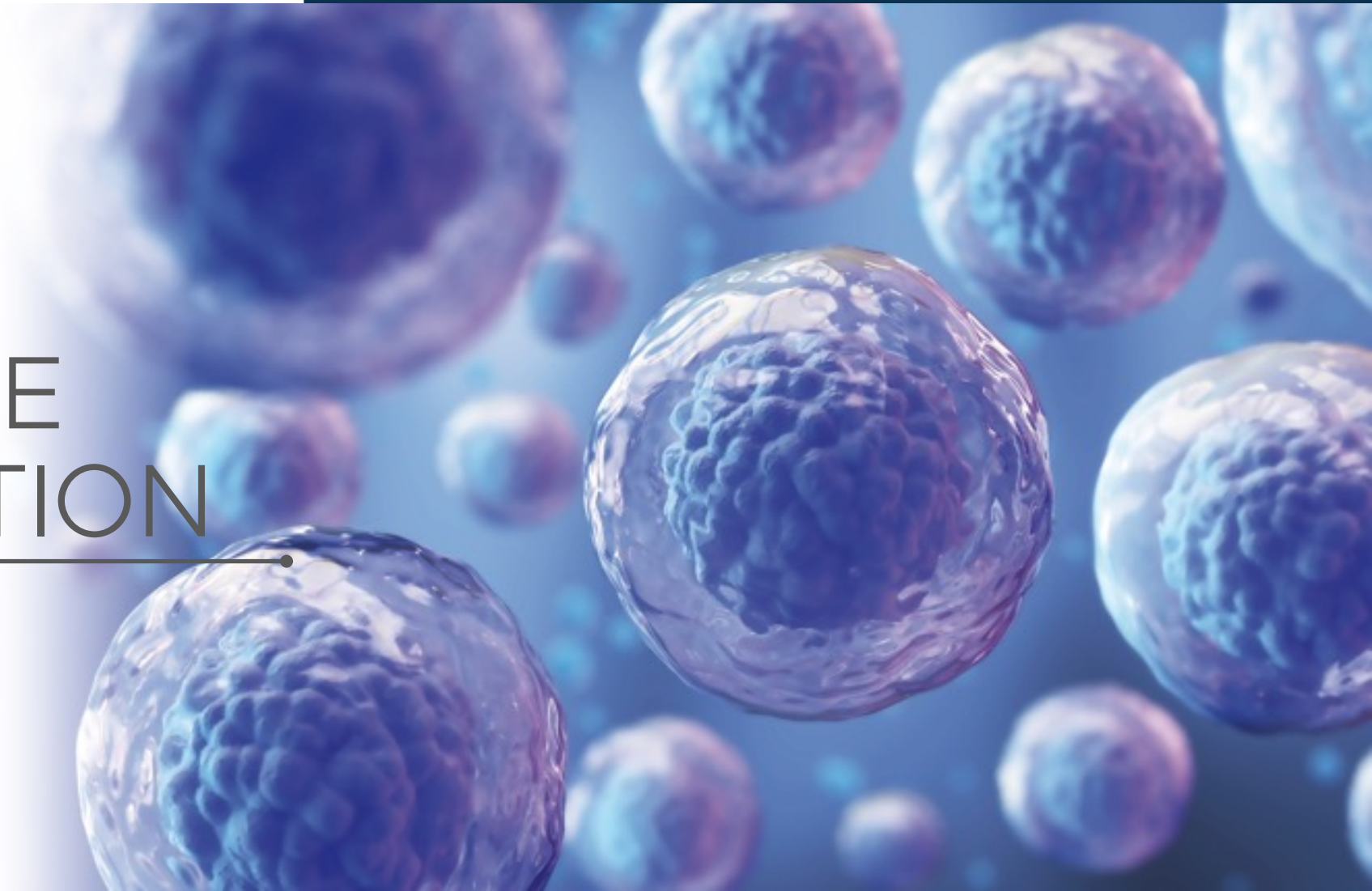




CORPORATE PRESENTATION

NASDAQ: GNTA

May 2024



Forward-Looking Statement

This presentation contains forward-looking statements. In some cases, you can identify forward-looking statements by terminology such as “may,” “will,” “should,” “expects,” “plans,” “anticipates,” “believes,” “estimates,” “predicts,” “potential” “intends” or “continue,” or the negative of these terms or other comparable terminology. All statements other than statements of historical facts contained in the presentation, such as statements regarding our potential future results of operations and financial position, prospective product candidates, availability of future funding, anticipated clinical trial results, timing of possible product approvals and expected regulatory pathways, future potential collaborations and matters concerning the timing and likelihood of success of plans and objectives of management for future operations, are forward-looking statements. Any such forward-looking statements are based on our current expectations and beliefs, as well as assumptions concerning future events. These statements involve known and unknown risks, uncertainties and other factors that could cause such matters to differ materially from those discussed in such forward-looking statements. We discuss many of these risks in our filings from time to time with the U.S. Securities and Exchange Commission, including under the heading “Risk Factors” in such documents. You should not rely upon forward-looking statements as predictions of future events.

Although we believe that the expectations reflected in the forward-looking statements are reasonable, we cannot guarantee future results, levels of activity, performance, or achievements. Except as required by law, we are under no duty to update or revise any of the forward-looking statements, whether as a result of new information, future events or otherwise, after the date hereof.

The background of the slide is a microscopic view of numerous cells, likely cancer cells, with prominent, dark, textured nuclei. The cells are set against a blue background with a bokeh effect of light spots. A semi-transparent white rectangular box is centered over the image, containing the title text in white, bold, uppercase letters.

TURNING A COLD TUMOR TO HOT

Leadership

PIERLUIGI PARACCHI

CEO & Co-Founder



- >\$200MM raised in Venture Capital (VC); \$400MM+ exits
- Co-Founder & Board Member **Altheia Science**, Founder & CEO of **Quantica SGR**, Co-founder of **Axon Capital** and **Aurora Science**, previously Venture Consultant at **Sofinnova Partners**. Member of the **Assobiotec Executive Committee**, the Italian Association for the development of biotechnology, Moderator of the "Working Table for the Internationalization of Biotechnology Sector" **Ministry of Foreign Policy and Italian Development Cooperation**

LUIGI NALDINI

Professor, M.D., Ph.D.,
Co-Founder & Executive Scientific
Board Chairman



- "Father" of Lentiviral Gene Therapy
- Director **SR-Tiget**, Professor of Cell and Gene Therapy at **San Raffaele University**, Milan, Italy
- He developed with Russo, as GSK exe, the **first ever gene therapy product approved** for commercialization, 2017 **Strimvelis®** for ADA-SCID

CARLO RUSSO

M.D., Chief Medical Officer &
Head of Development



- Former **GSK** head of R&D Rare Disease Unit; Head of Devel. R&D Biopharma Unit
- Former **Merck** Head of Regulatory for New Vaccines, including Gardasil and Rotateg

RICHARD B. SLANSKY

Chief Financial Officer



- 30+ years life science executive experience including at **Biological Dynamics**, **GenMark** and **CN Bioscience**
- Raised \$500MM+ in equity and debt capital in public and private offerings

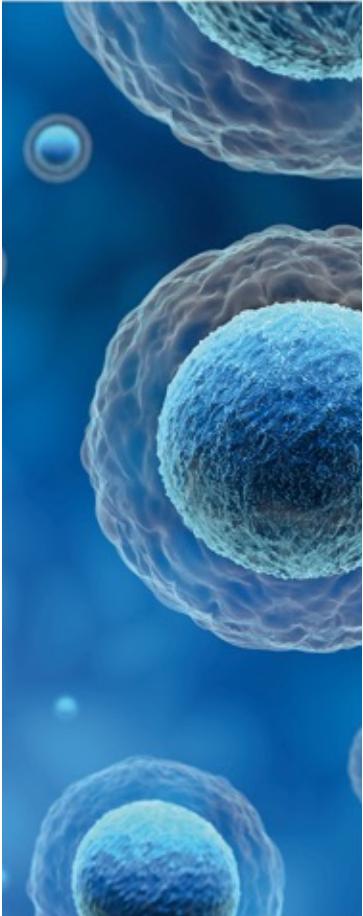
STEFANIA MAZZOLENI

Ph.D., Director of Program
Development



- 15+ years experience in R&D, oncology and project management, in both academia and industry

Genenta Built on Strong Foundations



**PROPRIETARY PLATFORM
TO PROVIDE DURABLE
AND SAFE TREATMENTS
FOR SOLID TUMORS**

**GENERATING CLINICAL
PROOF OF CONCEPT
FOR BREAKING
IMMUNE TOLERANCE**

**ROBUST FUNDING
AND PARTNERSHIPS
TO TAKE
TO NEXT STAGE**

- **One-time cell therapy** designed to break tumor-induced immune suppression by enabling sustained targeted expression of **therapeutic payload inside the TME¹** minimizing potential systemic toxicity
- **Tumor and antigen agnostic**
- Highly synergistic in **combination with I/O**
- Lead product candidate precisely delivers **IFN- α to the tumor microenvironment** aiming to break immune tolerance
- Phase 1/2a clinical data: favorable initial biological data with **no drug limiting toxicities**

- **Expected cash runway to Q2 of 2025**; ~\$10M Tax Credit accumulated to date
- Research engine through partnership with **SR-TIGET²**

¹ TME: tumor microenvironment

² SR-TIGET is a world leading cell and gene therapy institute founded by San Raffaele Research hospital, a co-founder and key shareholder of Genenta, and non-profit organization Telethon

Temferon™ Designed to Address Three Major Challenges in I/O Treatment of Solid Tumors

TEMFERON

ACHIEVE DURABLE RESPONSES

ENABLE A DURABLE THERAPEUTIC POTENTIAL

Engineering of hematopoietic stem and progenitor cells (HSPCs) creates a **living drug stable reservoir** that may ensure a persistent response (HSCS ex-vivo Lentiviral strategy).

