Harnessing the Power of Gamma-Delta T Cells

July 2024

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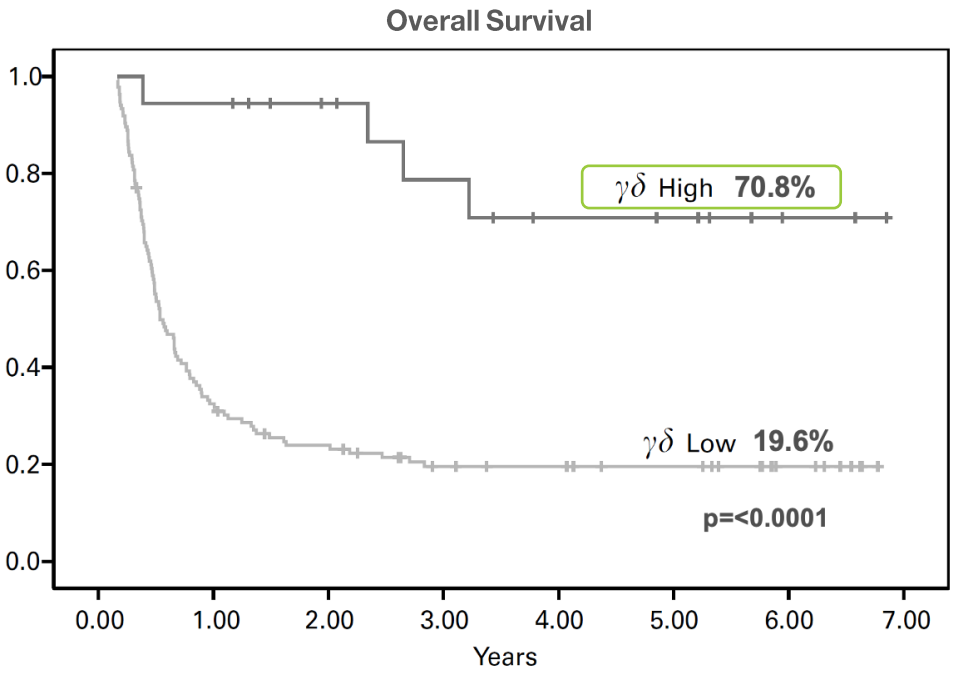
IN8bio Leading the Fight Against Cancer

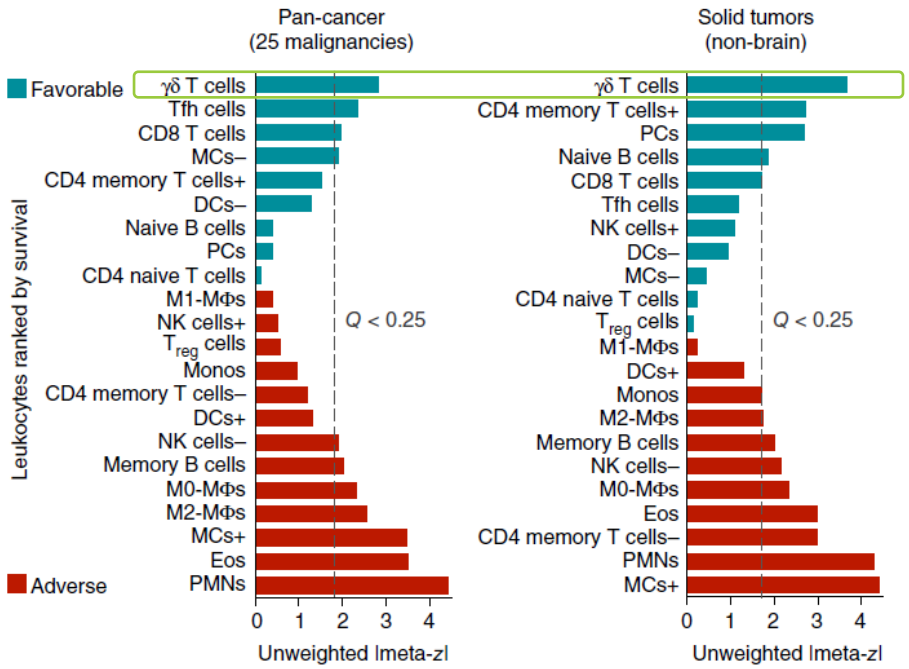
- At IN8bio, our pioneering approach has achieved long-term remissions exceeding 3 years in patients with Acute Myeloid Leukemia (AML) and Glioblastoma (GBM) through two groundbreaking clinical trials
- Unconventional Strategies in the "*War on Cancer*"
  - **Harnessing the Power of Immune Cells:** Our γδ T cells are a "Special Operations Force" that act as direct cancer killers while orchestrating a comprehensive immune response
  - **Precision and Safety:** These cells coordinate and direct the actions of the immune system and identify the locations of friendly forces, enemies, and civilians on the battlefield, which helps to reduce the risk of adverse events and toxicities
  - **Durable Remissions:** With over 30 years of expertise in γδ T cell research, we have pioneered the field; achieving long-term remissions against challenging cancers with significant unmet needs
- Mission **Cancer Zero**™ - Driven by our goal to safely eradicate residual cancer cells, we employ innovative and unconventional strategies to transform treatment outcomes
- IN8bio is redefining cancer treatment with our innovative and novel approaches. Join us in our mission to achieve **Cancer Zero**™ and transform cancer care

γδ T Cells are Key to Better Survival

Leukemia Post-HSCT:
Improved Patient Survival



Pan-Cancer:
Improved Patient Overall Prognosis



Human data demonstrate that γδ T cell levels strongly correlate with improved clinical outcomes

Source: Gentles et al. 2015, Godder et al. (Lamb), 2007 and Meraviglia et al. 2017

IN8bio's Thesis for a Successful Cellular Therapy

Our three-pronged approach to targeting cancers:

Durability

Meaningful **duration of response** can be achieved by increasing the **depth of response** through novel **synergistic combinations**.

Tolerability

Utilize **novel cell types** with a natural ability to identify and kill malignant cells while **preserving healthy tissue** to avoid toxicities seen with other cell therapy approaches.

Heterogeneity

Employ an approach that can leverage **endogenous immune mechanisms** to **cover tumor heterogeneity** and drive broader immune activation.

Robust Pipeline with Multiple Near-Term Clinical Readouts

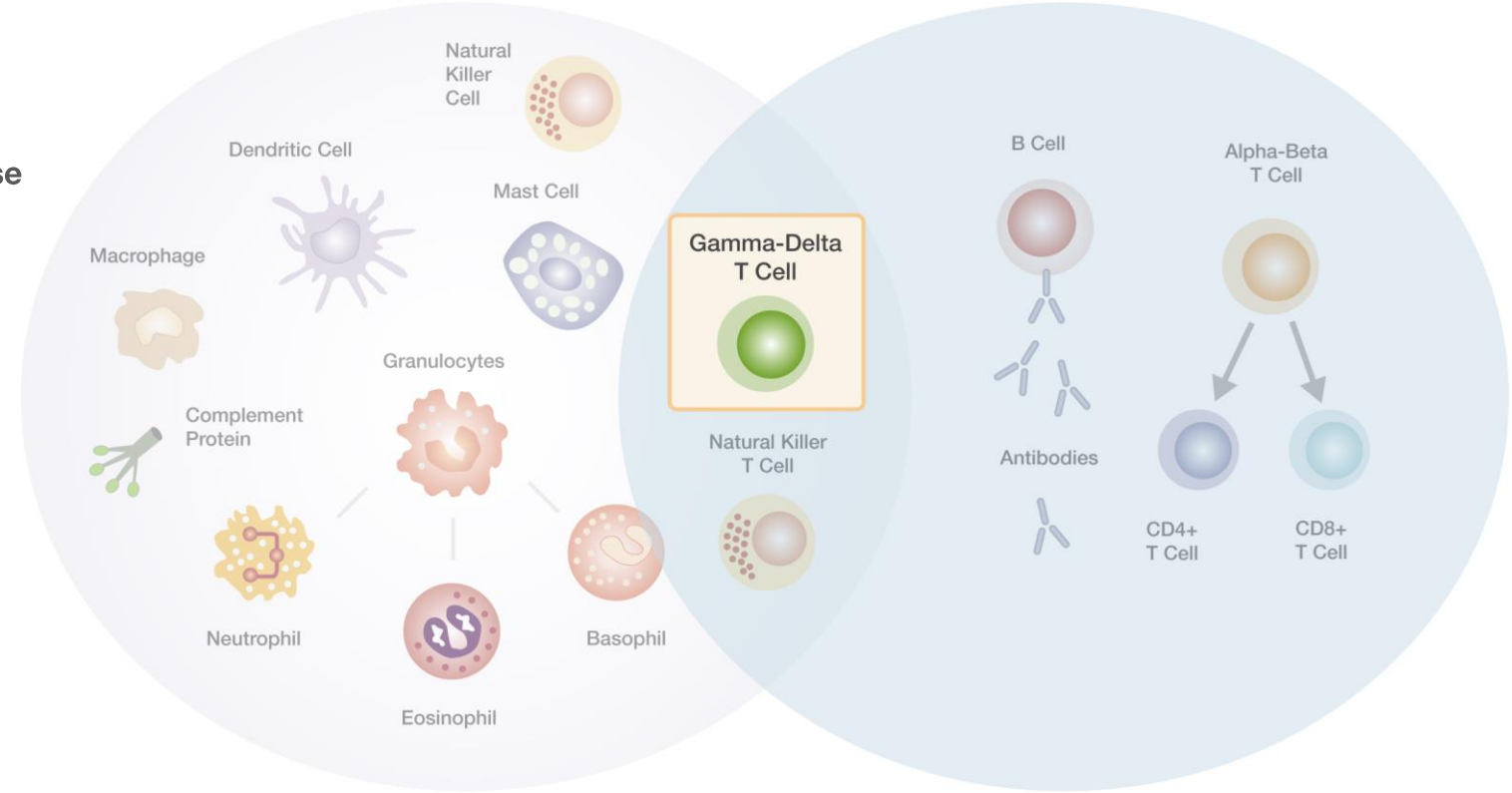
Product Candidate	Approach	Key Indications	Preclinical	Phase 1	Phase 2	Phase 3	Next Anticipated Milestone(s)^
Hematologic Malignancies (Allogeneic)							
INB-100	DeltEx	AML, MDS					• Enroll patients in expansion cohort at DL 2 • Long-term follow-up at medical meetings in 2024 starting at EHA in June 2024 • Potential submission of IND for Phase 2 RCT trial
Solid Tumors (Autologous)							
INB-200	DeltEx DRI*	GBM (1L)**					• Completion of Phase 1 enrollment 1H24 • Long-term follow-up at medical meetings in 2024 starting at ASCO in June 2024
INB-400	DeltEx DRI	GBM (1L)					• Data update at medical meetings in 2025
Solid Tumors (Allogeneic)							
INB-400	DeltEx DRI	GBM (relapsed & 1L)					• Potentially submit IND for Allo Phase 1b in relapsed GBM in 2024
In Development							
INB-300	Non-signaling CAR-T (nsCAR)	TBD					• Updated proof-of-concept data on nsCAR platform targeting AML starting at AACR 2024
INB-500	γδ iPSC T cells	TBD					

* 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

Overview of γδ T Cells

γδ T Cells – Leveraging the Nexus of the Immune System

Innate Immune Response



Adaptive Immune Response

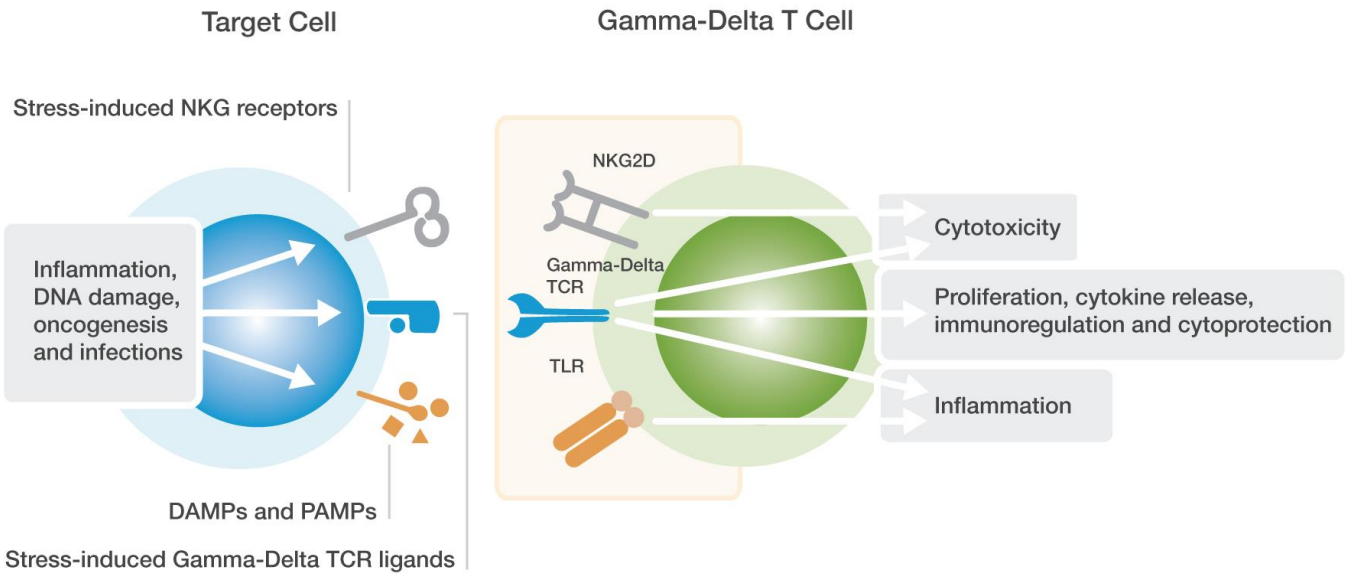
Key Advantages of Gamma-Delta T Cells

- **Persistence of αβ T cells without the toxicities**
- **Safety, recognition and killing abilities of Natural Killer (NK) cells with better durability**
- **Recognizing between healthy and tumor tissues**

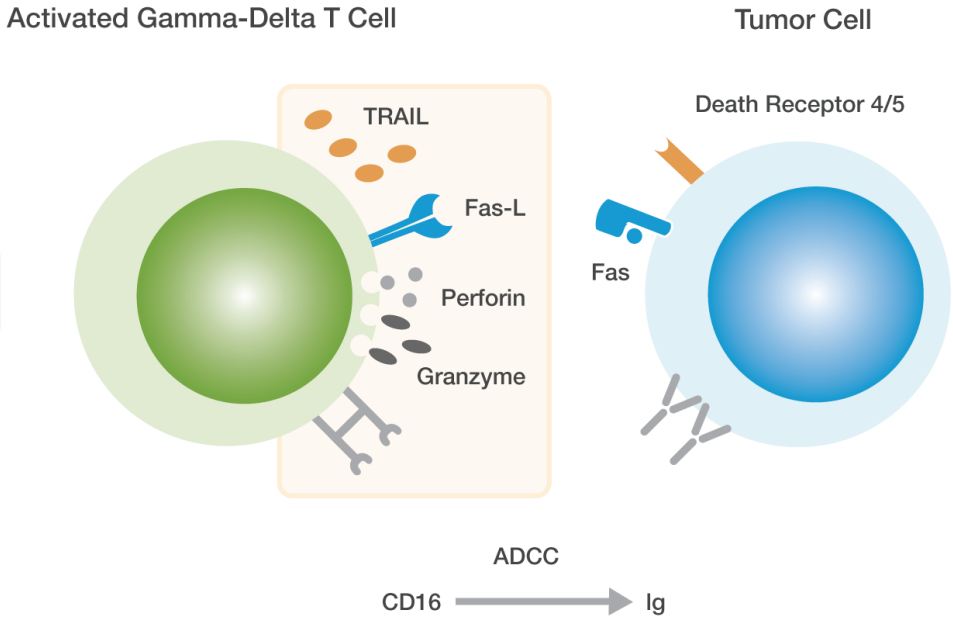
Source: IN8bio – adapted with permission from Dranoff et al. Nature Rev. Can., Jan. 2004, fig 1.

