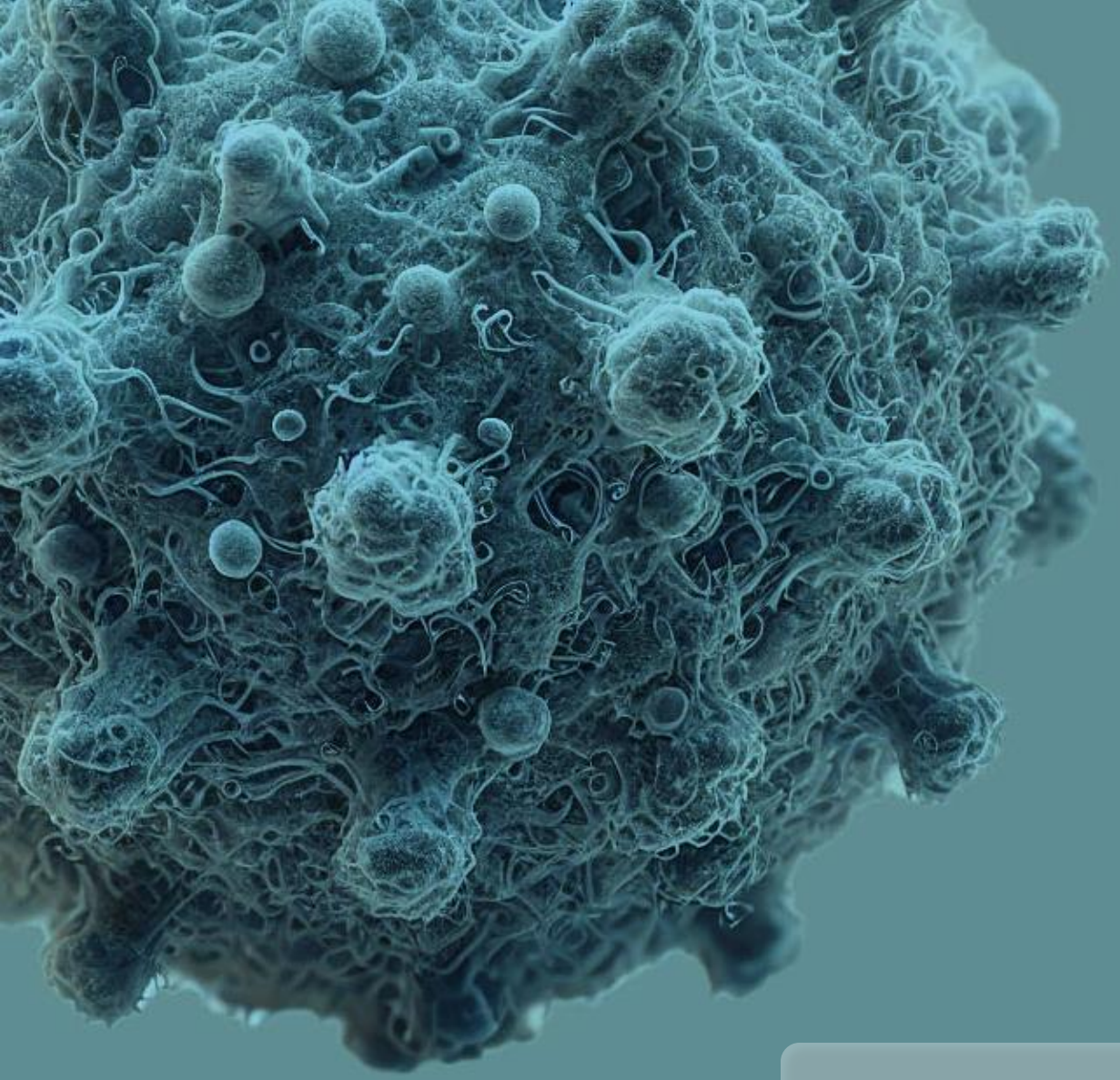

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# Corporate Presentation

NASDAQ: GNTA

January 2025

# Forward-Looking Statements and Other Notices

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.

This presentation discusses product candidates that are under preclinical or clinical evaluation and that have not yet been approved for marketing by the U.S. Food and Drug Administration (FDA) or any other regulatory authority. Until finalized in a clinical study report, clinical trial data presented herein remain subject to adjustment as a result of clinical site audits and other review processes. No representation is made as to the safety or effectiveness of these product candidates or the use for which such product candidates are being studied. TEM GBM and TEM GU are investigational product candidates for which the effectiveness and safety have not been established. In addition, neither TEM GBM nor TEM GU are approved for use in any jurisdiction.

# Leadership

## 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 **Axòn Capital**, Venture Consultant at **Sofinnova Partners.** $400MM+ exits; >$200MM raised as VC.

## LUIGI NALDINI

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

**Professor of Cell and Tissue Biology and of Gene and Cell Therapy at the San Raffaele University School of Medicine and Scientific Director of the San Raffaele Telethon Institute for Gene Therapy** (Milan, Italy). Pioneer of the development and the applications of lentiviral vectors for gene therapy and he has continued to investigate new strategies to overcome the major hurdles to safe and effective gene transfer, bringing about innovative solutions that are not only being translated into new therapeutic strategies for genetic disease and cancer, but have also allowed novel insights into hematopoietic stem cell function, induction of immunological tolerance, and tumor angiogenesis.