AGNOSTIC BUT SELECTIVE DELIVERY AND SWITCH COLD TO HOT

DELIVER TREATMENTS TO SOLID TUMOR AND BREAK TUMOR INDUCED TOLERANCE

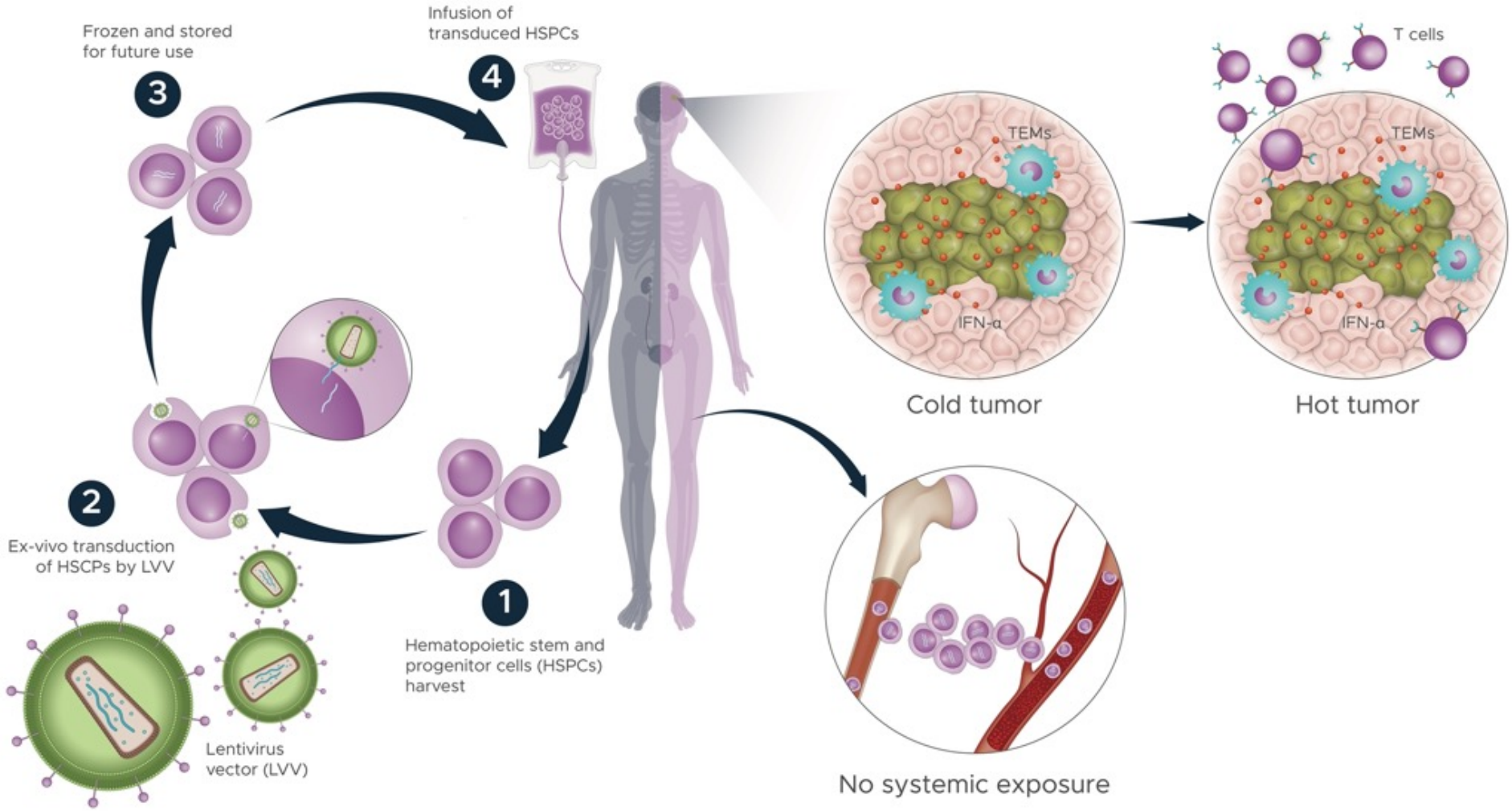
Tie2+ expressing monocytes (TEMs), naturally recruited by growing tumors, **infiltrate and deliver the payload.**

AVOID SYSTEMIC TOXICITY

LIMIT EXPRESSION OF THERAPEUTIC TO TUMOR MICROENVIRONMENT

Proprietary transgene expression technology designed to ensure **precise intra-tumor expression of payload therapy avoiding systemic toxicity** (microRNA regulation).

Temferon: Hematopoietic stem cell-based single treatment for solid tumors



Temferon Agnostic Efficacy Designed to Be Suitable For Treatment of Large Number Of Solid Tumors¹

| INDICATION | MARKET SIZE U.S. INCIDENCE ² | UNMET NEED 5Y SURVIVAL ³ |
|---|---|-------------------------------------|
| Glioblastoma Multiforme | ~3,721 ⁴ | 8.3% ⁵ |
| Renal cell carcinoma | ~81,800 | 78% |
| Melanoma (Stage 4) | ~4,880 | 35% |
| High Grade Osteosarcoma | 3,970 | 69% |
| NSCLC (Stage 4) | ~126,320 | 8% |
| Breast Cancers (Stage 4) | ~17,867 | 30% |
| Squamous cell carcinoma (SCC) head and neck: (Stage IV) | ~45,000 | 20-50% |
| Bladder cancer (Stage 4) | ~4,114 | 8% |
| Liver & Intrahepatic Bile Duct Cancer | ~41,210 | 22% |
| Gastroesophageal adenocarcinoma/SCC | ~21,560 | 22% |
| Mesothelioma | ~3,000 | 12% |
| Liver metastases (e.g., colorectal, breast, urothelial, melanoma) | ~123,000 | 15% at 1 year |
| Epithelial ovarian cancer | ~19,710 | 51% |

1 - Combo with Immune Checkpoint Inhibitors - I/O, Tie2+ Expressing Monocytes Presence, Tumor Microenvironment access pre- & post-treatment

2 - SEER Database - Estimated new cases in 2023 ; 3 - SEER Database 5-year survival rate 2013-2019;

4 - SEER Estimated New cases in 2023 adjusted on Glioblastoma frequency (15%) over all the primary brain tumor (Omstrom et al., 2019 - <https://doi.org/10.1093/neuonc/noz150>) ; 5- Stupp et al, 2009 SCC:

Squamous Cell Carcinoma

Temferon Agnostic Efficacy in Treating Solid and Hematological Tumors

SOLID TUMORS

HEMATOLOGICAL MALIGNANCIES

GLIOBLASTOMA

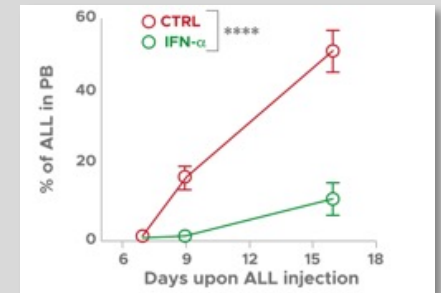
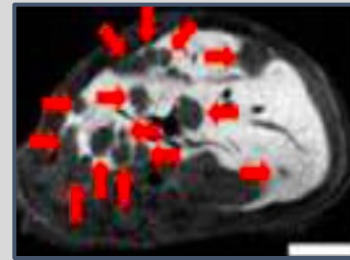
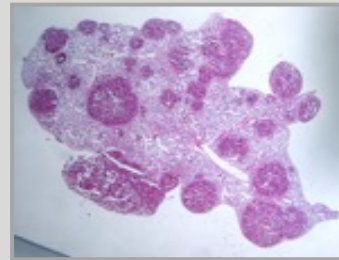
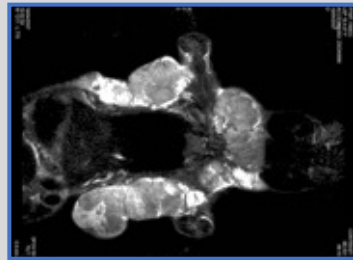
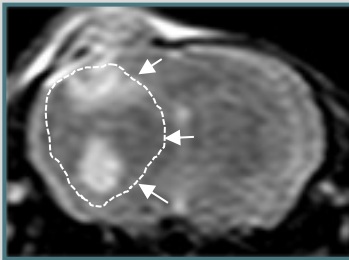
BREAST

LUNG METASTASIS

CRC LIVER METASTASIS

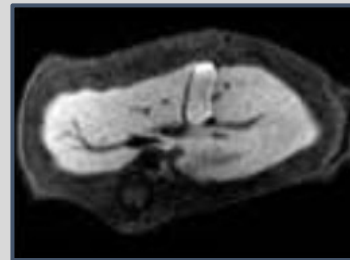
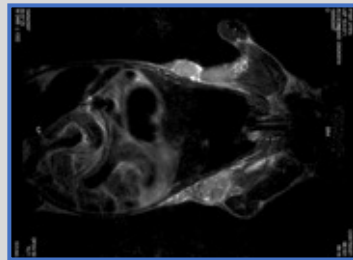
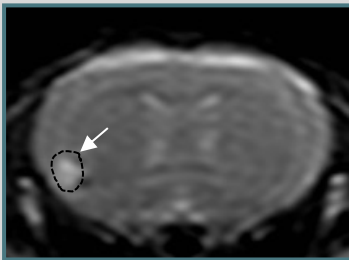
B-ALL MURINE B-ALL CELLS

CONTROL

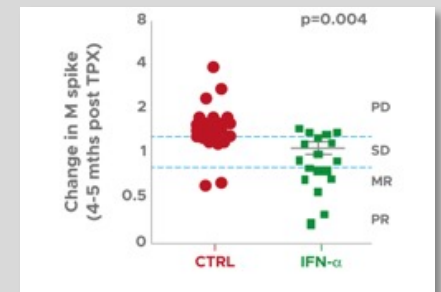


Escobar et al., Nature Communication 2018

IFN-α
IMMUNO-GENE
THERAPY



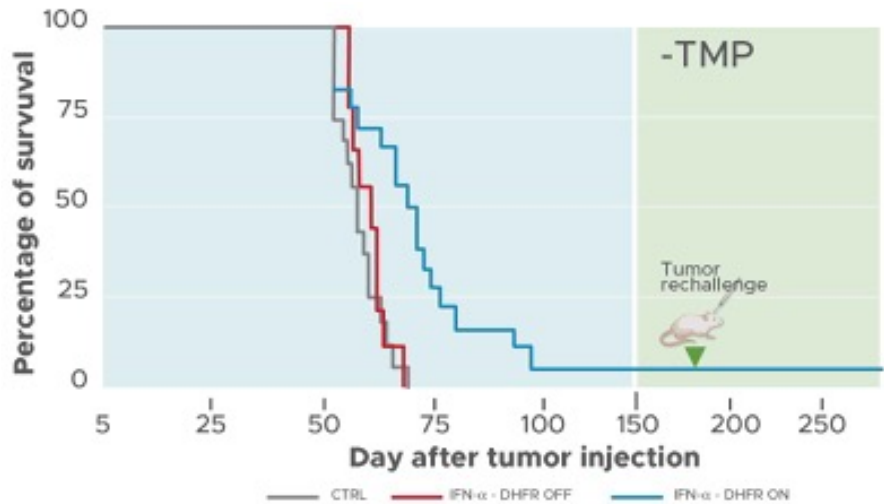
MULTIPLE MYELOMA VK*MYC MICE



B. Gentner Unpublished

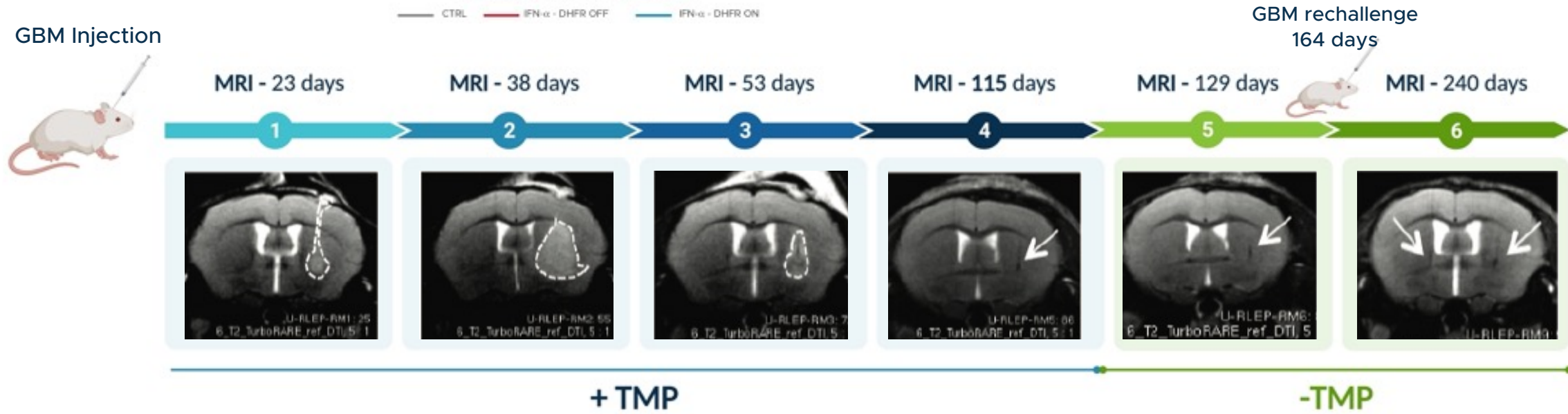
De Palma et al., Cancer Cell 2008; Escobar et al., Sci Transl Med 2014;
Catarinella et al., EMBO Mol Med 2016; Escobar et al., Nature Communication 2018

