Treatment Arms

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

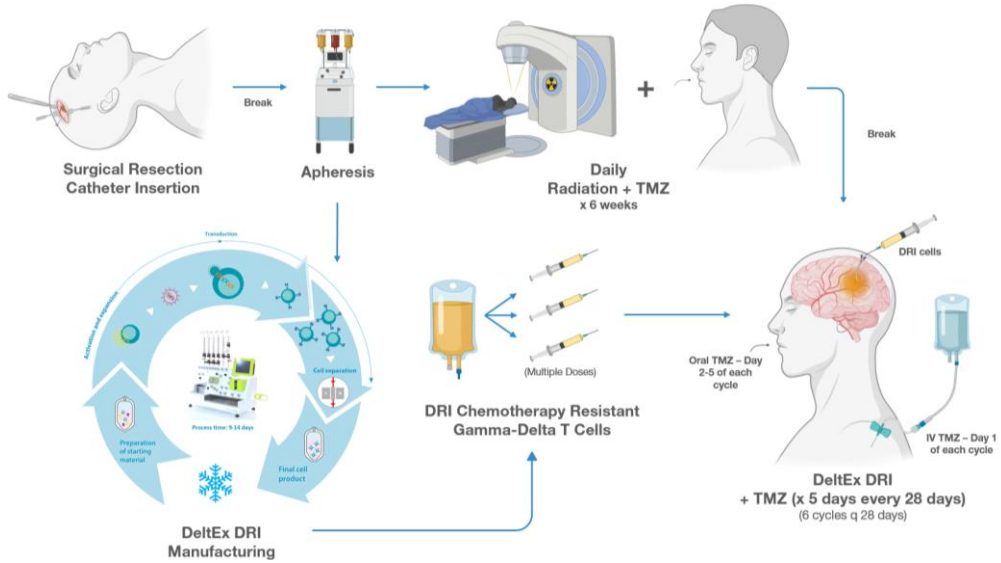
DL1: N = 3 (up to 6) patients, single dose of 1 x 10^7 cells on C1D1
DL2: N = 3 (up to 6) patients, three doses of 1 x 10^7 cells, one dose every 28 D1 of C1-C3
DL3: N = 3 (up to 6) patients, six doses of 1 x 10^7 cells, one dose every 28 days on D1 of C1-C6

Treatment Regimen & Timing

Surgical resection followed by apheresis

6 weeks induction TMZ + radiation

6 cycles maintenance TMZ + DRI



Primary Endpoints

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

Secondary Endpoints

- Time to progression
- Overall survival
- Biologic response

Site

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UAB THE UNIVERSITY OF ALABAMA AT BIRMINGHAM

UAB HEERSINK SCHOOL OF MEDICINE

Source: IN8bio, image created with biorender.com

Patient Demographics

Subject	Age / Sex	Cytogenetics	Dose level	Resection	TMZ Maint. Cycles Received
001	68 / M	IDH-WT, MGMT-unmethylated	1	Total	5
003	74 / F	IDH-WT, MGMT-methylated	1	Total	6
004	21 / F	IDH-WT, MGMT-unmethylated	1	Total	3
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
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

- Median age: 66
- 54% **unmethylated**
- 46% **subtotal** resection
- 23 enrolled, 13 treated, 4 remain in follow-up
- 9 deaths:
  - Due to progression or disease-related issues
  - Other
    - Cardiac event (007)
    - Pulmonary Emboli (014)

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

Treatment Emergent Adverse Events (N=13)