## CARLO RUSSO

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

Senior Executive in the pharmaceutical and biotech industry with extensive expertise in clinical development in oncology, gene therapy, cardiovascular, metabolic diseases, infectious and rare diseases. A proven track record of bringing products through Phase 1-3 clinical development to regulatory approval. Senior roles in large pharma companies **GSK, Merck** and Biotech companies **Adverum, Annapurna, VaxInnate Corporation** and a number of senior positions at research institutions including Cornell University Medical College, Columbia University and Scripps Research Institute.

## RICHARD B. SLANSKY

Chief Financial Officer

+ 30 years of experience as Chief Financial Officer in various biopharmaceutical diagnostic and life science companies, including **Biological Dynamics, Oncosec Medical, GenMark Diagnostics** and **C-N Biosciences** (now part of Merck). He also serves on the Board of Directors of several private companies, including Nuclear RNA Networks, an early-stage RNA gene transcription therapeutics company. Raised $500MM+ in equity and debt capital in public and and private offerings.

## STEFANIA MAZZOLENI

Ph.D., Director of Program Development

20 years of experience in life sciences R&D encompassing oncology, drug development, and cell and gene therapy — she has collaborated with various pharmaceutical companies and academic institutions. She earned a Master's degree in Medical Biotechnology and a **second-level vocational Master's in Pharmacy and Pharmaceutical Oncology** from the University of Milan. Additionally, she completed a Ph.D. in **Molecular and Cellular Biology** at Vita-Salute San Raffaele University. She is also a member of the European Academy of Tumor Immunology (EATI).



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# Genenta: Developing a First in Class Cell Therapy

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

**Temferon ™ is a one-time cell therapy** designed to break the tumor-induced immune suppression by enabling sustained targeted expression of **therapeutic payload inside the tumor microenvironment (TME).**

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

**TEM GBM** Phase 1/2a study:

- Phase 1 dosing completed;
- Favorable initial evidence of reprogramming of the tumor microenvironment;
- Potential ability to activate T cells which could then be enhanced by the use of immune checkpoint inhibitors.

**TEM GU** Phase 1 study:

- Enrollment started in Q4 2024, combination treatment option with immune checkpoint inhibitors and TKIs.

**PARTNERSHIPS TO TAKE TO NEXT STAGE**

Research engine through partnership with **SR-TIGET** a world leading cell and gene therapy institute founded by **San Raffaele Research Hospital**, a co-founder and key shareholder of Genenta.



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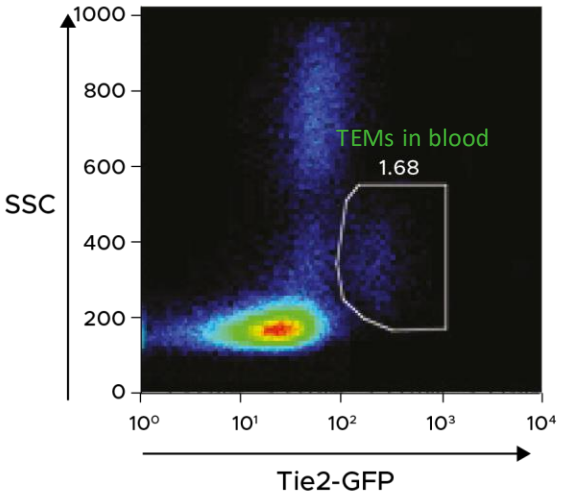
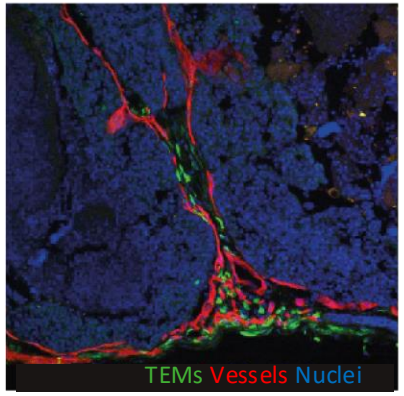
# Harnessing the power of Stem Cells while incorporating miRNA

