



Tri-Specific NK Cell ENGAGERS (TriKE[®])

**Targeted NK Cell Therapies to Treat Cancer and
Autoimmune Disease**

GT Biopharma (Nasdaq: GTBP)
Corporate Presentation – May 2026

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Investment Opportunity – Next Generation of NK Cell ENGAGERS

Proprietary TriKE® Platform – Camelid Nanobodies

- TriKE® are tri-specific NK cell ENGAGERS
- Incorporate Camelid “nanobodies”

NK Cell ENGAGERS – Safer than T Cells¹

- Protein therapeutics to harness the natural killing power of NK cells with – NOT NK cell therapy
- Activates NK cells via CD16A and IL-15 while targeting tumor antigens
- Potentially safer than T-cell immunotherapy

POC Established and Broad Applicability

- GTB-3550 (targeting CD33) showed POC in Phase 1 in AML patients
- GTB-3650 will supplant 3550 as 2nd generation TriKE® with several advantages
- TriKE®s target multiple tumor antigens including B7H3, HER2, CD33, PDL1

Multiple Catalysts

- 6+ pipeline assets in preclinical development, both solid tumors and hematological malignancies
- GTB-3650 IND accepted in June 2024, first patient dosed Q1 2025
- GTB-5550 IND accepted in Jan 2026, Phase 1 dose escalation basket trial, first patient dosed in May 2025

Broad Indication Potential

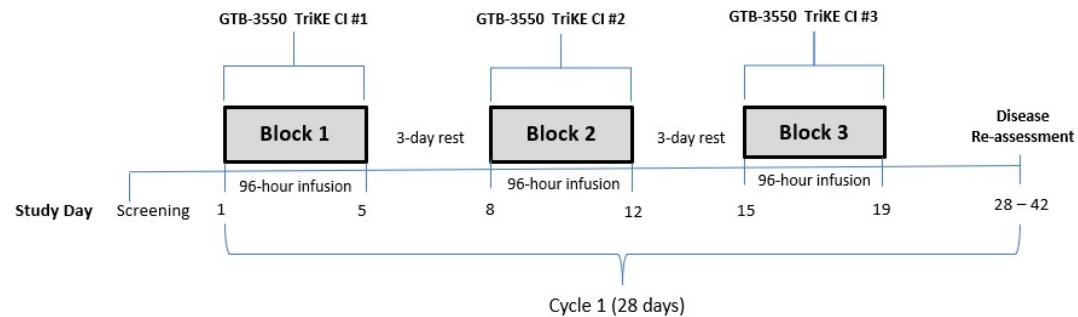
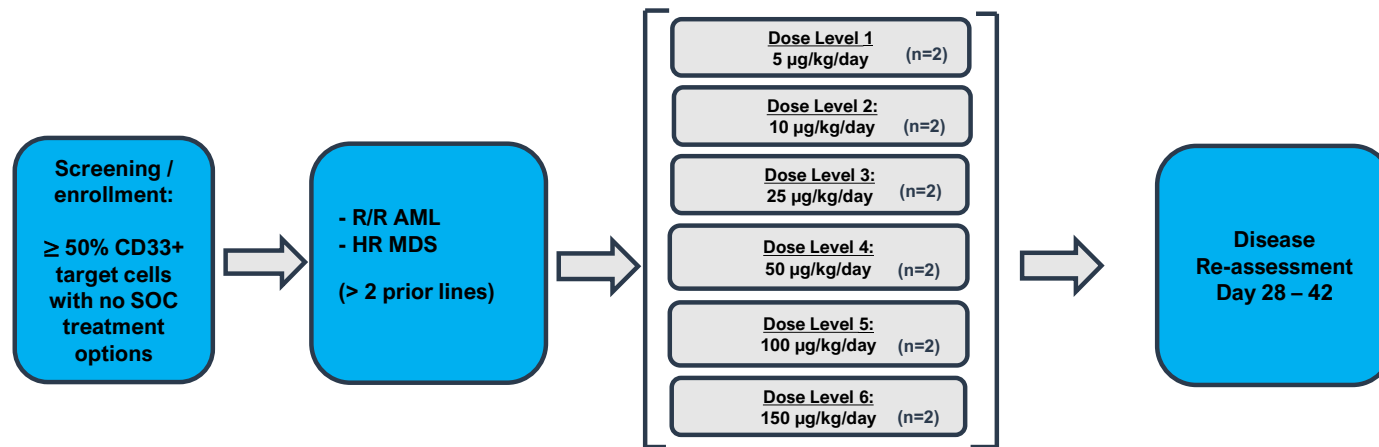
- GTB-7550 TriKE® candidate in development for the treatment of lupus, other autoimmune disorders
- Exploring manufacturers for GTB-7550