Multiple Weapons, Multiple Targets for Cancer Treatment

Sensing Cellular Stress with Gamma-Delta T cells

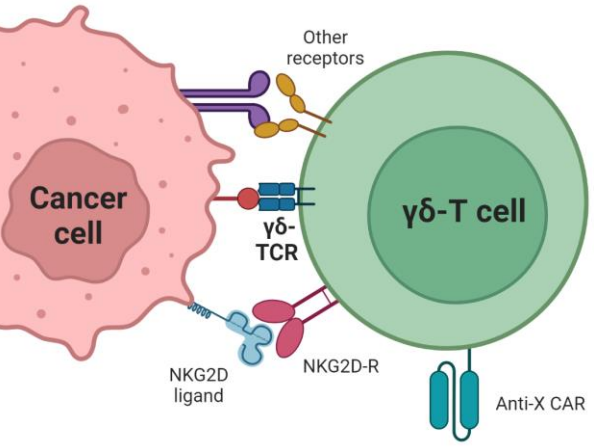


Effector Functions of Gamma-Delta T cells

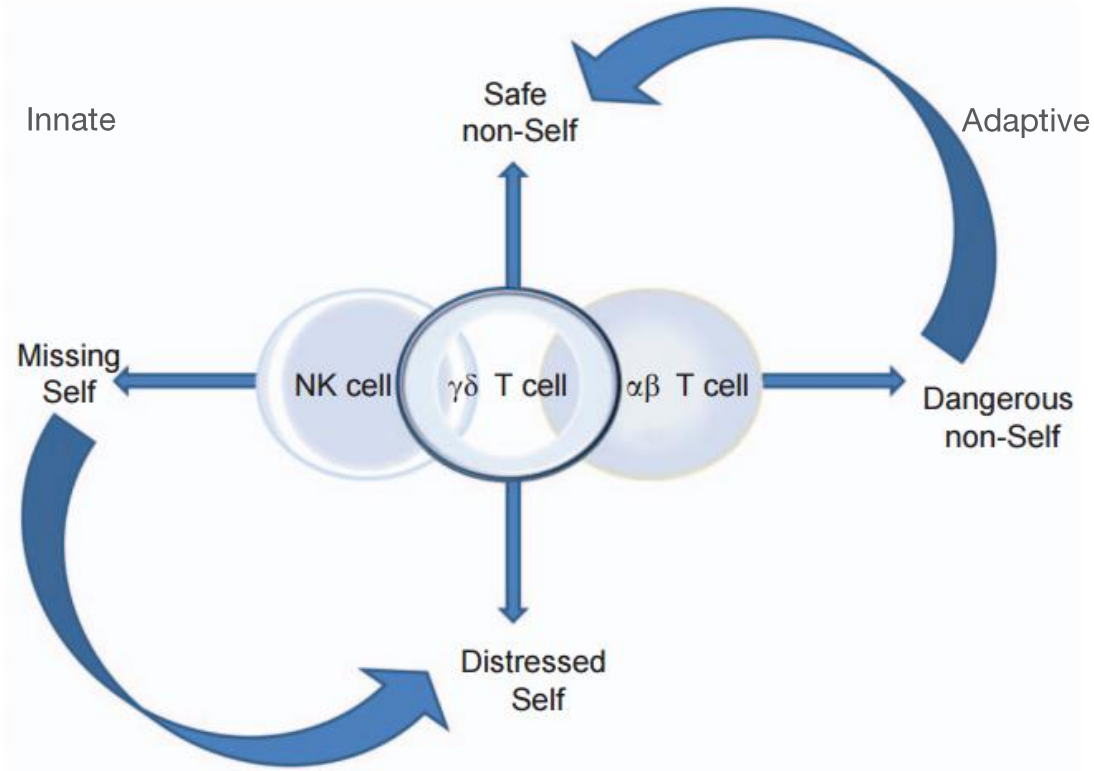


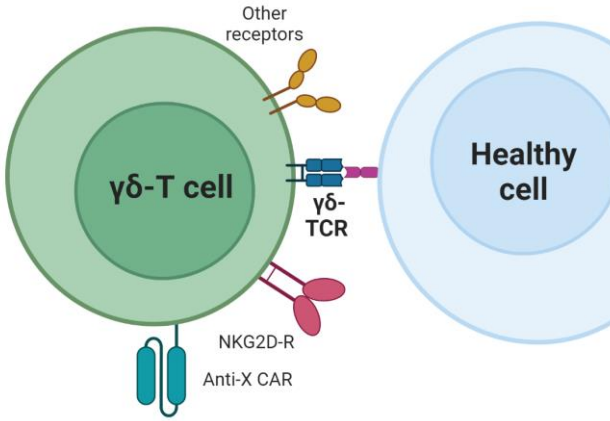
Source: IN8bio – adapted with permission from Dranoff et al. Nature Rev. Can., Jan. 2004, fig 1. and Lafont, V et al. Frontiers in Immunology 2014, Fig 1.

γδ T Cells Possess Unique Capability to Distinguish Healthy Cells



Kill





No-Kill

Potentially widens the therapeutic index, which will be required to successfully target solid tumors

Source: Adapted from Hayday et al., Gamma-delta T Cell Conference, Lison 2023; Kablitz et al. Cellular & Molecular Immunology, 2013, 10, 21-29 fig 1.

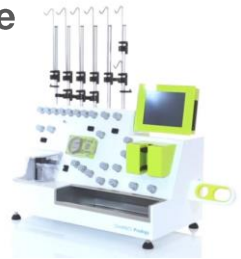
Our DeltEx Platform

γδ T Cell Engineering and Manufacturing

Manufacturing Primary γδ T Cells

Clinical Manufacturing for INB-100, -200, -400

- Automated, robust and scalable cell manufacturing in a single, closed system to increase output and reduce risks of contamination
- Designed for efficient scaling for clinical trials and commercial capabilities
- IN8bio's technology can generate **autologous, allogeneic and/or genetically modified GMP gamma-delta T cells at clinical scale**



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Process Steps:

Obtain peripheral blood

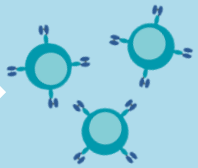
A

Preparation of starting material

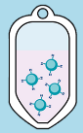
Activation

Transduction (Optional)

Expansion



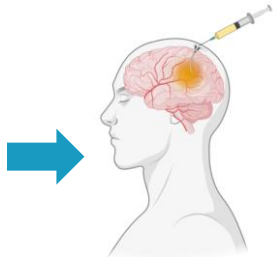
Cell separation



DeltEx cell product

B

Cryopreservation



Patient infusion

Process time: 9-14 days on average

Source: IN8bio, Miltenyi, and biorender.com

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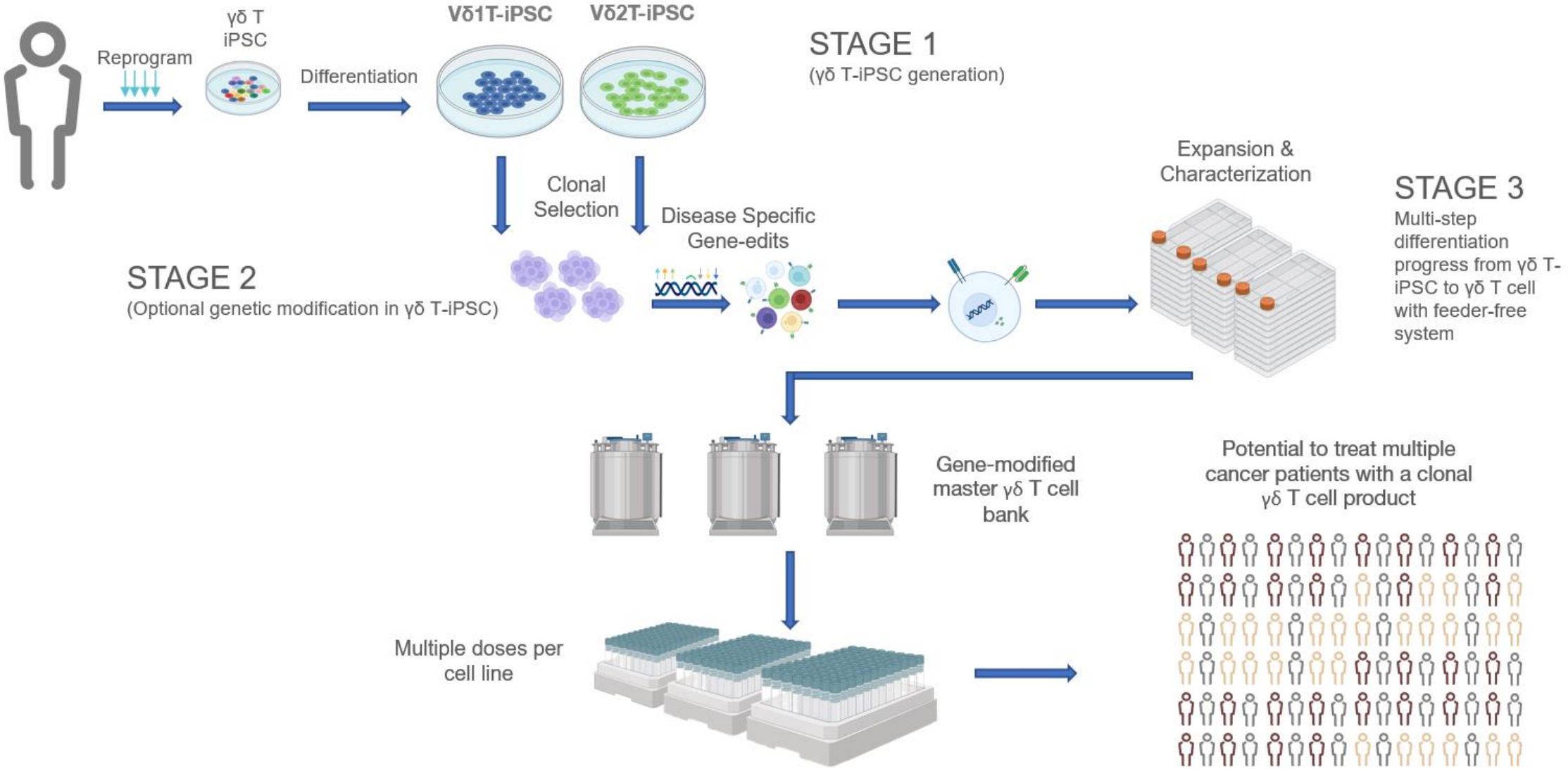
Manufacturing iPSC γδ T Cells



Source: IN8bio; created with biorender.com

IN8bio Possesses a Comprehensive γδ T Cell Platform

γδ T Cell Sourcing

Vδ1+

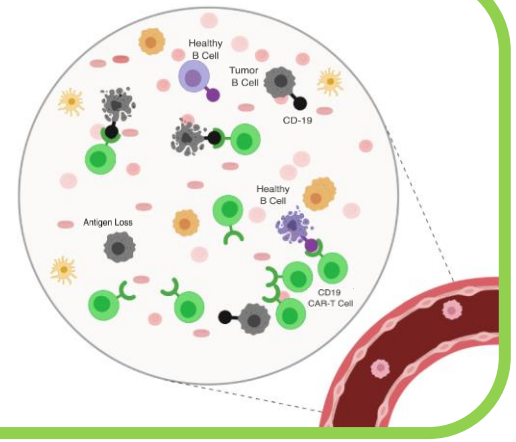
Vδ2+

iPSC

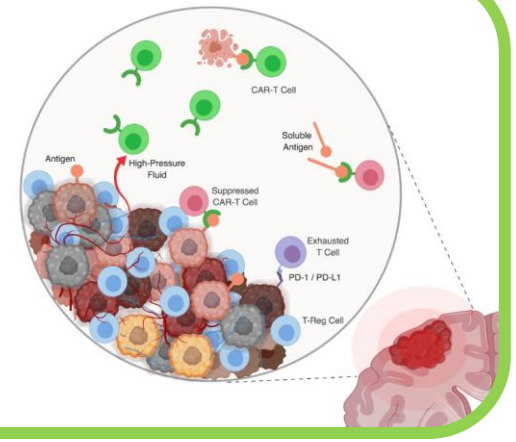
γδ

Tumor Targeting

Hematological Cancers



Solid Tumor Cancers



Source: IN8bio, image created with Shutterstock and biorender.com

INB-100

Haploidentical Stem Cell Transplantation (HSCT)

Relapse is the biggest HSCT problem

- Haploidentical transplants and reduced intensity conditioning (RIC) regimens have expanded access to stem cell transplantation
- **Relapse remains the biggest risk post-transplant with a ~51% risk of relapse at 1-year**
- Gamma-delta (γδ) T cells are an inherent anti-cancer immune cell that may be able to preempt relapse in the post-transplant setting
- γδ T cells respond to stress ligands expressed on tumor cells to eliminate residual leukemia

HLA-Haploidentical Bone Marrow Transplantation for Hematologic Malignancies Using Nonmyeloablative Conditioning and High-Dose, Posttransplantation Cyclophosphamide

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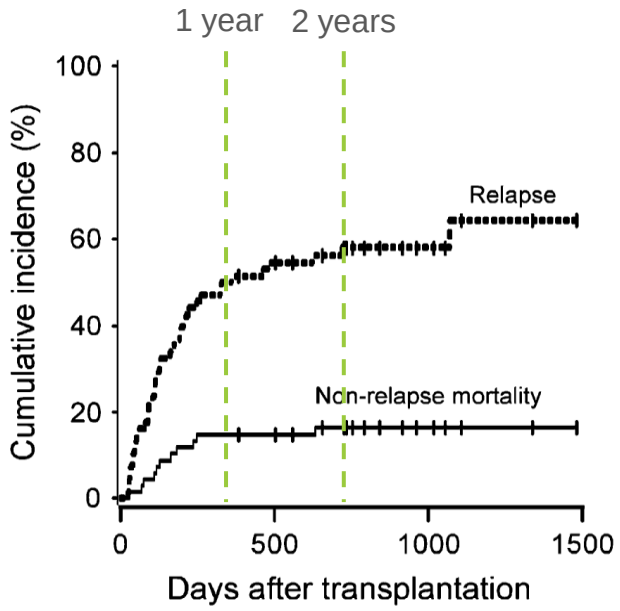
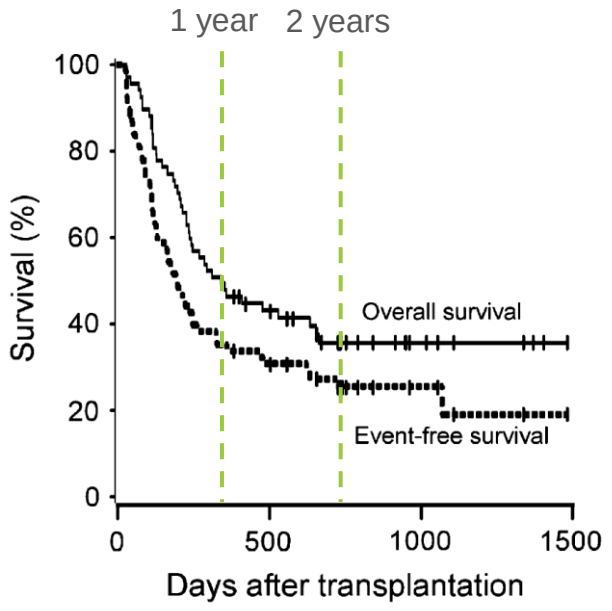
[1] Sidney Kimmel Comprehensive Cancer Center at Johns Hopkins, Baltimore, Maryland; [2] Fred Hutchinson Cancer Research Center, Seattle, Washington; and [3] University of Washington School of Medicine Seattle, Washington





Source: 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: An Allo Therapy to Reduce Leukemic Relapse

Single-center, 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
3. ~~N = 3 (up to 6) patients, single dose of 1 x 10^7 cells/kg~~

RP2D*

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
- Maximum tolerated dose (MTD) 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