## Tie2-Expressing Monocytes (TEMs)

Circulate low frequency



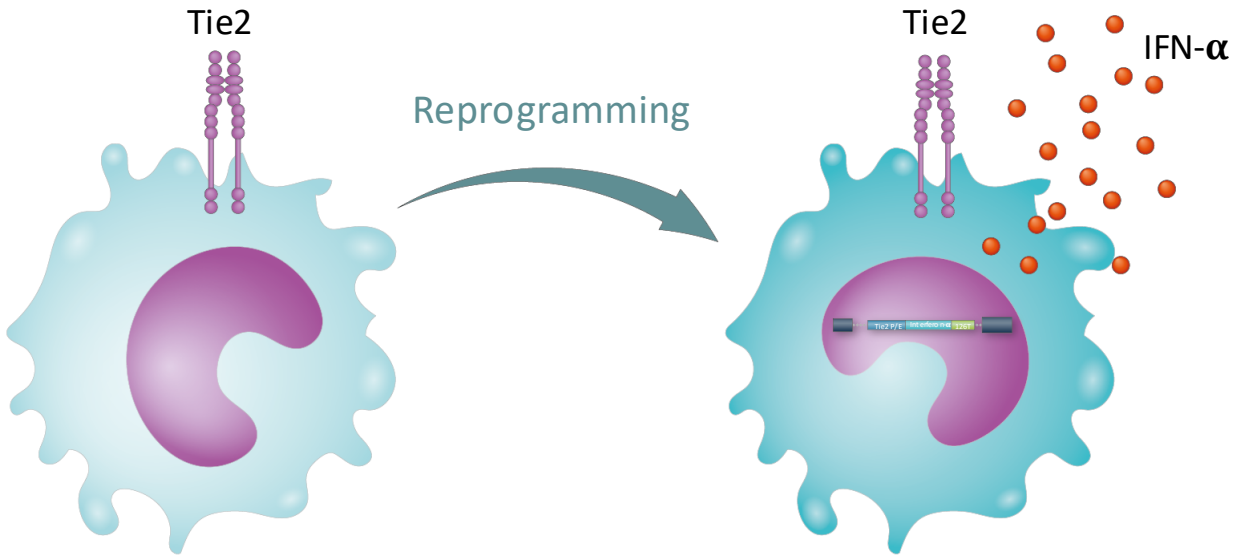

Recruited into tumors




De Palma et al. Nature Med 2003;
De Palma et al. Cancer Cell 2005 & 2008;
Pucci et al. Blood 2009
Mazzieri et. al. Cancer Cell 2011

- Pro tumoral associated macrophage subset.
- Perivascular localization.
- Angiogenic & tissue remodeling function.
- Genetic ablation curbs tumor growth.

## Use TEMs as vehicles to deliver IFN-α into the TME


Tissue remodeling macrophage (TEM)

Immune-activating macrophage («synthetic» TEM)



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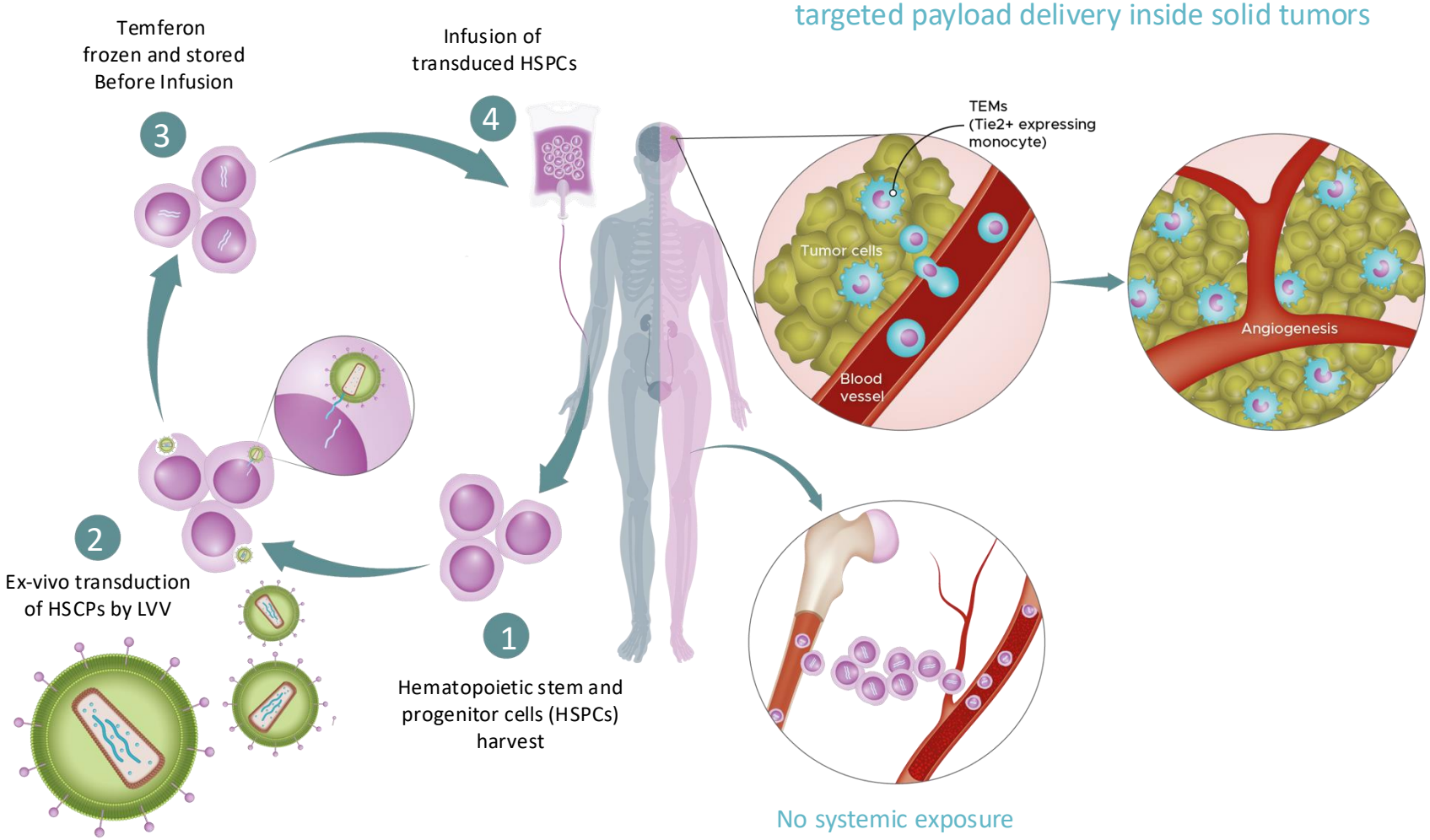
# Temferon delivers IFN-α within the Tumor Microenvironment

## Single Temferon treatment potentially renders solid tumors visible to the immune system

Tie 2 monocytes coupled with miRNA126 enable targeted payload delivery inside solid tumors




## TEMFERON AT A GLANCE

### TEMFERON

Frozen autologous hematopoietic stem & progenitor cells (CD34+) transduced ex-vivo with a third generation LVV to drive myeloid-specific IFN-α2 expression.