Well-funded Experienced Leadership

- Management team with deep expertise in all stages of oncology drug development
- Approx. \$9M in cash as of 3/31/2026 (unaudited), anticipated to be sufficient to fund operations through Q4 2026

1. [Demaria, et.al. Eur J. of Immun; \(2021\)51:8; 1934](#)

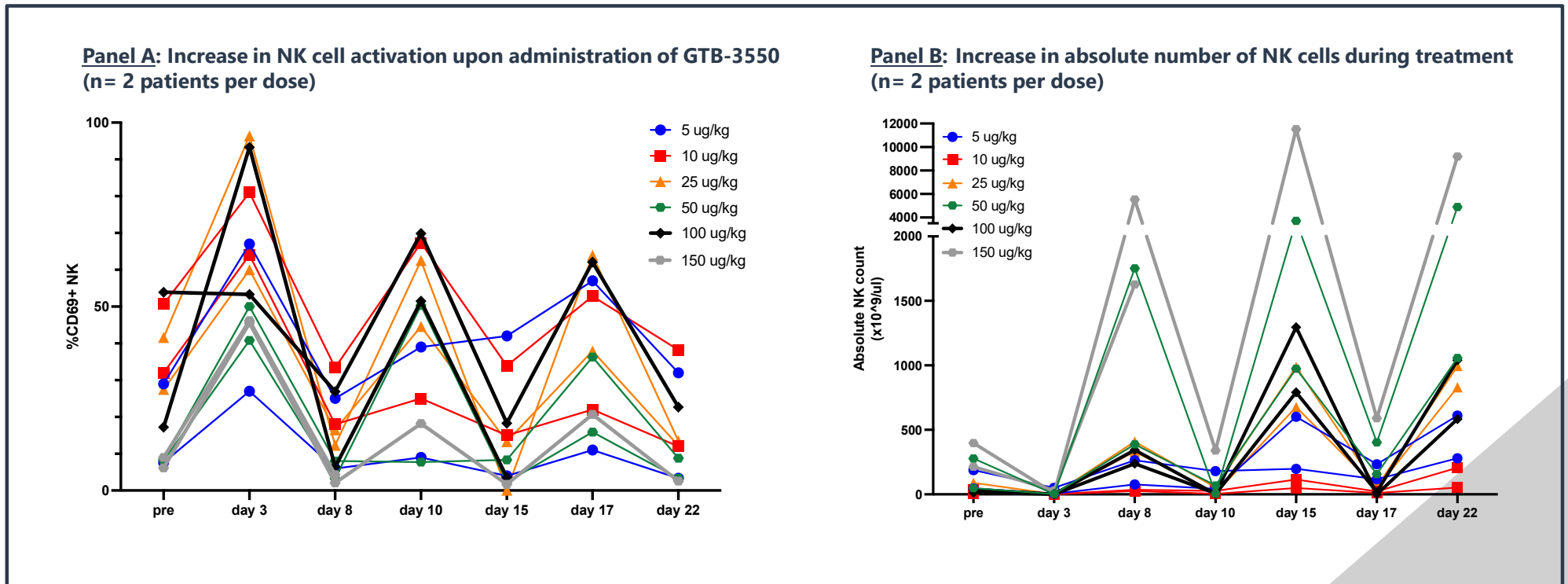
GTB-3550 AML/MDS Phase 1 Study Design



Phase 1 (safety and dose finding)

- Six dose levels
- Escalation based on continual reassessment method; Cohorts of 2 subjects
- Day 28 (end of DLT assessment period)

GTB-3550 Activation of Endogenous NK Cells



Source: Data on File, GT Biopharma, Inc.