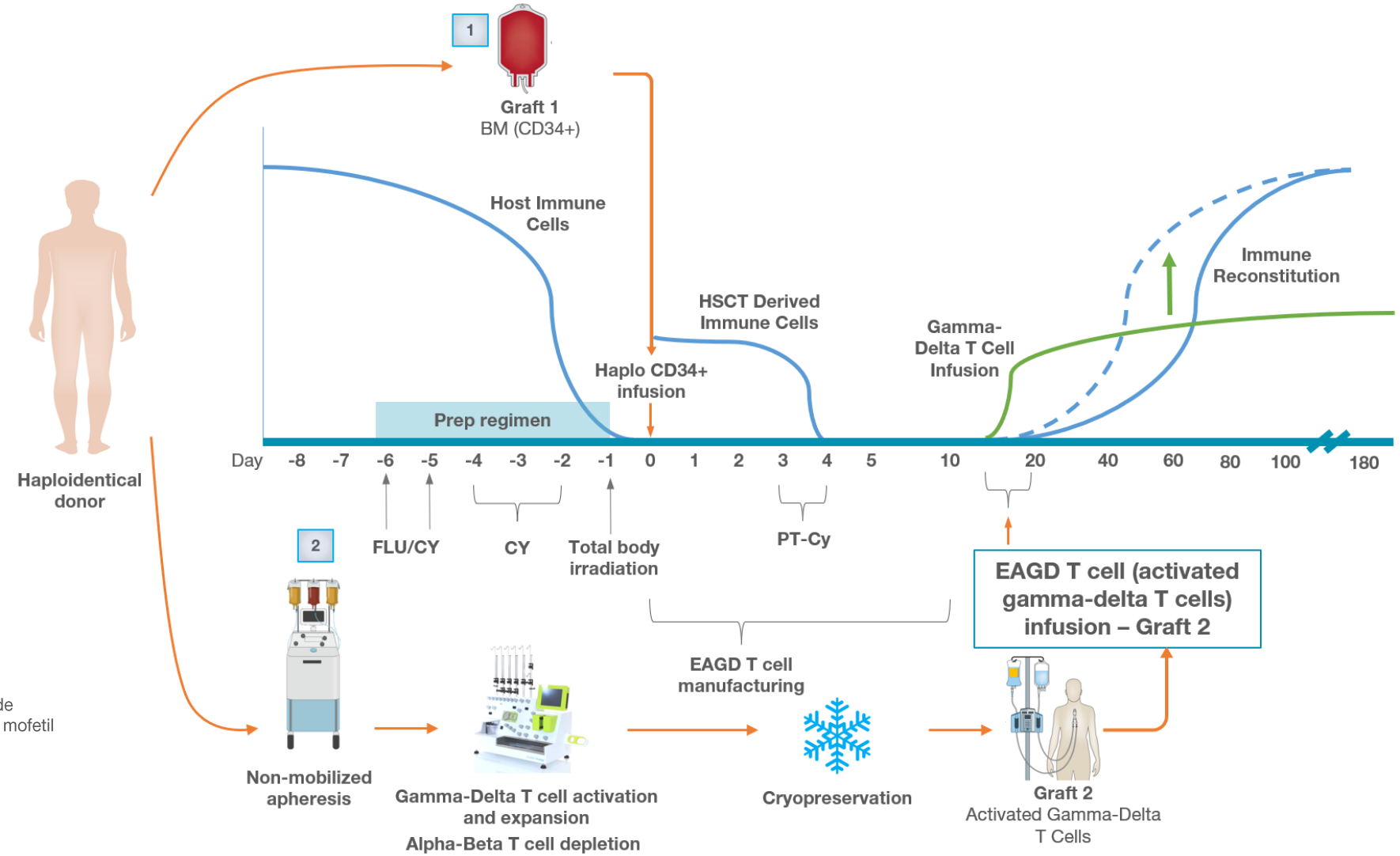
Site

THE UNIVERSITY OF KANSAS CANCER CENTER

*RP2D = Recommended Phase 2 Dose

Potential to Provide Protection During a Vulnerable Period

Expanded + activated gamma-delta T cells (EAGD) to prevent leukemic relapse



FLU = Fludarabine
CY = Cyclophosphamide
MMF = Mycophenolate mofetil

Patient Demographics and Summary

Patient	Dose Level	Age / Sex	Prior Therapies	Disease	Acute / Chronic GvHD	CR (mos)	OS (mos)
002	1	63 / female	Idasanutlin + 7+3	**High-risk AML trisomy 8+ and del7, FLT3 TKD**	Acute G2 skin GvHD Chronic limited mild skin GvHD	**49.6+**	**Alive**
003	1	44 / female	7+3	**High-risk AML trisomy 8+ and del7, IDH2**	Acute G2 GI, Acute G2 rash GvHD	**42.4** **LTFU**	**Alive**
006	1	66 / male	7+3 IDAC	**High-risk relapsed AML**	Acute G2 rash GvHD Chronic extensive GvHD	**35.5+**	**Alive**
007	1	71 / male	Ven/Aza+Pembrolizumab	AML	Acute G2 rash GvHD Chronic limited mod GvHD	**15.5+**	**15.5 died due to IPF**
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 rash GvHD	**14.7**	**Alive at 19.1+**
010	2	63 / female	7 cycles Venetoclax/Aza	AML	Acute G2b rash - GvHD	**18.9+**	**Alive**
011	2	68 / male	Hydrea/Peg-IFN	ET with **MDS/MPN overlap; TP53 mutated**	Acute G1 rash - not GvHD Acute G1 diarrhea - not GvHD	**12.5**	**Alive at 16.0+**
012	2	69 / male	2 cycles Venetoclax/Aza	AML		**12.5+**	**Alive**
013	2	71 / female	1 cycle Ven/aza/gliteritinib 2 cycles Venetoclax/Aza	AML, **FLT3**	Acute G1 diarrhea - not GvHD Oral sensitivity- not GvHD	**12.2+**	**Alive**
014	2	71 / male	Venetoclax/Dacogen	AML, del20, -Y	Acute G1 diarrhea - not GvHD Acute G1 rash - not GvHD	**11.8+**	**Alive**

Average patient age ~68 y/o

Majority have AML

Received up to 7 prior therapies

14 enrolled, n=10 dosed and evaluable for safety

- 1 patient expired prior to dosing
- 1 patient received an out of specification product at 6 x 10^5 EAGD/kg
- 1 manufacturing failure
- 1 screen failure due to relapse prior to treatment

Median follow-up = 17.4 mos

Note: As of May 31, 2024; Early trial results are not indicative of future results, including the outcome of this trial.

Treatment Emergent AE's in ≥ 20% of Patients (n=10)

Adverse Events	Total (%)	Grade 1/2 (%)	Grade 3 (%)	Grade 4 (%)
Platelet count decreased	100	40	60	
WBC decreased	90	50	30	10
ANC decreased	80	40	10	30
ALC decreased	60		40	20
Anemia	90	50	40	
Hypomagnesemia	60	60		
Creatinine increased	50	50		
Hyperglycemia	20	10	10	
Hypokalemia	40	40		
Hyponatremia	40	40		
Hypertension	30	30		
Hypotension	20	20		
Nausea	20	10	10	
Vomiting	20	20		
Diarrhea	20	20		
Dry Mouth	40	40		
Decreased appetite	20		20	
Peripheral edema	20	20		
Peripheral sensory neuropathy	20	20		
Dyspnea	30	30		
Insomnia	20	20		
Pollakiuria	20	20		
Rash maculopapular	60	50	10	

No DLT's, CRS or ICANS to date

2 patients with CMV reactivation

Treatment-related SAE's:

- G2 Rash maculopapular
- G3 Nausea (aGvHD 2B GI)

Other non-treatment related SAE's include:

- G3 Acute Kidney Injury
- G3 Anemia
- G3 CMV reactivation
- G3 Fall
- G3 Decreased appetite

Low rates of infections

No treatment-related deaths

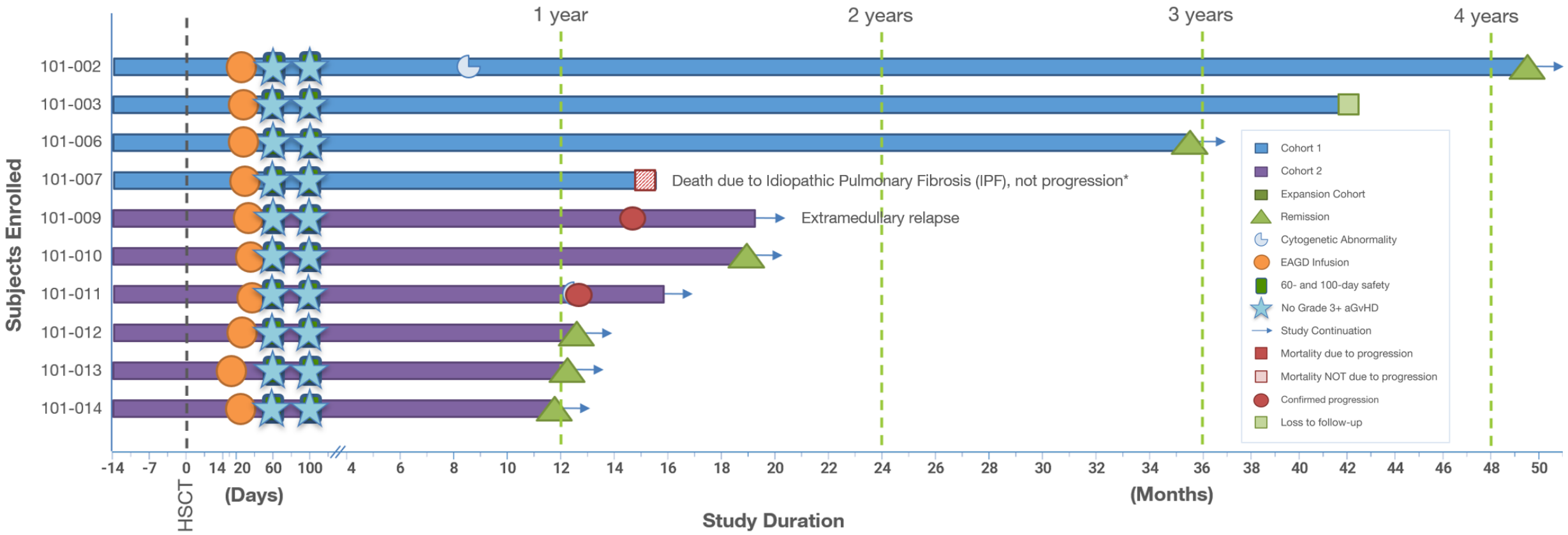
No SUSAR's or unexpected safety events

No change in AE profile from DL1 to DL2

Note: As of May 15, 2024 with no new safety signals noted since May 15th DCO; Early trial results are not indicative of future results, including the outcome of this trial.

100% Patients Remained in Morphologic CR ≥ 12 Months*

Three patients with high-risk disease remain relapse free for >35 months with median follow-up 17.4 months



Note: *POD = progression of disease;
*As of May 31, 2024; Early trial results are not indicative of future results, including the outcome of this trial.

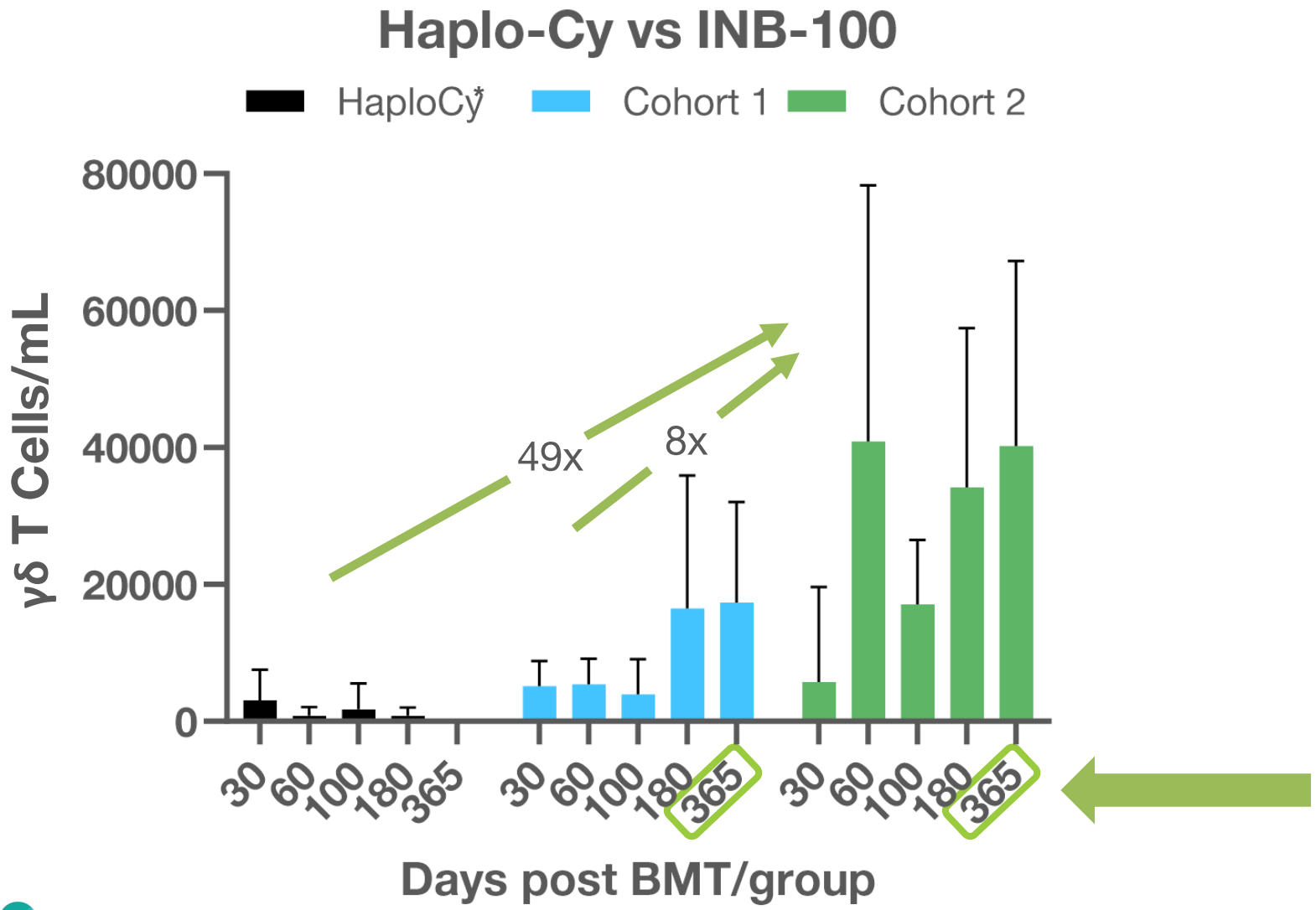
Chimerism Data Confirms 1-year RFS for 10/10 Patients

	Dose Level 1				Dose Level 2 - RP2D					
	101-002	101-003	101-006	101-007	101-009	101-010	101-011	101-012	101-013	101-014
Infusion	●	●	●	●	●	●	●	●	●	●
Day 30	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓
Day 60	✓	✓	✓	✓	✓	✓	✓	✓	na	✓
Day 100	✓	✓	na	✓	✓	na	✓	✓	✓	✓
Day 180	⬢	na	✓	✓	✓	✓	✓	✓	✓	✓
Day 365	✓	na	✓	✓	✓	✓	⬢	✓	✓	✓
Morphologic CR @ 1yr	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓

- Cohort 1
- Cohort 2
- EAGD Infusion
- Cytogenetic CR
- Morphologic CR
- Mixed chimerism requiring clinical intervention

Note: *As of May 31, 2024; Early trial results are not indicative of future results, including the outcome of this trial.