### FORMULATION

Cryopreserved intravenous injectable solution.

### DURABILITY OF RESPONSE

Potentially life-long.

### INDICATIONS

Solid tumors: uMGMT GBM & mRCC.

### MECHANISM OF ACTION

Direct: anti-proliferative, anti-angiogenic;
Indirect: immune system re-programming, CD8+ T cells recruitment, T cells exhaustion counteraction.



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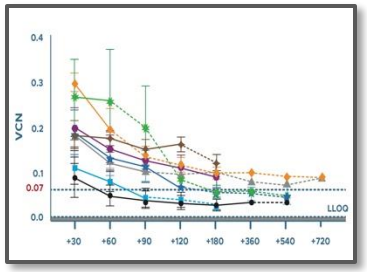
# Temferon designed to address some major challenges in Immuno-Oncology

## DEMONSTRATED DURABILITY

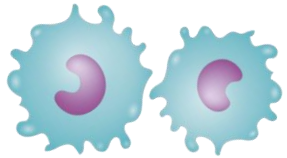
CD14



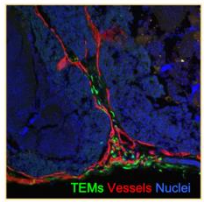

HSCs enable the maintenance of a long-term tumor protection by creating a potential life-long drug reservoir.

## SELECTIVE DELIVERY WITHIN THE TUMOR MICROENVIRONMENT

Tie2 macrophages (TEMs)



Recruited into Tumors

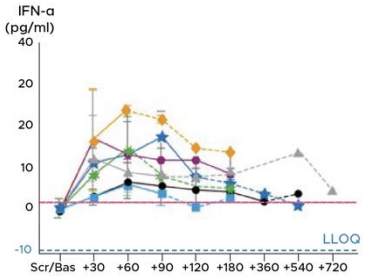



De Palma et al, Nature Med 2003;
De Palma et al, Cancer Cell 2005 & 2008
Pucci et al, Blood 2009
Mazzieri et al, Cancer Cell 2011

TEMs, being recruited by growing tumors, enable the payload delivery within the TME.

## PREVENT SYSTEMIC TOXICITY

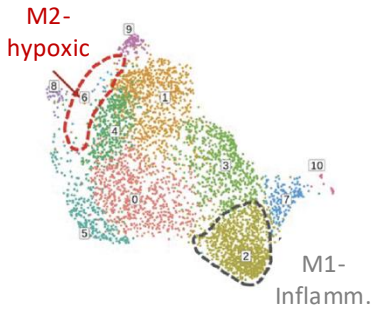
Peripheral Blood




The miRNA post-transcriptional regulation limits the systemic payload exposure.

## ACTIVATE IMMUNE SYSTEM

Genenta cell-based platform (IFN-α)




IFN-α deployment by TEMs, both in pre-clinical and clinical setting favors the myeloid proinflammatory state.

Temferon is tumor agnostic and demonstrates immune activation, which would be ideal for combination approaches.