GTB-3550 First in Human Phase 1 Clinical Trial – Individual Results



| Subject | Dose level (µg/kg/d) | Disease and Prior Treatment History | Disease Characteristics Before GTB-3550 Therapy | Disease Characteristics After GTB-3550 Therapy | Response Post Cycle 1 |
|---------|----------------------|--|---|--|---|
| 1 | 5 | r/r AML. Triple Hit Lymphoma - 6 therapies: 1. R-EPOCHx6, 2. RICE x3, 3. XRT to abdominal lymphadenopathy, 4. NAM-NK Clinical Trial, 5. CAR-T, 6. anti-CD20 and Anti-CD3 monoclonal antibody clinical trial | Cellularity: 10% Blast: 5 – 10% | Cellularity: 10 – 30% Blast: 10% | Stable AML with improved platelet transfusion needs |
| 2 | 5 | r/r AML. AML- 3 therapies before TriKE: 1. Vyxeos + Midostaurin 2. FLAG-IDA + midostaurin 3. Decitabine + Gilteritinib | Cellularity: 70 – 80% Blast: 7% | Cellularity: 90 – 95% Blast: 94% | Progression |
| 3 | 10 | r/r AML. AML- 3 therapies before TriKE: 1. Azacitidine, 2. Enasidenib, 3. Hydrea | Cellularity: 100% Blast: 85% | Cellularity: 100% Blast: 92% | Stable AML |
| 4 | 10 | t-MDS. Multiple Myeloma - 5 therapies: 1. CyBorD, 2. Bortezomib, 3. Dexamethasone + lenalidomide + idazomib, 4. Daratumumab + Pomalidomide + Dexamethasone, 5. Dara maintenance | Cellularity: 5% Blast: 5.5% | Cellularity: 5% Blast: 20% | Stable MDS |
| 5 | 25 | Secondary AML, progressed from MDS. 33% reduction in blast count | Cellularity: 10 – 15% Blast: 18% | Cellularity: 20% Blast: 12% | Blast count reduction, improved platelet needs |
| 6 | 25 | r/r AML. 2 therapies before TriKE: 1. 7+3 with CR1 then relapse, 2. Azacitidine + Venetoclax | Cellularity: 10 – 20% Blast: 29% | Cellularity: 10 – 20% Blast: 35% | Mild blast increase |
| 7 | 50 | HR MDS. MDS - 3 therapies: 1. Decitabine, 2. Luspatercept, 3. Decitabine 10 day 61.7% reduction in blast count | Cellularity: 70 – 80% Blast: 12% | Cellularity: 60% Blast: 4.6% | Partial remission |
| 8 | 50 | HR MDS. MDS - 3 therapies before TriKE: 1. Azacitidine, 2. NMA DUCBT, CR1 for 7 years before relapse 3. Azacitidine - CR2 then relapse | Cellularity: 20% Blast: 12% | Cellularity: 30% Blast: 19% | Mild blast increase |
| 9 | 100 | High Grade MDS- 1. Azacitidine. 2. Decitabine, 3. 7+3, 4. Allo transplant with CR then relapse and progression to AML then no response to Decitabine + Venetoclax 63.6% reduction in blast count | Cellularity: 20% Blast: 22% | Cellularity: 10 – 20% Blast: 8% | Partial remission |
| 10 | 100 | r/r AML. Breast Cancer: 4 therapies: 1. Mastectomy/LN dissection, 2. XRT, 3. Adriamycin/Cyclophosphamide, 4. Taxol. | Cellularity: 10% Blast: 17% | Cellularity: 40% Blast: 31% | Stable AML |
| 11 | 150 | DLBCL - 3 therapies 1. R-DA-EPOCH, 2. Auto Transplant, 3. ADAM-17+Rituximab, Therapy-related MDS: 2 therapies: 1. Azacitidine, 2. Allo transplant -- CR, Relapse/transformed to AML (bi-phenotypic) - 1 therapy before TriKE: 1. Venetoclax + Decitabine x 2 cycles 50% reduction in CD33+ blast count | Cellularity: 25% Blast: 80% | Cellularity: 80% Blast: 73% | Blast reduction by FLOW |
| 12 | 150 | r/r AML - 2 therapies: 1. FLAG-IDA + venetoclax, 2. Decitabine | Cellularity: 30 – 40% Blast: 36% | Cellularity: 60 % Blast: 64% | Disease Progression |

TriKE[®] Nanobody Pipeline



| TriKE [®] Product Candidates | Approach | Target | Indication | Pre-Clinical | IND-Enabling/ GMP Manufacturing | Phase 1 | Phase 2 |
|---|-------------------------------|--------|------------------------------------|---------------------|------------------------------------|---------|---------|
| GTB-3650 2 nd Generation Camelid | Monotherapy | CD33 | Leukemia – AML, MDS | [Dark Blue Arrow] | | | |
| | Combination with Chemotherapy | CD33 | Leukemia – AML, MDS | [Dark Blue Arrow] | | | |
| GTB-5550 | Monotherapy & Combination | B7H3 | Solid Tumors | [Dark Blue Arrow] | | | |
| GTB-6550 | Monotherapy & Combination | HER2 | Solid Tumors | [Light Green Arrow] | | | |
| GTB-7550 | Monotherapy & Combination | CD19 | B-Cell Malignancies | [Light Green Arrow] | | | |
| GTB-1050 | Monotherapy & Combination | | HIV | [Light Green Arrow] | | | |
| Undisclosed Candidates | Monotherapy & Combination | | Solid & Hematological Malignancies | [Light Green Arrow] | | | |
| GTB-3550 | Monotherapy | CD33 | Leukemia – AML, MDS | [Dark Blue Arrow] | | | |