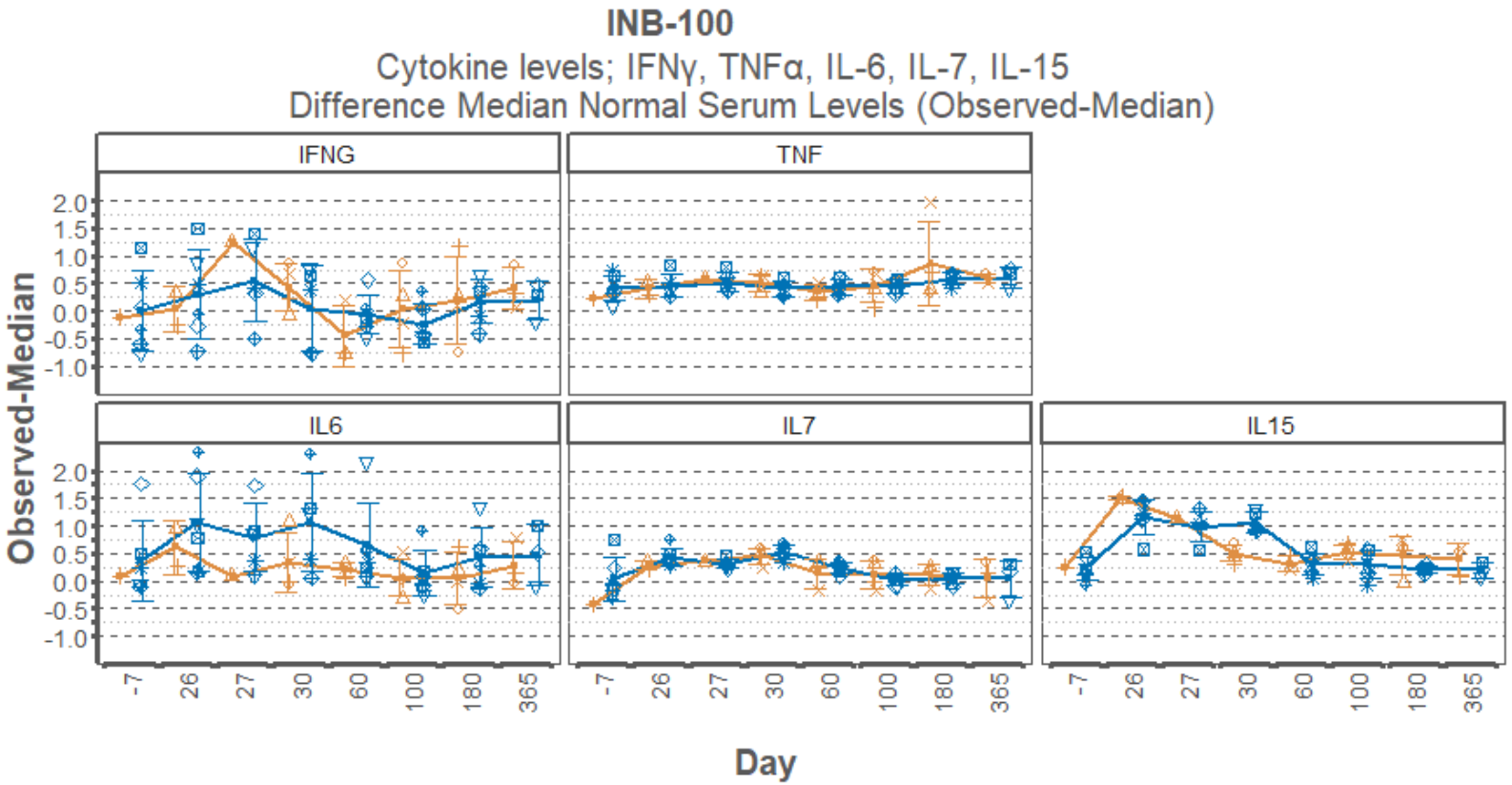
One-Year *In Vivo* Persistence and Expansion of γδ T Cells



- Comparison of γδ T cell count recovery between patients who received haploidentical BMT + post-BMT Cy without γδ T cell infusion and INB-100 patients from Cohort 1 and Cohort 2
- Dose dependent increase of circulating γδ T cells at Days +60, +100, +180 and +365 for INB-100 treated patients
- Despite Cohort 2 patients receiving 3x the γδ T cell dose as Cohort 1, an 8x increase in γδ T cells was observed at 60 days
- Continued presence at 365 days suggests **in vivo expansion AND persistence** of cells

Source: IN8bio, Inc. and UAB *previously unpublished data from laboratory of Dr. Lawrence Lamb; as of May 31, 2024

Immune Recovery: Serum Cytokine Profile



- Following infusion of the γδ T cells, there is a decrease in IL-6, and IL-7, which increases post-BMT, indicating a positive impact of γδ T cells on immune function and a reduction in inflammatory cytokines.
- IFN-γ levels increase following γδ T cell infusion, suggesting immune activation and activity

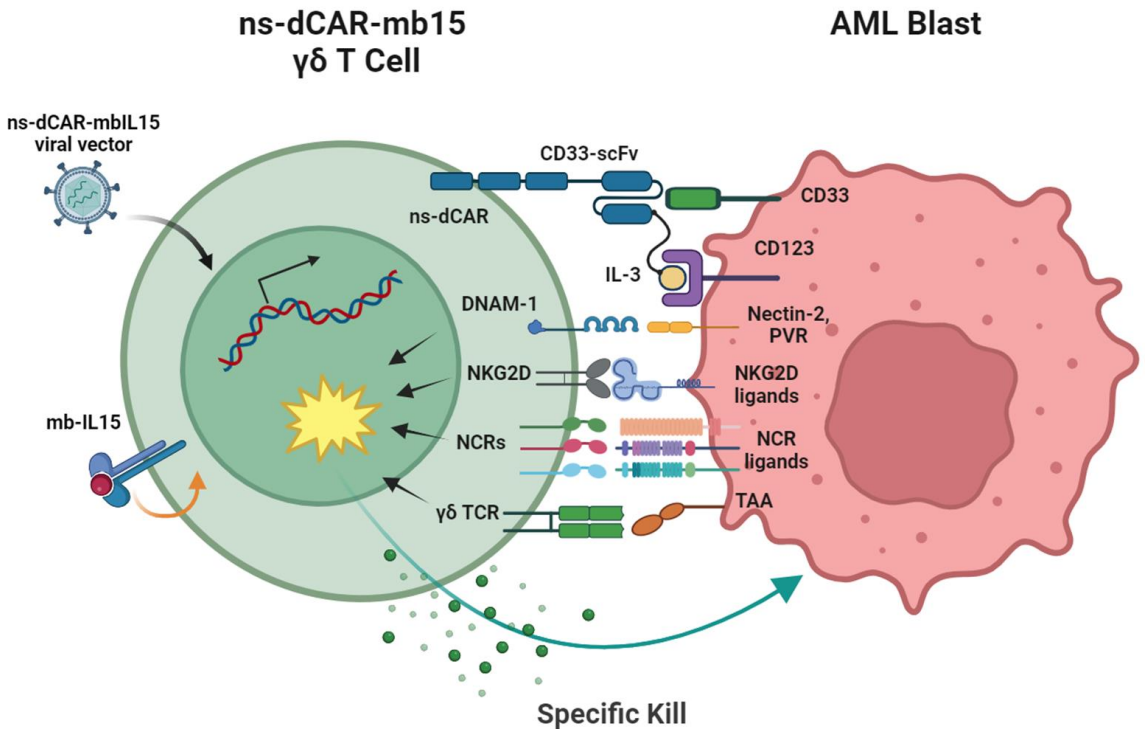
Source: IN8bio, Inc.; as May 31, 2024

INB-300

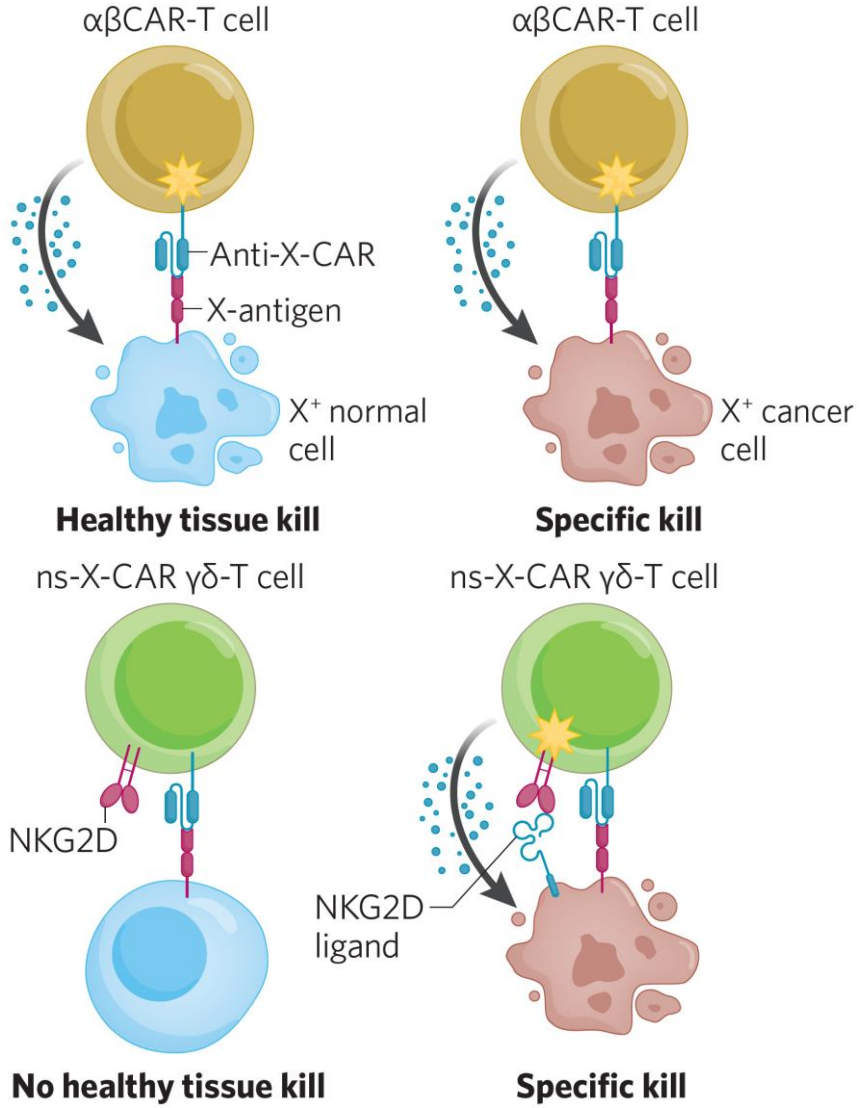
Novel nsCAR γδ T Cell Platform

A Unique CAR-T Platform that Spares Healthy Tissue

Novel Non-Signaling γδ CAR-T Platform (ns-CAR)

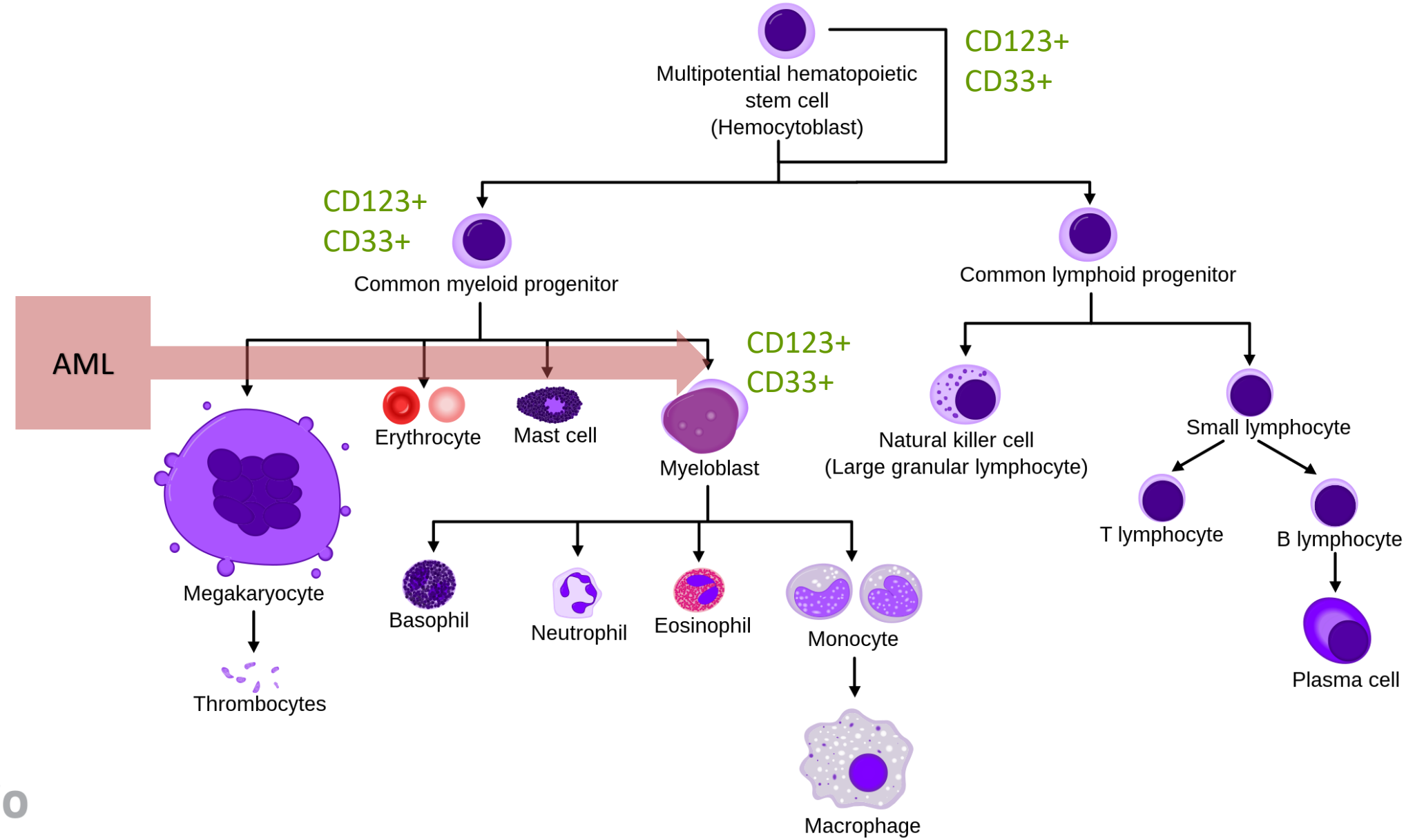


- γδ T cells have a broad-based MHC unrestricted receptor repertoire that can identify and distinguish healthy from stressed cells (infected or transformed) to be targeted for killing



(1) Dardevet et al., Toxins (Basel), 2015 Apr; 7(4): 1079-1101 and Cancer Res November 1 1998 (58) (21) 4871-4879; Nature Biopharma Dealmakers 2023

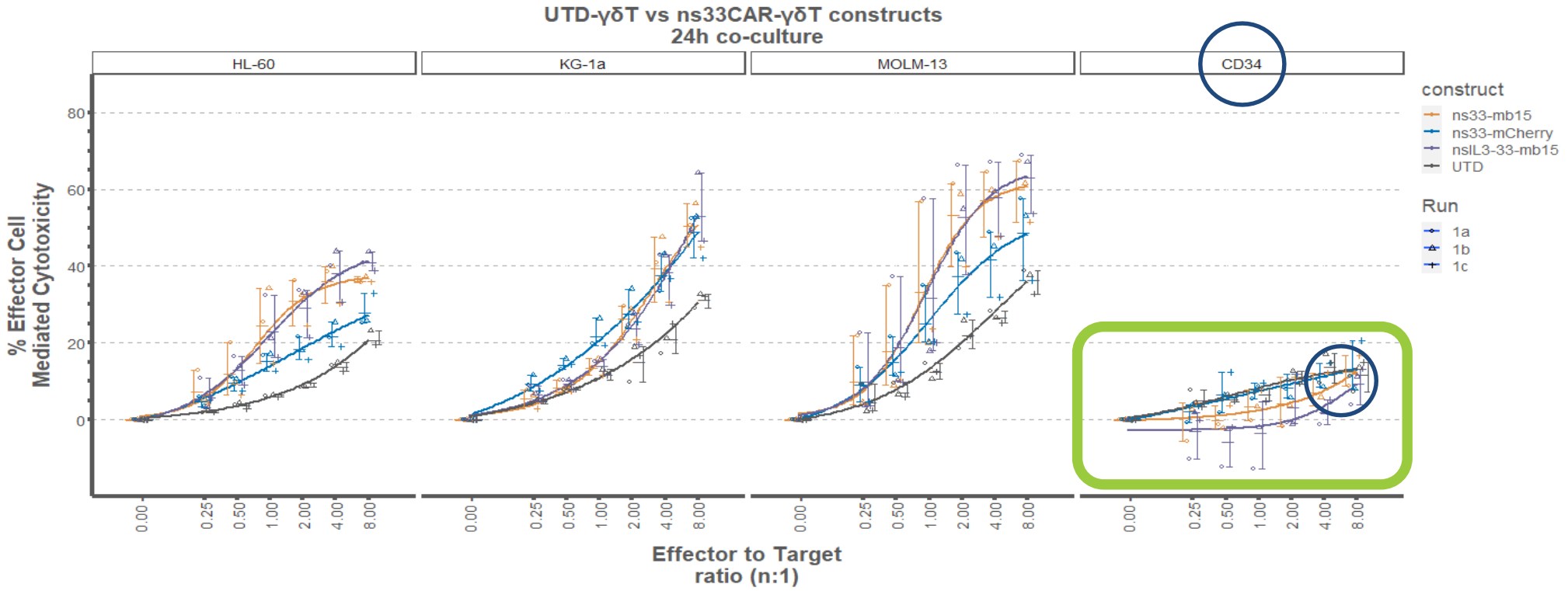
Overview: Hematopoiesis and AML



ns-γδ T CARs Do Not Increase Killing vs. Healthy Cells

Presented at AACR 2024 - CD34+ HPC, HL-60, KG-1a, MOLM-13 are all CD33+ cells

- Cytotoxicity of nsIL3-33mb15 nsCAR against AML cell lines was 5.5x greater than against healthy CD34+ hematopoietic progenitor cells (HPCs)
- Experiments run in triplicate
- nsCAR constructs demonstrated an average 1.8x increase in killing across three AML cell lines at peak
- nsCAR killing was less than untransduced control γδ T cells across all constructs



Source: IN8bio, Inc.