The Temferon Triggered Hot TME Status Results in a Long-Term Immunoprotection

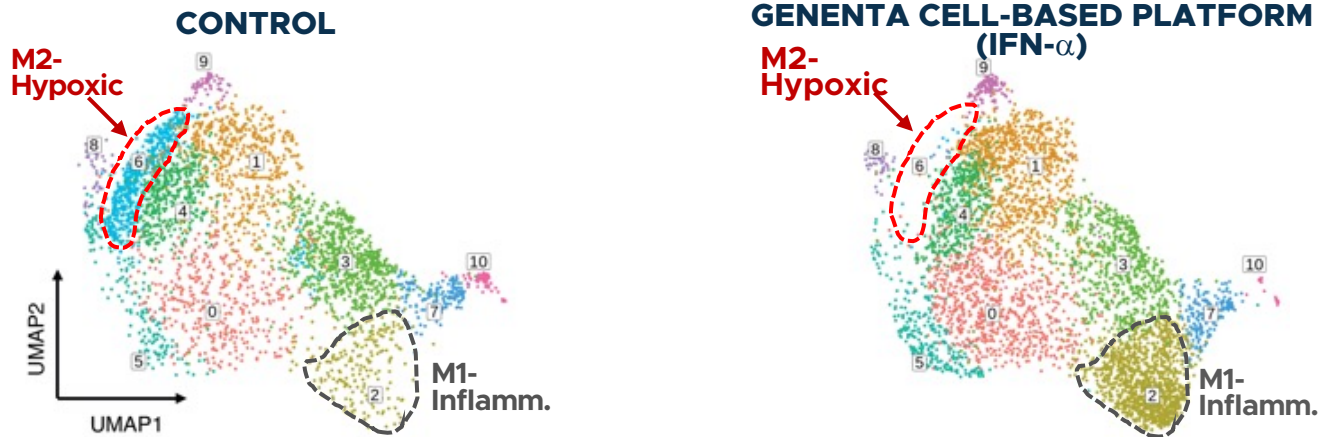


IFN- α cytokine was fused with a destabilizing domain (DD) which cause its proteasome degradation.

Trimethoprim (**TMP**) binds to the DD destabilizing domain and allows IFN- α to be expressed.

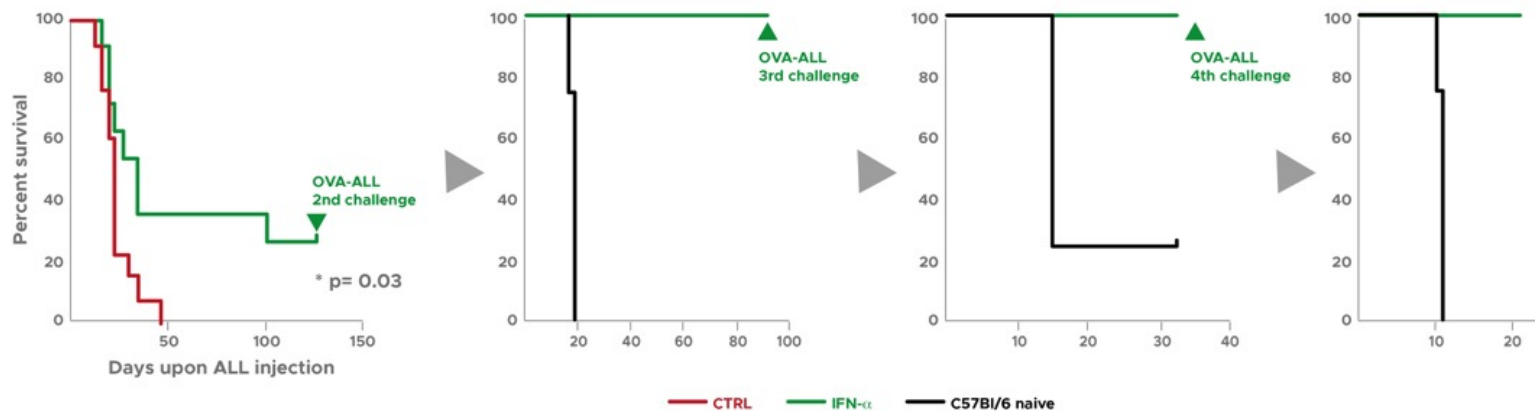


Temferon reprograms the TME and Switches Tumor State from Cold to Hot



SWITCH the TME MYELOID CELL COMPARTMENT...

- imposes an ISG-driven immunostimulatory
- programs to non-classical monocytes associated with M1 skewing of the myeloid populations
- activates tumor-infiltrating DCs



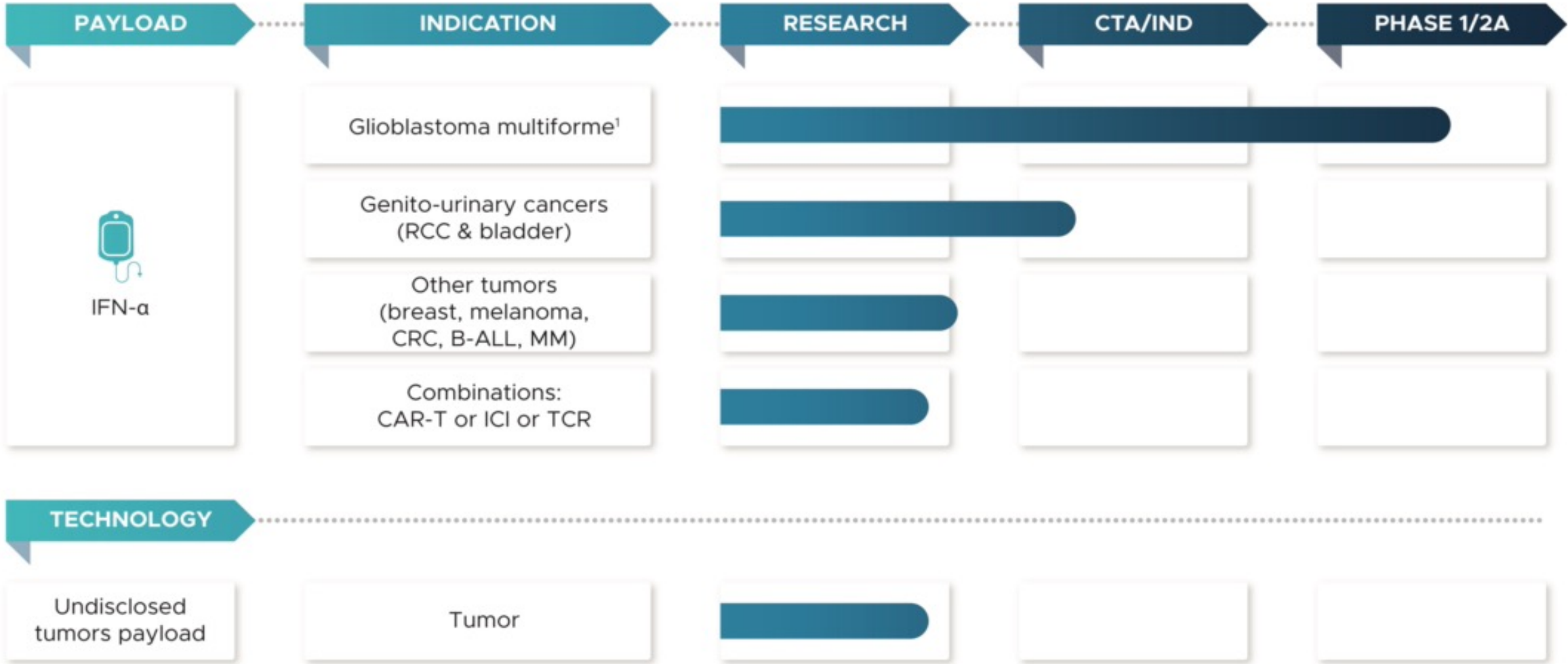
...TO GET a HOT T CELL COMPARTMENT

- increases active CD8+ T cells infiltration
- Induces T cells clonal expansion including cytotoxic CD8+ T cells and tumor-reactive T cell clones
- reduces percentage of exhausted CD8+ T cells
- increases the central memory T cells

Graphs have been faithfully reproduced by the original article

De Palma et al., Cancer Cell, 2008; Escobar et al., Sci Transl Med, 2014; Catarinella et al., EMBO Mol Med, 2016; Escobar et al., Nature Communication 2018; Birocchi et al., Sci Transl Med, 2022