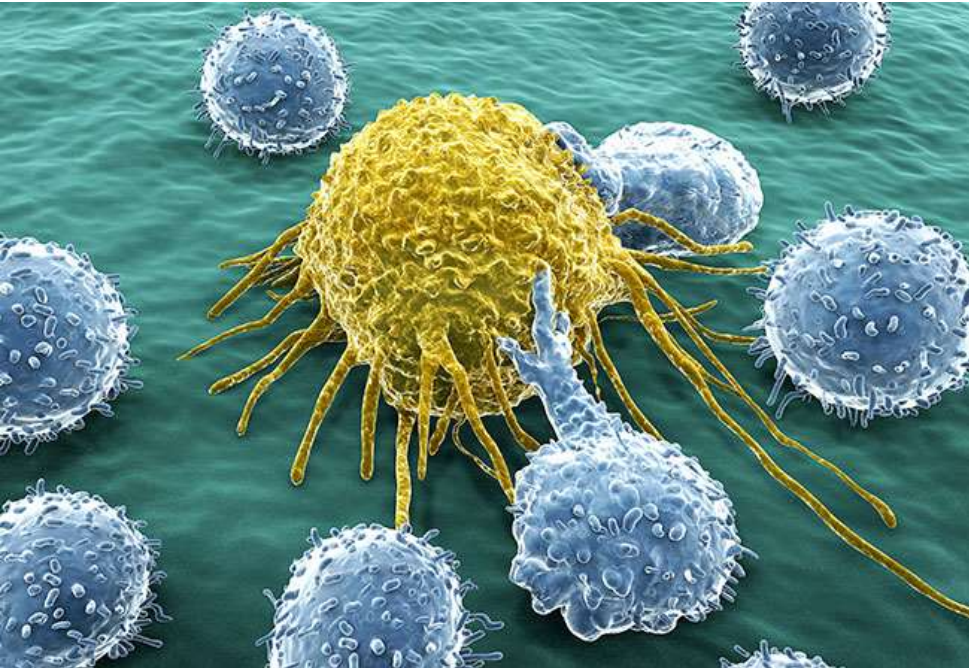
GTB-3650
Phase 1 trial,
50% of
patients dosed

GTB-5550 IND accepted
Jan 2026, Phase 1 dose
escalation basket trial, first
patient dosed in May 2026

GTB-3550
supplanted
by second
generation
GTB-3650

Clinical Product Candidates [Dark Blue Arrow] Preclinical Product Candidates [Light Green Arrow] Development Abandoned [Light Blue Box]

Natural Killer Cell ENGAGERS to Fight Cancer



Natural Killer Cells

- Cytotoxic lymphocytes in the innate immune system
- Recognize and kill cancer cells
- Mediate antibody-dependent cellular cytotoxicity (ADCC) via the highly potent CD16 activating receptor

NK Cell ENGAGERS

- TriKE[®] nanobody platform designed to activate endogenous NK cells to target specific cancer cells
- Potential for less toxicity than other cellular therapies such as CAR-T therapy
 - Less cytokine release syndrome (CRS)
 - Fewer neurological complications

Source: Levy R. Paths of Progress 2019, Natural Killer Cells: How the immune system's first wave of defense may play a newfound role in cancer care; accessed: 6 September 2021

www.dana-farber.org/newsroom/publications/paths-of-progress-2019/natural-killer-cells/

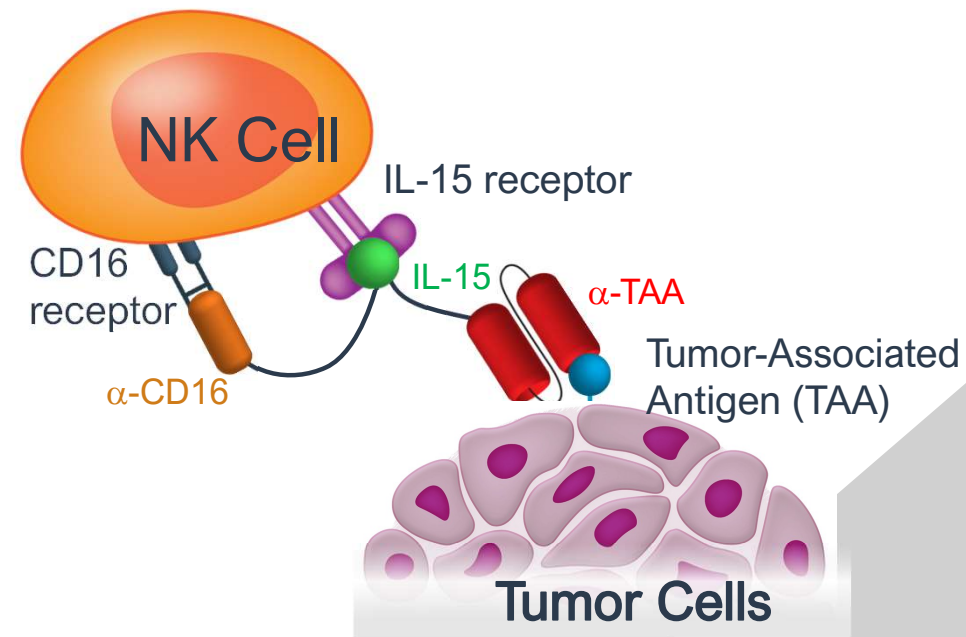
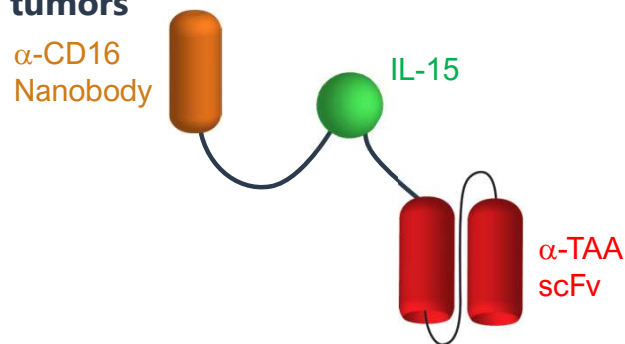
TriKE®: Tri-Specific Natural Killer (NK) Cell ENGAGERS - A Modular Platform