	SAEs	>1 Gr. 1-2	Grade 3	Grade 4	Grade 5	All Grades
WBC count decreased	SAE	2 (15.4)	1 (7.7)	0	0	3 (23%)
Platelet count decreased	SAE		2 (15.4)	1 (7.7)	0	3 (23%)
Hydrocephalus	SAE	1 (7.7)	1 (7.7)	0	0	2 (15.4)
Deep vein thrombosis	SAE	1 (7.7)	1 (7.7)	0	0	2 (15.4)
Dysarthria	SAE		1 (7.7)	0	0	1 (7.7)
Hypertension	SAE		1 (7.7)	0	0	1 (7.7)
Cardiac arrest	SAE		0	0	1 (7.7)	1 (7.7)
Cardiac disorder	SAE		1 (7.7)	0	0	1 (7.7)
Pulmonary embolism	SAE		0	1 (7.7)	0	1 (7.7)
Fall	SAE		1 (7.7)	0	0	1 (7.7)
Cyst drainage	SAE		1 (7.7)	0	0	1 (7.7)
Lymphocyte count decreased			1 (7.7)	0	0	1 (7.7)
Neutrophil count decreased			0	1 (7.7)	0	1 (7.7)
Hypotension			1 (7.7)	0	0	1 (7.7)
Appendicitis			1 (7.7)	0	0	1 (7.7)
Balance disorder		2 (15.4)	0	0	0	2 (15.4)
Urinary tract infection		2 (15.4)	0	0	0	2 (15.4)
Asthenia		2 (15.4)	0	0	0	2 (15.4)
Fatigue		2 (15.4)	0	0	0	2 (15.4)
Headache		2 (15.4)	0	0	0	2 (15.4)
Arthralgia		2 (15.4)	0	0	0	2 (15.4)
Decreased appetite		2 (15.4)	0	0	0	2 (15.4)

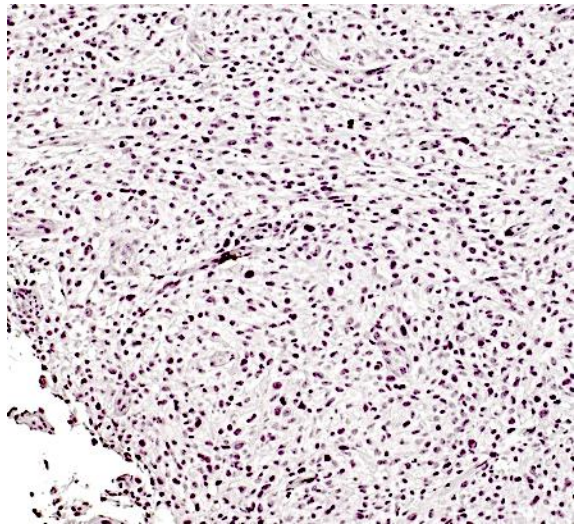
- No DRI-related toxicity
- **No DLTs to date**
- **No ICANS/CRS**
- Majority of toxicities are grade 1 or 2 and attributable to TMZ
- Unrelated TESAE's of cardiac arrest, pulmonary embolus, temporal cyst drainage, dysarthria, hydrocephalus
- **No treatment-related deaths**
- Repeat dosing **DOES NOT** demonstrate change in toxicity profile to date

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

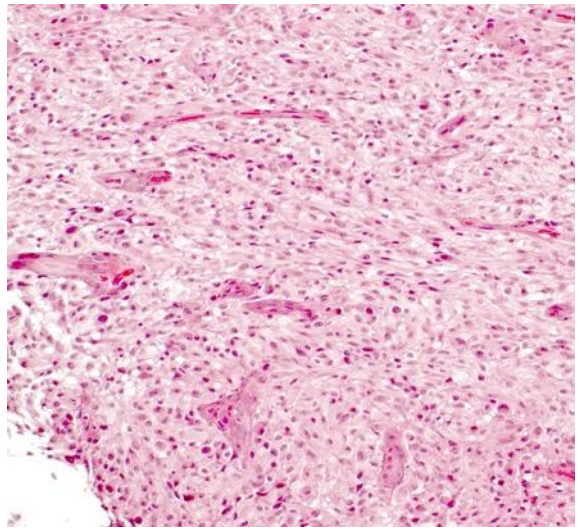
INB-200 Drives Immune Cells into a ‘Cold’ Tumor