Pipeline



¹ODD status in US and EU

Temferon Dose Ranging Phase 1/2a Trial in uMGMT GBM

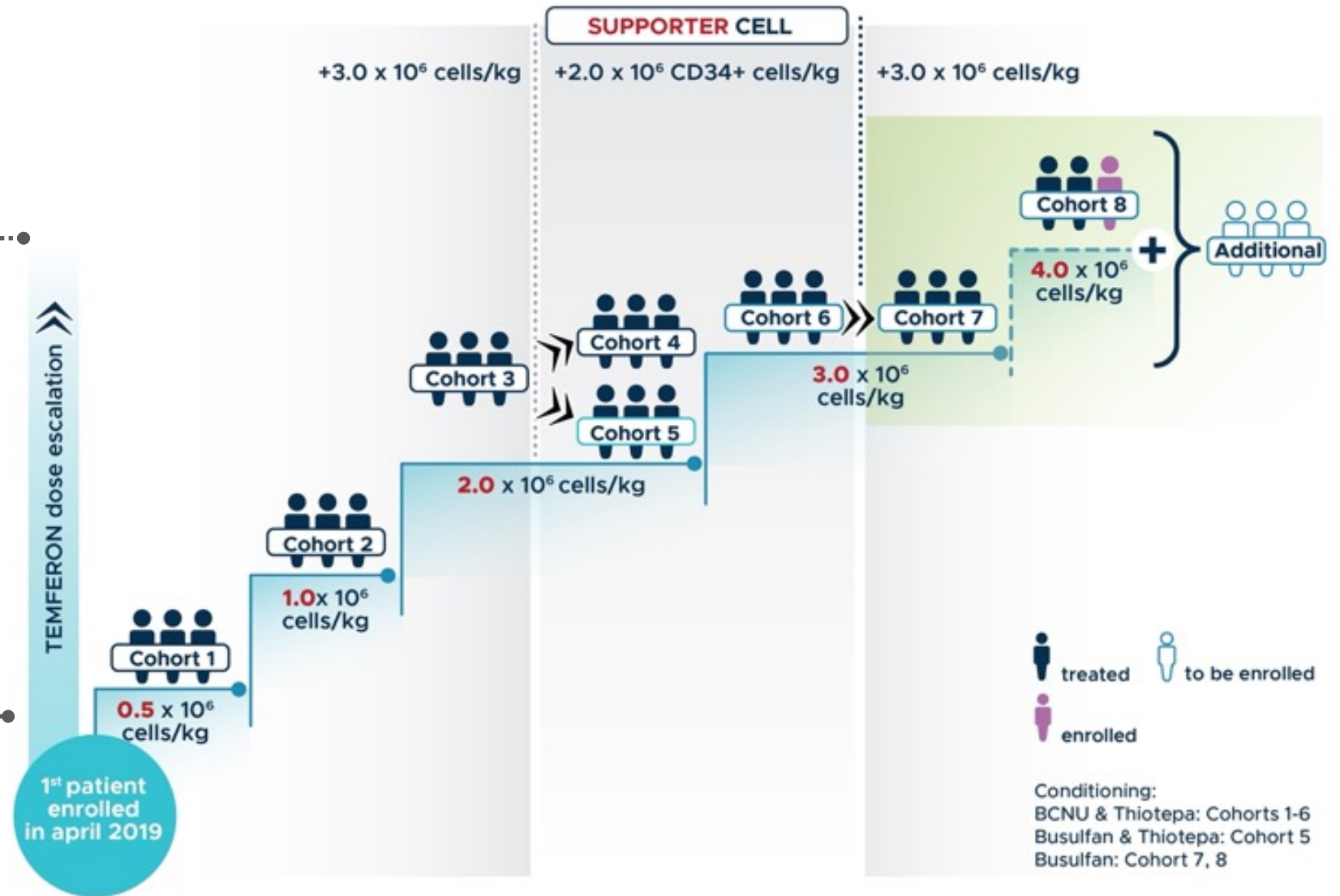
A multi-center, open-label, dose escalation & extension study in newly diagnosed GBM patients with unmethylated MGMT promoter following standard of care. DMC¹ at completion of each cohort

PRIMARY ENDPOINTS:

Tolerability and safety at 90 days

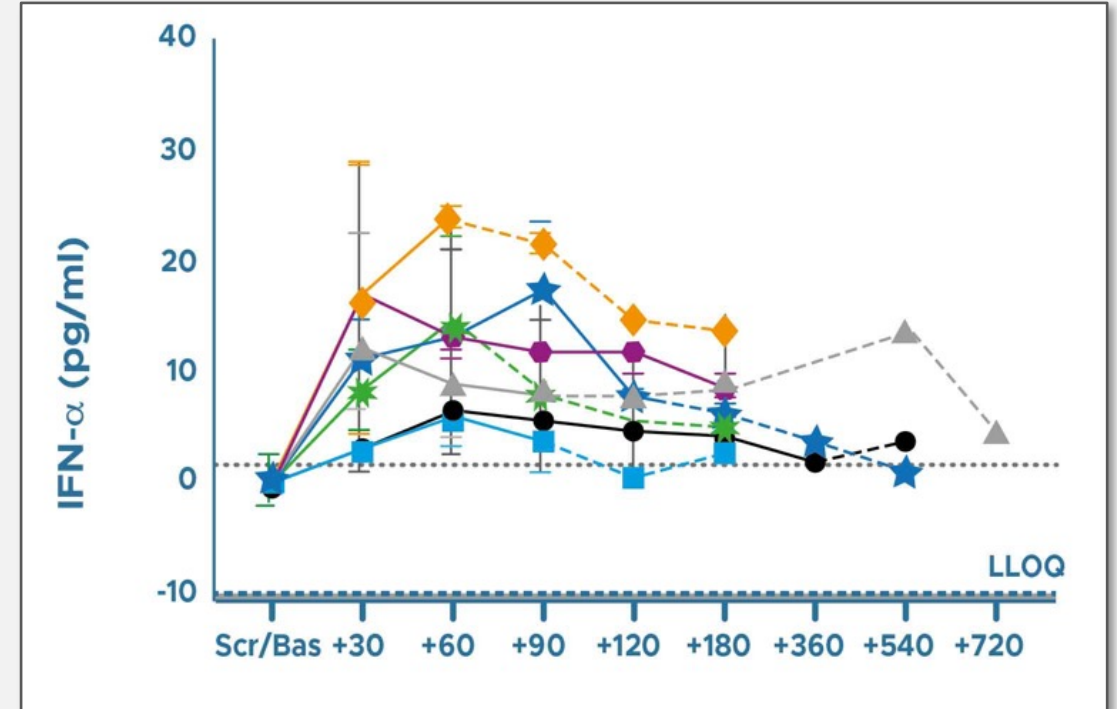
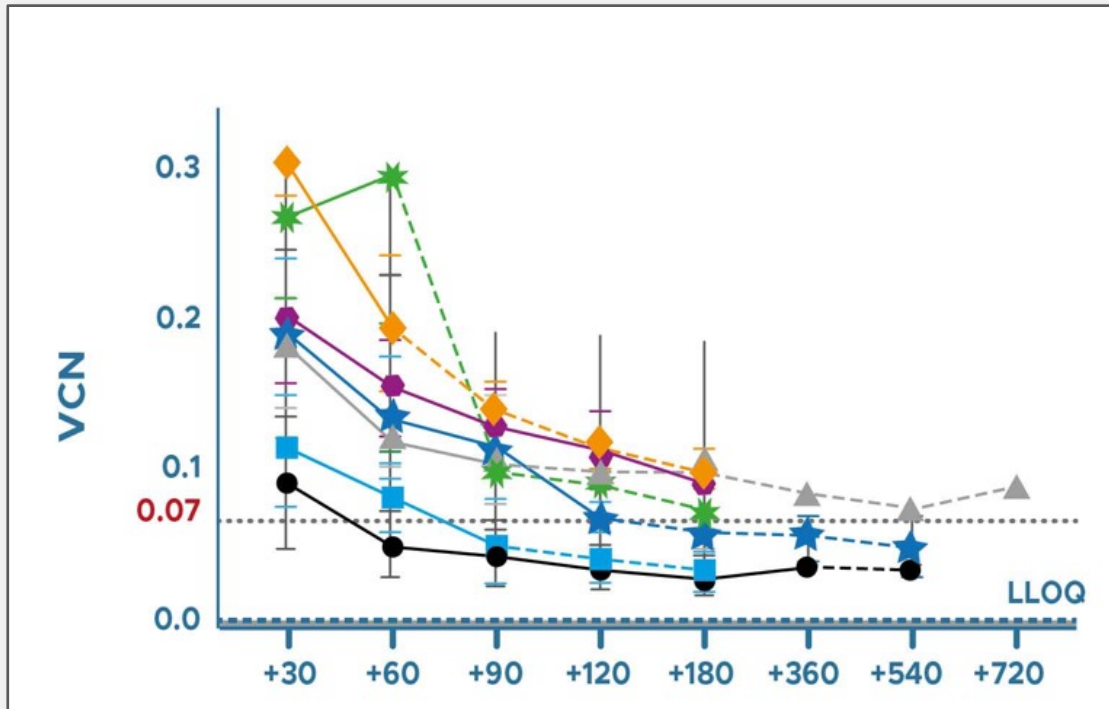
SECONDARY ENDPOINTS:

Long-term tolerability, safety and efficacy including PFS and OS up to 2 years



*1 DMC: Data Monitoring Committee

Temferon Single Dose is Durable and Well Tolerated



● Cohort 1 ■ Cohort 2 ▲ Cohort 3 ◆ Cohort 4 ★ Cohort 5 ◇ Cohort 6 ★ Cohort 7

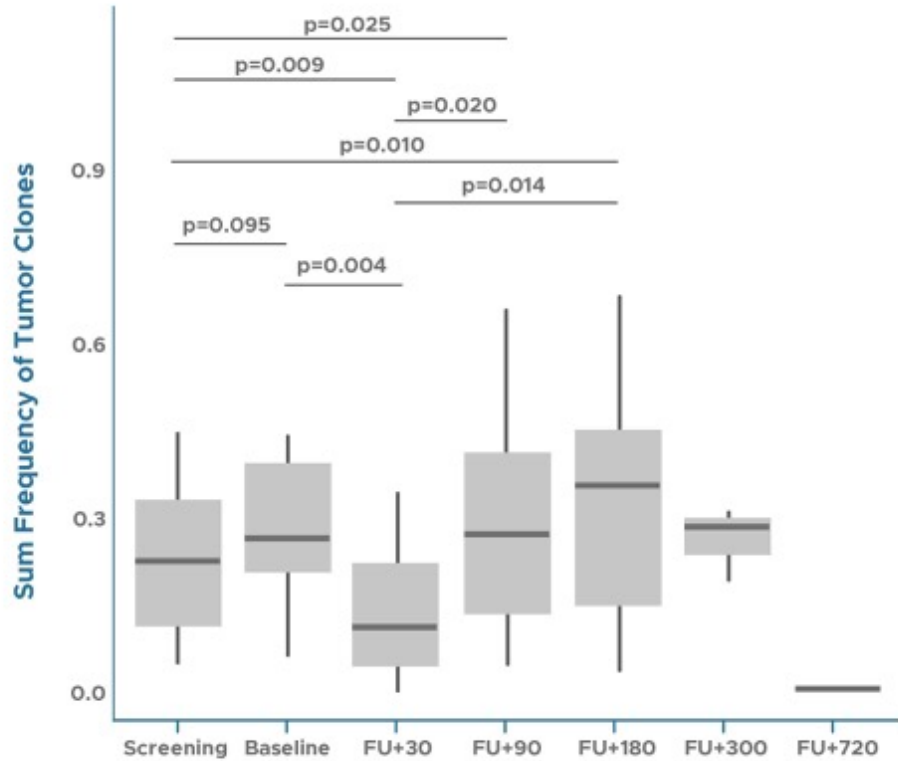
Dashes line connect timepoints with less than 3 measurements

Temferon Breaks Tolerance

Allowing Intra Tumor Infiltration of T Cell Clones

T CELL CLONES FROM THE TUMOR CAN OFTEN BE DETECTED IN THE BLOOD

- Contraction at day +30 in the blood (ASCT conditioning)
- Rebound after 3-6 months