Targeting Solid Tumor Cancers

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

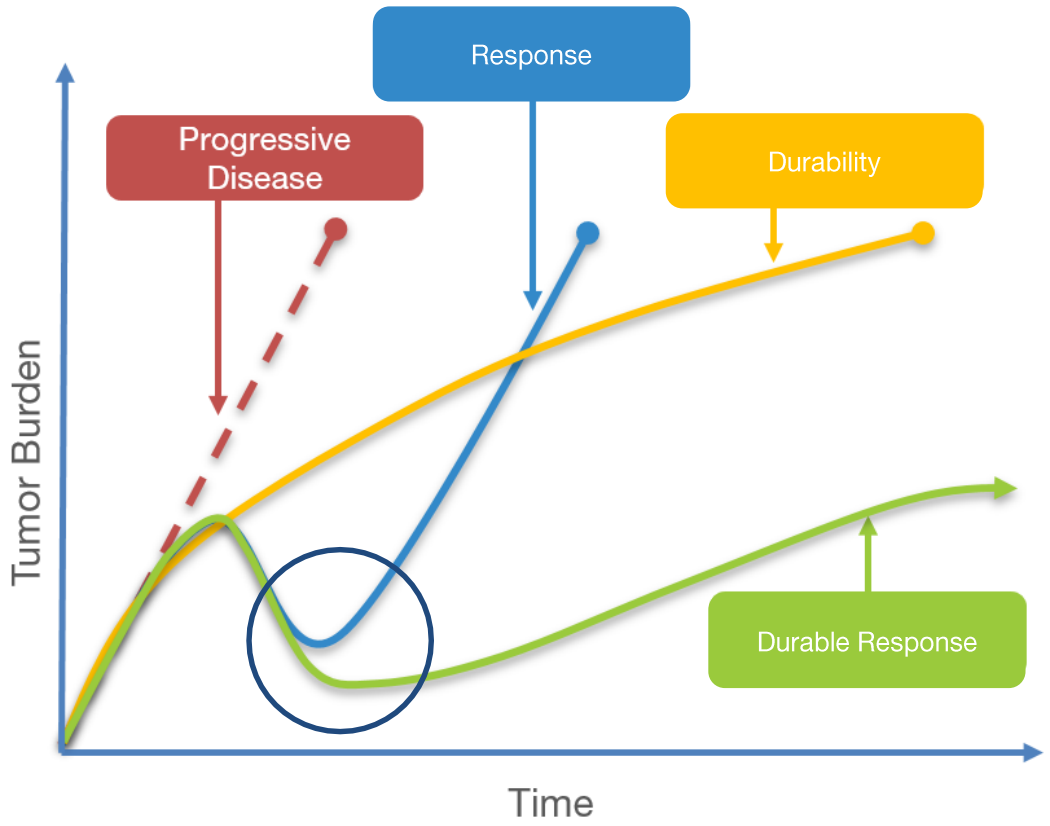
Trafficking of T cells into Tumors

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

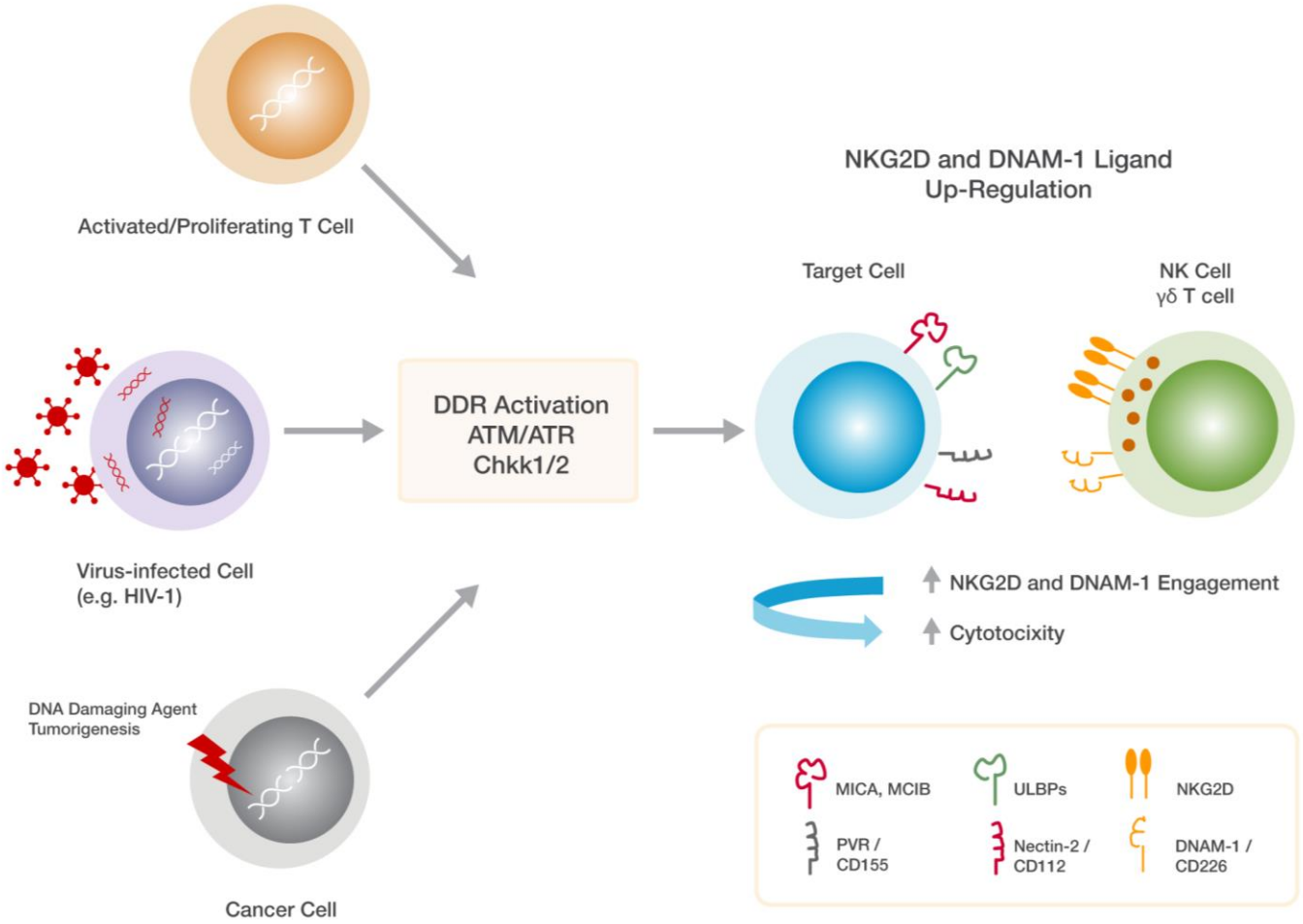
Targeting Cancers by Driving Deeper Responses



γδ T cells Genetically Engineered to Survive Chemotherapy Induced Cell Death

Source: Adapted from Madan et al. Sem in Onc. 2012, Vol 39, No 3 and Ribas et al. Clin Canc Res, 18(2), Jan. 2012

Stimuli that Can Up-regulate NKG2D AND DNAM-1 Ligands

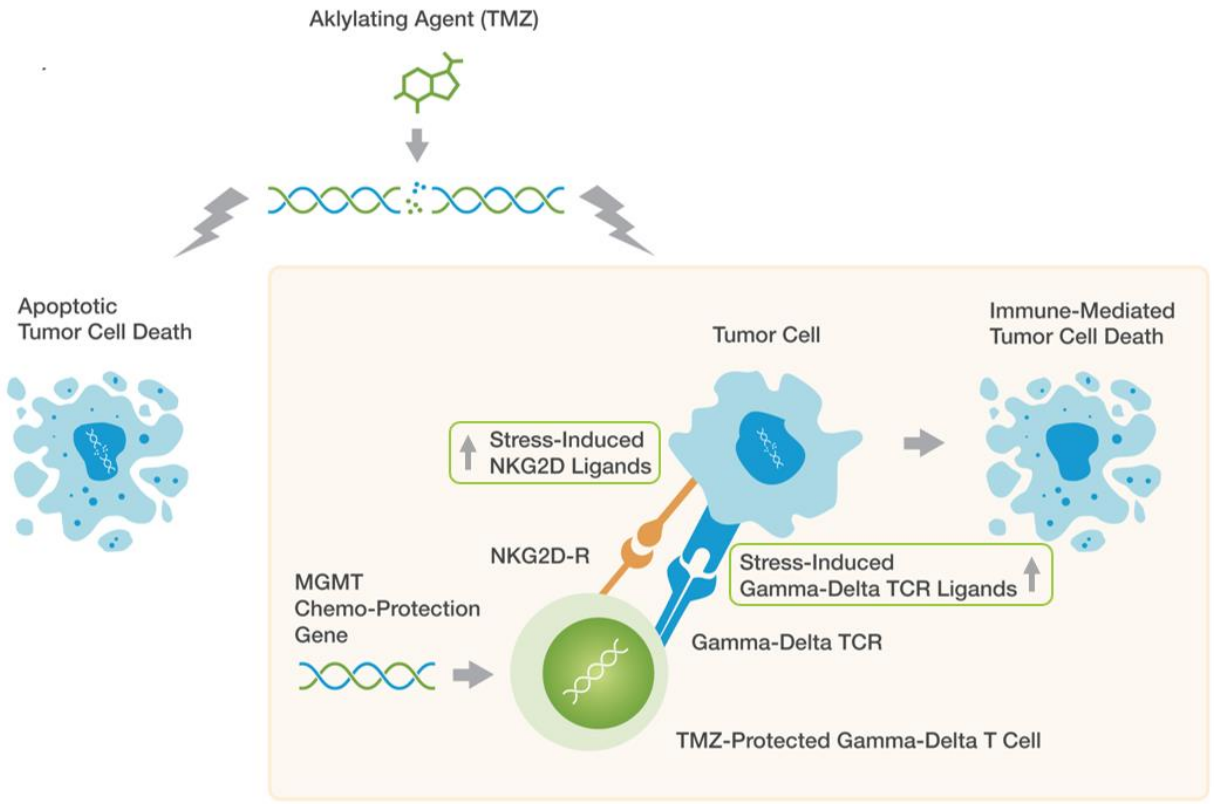


- SCHEMATIC REPRESENTATION OF THE VARIETY OF STIMULI THAT CAN UP-REGULATE NKG2D AND DNAM-1 LIGANDS. There is evidence that both in normal cells (e.g., antigen-activated T lymphocytes), as well as in pathological conditions, including virally-infected cells (in particular with HIV-1) and cancer cells, a major regulatory pathway involved in ligand up-regulation is the DNA damage response (DDR), activated by different stimuli. The increased expression of activating ligands has been shown to be implicated in the recognition and elimination of “stressed” cells by NK cells, and presumably also by other cytotoxic cells (i.e., γδ T cells and CD8+ T cells).

Source: Adapted from Cerboni et al., Front. Immunol., 07 January 2014, https://doi.org/10.3389/fimmu.2013.00508

Targeting the DNA Damage Response (DDR) to Kill Tumors

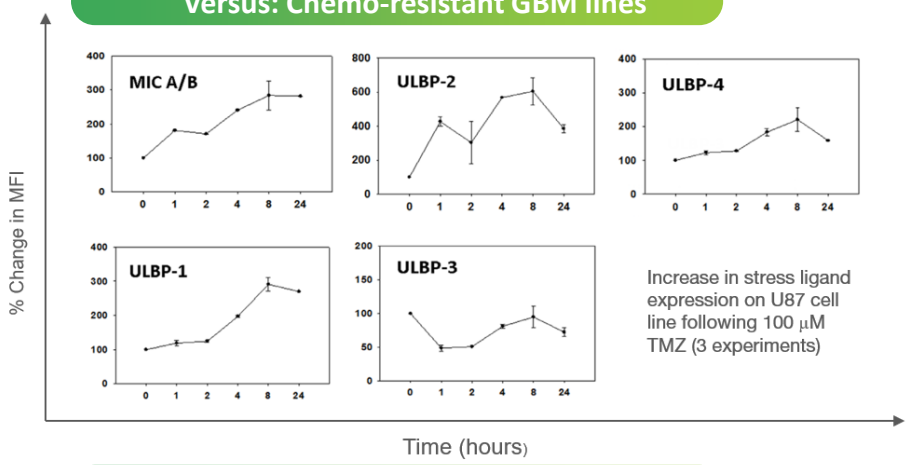
DDR is a biological process that can detect and eliminate cells with DNA damage through increased avidity



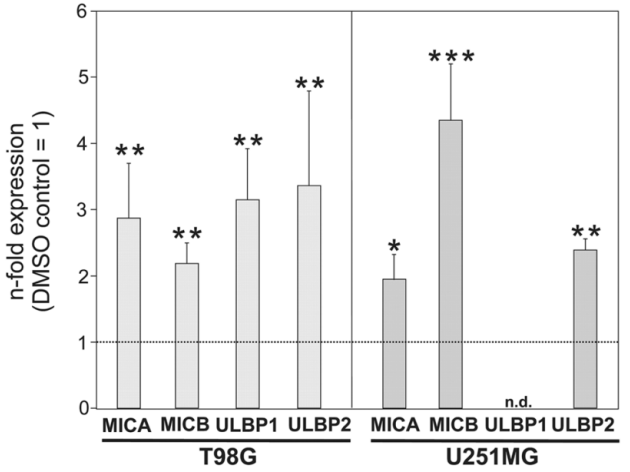
DRI gamma-delta T cell mechanism overview

TMZ Increases NKG2D-L Expression:

Versus: Chemo-resistant GBM lines



Versus: Glioma stem-like cells

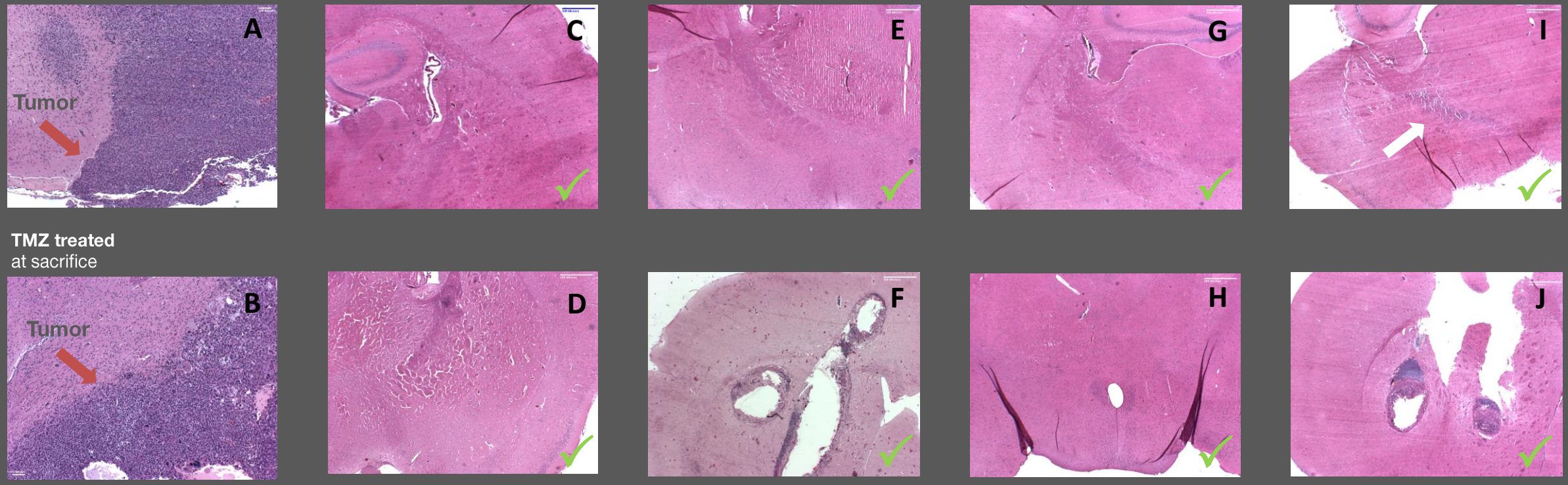


Source: IN8bio, Lamb et al. Plos One, (2013) Vol 8., Issue 1, Held-Feindt et al. Histochemistry and Cell Biology, (2018) 149: 2019-233; https://doi.org/10.1007/s00418-018-1633-5

Targeting the DDR Pathway Eliminates Residual GBM

Untreated
+14 days

TMZ + DRI gamma-delta T cells treated - surviving mice (n=8)



✓ - Long-Term Survival (+150 days)

Figure 1: Digital light microscopic images from athymic nude mice that received intracranial injection of 5 x 10^5 GBM tumor cells derived from classical human patient GBM xenograft JX12P. Images (A) show hematoxylin and eosin (H&E) staining of tumor growth (dark purple - noted by red arrow) from untreated mice at +14 days following stereotactic tumor placement in the left caudate nucleus (4x); (B) was obtained at euthanasia following fatal tumor progression from a mouse treated with temozolomide (TMZ) (4x); (Images: C - J) surviving mice treated with TMZ + DRI γδ T cells, 80% (n=8) demonstrated long-term survival (+150 days) following tumor placement, at the time of euthanasia these mice demonstrated improved survival with no observable neurologic dysfunction and are negative for H&E staining and the white arrow (I) points to scarring where residual necrotic tumor has been cleared.