MGMT-unmethylated and sub-total resection at diagnoses - preserved **γδ** T cells confirmed following six does of DRI infusion + TMZ with presence of necrotic tissue and prominent **γδ** T cell infiltration of relapsed tumor

Biopsy: at diagnosis

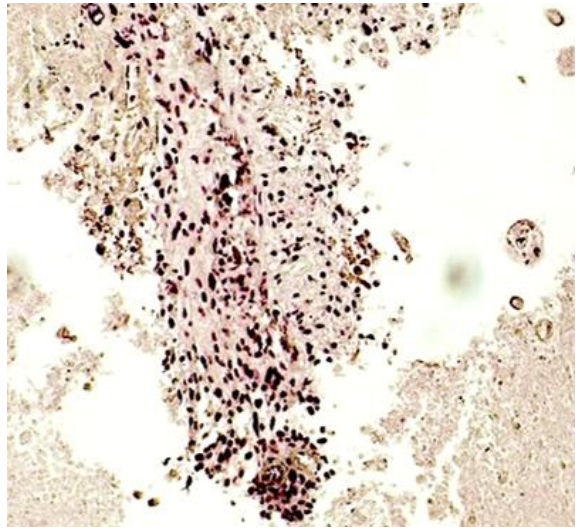


γδ T cell stain (brown)

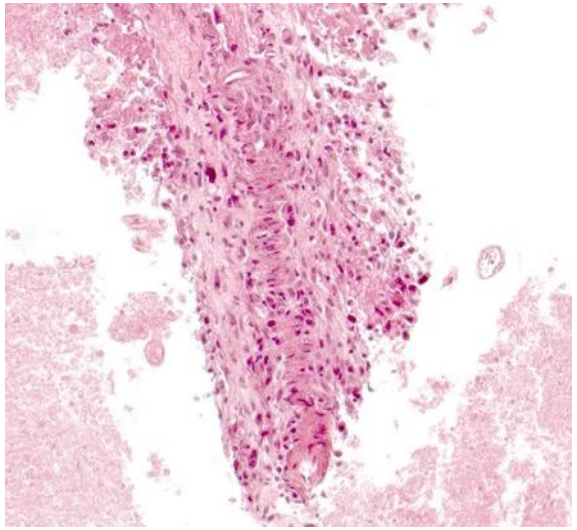


H&E stain

Biopsy: at relapse



γδ T cell stain (brown)