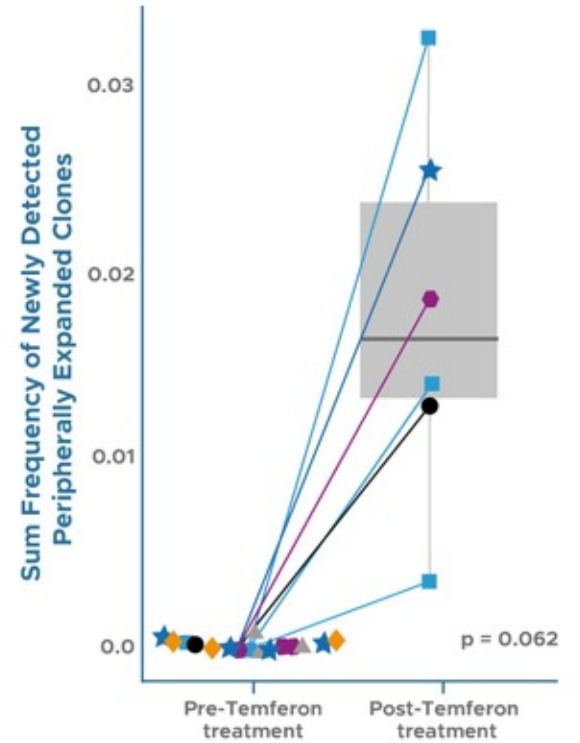
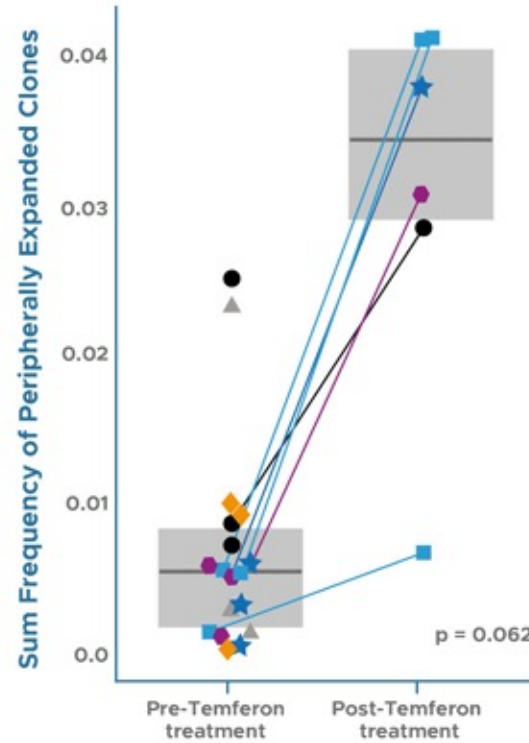


p-values from uncorrected paired Wilcoxon tests

EXPANDED PERIPHERAL T CELL CLONES ARE INCREASED AT 2nd SURGERY

Pre-existing peripheral T cells clones

Newly-detected peripheral T cells clones



● Cohort 1 ■ Cohort 2 ▲ Cohort 3 ◆ Cohort 4 ★ Cohort 5 ◆ Cohort 6

Preliminary Clinical Data in uMGMT GBM: Safe, Well Tolerated and Biologically Active



SAFETY

Detectable but very **low level of IFN- α** (pg/ml range) in the plasma. Expected and **manageable** adverse events and serious adverse events¹ associated with autologous stem cell transplantation and glioblastoma.

TOLERABILITY

No dose limiting toxicities to date.

Rapid engraftment and **hematological recovery** observed in all patients treated (n=22).

BIOLOGICAL ACTIVITY

Current data shows median **overall survival of 17 months² with an interim² 2-year survival rate higher than what is historically reported.**

Temferon-derived differentiated cells were evident within the peripheral blood 14 days after infusion and were still **detectable at more than 24 months.**

Evidence of a **pro-inflammatory state** in patients that required a second surgery.

1 - The reported serious adverse events (SAEs) for Cohorts 1 to 6 were of the type typically associated with transplant procedures (pneumonia, pulmonary embolism, febrile neutropenia, fatigue, C.diff infection, CMV reactivation, sepsis, anemia due to CMV reactivation) or underlying disease GBM (worsening left hemiparesis, seizure, brain abscess, sudden death). A suspected unexpected serious adverse reaction (SUSAR) of elevated gamma glutamyl transferase was also reported (spontaneously resolved).

2 - Cutoff date – December 21, 2023

Temferon 2nd Solid Tumor Indication Urinary Cancers

Genenta selected **metastatic clear cell renal cell carcinoma** (mRCC) and **metastatic urothelial carcinoma** (mUC) as additional solid tumor indications for Temferon clinical development

RATIONALE

Strong historical evidence to support potential Temferon efficacy

- Until 2005, systemic administration of IFN- α and IL-2 were SOC treatment for mRCC.
- IFN- α in mRCC was associated with a survival benefit compared to controls [644 mRCC pts: OR for death at 1 year=0.56 (95%CI 0.40 to 0.77)¹]
- Systemic IFN- α use limited in the past by the systemic toxicities
- IFN- α as an intravesical therapy for UC has been evaluated over many decades with variable results. Short drug exposure time rather than inherent lack of anti-tumour activity²

SOLUTION

Temferon has been designed to attenuate systemic toxicity associated with IFN- α and to achieve greater therapeutic benefit within the TME

Targeted IFN- α delivery as an innovative and clinically relevant approach



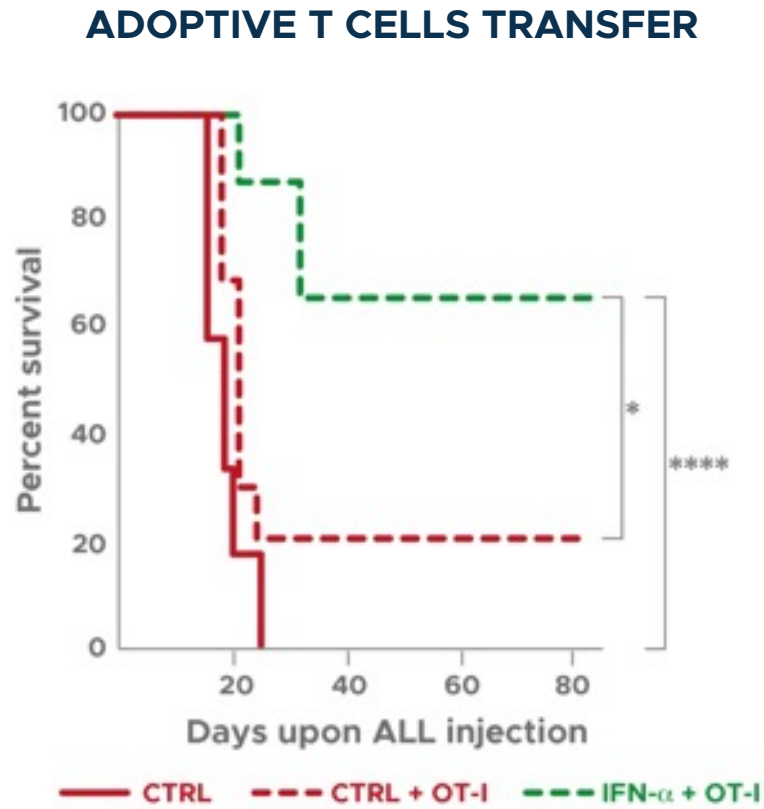
BCG-unresponsive non-muscle invasive bladder

- CR in 51% of patients with *in situ* carcinoma by three months³

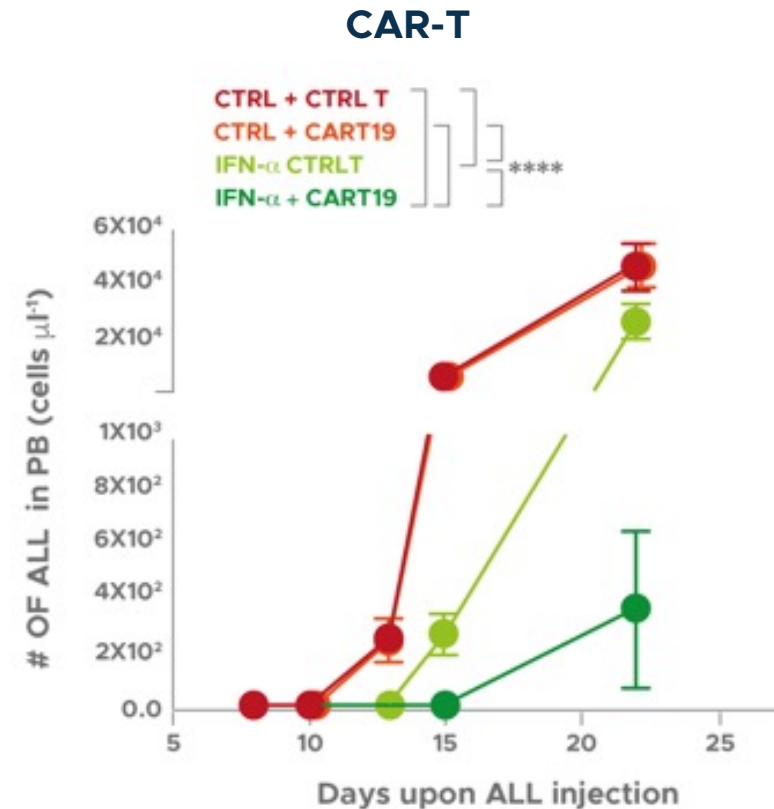
Temferon reprograms the TME and acts directly on tumors cells and neo-angiogenesis

Combination of Temferon with I/O Treatments Increases OS and Tumor Control

Temferon boosts/rescues other I/O therapies



OT-I = Ovalbumin transgenic mice



Escobar et al., Nature Communication 2018

Graphs have been faithfully reproduced by the original articles

Summary: Working Towards a Transformative Single and Permanent Treatment of a Broad Range of Solid Tumors



- Transformative proprietary platform designed to overcome the challenges of immunoncology therapy: infiltrating solid tumors, limiting toxicity and achieving a durable response
- Supported by preclinical and initial clinical data including engineered cells in patients from 14 days until 18 months+ after treatment, and payload expression without systemic toxicity
- Tumor and antigen agnostic; potential for broad combination use
- Combination use with I/O confirmed in preclinical animal models
- Upcoming catalysts:
 - ▶ Part 1 of Phase 1/2a for GBM completed enrollment in 1Q'24
 - ▶ Amendment Submission for the Phase 2 GBM study in H2'24
- Expected cash runway to Q2 of 2025, with no debt or warrants¹
- In 1H 2023, the U.S. Food and Drug Administration (FDA) and the European Commission have granted Orphan Drug Designation (ODD) to Temferon for the treatment of glioblastoma multiforme.
- Refractory Advanced Genitourinary Malignancies have been selected as the additional solid tumor indications for Temferon clinical development

1 - Except normal payables, accruals and underwriters' warrants

Anticipated Pipeline Development Milestones *

- Pre-CTA Regulatory Meeting for GU indications
- Complete enrollment TEM-GBM Phase 1 trial
- CTA filing for Advanced Genitourinary Malignancies (TEM-GU study)

H1 2024

- TEM-GBM study Phase 2 Amendment Submission
- Start of the TEM-GU Phase 1 trial

H2 2024

- Start of the TEM-GBM Phase 2 trial

H1 2025

- Review of TEM-GBM Phase 2 preliminary data
- Start of the TEM-GU Phase 2 trial

H2 2025

* Our anticipated pipeline development milestones constitute forward-looking statements as discussed in greater detail on slide 2. You should not rely upon forward-looking statements as predictions of future events.