IN8bio

Source: Lamb et al. Scientific Reports (2021) 11:21133 - https://doi.org/10.1038/s41598-021-00536-8

INB-200

DeltEx DRI Auto for GBM

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.

INB-200: Study Design and Treatment Schema

Treatment Arms

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

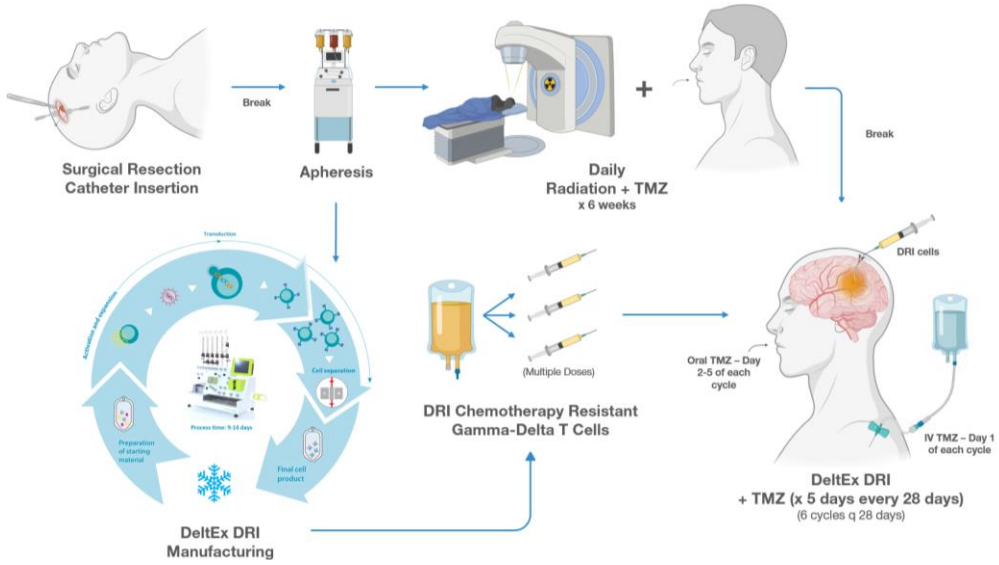
DL1: N = 3 (up to 6) patients, single dose of 1 x 10^7 cells on C1D1
DL2: N = 3 (up to 6) patients, three doses of 1 x 10^7 cells, one dose every 28 D1 of C1-C3
DL3: N = 3 (up to 6) patients, six doses of 1 x 10^7 cells, one dose every 28 days on D1 of C1-C6

Treatment Regimen & Timing

Surgical resection followed by apheresis

6 weeks induction TMZ + radiation

6 cycles maintenance TMZ + DRI



Primary Endpoints

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

Secondary Endpoints

- Time to progression
- Overall survival
- Biologic response

Site

Source: IN8bio, image created with biorender.com

Poor Survival and Standard of Care Hasn't Changed in 18 Years

The NEW ENGLAND JOURNAL of MEDICINE

ORIGINAL ARTICLE

Radiotherapy plus Concomitant and Adjuvant Temozolomide for Glioblastoma

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., Alba A. Brandes, M.D., Christine Marosi, M.D., Ulrich Bogdahn, M.D., Jürgen Curschmann, M.D., Robert C. Janzer, M.D., et al., 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*

- N = 573
- Median age 56 (range 19-71)
- PS 2 only 12%
- RT+TMZ median OS 14.6 months
- RT+TMZ median PFS 6.9 months (95% CI 5.8-8.2)
  - MGMT methylated 10.3 months
  - **MGMT unmethylated 5.3 months**

ORIGINAL ARTICLE

Short-Course Radiation plus Temozolomide in Elderly Patients with Glioblastoma

James R. Perry, M.D., Normand Laperriere, M.D., Christopher J. O'Callaghan, D.V.M., Alba A. Brandes, M.D., Johan Menten, M.D., Claire Phillips, M.B., B.S., Michael Fay, M.B., Ch.B., Ryo Nishikawa, M.D., J. Gregory Cairncross, M.D., Wilson Roa, M.D., David Osoba, M.D., John P. Rossiter, M.B., B.Ch., et al., for the Trial Investigators*

- N = 562
- Median age 73 (range 65-90)
- PS 1 – 54%; PS 2 – 23%
- RT+TMZ median OS 9.3 months
- RT+TMZ median PFS 5.3 months
  - MGMT methylated 7.9 months
  - **MGMT unmethylated 4.8 months**

IN8bio

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

Demographics and Efficacy

Subject	Age / Sex	Cytogenetics	Dose level	Resection	TMZ Maint. Cycles Received	Response	PFS (mos)	OS (mos)
001	68 / M	IDH-WT, MGMT-unmethylated	1	Total	5	SD	8.3	15.6 Died from sepsis
003	74 / F	IDH-WT, MGMT-methylated	1	Total	6	SD	11.9	17.7
004	21 / F	IDH-WT, MGMT-unmethylated	1	Total	3	SD	7.4	9.6
007	74 / M	IDH-WT, MGMT-unmethylated	2	Total	2	Unevaluable	-	5.1 Died w/out progression
009	32 / M	IDH-mutant, MGMT-methylated	2	Total	12	SD	34.9+	Alive
011	56 / F	IDH-WT, MGMT-methylated	2	Total	6	SD	22.2	28.6
014	73 / F	IDH-WT, MGMT-unmethylated	2	Subtotal	6	SD	8.7	8.7 Died w/out progression
015	73 / M	IDH-WT, MGMT-methylated	3	Subtotal	5	SD	7.1	11.8
017	74 / F	IDH-WT, MGMT-methylated	3	Subtotal	3	SD	12.7+	Alive
020	66 / M	IDH-WT, MGMT-methylated	3	Subtotal	6	SD	10.8+	Alive
021	57 / M	IDH-WT, MGMT-unmethylated	3	Total	5	SD	9.2+	Alive
022	53 / M	IDH-WT, MGMT-unmethylated	3	Subtotal	3	SD	6.4+	Alive
023	52 / M	IDH-WT, MGMT-unmethylated	3	Subtotal	1	PD	4.2	5.4

*As of May 1, 2024; Early trial results are not indicative of future results, including the outcome of this trial.

- **Median age: 68**
- **54% unmethylated**
- **23 enrolled, five products unable to be manufactured**
- **Of 13 treated, 5 remain in follow-up**
- **8 deaths:**
  - 7 due to PD or disease-related issues
  - Other:
    - Cardiac event (007)

Patient 009 – Surpassing Expectations for IDH-mut Glioma

The NEW ENGLAND JOURNAL of MEDICINE

RESEARCH SUMMARY

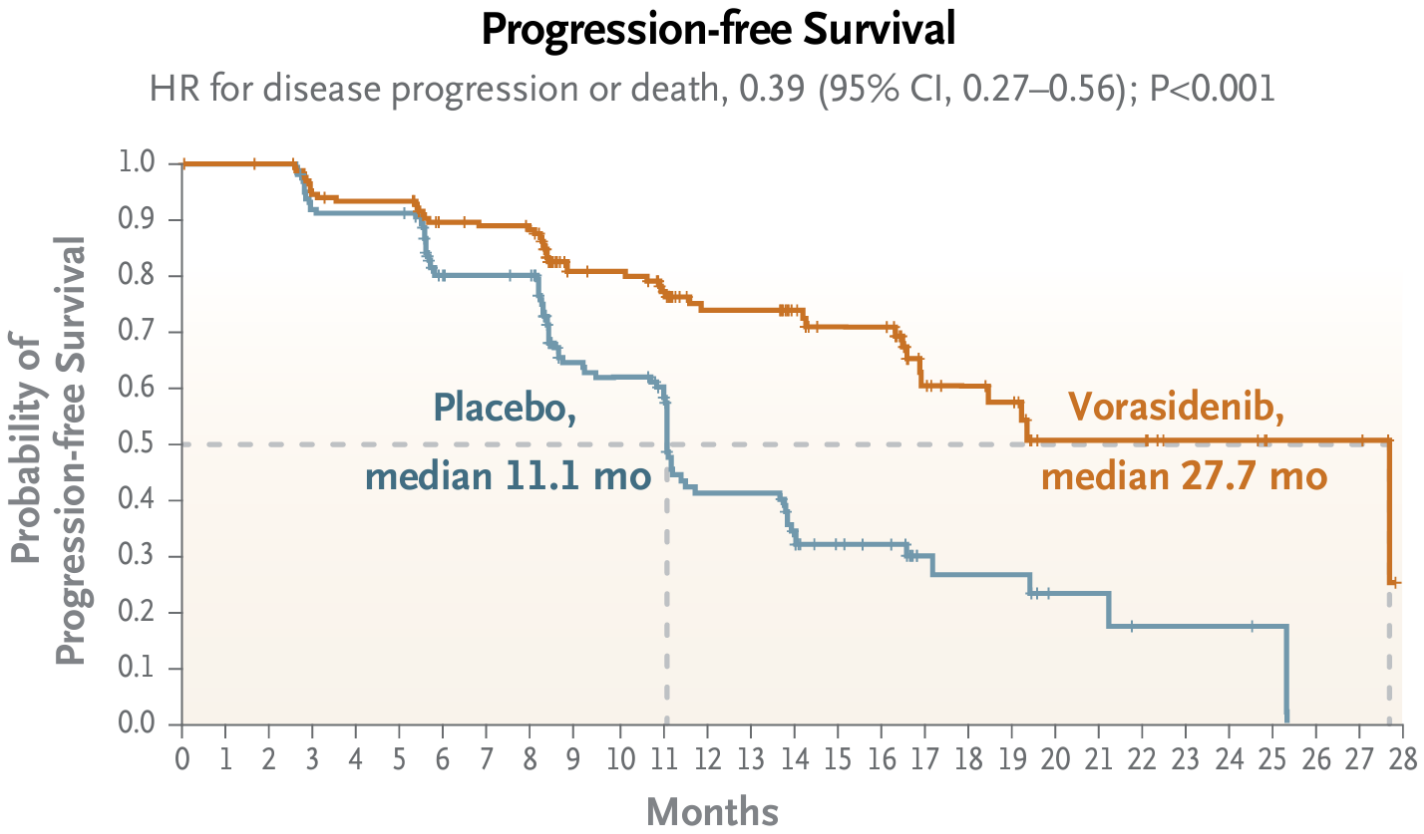
Vorasidenib in IDH1- or IDH2-Mutant Low-Grade Glioma

Mellinghoff IK et al. DOI: 10.1056/NEJMoa2304194

CLINICAL TRIAL

Design: This phase 3, double-blind, randomized, placebo-controlled trial tested the clinical effects of vorasidenib — an oral brain-penetrant inhibitor of mutant IDH1 and IDH2 enzymes — in patients with residual or recurrent grade 2 IDH-mutant glioma who had undergone surgery as their only previous treatment.

Intervention: 331 patients were assigned to receive oral vorasidenib (40 mg once daily) or matched placebo in 28-day cycles. The primary end point was imaging-based progression-free survival.



Safety and Adverse Events (n=13)

Serious Adverse Events	All Grades	≥ Grade3
Cardiac Arrest	7.7%	7.7%
Cardiac Disorder	7.7%	7.7%
Platelet Count Decreased	15.4%	15.4%
WBC Count Decreased	7.7%	7.7%
Hydrocephalus	15.4%	7.7%
Dysarthria	7.7%	7.7%
Pulmonary Embolus	7.7%	7.7%
Cyst Drainage	7.7%	7.7%
Deep Vein Thrombosis	7.7%	7.7%
Fall	7.7%	7.7%

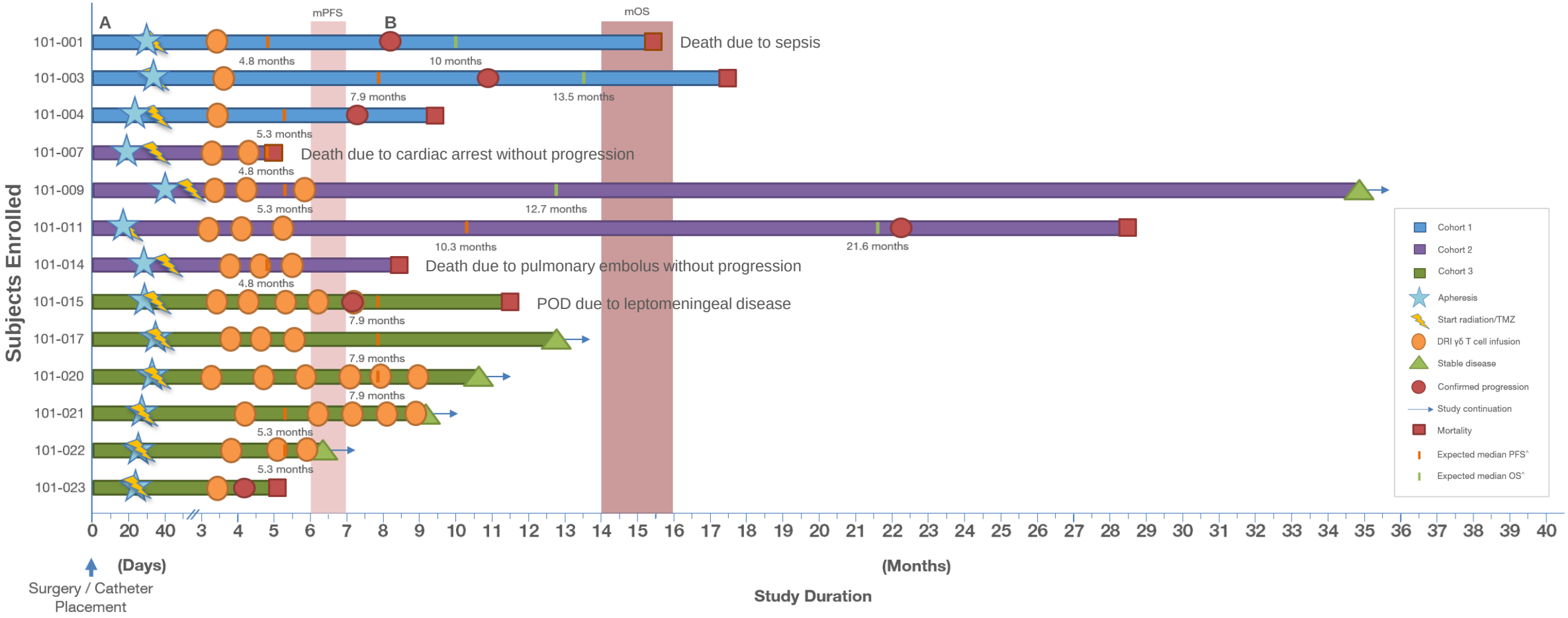
Adverse Events	All Grades	≥ Grade3
Decreased Appetite	15.4%	
Balance Disorder	15.4%	
Headache	15.4%	
Hydrocephalus	15.4%	7.7%
Platelet count decreased	23.1%	23.1%
WBC count decreased	23.1%	7.7%
Lymphocyte count decreased	7.7%	7.7%
Neutrophil count decreased	7.7%	7.7%
Asthenia	15.4%	
Fatigue	15.4%	
Urinary tract infection	15.4%	
Deep Vein Thrombosis	15.4%	

- No DRI-related toxicity
- **No DLT's 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**
- **No change in safety profile observed to date following repeat administration of up to six doses**

*As of May 1, 2024; Early trial results are not indicative of future results, including the outcome of this trial.