H&E stain

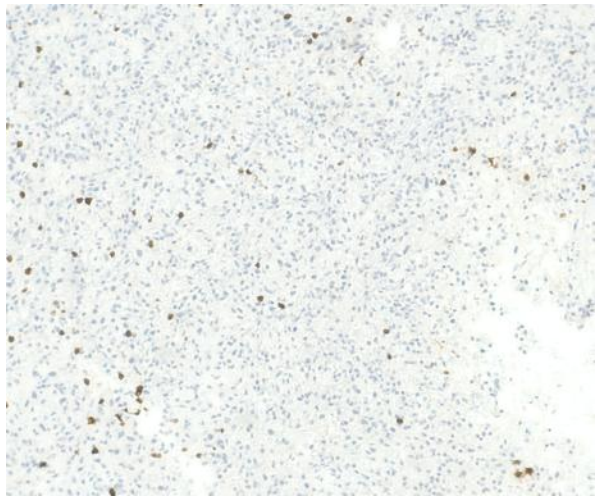
Source: IN8bio and UAB

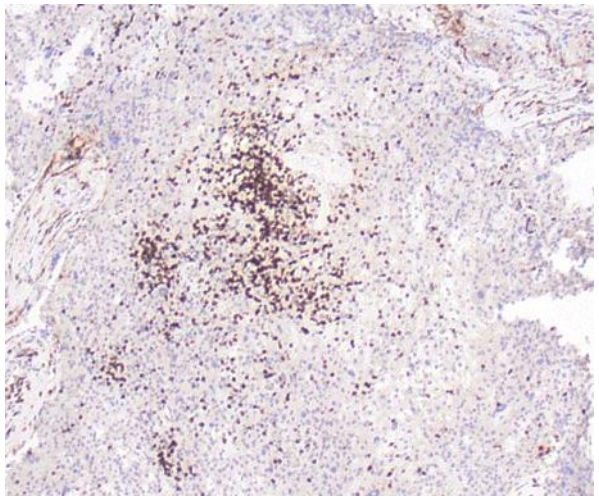
Marked Differences in Immune Infiltration Broadly

Biopsy: at **diagnosis**

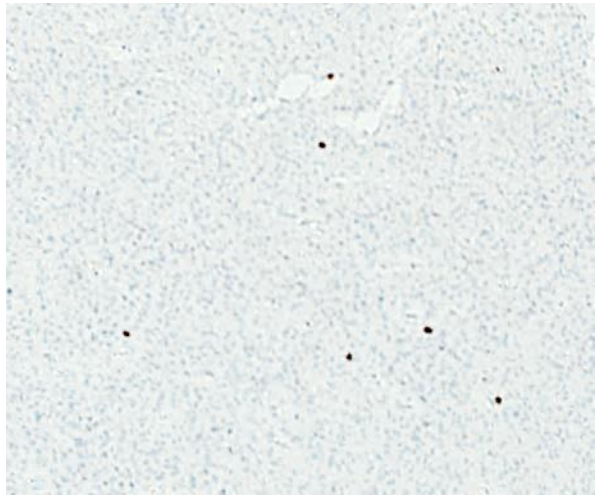
Biopsy: at **relapse**

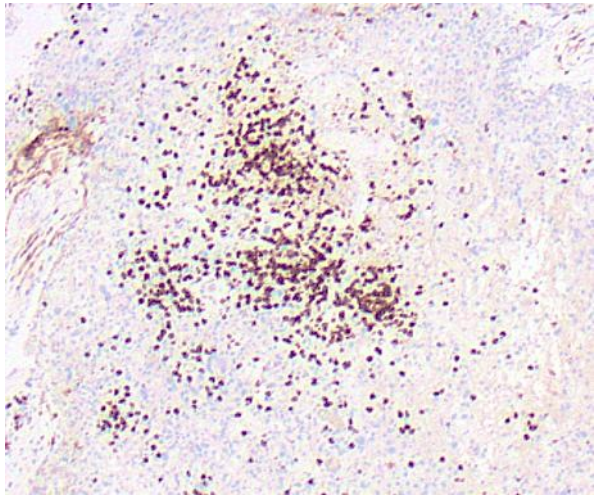
CD3+ T cells





CD8+ T cells





Source: IN8bio and UAB

Results from one patient are not indicative of future results including the outcome of this trial