Proprietary platform utilizing camelid nanobody technology designed to bridge NK cells to tumor cells while inducing NK cell activation and expansion at the site of the tumor to enhance killing

Tri-specific Modular Platform with Nanobody Technology

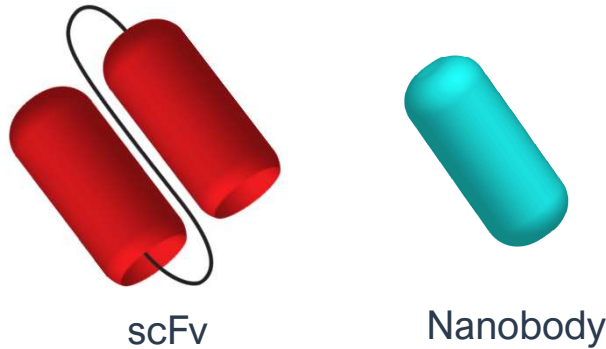
- **Anti-CD16 nanobody*** – binds CD16 receptor on NK cells, triggering antibody directed cell-mediated cytotoxicity (ADCC)¹
- **IL-15** – crosslinker that binds IL-15/IL-2 receptor on NK cells to induce self-sustaining expansion and extended survival^{2,3}
- **Anti-TAA scFv** – scFv domain binds to various tumor-associated antigens on tumors



* 1st generation TriKE GTB3550 utilizes scFv for a-CD16 1. [Semin Immunol. 2017 Jun; 31: 64–75.](#)

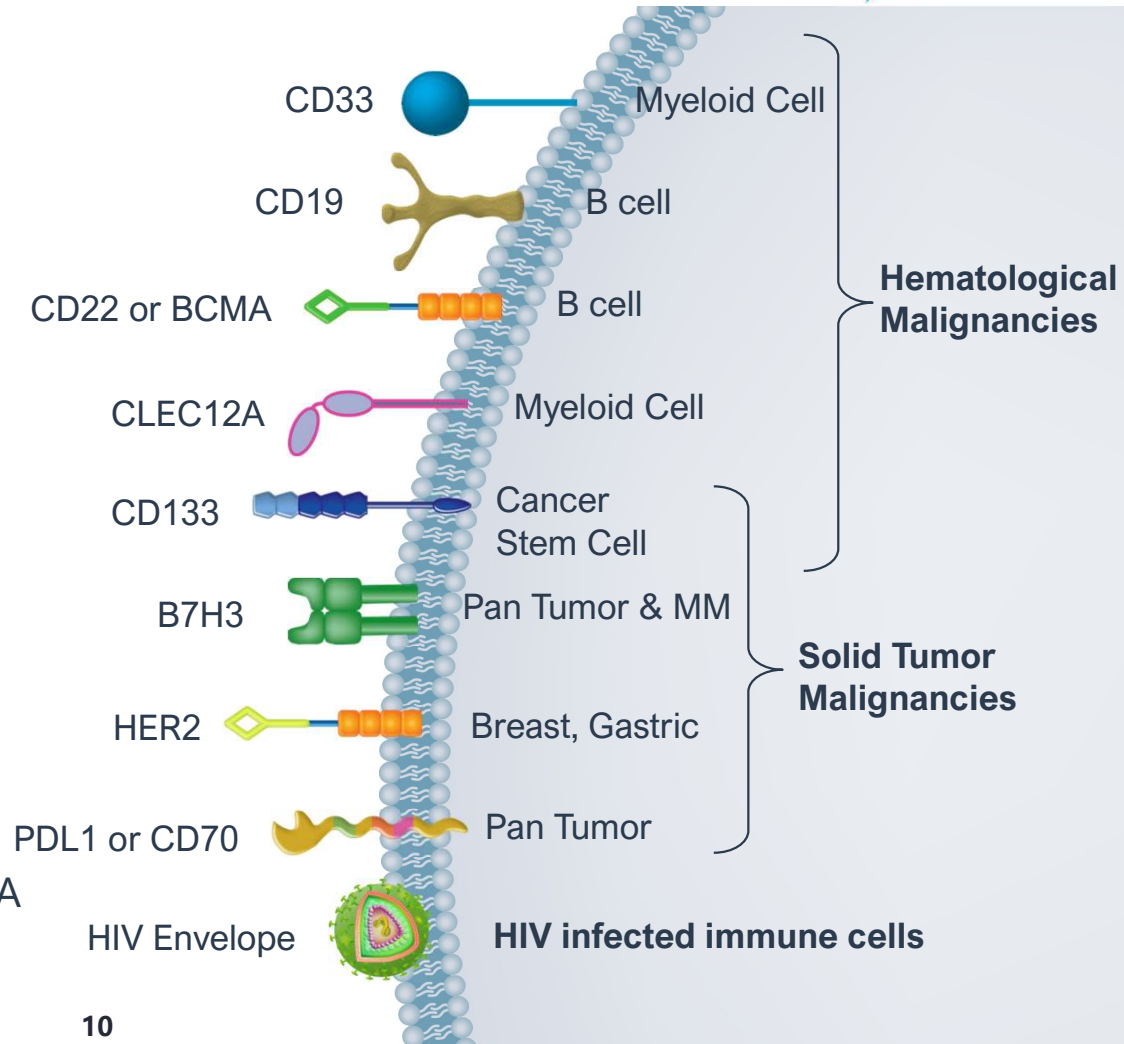
2. [J Exp Med. 1994 Oct 1; 180\(4\): 1395–1403.](#) 3. [Vallera et. al. Clin Cancer Res. 22\(14\) July 15, 2016](#)