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# Pipeline


1 Orphan Drug Designation status in U.S. and EU

[2] Combination study



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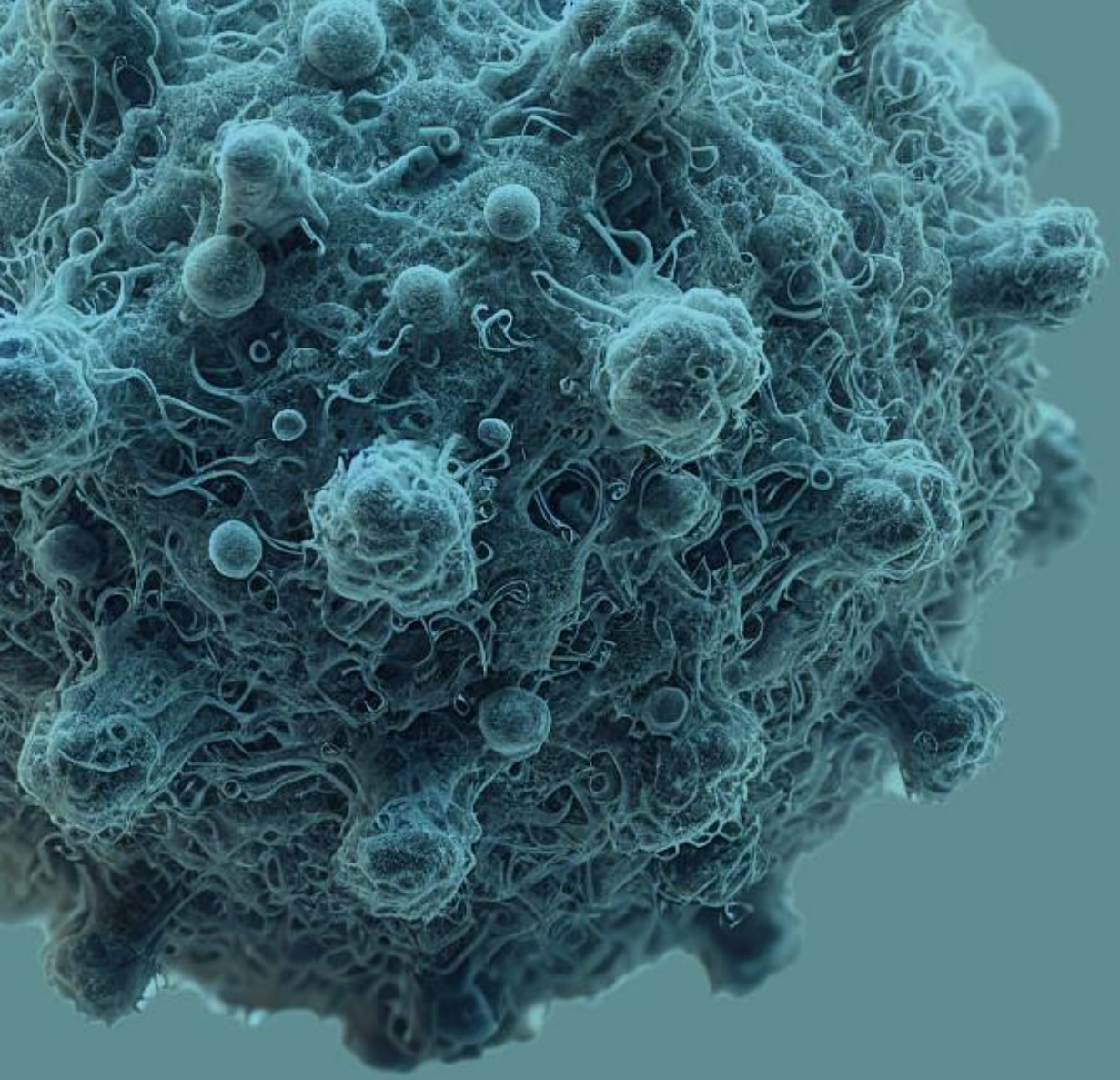

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# Harnessing the Power of Stem Cells

- Phase 1/2a uMGMT GBM Study
- Clinical Data
- Immune Activation Data

# Preliminary clinical data in uMGMT GBM:
## well tolerated and biologically active

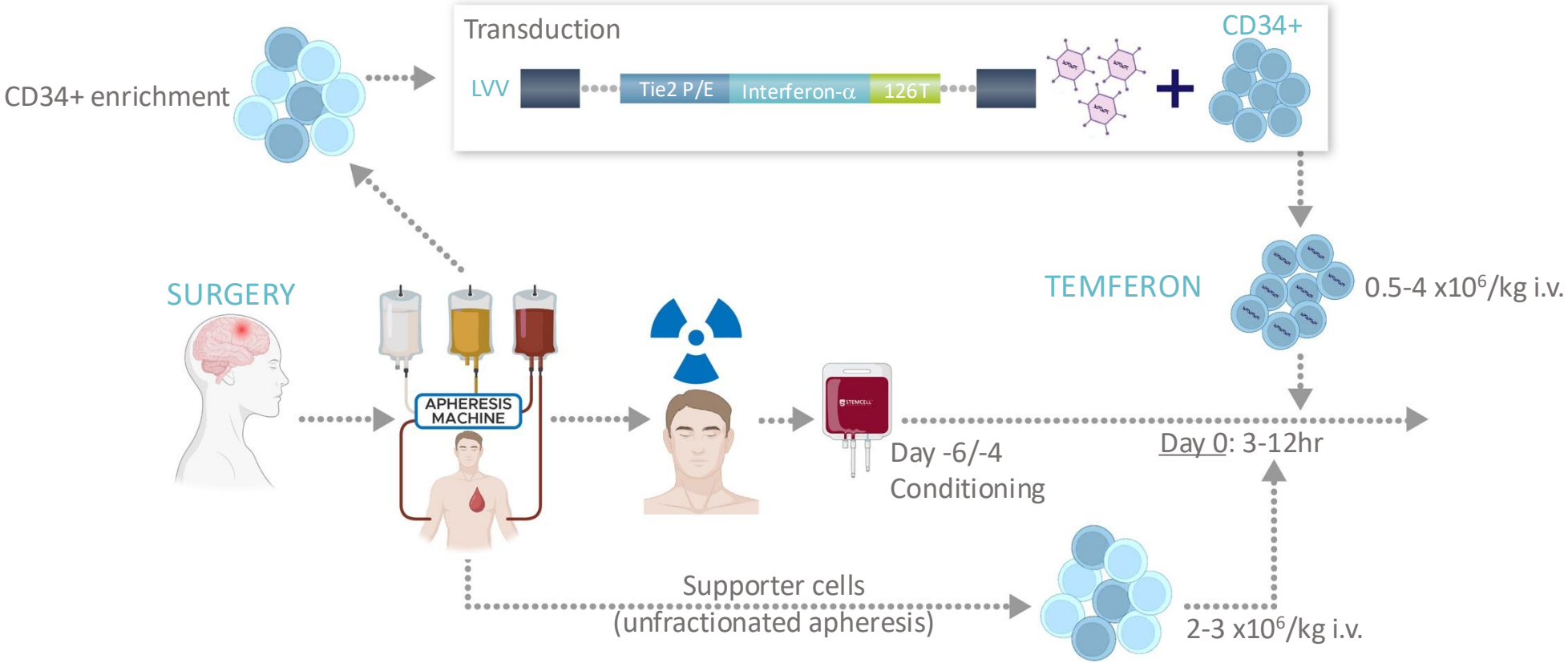
### SAFETY AND TOLERABILITY

- Detectable and as expected at very **low level of IFN-α** (pg/ml range) in the plasma;
- **Manageable** adverse events and serious adverse events generally commonly associated with autologous stem cell transplantation and glioblastoma;
- **No dose limiting toxicities** observed to date;
- Rapid engraftment and **hematological recovery** observed in all patients treated (n=24).