Repeated INB-200 Doses is Extending Progression Free Survival

Poor Survival and Low Median PFS for the Past 20 Years

The NEW ENGLAND JOURNAL of MEDICINE

ORIGINAL ARTICLE

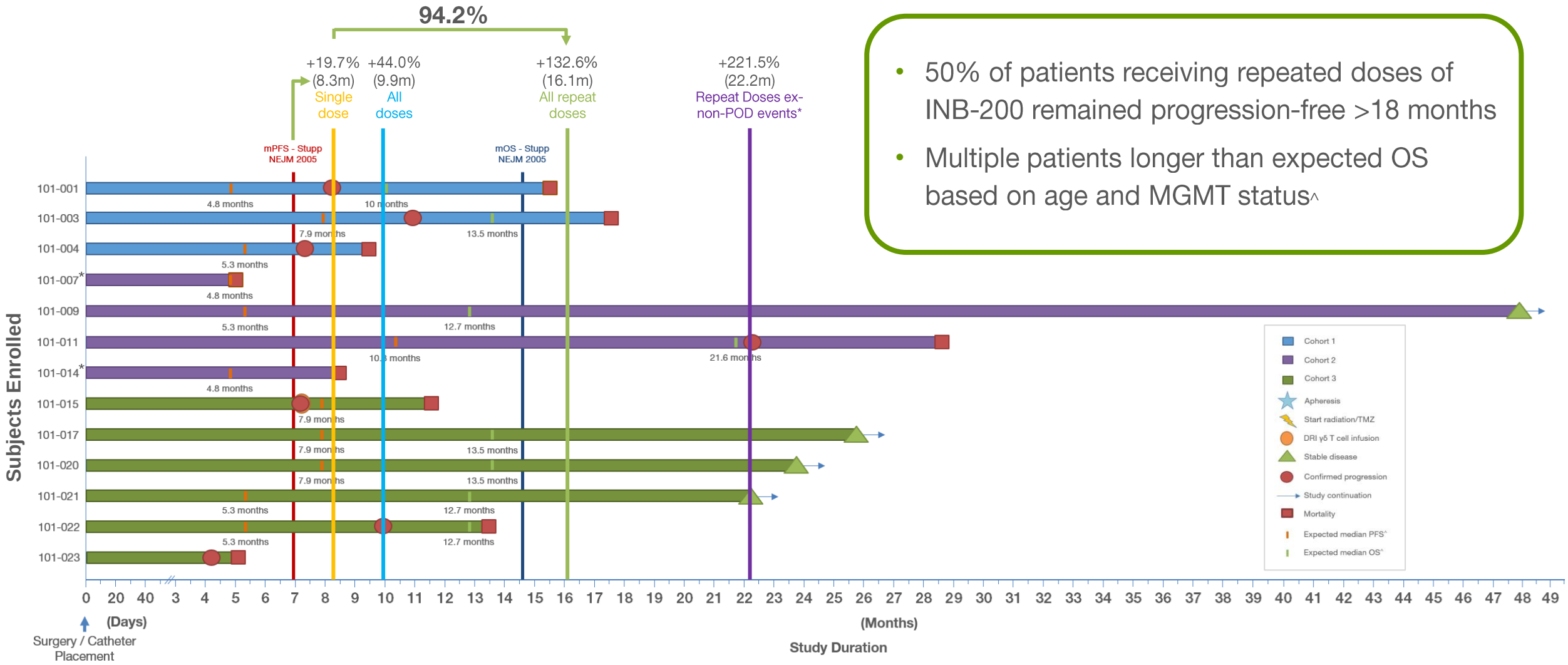
Radiotherapy plus Concomitant and Adjuvant Temozolomide for Glioblastoma

Authors: Roger Stupp, M.D., Warren P. Mason, M.D., Martin J. van den Bent, M.D., Michael Weller, M.D., Barbara Fisher, M.D., Martin J.B. Taphoorn, M.D., Karl Belanger, M.D., +12 , for the European Organisation for Research and Treatment of Cancer Brain Tumor and Radiotherapy Groups and the National Cancer Institute of Canada Clinical Trials Group* Author Info & Affiliations

Published March 10, 2005 | N Engl J Med 2005;352:987-996 | DOI: 10.1056/NEJMoa043330 | VOL. 352 NO. 10

Source: NEJM 2005; 352:987-996 DOI: 10.1056/NEJMoa043330; NEJM 2017; 376:1027-1037 DOI: 10.1056/NEJMoa1611977

INB-200 Improvement Over Stupp mPFS of 6.9 Months^



- 50% of patients receiving repeated doses of INB-200 remained progression-free >18 months
- Multiple patients longer than expected OS based on age and MGMT status^

Note: As of May 31, 2025; 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.