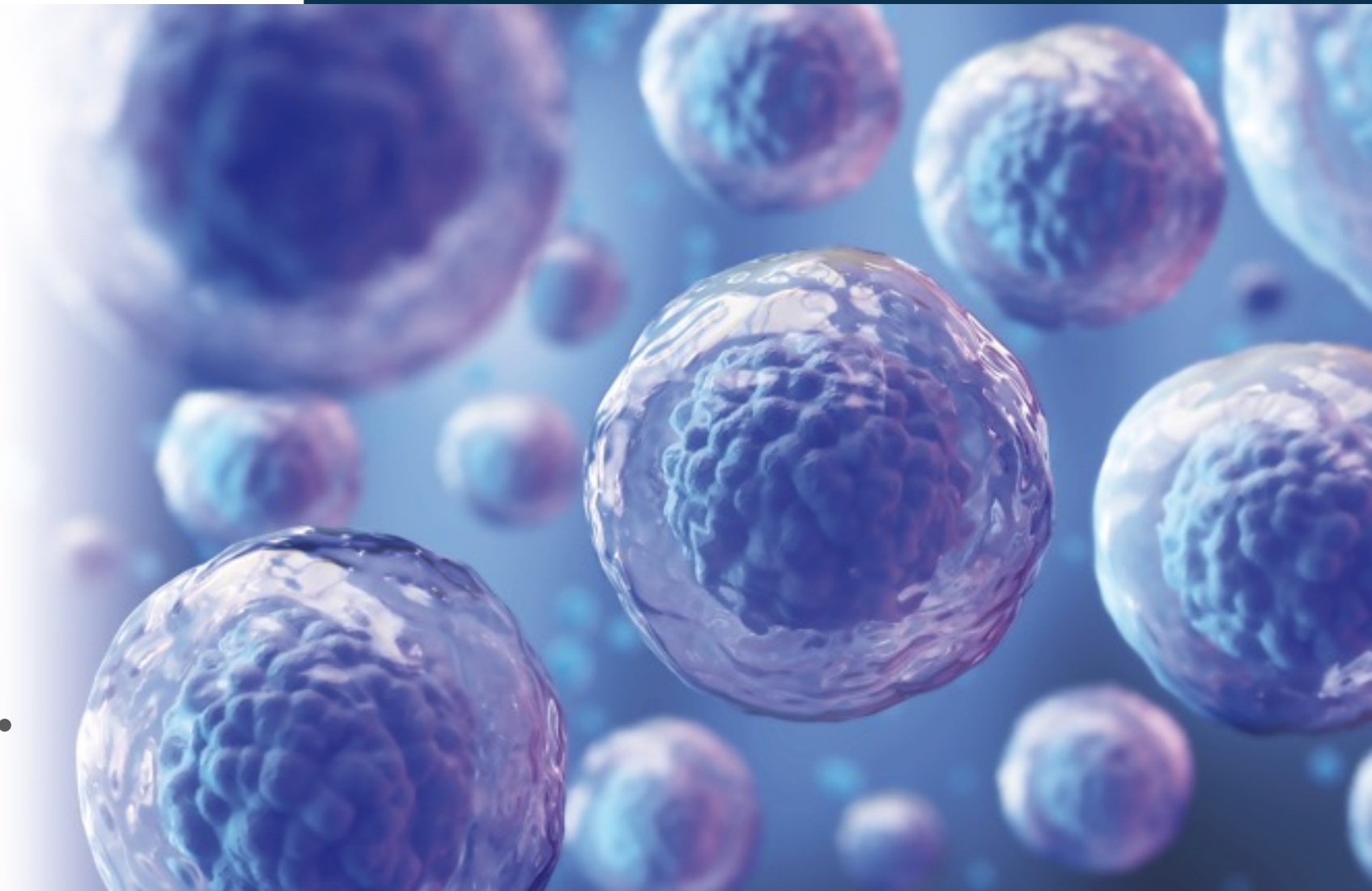
Pierluigi Paracchi, CEO

pierluigi.paracchi@genenta.com

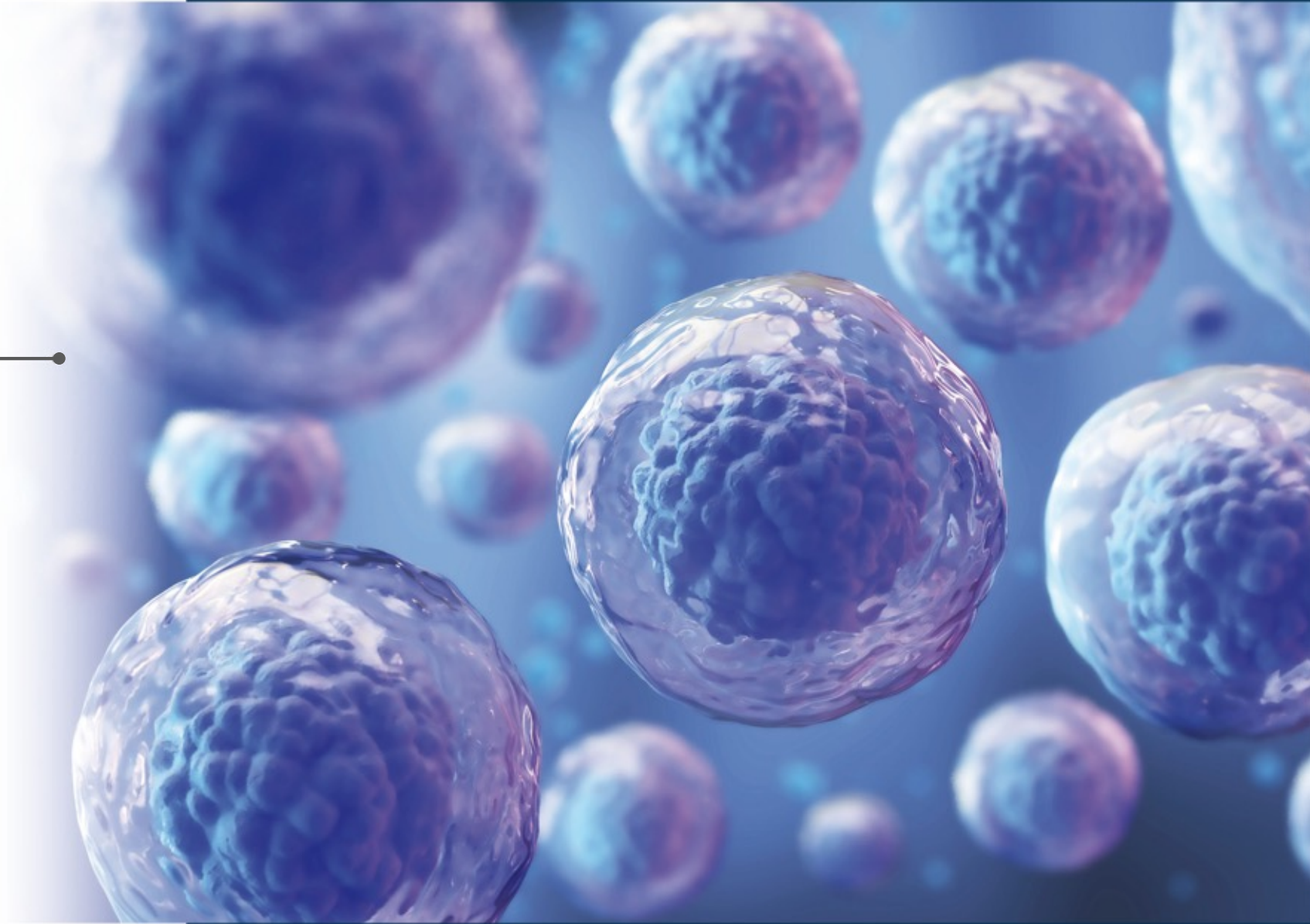
www.genenta.com

Dibit 1 - San Raffaele Hospital, via
Olgettina 58 - 20132 Milano, Italy

LaunchLabs - Alexandria Center, 14th
Floor - 430 East 29th Street
New York, NY 10016, USA



APPENDIX

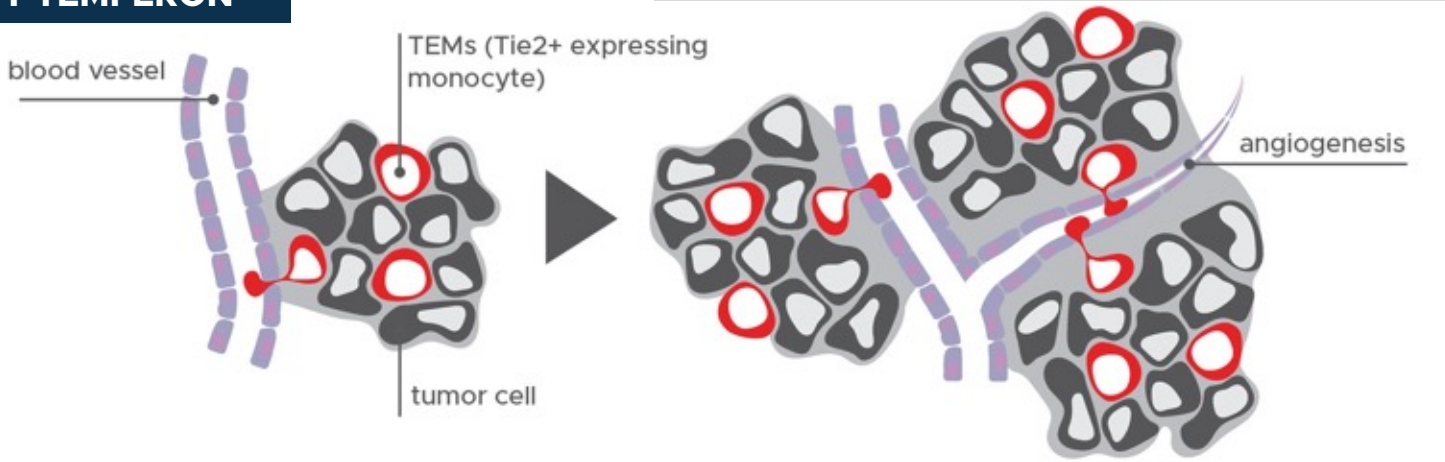




MECHANISM OF ACTION

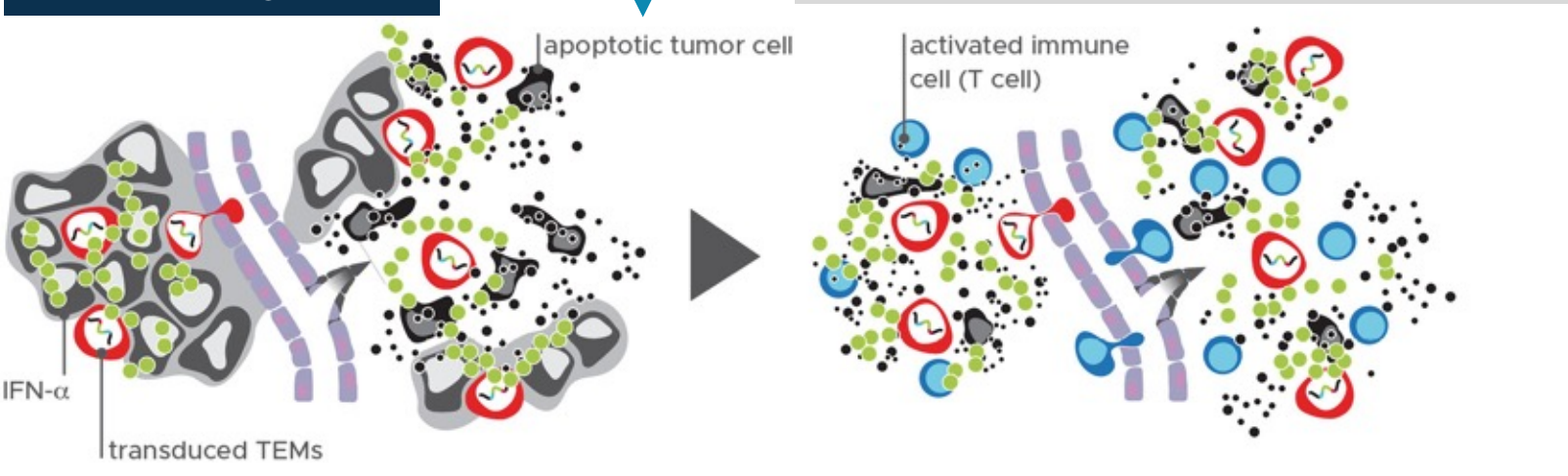
Temferon Designed to Deliver IFN- α in the Tumor Microenvironment to Break Immune Tolerance