92%* Exceeding Stupp Regimen Median PFS of 7 months

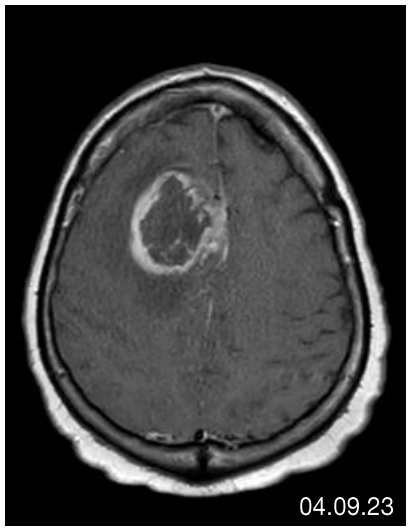
Median Follow-up: 10.8 months

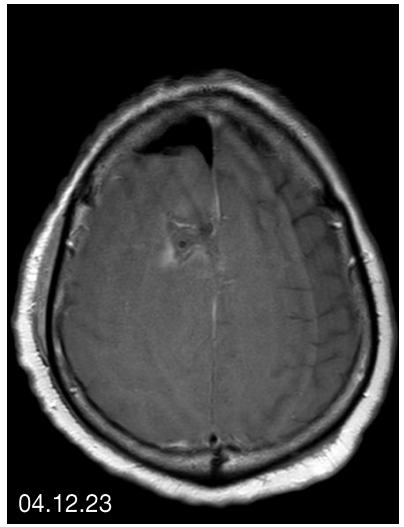


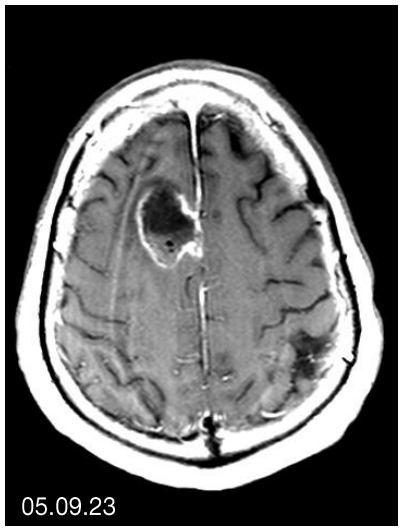
Note: ***Of Evaluable Subjects; POD = progression of disease***; As of May 1, 2024; Source: ^NEJM 2005; 352:987-996 & 352:997-1003 DOI: 10.1056/NEJMoa043330, DOI: 10.1056/NEJMoa043331; NEJM 2017; 376:1027-1037 DOI: 10.1056/NEJMoa1611977; ˣ Not yet treated; Early trial results are not indicative of future results, including the outcome of this trial.

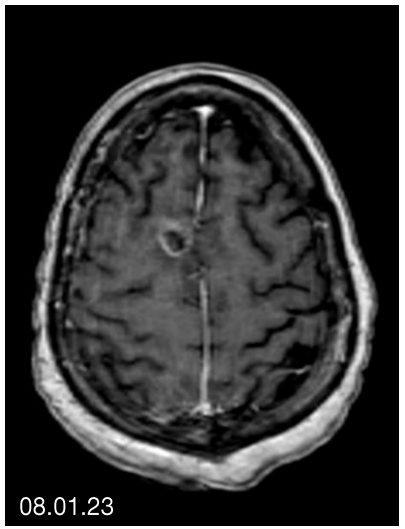
Patient 017 – Female 77y, IDH-wt, MGMT-methylated

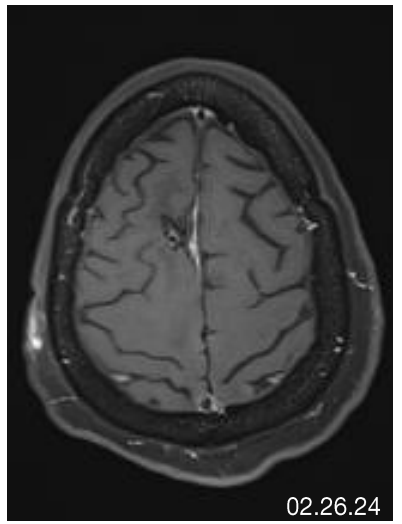
Remains alive and relapse-free at 12.7+ months; “Demonstrated continued slight decrease in size of heterogenous enhancing lesions and decrease in size of nodular enhancing component”

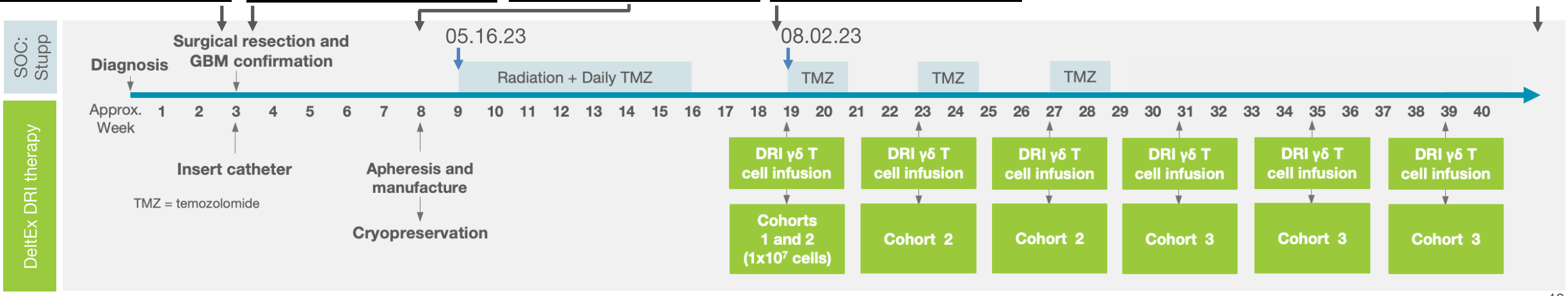










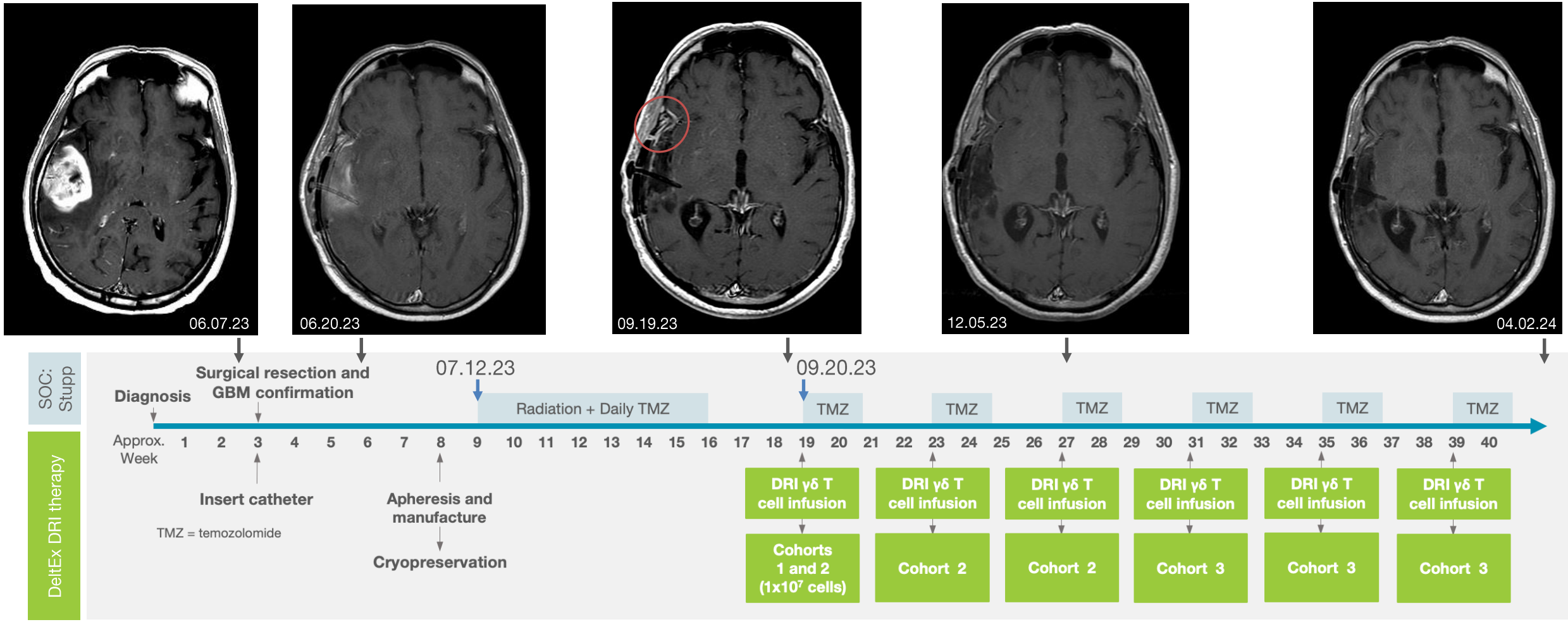


Results from one patient are not indicative of future results including the outcome of this trial

Source: IN8bio and UAB

Patient 020 – Male 66y, IDH-wt, MGMT-methylated

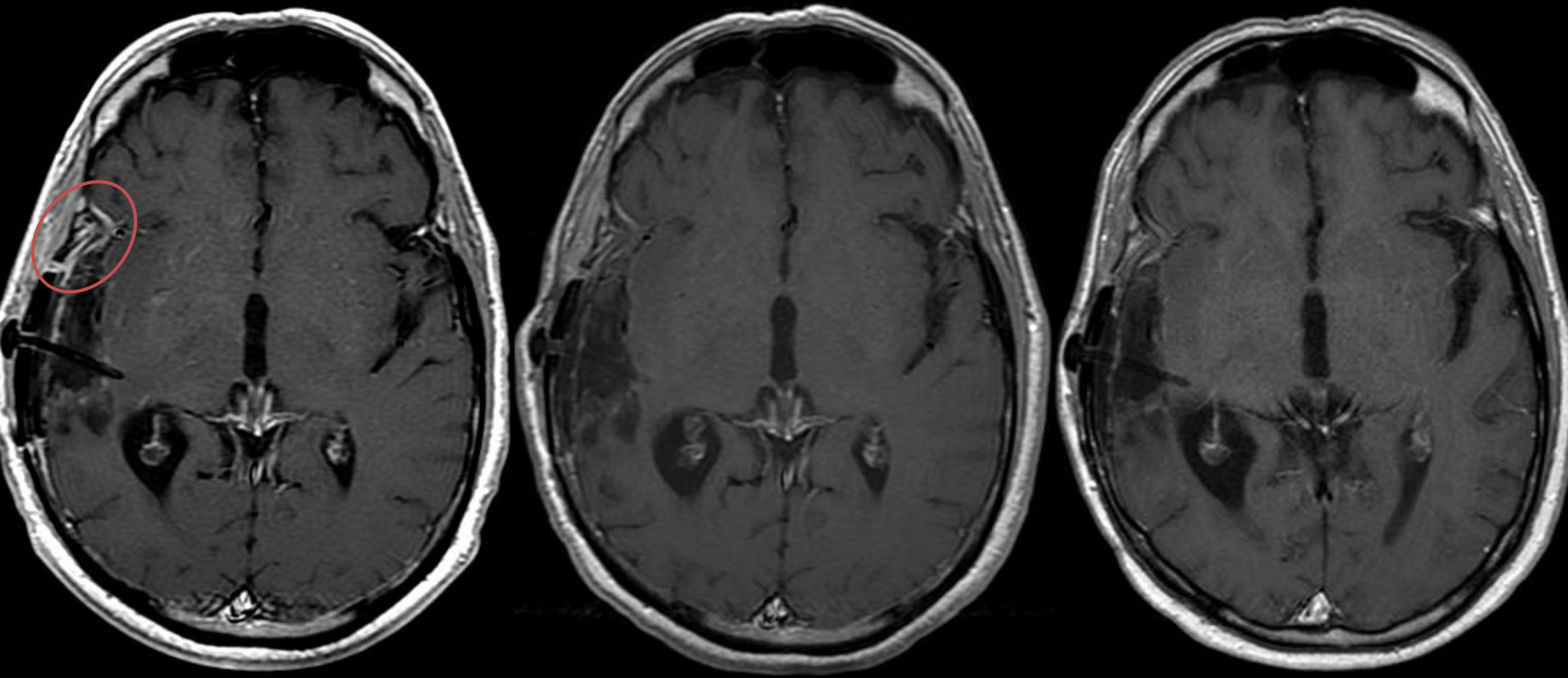
Remains alive and relapse-free at 10.8+ months; “Anterior and medial portion of R temporal lobe showing enhancement suggestive of post-treatment change at off-treatment scan”



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

Source: IN8bio and UAB

Third Patient Enrolled and Treated – 020

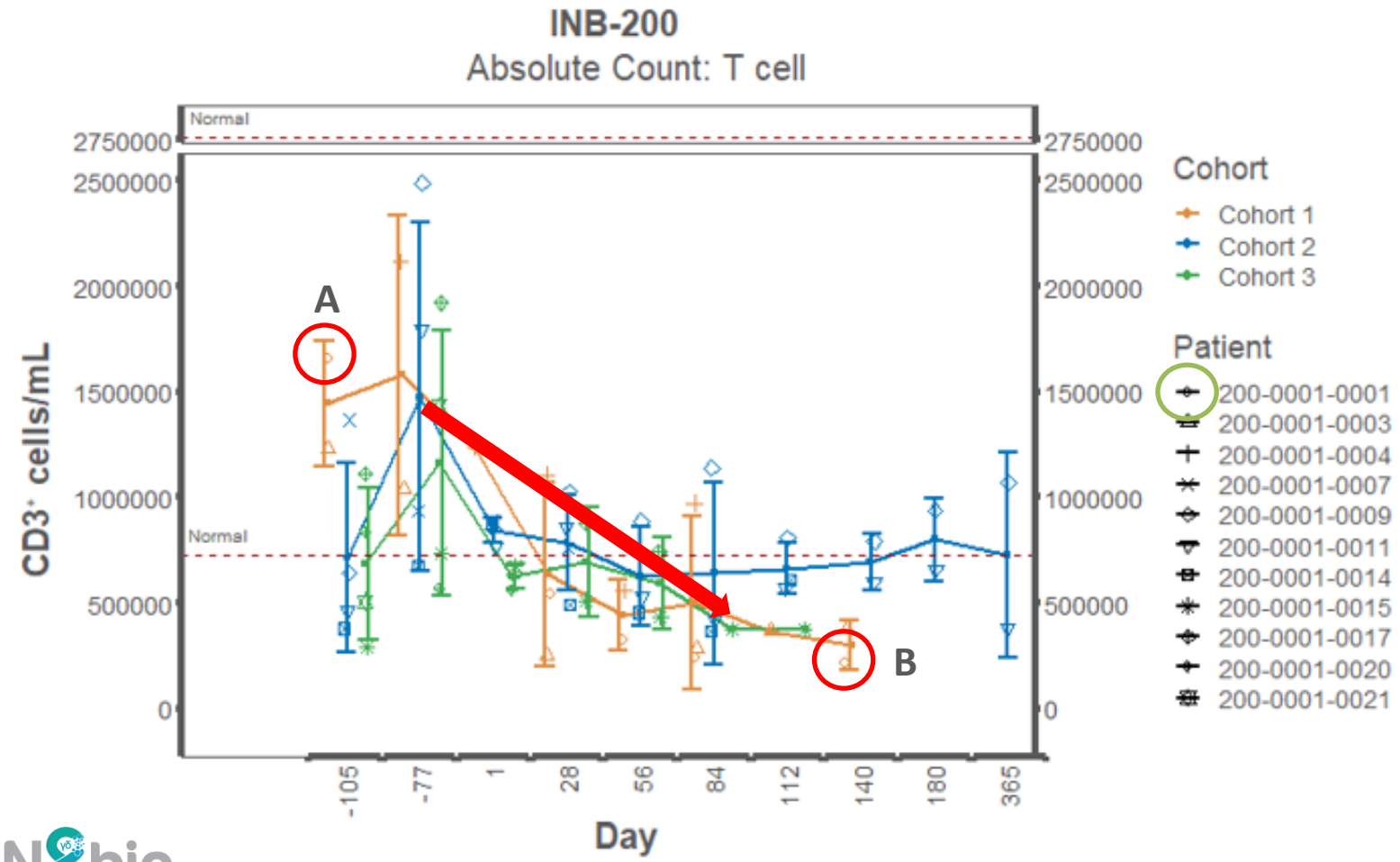


See Slide 47 for further information; Results from one patient are not indicative of future results including the outcome of this trial