### BIOLOGICAL ACTIVITY

- Temferon-derived cells were **detectable** at more than 24 months **post infusion**;
- **Temferon** progeny found **inside the GBM** tumor;
- Intra tumor **IFN-α release**;
- Evidence of a pro-inflammatory state in recurrent tumors from patients that required second surgery;
- **Reprogramming** of the myeloid compartment.

# Phase 1/2a Study in Glioblastoma Multiforme, 1st line




## KEY INCLUSION CRITERIA

- Histologically confirmed, **newly diagnosed supratentorial glioblastoma** with **unmethylated MGMT** gene promoter;
- Patients have undergone **complete** or **partial tumor resection** and are eligible for adjuvant radiotherapy.



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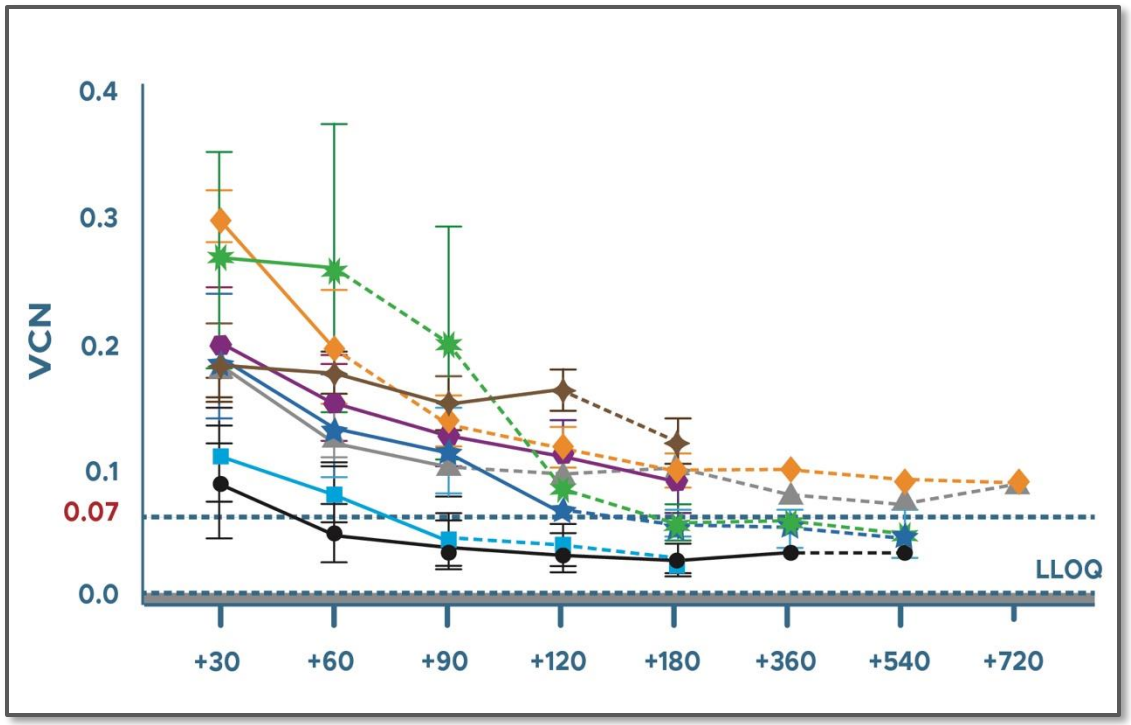
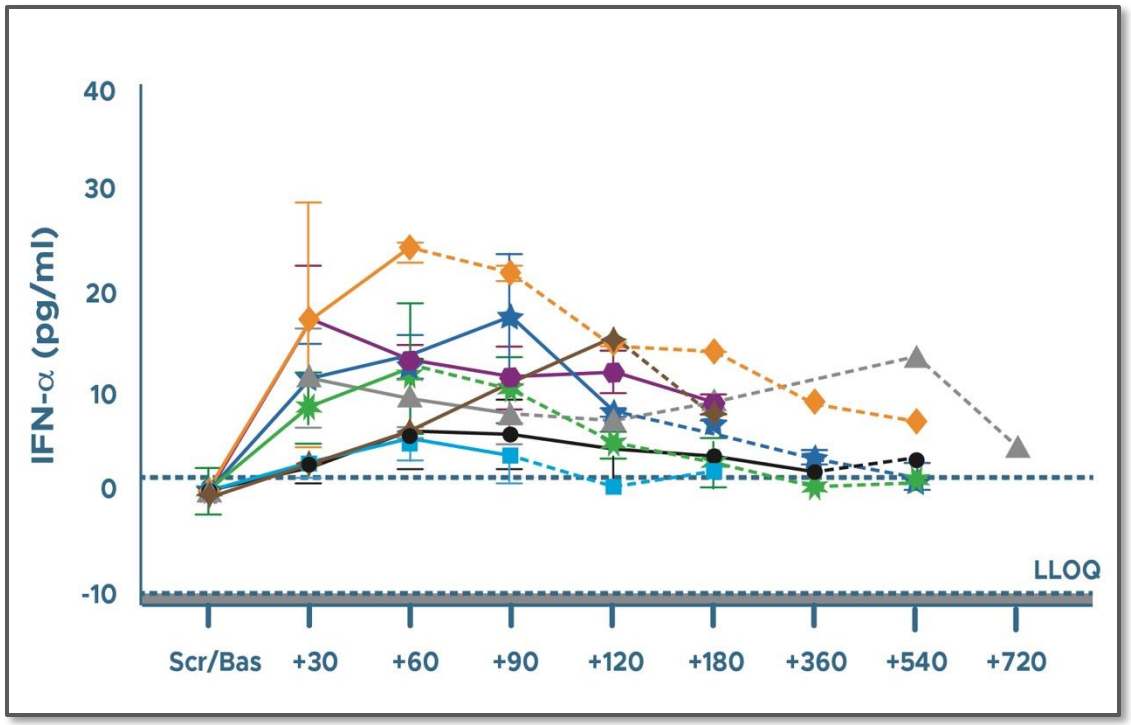
# Temferon single dose observed to be durable and well tolerated