WITHOUT TEMFERON



TEMs* are natural tumor infiltrating monocytes associated with angiogenesis in tumors

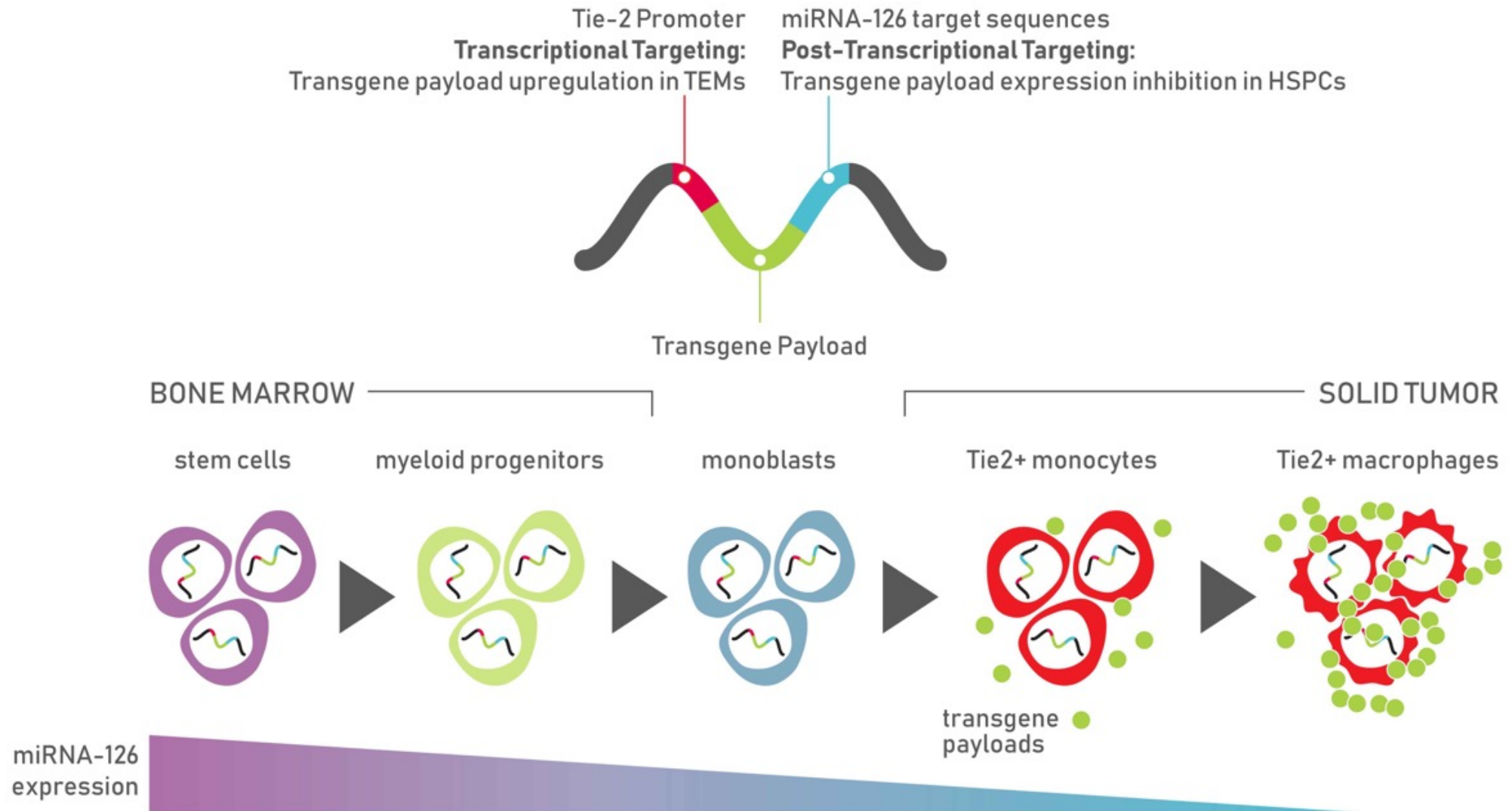
WITH TEMFERON



IFN- α targets tumor proliferation via anti-angiogenic impact and reprogramming the immune system

Proprietary mRNA Mediated Control of Payload Expression

Interaction of miRNAs with their miRNA-targets regulates gene expression via mRNA degradation and translational repression



Clear Development Rationale for Temferon in GBM

ROBUST SCIENTIFIC GROUNDING

- Characterized by a **highly suppressive tumor microenvironment** induced by a subset of tumor associated macrophages
- Temferon is designed **to break immunosuppression**

LIMITED AVAILABLE TREATMENTS

- Temferon may be offered as **1st line monotherapy** after 1st surgery enabling patient's **uncompromised immune system to be harnessed**
- Enables impact of Temferon to be **seen in isolation**
- Temferon **ODD status in US and EU**

FAVORABLE PRECLINICAL DATA

- Temferon demonstrates **control of GBM** pathology despite its aggressive nature
- Temferon creates a **pro-inflammatory state** that induces an immune system reset, **breaking tumor tolerance**

ABOUT GBM

Estimated market size by 2032: \$3.4B¹

Annual incidence: 3 per 100,000 adults², ~60% with uMGMT promoter status³ (target population)

Median survival: ~≤ 15 months³; 5-year survival: 5.5%⁴

Intellectual Property

- Potential 12-year market exclusivity for new biological products (U.S.)
- Key patents already granted

| | US | EU | China | Japan | ROW | Expiration |
|--|-----------|-----------|--------------|--------------|------------|-------------------|
| Gene vector comprising mi-RNA | ✓ | ✓ | ✓ | ✓ | ✓ | 4/30/2030 |
| mi-RNA regulated vectors | ✓ | ✓ | ✓ | ✓ | ✓ | 5/26/2026* |
| Monocyte cell (Tie-2) activation process | ✓ | ✓ | ✓ | ✓ | ✓ | 10/5/2027 |
| Method for Genetic Modification | ✓ | ✓ | ✓ | ✓ | ✓ | 10/24/2034* |
| Vector Production | ✓ | pending | pending | ✓ | pending | 7/13/2035 |
| Type 1 IFN gene therapy | pending | pending | pending | pending | pending | 4/20/2038** |

*Later expiration for certain U.S. patents pursuant to patent term adjustment (35 U.S.C. §154(b))

** Application pending, anticipated expiration based on 20 years patent term.

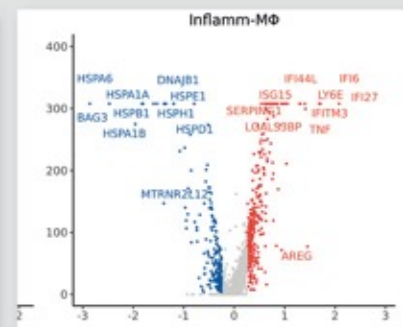
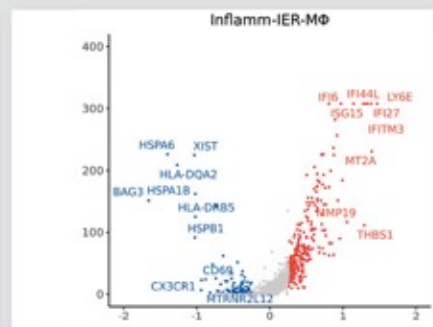
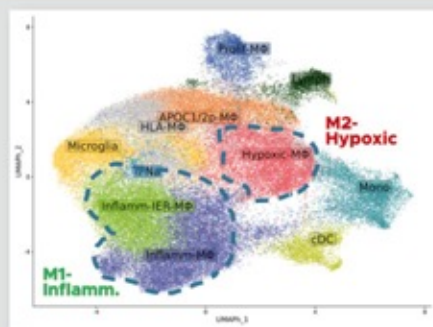
scRNAseq of GBM TME in Temferon and SOC Treated Patients

scRNAseq of GBM TME: Comparison of 5 Temferon patients and 6 SOC control relapses pts

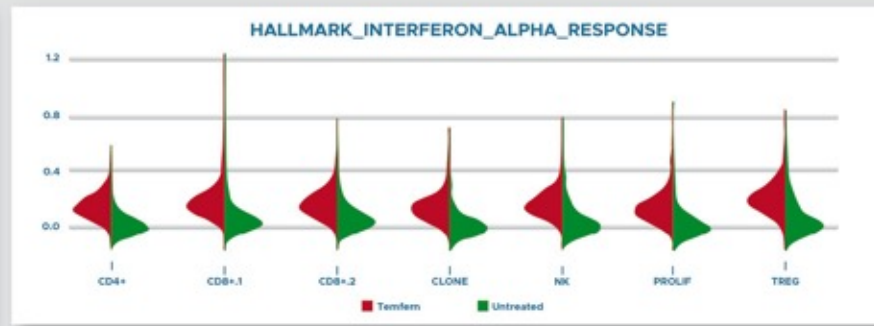
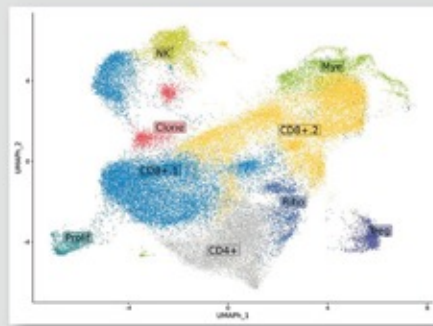
- Temferon patients who underwent second were compared to six recurrent glioblastoma patients treated as per the current first line standard of care (SOC – Temozolomide + Radiotherapy).

- Patients who received Temferon showed a general upregulation of IFN- α response hallmarks in the myeloid and T cells compartment of hematopoietic origin (CD45+ cells) and in the tumor cells (CD45-).