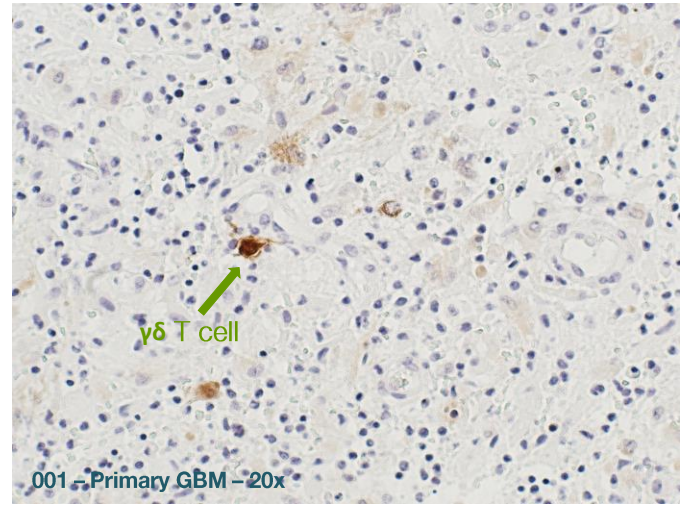
Source: IN8bio and UAB

γδ T Cells are Infiltrating and Persisting in Tumor Tissue

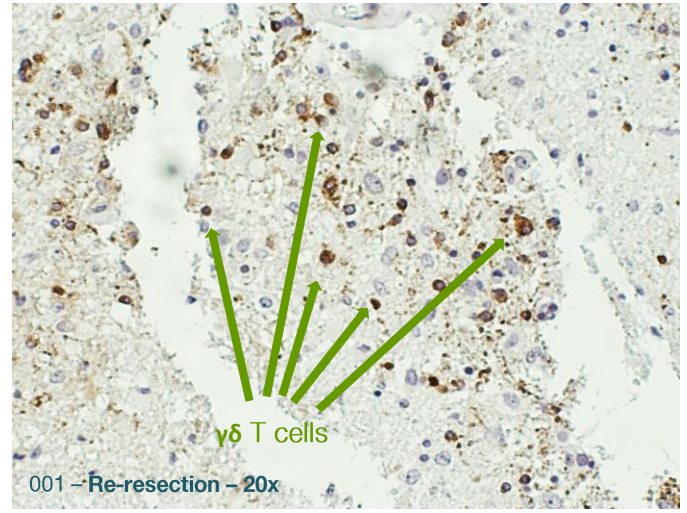
Preserved γδ cells in relapsed tumor 148 days post-DRI infusion despite significant peripheral lymphodepletion in patient 001



Biopsy A: at diagnosis



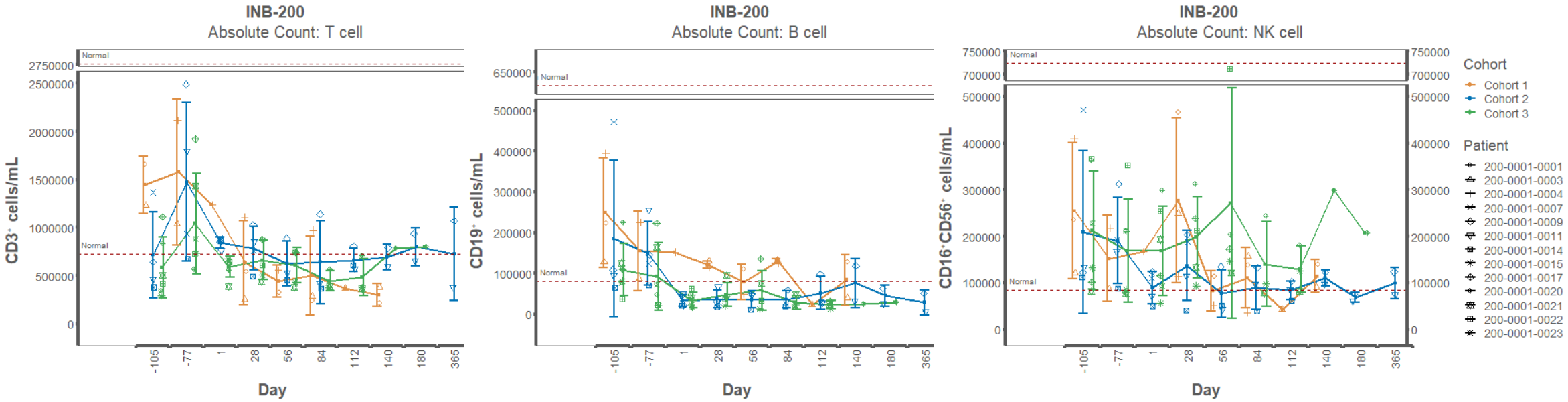
Biopsy B: at relapse, 148 days after single dose



Source: IN8bio and UAB

Peripheral Immunophenotyping; T, B & NK

TMZ is an effective lymphodepleting agent for cell therapy



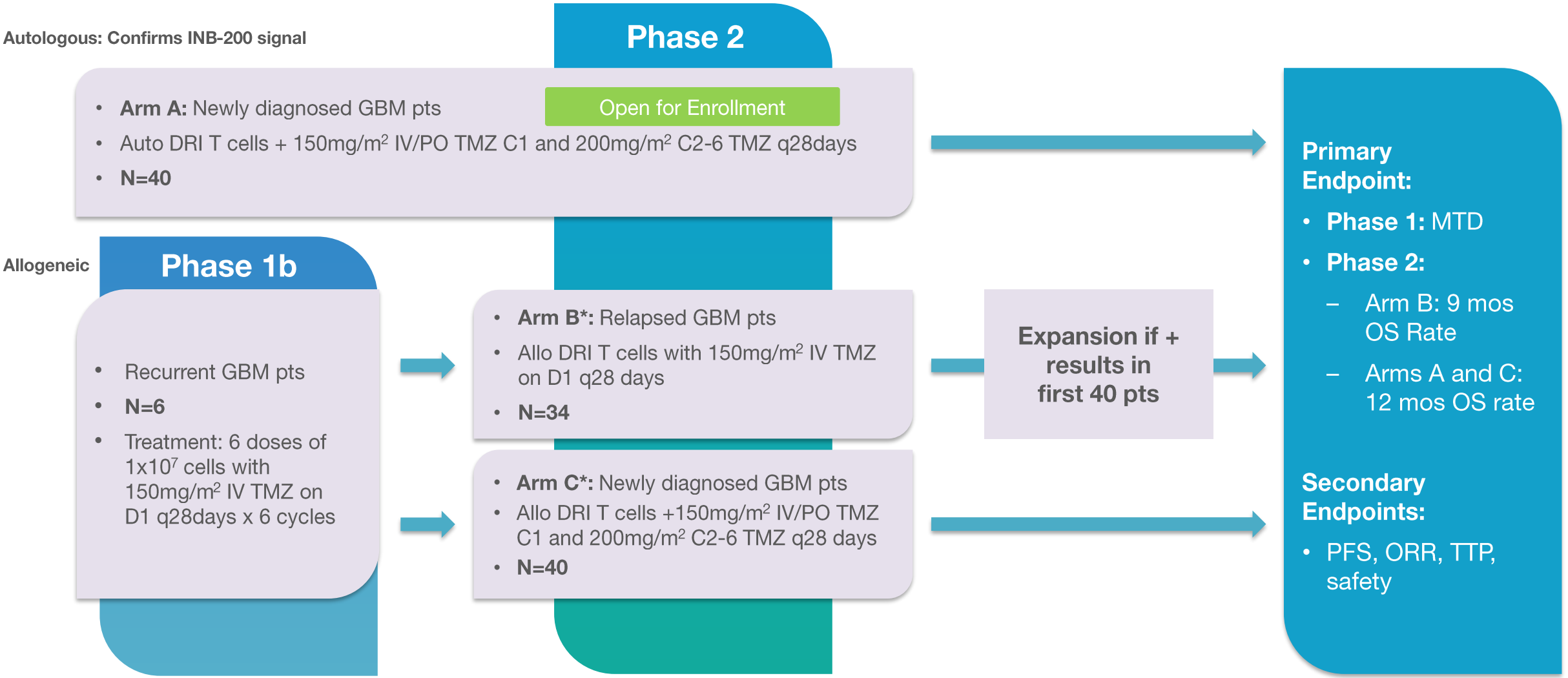
- During TMZ treatment, as expected T, B and NK levels drop to low normal or below low normal values
- The main CD8+ T cells profile is Naïve and Central Memory

INB-400

DeltEx DRI for GBM

Phase 2 – “Arm A” Enrolling Newly Diagnosed GBM Patients

INB-400: Study Design and Treatment Schema



*Arm B and C subject to additional IND for allogeneic drug product (INB-400) as per FDA Guidance for Industry updated Nov. 2022 (https://clinicaltrials.gov/ct2/show/NCT05664243)

Corporate

Deep Experience Across Development and Biotechnology

William Ho
Co-Founder, President and Chief Executive Officer

Lawrence Lamb, PhD
Co-Founder and Chief Scientific Officer

Patrick McCall, CPA
Chief Financial Officer

Trishna Goswami, MD
Chief Medical Officer

Kate Rochlin, PhD
Chief Operating Officer

Glenn Schulman, PharmD, MPH
Head IR and Corporate Communications

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

- Diverse 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
- Proven and measurable successes in bringing high-profile candidates to market, including Stemline, Immunomedics and Gilead Sciences

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Historical & Anticipated Milestones Across Pipeline^

Balance Sheet

(as of March 31, 2024)

- Cash of ~$13.0M
  - Provides runway into 1Q25
  - Potential for up to ~$33M in additional capital at increasing valuations from convertible securities issued in 4Q23
  - $0 debt
- $99.8M accumulated deficit on $117.3M raised

- Ticker: **INAB**
- 44.1 million common shares outstanding as of May 6, 2024

2023 2H

INB-100	100% of Patients in Phase 1 leukemia trial in mCR (ASH Dec. 11, 2023)
INB-200	Additional Phase 1 data (cohorts 2 & 3) in GBM (SNO Nov. 17, 2023)
INB-300	Positive preclinical data demonstrated proof-of-concept of nsCAR CD33 platform @ R&D Day
INB-400	Initiation of enrollment of first patient in 2H23
INB-500	iPSC development update (SITC Nov. 4, 2023)

2024

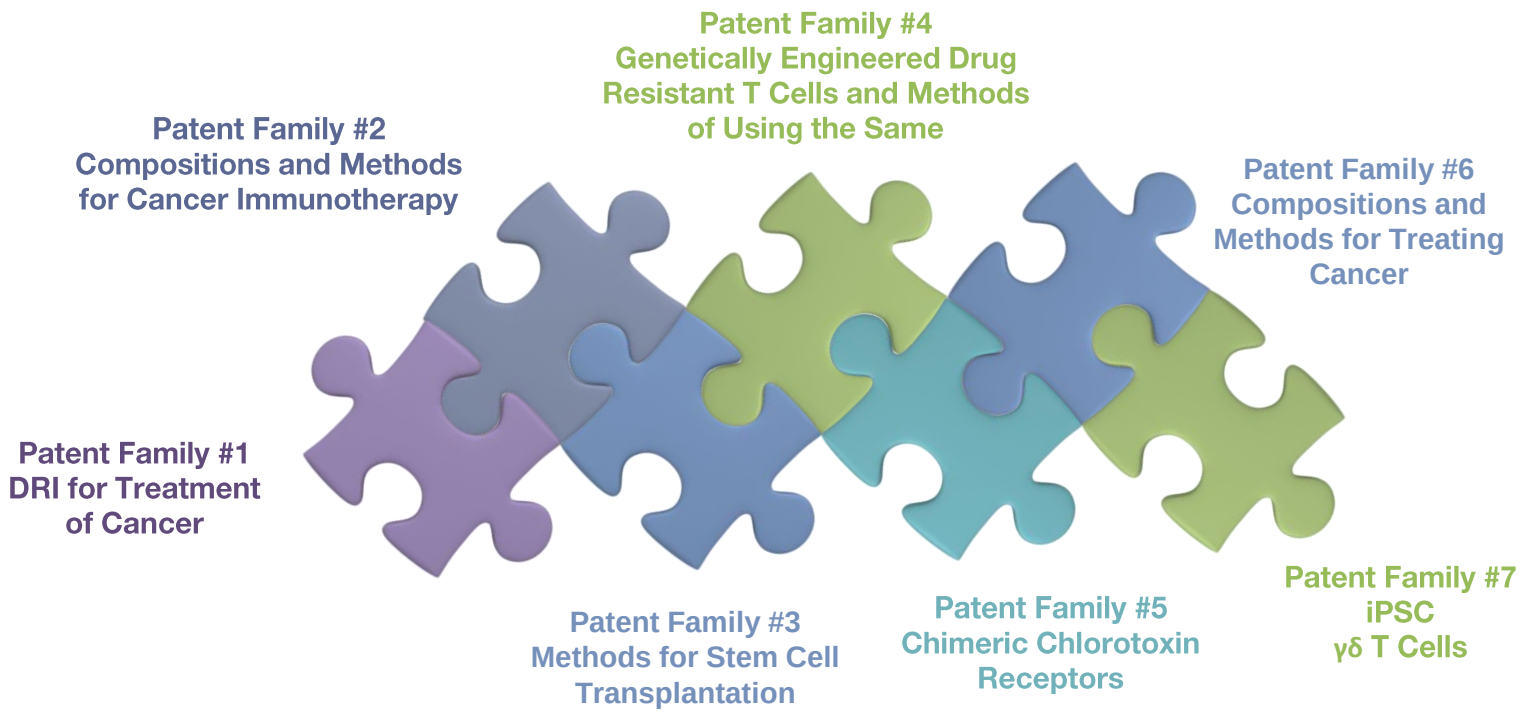
INB-100	Enroll patients in expansion cohort at DL 2
	Report long-term follow-up results at multiple medical meetings in 2024
	Potentially submit IND for registrational RCT trial
INB-200	Completion of Phase 1 enrollment
	Long-term follow-up results at multiple medical meetings in 2024
INB-300	Updated proof-of-concept data on nsCAR platform targeting AML at American Association for Cancer Research (AACR) 2024
INB-400	Dose first patient in 1H24
	Potentially submit IND for Allo Phase 1b in relapsed GBM in 2024

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

A Robust Intellectual Property Portfolio

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

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



Harnessing the Power of Gamma-Delta (γδ) T Cells…

Unique Platform

We are using γδ T cell therapy in a differentiated way, focusing on synergistic combinations

Approach based on biology unique to γδ T cells

Most comprehensive in the industry, with proprietary genetic engineering and cell-type specific manufacturing capabilities

Platform to be applied across multiple indications

Robust Pipeline

Most advanced and deepest γδ T cell pipeline targeting multiple oncologic indications

3 clinical stage candidates
- INB-100 in leukemias
- INB-200 in GBM
- INB-400 in GBM

2 preclinical platforms, with multiple planned INDs over the next few years^
- INB-400 – allogeneic in GBM
- INB-100 – Potential registrational trial in leukemia

Multiple clinical milestones in 2024
- INB-100 in leukemias
- INB-200 in GBM

Strong Expertise

Experts in γδ T cell development

Team's acumen and experience have significantly de-risked our CMC processes and procedures

Successfully advanced a novel approach to the use of gamma-delta T cells as part of a synergistic immunotherapy approach

Recognized leaders with seminal contributions to the development and manufacturing of γδ T cells

Seasoned management team with strong drug development expertise

Market Leader

First to bring genetically modified γδ T cells into the clinic

First to bring allogeneic γδ T cells into the clinic through the FDA

Pursuing rigorous science to achieve better patient outcomes

Standing up for patients with limited to no treatment options

Working to achieve our mission of "Cancer Zero™" the complete removal of cancer cells in patients

^Timing of Next Anticipated Milestones are estimates based on the successful raise of additional capital to fund our programs

Harnessing the Power of γδ T Cells

- Utilizing innovative approaches to efficiently advance our programs
- Demonstrating the ability to execute and to build our business methodically and intentionally
- Pursuing rigorous science to achieve better patient outcomes
- Completed enrollment in INB-100 and INB-200 Phase 1 trials
- Actively enrolling patients in INB-400 Phase 2 trial
- Near-term value creating milestones with presentations and clinical data updates at medical meetings throughout 2024

The Unmet Need in Oncology Trials is Significant

"When I was first diagnosed with AML, we (my wife and I) were updating the will and planning for the worst. Dr. McGuirk and his team discussed the gamma-delta clinical trial and asked if I wanted to participate. I was hoping for a cure, but I figured if I were not to make it, others might learn something from my participation in the trial. We were resigned for the worst but Dr. McGuirk and this trial gave us hope. Today we are living a pretty normal life with people in our community, the church and family. They prayed for us and for a successful treatment. Right now I am feeling good and we are so thankful." – INB-100 patient

*Early trial results are not indicative of future results, including the outcome of this individual patient or trial.

Join Us on Our Mission to Achieve…

Cancer Zero™

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