CD14




PLASMA




Dashes line connect timepoints with less than 3 measurements

Cohort 1 Cohort 2 Cohort 3 Cohort 4 Cohort 5 Cohort 6 Cohort 7 Cohort 8

Engineered myeloid cells stabilize by day +90 and persist in blood with tightly regulated IFN-a expression (as shown by barely detectable IFN-a levels in the blood plasma in the pg/ml range).



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# 2 years Survival in TEM-GBM and in INCB Registry[1]

20 June 2024

| Number of Treated Patients | 24 |
|---|---|
| Number of Patients survival > 2 years | 6 |

| Number of deceased Patients or with FU > 1 year (considered for the % calculation) | 22 Patients (2 patients excluded[3]) |
|---|---|
| % of Patients surviving beyond 2 years[2,4] | **27%** (6 out 22) |
| % of Patients with progression free survival >8.3 months[2] | **41%** (9 out 22) |

## INCB GBM registry

159 Patients included with the following criteria

- 18-70 age
- KPS > 70
- Complete or partial tumor resection

## Clinical outcome

- 14.4% of Patients alive at 2 years[4]

1 - Istituto Neurologico Carlo Besta, Milan
2 - % of long-term survival patients calculated since 1st surgery
3 - FU < 1 year
4 – the data from TEM-GBM study includes only patients who completed radiotherapy, whereas the INCB registry data include patients who did not complete radiotherapy.



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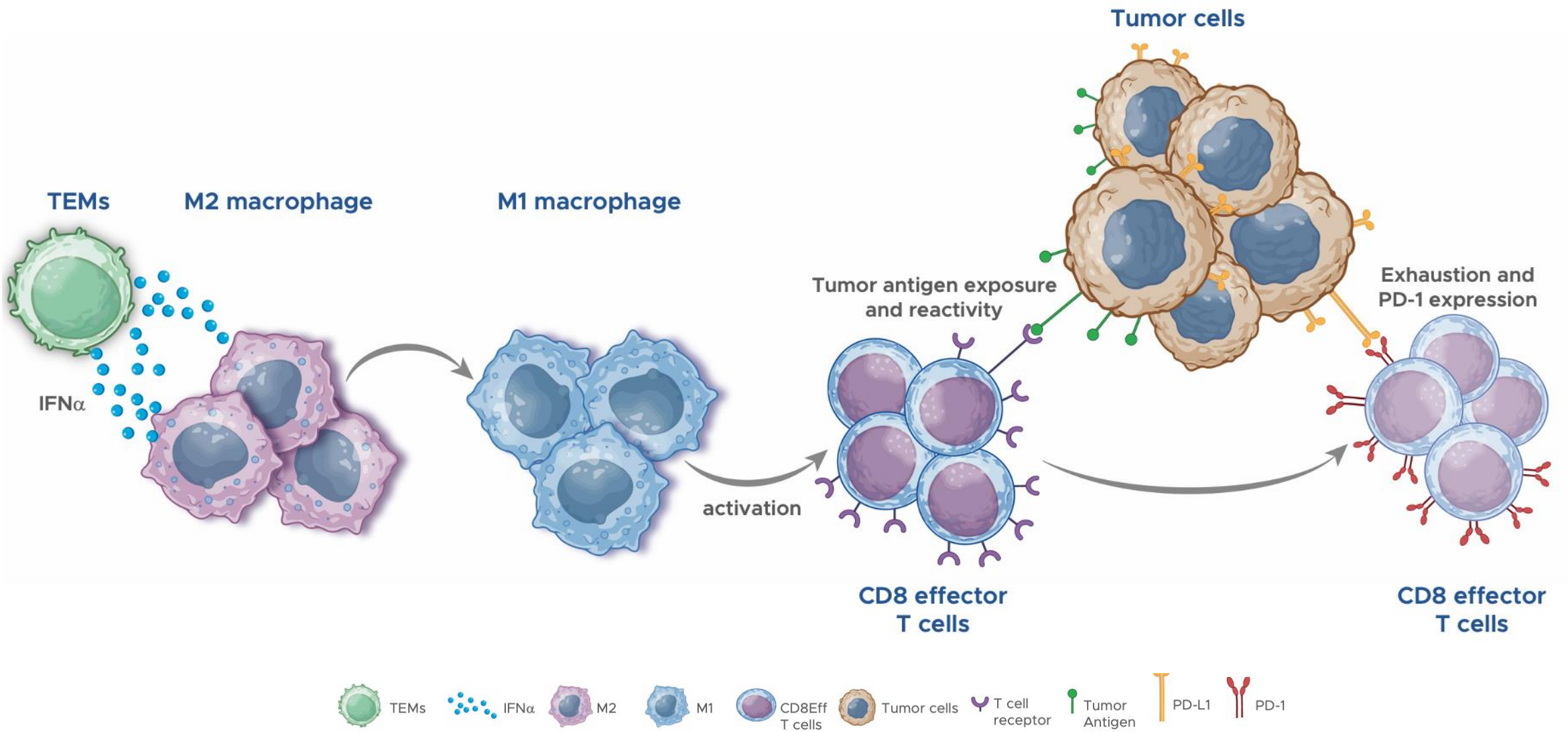
# Temferon TME reprogramming may favor PD-1 activity