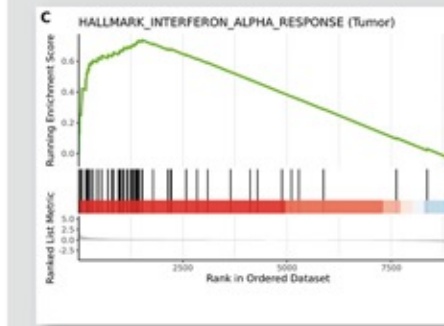
MYELOID COMPARTMENT



T CELL COMPARTMENT

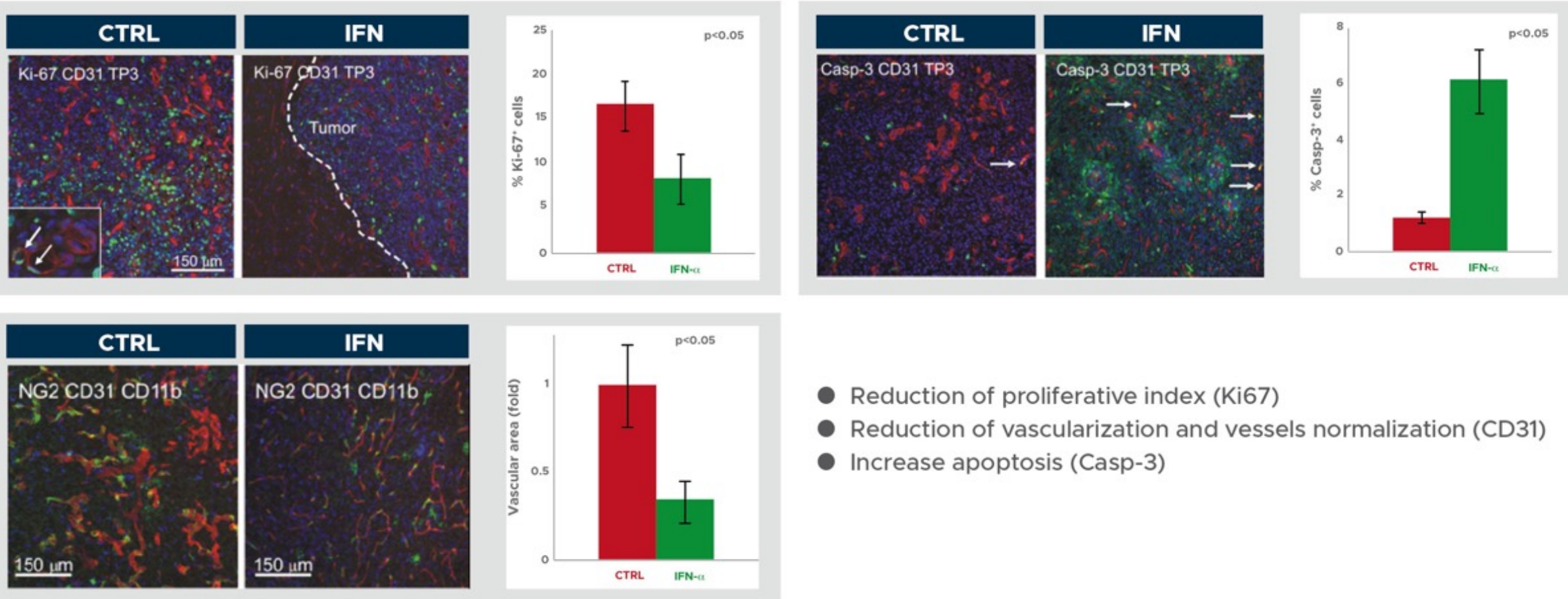


TUMOR CELLS



TEMs Cells Therapy Counteract Tumor Growth by Acting on Proliferation and on Vascularization

Tumor growth inhibition by TEMs IFN cell therapy in immunocompromised mice demonstrated the direct targeting at host-derived components like tumor cells and vessels



- Reduction of proliferative index (Ki67)
- Reduction of vascularization and vessels normalization (CD31)
- Increase apoptosis (Casp-3)

Board of Directors

PIERLUIGI PARACCHI

Chairman, CEO & Co-Founder



- Moderator of the **National Working Table for the Internationalization of Biotechnology Sector**, promoted by the **Foreign Ministry**. Member of the **Assobiotec** Executive Committee, the National Association of biotech companies. Co-Founder & Board Member **Altheia Science** and **Aurora Science**, Chairman **Lipogems International**. Previously, Founder & CEO of **Quantica SGR**, Co-founder of **Axon Capital**, Venture Consultant at **Sofinnova Partners**.
- \$400MM+ exits; >\$200MM raised as VC

JOHN L. CANTELLO

Ph.D.



- John is an independent advisor to the biopharma industry with over 20 years of experience. Former VP and Head of Business Development, Oncology Therapy Area at **GlaxoSmithKline** and VP and Head of BD, Respiratory & Immune Diseases at **AstraZeneca**.
- John has led teams accountable for assessing, valuing and transacting M&A, pipeline & commercial asset deals covering oncology, respiratory, inflammation, metabolic and rare diseases. He has a track record of closing deals (transacting >\$30B in deal value) representing primary care, specialty care and rare diseases.

LAUREN H. CHUNG

Ph.D.



- Lauren has over 20 years of operating experience spearheading agile investment management strategies and tactical asset allocation in the healthcare industry. As the founder and CEO of **Minleigh LLC**, a healthcare focused strategic advisory firm, Lauren has advised leadership, boards, and investment firms on global strategic plans, M&A, integration.
- Previously, Lauren co-founded **Tokum Capital Management**, a global institutional healthcare fund, and successfully managed its merger with **Perella Weinberg Partners**. Lauren serves on public and private company boards.
- She has a Ph.D. in Biomedical Sciences from Columbia University Vagelos College of Physicians and Surgeons, an M.B.A. from Columbia Business School, and a B.A. in Biochemistry and Economics with Honors from Wellesley College.

ARMON R. SHAREI

Ph.D.



- Armon is Founder and CEO of **Portal Bio** and formerly CEO and Founder of **SQZ Biotechnologies** (NYSE: SQZ), led company from invention to post-IPO with over \$300M in equity financing, \$1Bn **Roche** collaboration, and three clinical trials.
- He graduated from Stanford University, Ph.D. at Massachusetts Institute of Technology and Post-Doctoral at Harvard Medical School.

TODD WIDER

M.D.



- Consultant to numerous entities in the biotechnology space Co-founder and Board Member **Xanadu Bio** and prior Executive Chairman Emendo **Biot therapeutics**, Board Member **Abeona Therapeutics**, **Arya Science Acquisition Corp**.
- Todd is an active, honorary member of the medical staff of Mount Sinai Hospital in NYC. He graduated from Princeton University, M.D. from Columbia University Vagelos College of Physicians and Surgeons, where he was Rudin Fellow, and an AB, with high honors and Phi Beta Kappa, from Princeton University.
- Todd is also a principal in Wider Film Projects, a documentary film company focused on producing films with sociopolitical resonance that have won Academy, Emmy and Peabody Awards.

Scientific Advisory Board

LUIGI NALDINI

Professor, M.D., Ph.D.

He has pioneered the development of lentiviral vectors for gene therapy. With over 280 scientific papers, he has a SCOPUS Author h-index of 101. He has won numerous awards: the Outstanding Achievement Award from the **American Society of Gene and Cell Therapy (ASGCT)** in 2014, President of ESGCT in 2015, the Beutler Prize from the **American Society of Hematology (ASH)** in 2017, the Jeantet-Collen Prize for Translational Medicine in 2019. He was nominated as "Grande Ufficiale" dell'Ordine "Al Merito della Repubblica Italiana", one of the highest ranking honors in Italy, by the President of the Republic in 2019 and he is "Socio Corrispondente - Classe di Scienze Fisiche, Matematiche e Naturali" at **Accademia Nazionale dei Lincei**.

BERNHARD GENTNER

Professor, M.D., Ph.D.

Co-Founder. He is Professor in Immuno-Oncology, attending **physician** in the **Oncology Department at Lausanne University Hospital, Medical Director for the T cell therapy platform** and heads of **HSC engineering** within the **Lausanne branch of the Ludwig Institute**. He was **Group Leader at SR-TIGET** and Staff **Hematologist at the San Raffaele Hospital**. Received a MD from the University of Heidelberg and trained at MD Anderson Cancer Center and Baylor College of Medicine, Houston, Erlangen University Hospital and at San Raffaele Vita-Salute University. He is Author of more than 60 scientific publications and the recipient of the Young Investigator Award of ESGCT.

KENNETH C. ANDERSON

Professor, M.D.

Kraft Family Professor of Medicine at Harvard Medical School and Director of the Lebow Institute for Myeloma Therapeutics and Jerome Lipper Multiple Myeloma Center at Dana-Farber Cancer Institute. He is a Doris Duke Distinguished Clinical Research Scientist and American Cancer Society Clinical Research Professor.

LISA COUSSENS

Professor, M.D., Ph.D., FAACR

Professor and Chairwoman, Cell, Developmental & Cancer Biology Department at Oregon Health & Science University. She also serves as Hildegard Lamfrom Endowed Chair in Basic Science and Associate Director for Basic Science, Knight Cancer Institute.

MICHELE DE PALMA

Professor, Ph.D.

Professor at EPFL (École Polytechnique Federal de Lausanne). He is known for his work on the role of macrophages in cancer progression and the discovery of Tie2-expressing angiogenic monocytes.

RICHARD FLAVELL

Professor, Ph.D., FRS

Sterling Professor of Immunobiology at Yale University School of Medicine, and an Investigator of the Howard Hughes Medical Institute.

WOLF-HERVÉ FRIDMAN

Professor, M.D., Ph.D.

Professor Emeritus of Immunology at the Paris Descartes University Medical School in Paris, France. Former head of the Immunology Lab. of European.

MIRIAM MERAD

Professor, M.D., Ph.D.

Director of the Precision Immunology Institute at Mount Sinai School of Medicine NYC and Director of the Mount Sinai Human Immune Monitoring Center. Elected member of the American Society of Clinical Investigation and the recipient of the William B. Coley Award for Distinguished Research in Basic and Tumor Immunology.

TRICK Y. WEN

Professor, M.D.

Professor, Neurology, Harvard Medical School Director, Center for Neuro-Oncology, Dana-Farber Cancer Institute, Boston.

Financial Profile

Cash & cash equivalents and marketable securities¹

€ 18.8 MM

Expected cash runway

Q2 of 2025

Debt and warrant²

0

Number of shares outstanding³

€ 18.3 MM

Average volume³

~ 4K shares

1 - As of December 31, 2023

2 - Except normal payables, accruals and underwriters' warrants

3 - As of March 31, 2024

Stock Ownership Info

Founders and Leadership

29%

San Raffaele Hospital¹

10%

Institutions/Large FOs/Sovereign Fund

19%

1 - San Raffaele Research hospital is a co-founder and key shareholder of Genenta; ongoing relationship through service contract for clinical research. San Raffaele in alliance with non-profit organization Telethon runs the leading gene therapy institute SR-TIGET