TriKE® Modular Platform Allows for Multiple Tumor-Associated Antigen



Anti-Tumor Associated Antigen

- Binds to well-known tumor-specific antigens
- Defines the specificity of each TriKE®
- Localizes NK cells at the site of the malignancy
- Utilizes scFv fragments for most TriKE® constructs
- Certain TriKE®s utilize nanobodies for the α -TAA



TriKE[®] – NK Cell-Driven Serial Killing of AML Tumor Cells

- First-in-class modular immune oncology protein therapeutic platform technology – not a cell therapy
- Target-directed antibody-dependent cellular cytotoxicity (ADCC) killing
- Integrated CD16 and IL-15 driven activation of NK cells:
 - ADCC activation for enhanced serial killing of cancer cells
 - NK cell proliferation
 - NK cell persistence
- Minimizes toxicities such as cytokine release syndrome (CRS) resulting from hyperactivation of T cells
- Can be used to treat BOTH solid tumors and hematological cancers

Source: [Sarhan D et al. Blood Adv 2018 Jun 26; 2\(12\): 1459–1469](#)



GTB-3550 Phase 1 Demonstrates Proof of Concept for CD33 TriKE® in AML/MDS



- GTB-3550 induces reproducible NK cell proliferation, activation and persistence in all patients at all dose levels with minimal clinically significant toxicity
- Minimal CRS resulting from hyperactivation of patient's T-cell population at doses 5–150 µg/kg/day
 - Fever (Grade 1 CRS) observed in Subject #12 (150 µg/kg/day); resolved upon acetaminophen treatment
- No loss in CD16 expression on patient's NK cells
- GTB-3550 significantly reduced CD 33+ bone marrow blast levels by 33.3%, 61.7%, 63.6%, 50% in Patient 5 (25 µg/kg/day), Patient 7 (50 µg/kg/day), Patient 9 (100 µg/kg/day), and Patient 11 (150 µg/kg/day), respectively
- After the end of infusion, GTB-3550 & IL-15 concentrations declined rapidly with overall geometric mean terminal phase elimination half-life (T_{1/2}) of 2.2 and 2.52 hours, respectively