Key Takeaway Points/Conclusions

Alkylating chemotherapy induces the DNA damage response (DDR) and the upregulation of stress-associated cell surface proteins (NKG2D-L and others) even on chemo-resistant glioma cells and stem cells

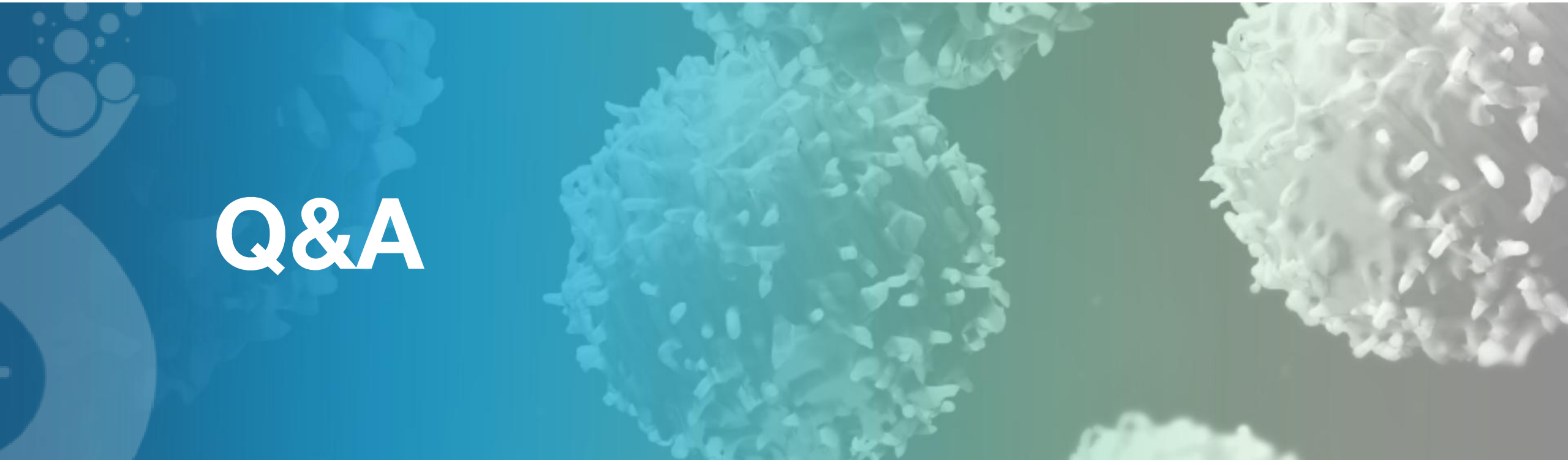
Repeated intracranial dosing with NKG2D-L targeting γδ T cells concomitantly with alkylating chemotherapy is feasible and can safely be used for treatment of newly diagnosed glioblastomas

IN8bio

Harnessing the Power of γδ T cells

- **Durable Long-Term PFS** - Repeated dosing of INB-200 achieved a median PFS of 16.1 months, more than double the 6.9 months typically observed with the standard-of-care (SOC) Stupp protocol
- **Well Tolerated** - INB-200 has been well tolerated, with no serious treatment related or dose-limiting toxicities, no cytokine release syndrome (CRS), or immune effector cell-associated neurotoxicity syndrome (ICANS) has been observed
- **Multi-Center Validation** - Preliminary data from three additional centers in the INB-400 Phase 2 trial are consistent with findings from the INB-200 Phase 1 study conducted at UAB, supporting multi-center feasibility and applicability
- **Encouraging Dose Response Signal** - Patients receiving multiple doses of INB-200 exceeded the historical median OS of 14.6 months with the Stupp protocol and 50% remained progression-free >18 months versus 0% of patients who received a single dose
- **Path Forward** – Actively exploring strategic partnerships, government funding and other opportunities to optimize and advance development of this novel therapy for frontline GBM

Q&A



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