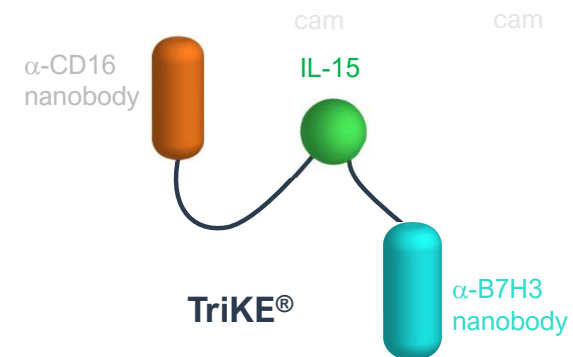
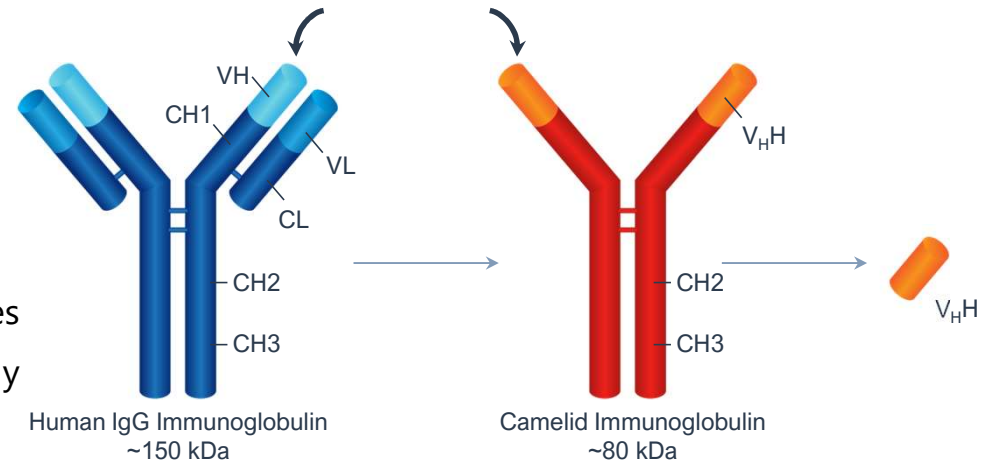
GTB 3650 for AML and MDS
GTB 5550 for Solid Tumors

**Second generation TriKEs® utilize
camelid nanobody technology**

Advantages of Camelid Antibodies – Nanobodies in 2nd Generation TriKEs®



- Camelidae family of mammals include llamas, camels, and alpacas
- Camelid antibody is made up of only 2 heavy chains
 - Heavy chain IgG (hclgG)
 - Do not contain the CH1 region
 - Retain an antigen binding domain – V_HH region
- V_HH are known as single domain antibodies or nanobodies
 - Contain only the V_HH region from the camelid antibody
- 2nd Generation TriKE®s utilize nanobodies
- Advantages over 1st Generation TriKE®s (GTB-3550) include:
 - Improved potency and enhanced binding affinity
 - Commercial manufacturing capabilities through Cytovance
 - Proprietary molecule wholly owned by GT Biopharma
 - Similar preclinical safety profile



Source: www.rndsystems.com/products/llamabody-camelid-antibodies

Our Approach – Co-Stimulation of CD16 and IL-15



TriKE® Competitive Differentiation

- The anti-CD16 component of the TriKE® binds FcRγIII with high affinity
- TriKE® does not result in proliferation of T-cells
- IL-15 provides NK cell specific proliferation with less bystander T-cell activity compared to the IL-15 protein itself
- IL-15 in TriKE® is less active surrounded by ENGAGERS than rhIL-15
- TriKE® can be targeted to heme malignancies, solid tumors and infectious diseases

NKp46/CD16

CD16A

NKG2D/CD16



- NK cell ENGAGER/antibody therapeutic strategies designed to engage CD16, NKG2D, or NKp46
- None of them co-stimulate CD16 and IL-15 simultaneously

CD123 in AML



- NK cell therapy
- Could be used in combination with TriKE®s

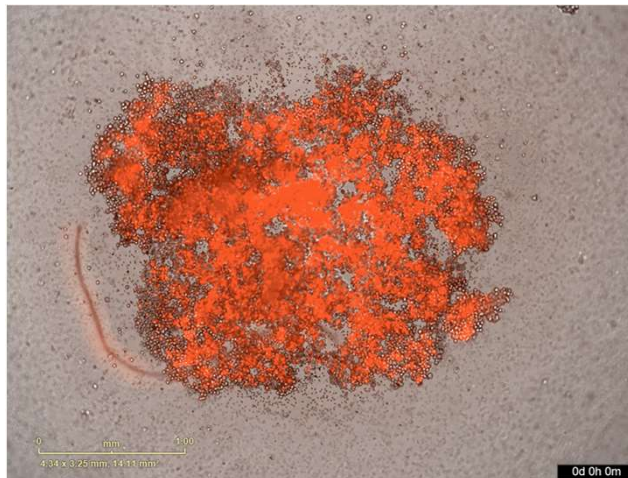
GTB 5550 - B7H3 TriKE Killing of Prostate Cancer



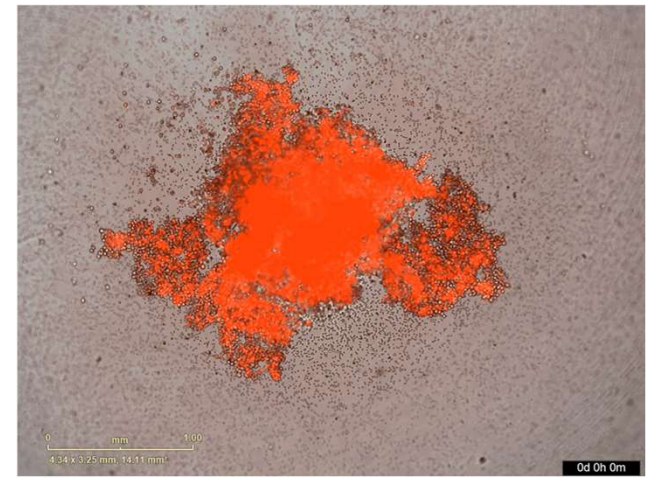
NK Cells Alone



NK Cells+IL-15



**GTB 5550
NK Cells+B7H3 TriKE**





GTB 7550 for Autoimmune Disease

Targeting CD19 for B-Cell Depletion In Vivo

GTB 7550 for Autoimmune Disease

- GTB-7550 TriKE[®] product candidate is in development for the treatment of lupus and other autoimmune disorders
- GTB-7550 TriKE[®] is a tri-specific molecule composed of a camelid nanobody that binds the CD16 receptor on NK cells, a scFv ENGAGER against CD19 on malignant and normal B cells, and a human IL-15 sequence between them
- Published data shows that GTB-7550 effectively targets CD19+ malignant cell lines and primary chronic lymphocytic leukemia (CLL)
- Preliminary data shows that GTB-7550 can target and eliminate normal B cells
- NSG mice will be used to test the ability of GTB-7550 to deplete normal B cells in vivo
- Exploring manufacturers for GTB-7550
- Quickest path to clinic may be testing safety in B cell malignancy first

Experienced Team With Deep Immuno-Oncology Experience



Proven Record in Biotech, Pharma, Product Development, Financing



Michael Breen, LL.B
Executive Chairman and
Chief Executive Officer



Alan Urban
Chief Financial Officer
CPA (Inactive)



Jeffrey Miller, MD
Consulting Senior
Medical Director ¹



Martin Felices, PhD
Consulting Scientist



Chris Hendry
Consultant, CMC and
Pharmaceutical Science



Hilary Kramer
Board of Directors
Nominating/Gov.
Committee Chair



David C. Mun-Gavin
Board of Directors
Compensation
Committee Chair



Charles J Casamento
Board of Directors
Audit Committee Chair





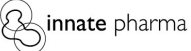







The University of Minnesota, pursuant to its license agreement with GT Biopharma, is entitled to receive royalties should commercial sales of GTB-3650 be realized. This interest has been reviewed and managed by the University of Minnesota in accordance with its conflict of interest policies.



1. Dr. Miller is the Consulting Senior Medical Director at GT Biopharma and holds stock and options in GTBP.

Recent M&A and BD Deals Highlight Value of NK Cell ENGAGERS and Immuno-Oncology

| | | | | | |
|------------------------|--|---|---|--|--|
| Innovator |  |  |  |  |  |
| Acquirer |  |  |  |  |  |
| Date | 8/27/2018 | 11/9/2020 | 12/21/2021 | 5/2/2022 | 12/19/22 |
| Deal Type | License Deal | Single Molecule Preclinical License Deal | Company Acquisition | Single Molecule Preclinical License Deal | Collaboration Expansion License Deal |
| Key Deal Terms | <ul style="list-style-type: none"> • \$96M upfront • \$5B in additional milestones | <ul style="list-style-type: none"> • \$60M upfront • \$2B in milestones | <ul style="list-style-type: none"> • \$1 billion upfront • \$225M in milestones | <ul style="list-style-type: none"> • \$300M cash upfront • Undisclosed milestones • 20% royalties | <ul style="list-style-type: none"> • €25M upfront • €1.3B in milestones • Royalties |
| Technology / Mechanism | Redirected Optimized Cell Killing (ROCK®) platform to generate both NK cell and T cell-engaging antibodies | ROCK® platform generates tetravalent, bispecific antibodies as innate cell ENGAGERS (ICE®) customized to target specific domains on hematologic and solid tumor cells | <ul style="list-style-type: none"> • Portfolio of T cell ENGAGERS using XTEN technology • Lead asset AMX-818 in pre-clinicals | <ul style="list-style-type: none"> • NK-cell ENGAGER • DF7001 is a TriNKET designed to activate and direct NK and cytotoxic T cell killing of cancer cells | <ul style="list-style-type: none"> • NK cell ENGAGER • Targeting B7H3 • ANKET™ platform • Option to add 2 additional targets |
| Rationale | Allowed Roche access to Affimed platform to explore range of ENGAGER constructs for multiple oncology applications | Grants Roivant a license to the preclinical molecule AFM32 | Combine Amunix's complementary molecules with Sanofi's immuno-oncology portfolio | Enhance Gilead's portfolio with complementary MOAs and scientific rationale for combination opportunities | Allogeneic NK cell immunotherapy is pillar of Sanofi's overall oncology strategy and using engineered lymphokines to stimulate NK cells is a key component |

Contact Us



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