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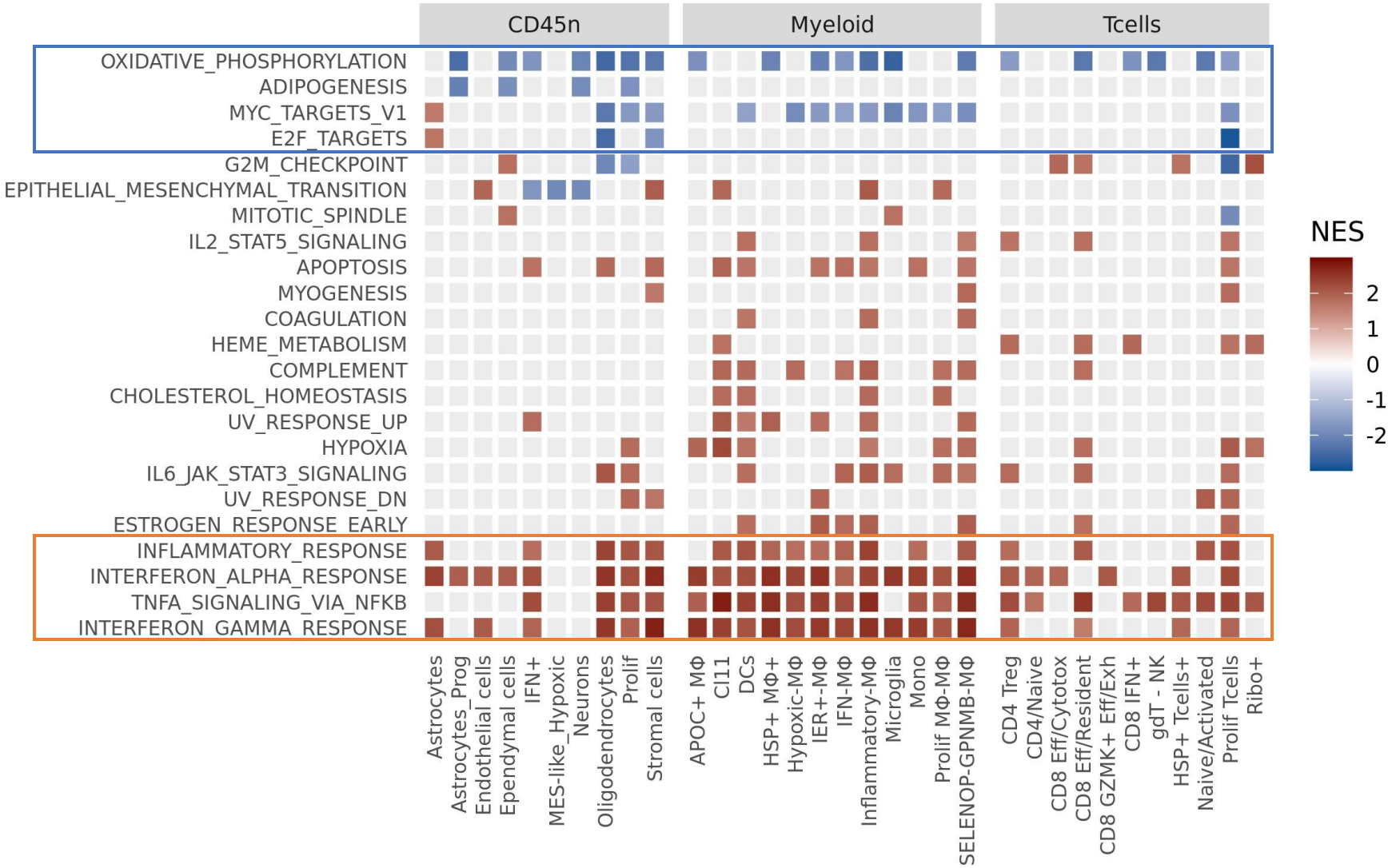
# Broad induction of Interferon responses across GBM/TME components




**Temferon and standard of care**
**Initial data suggests:**

- Widespread IFN and immune-activating response;
- Reduced proliferation, oxidative metabolism down;
- Hypoxia response up;
- Apoptosis, stress response up;
- Many expected consequences of interferon exposure.



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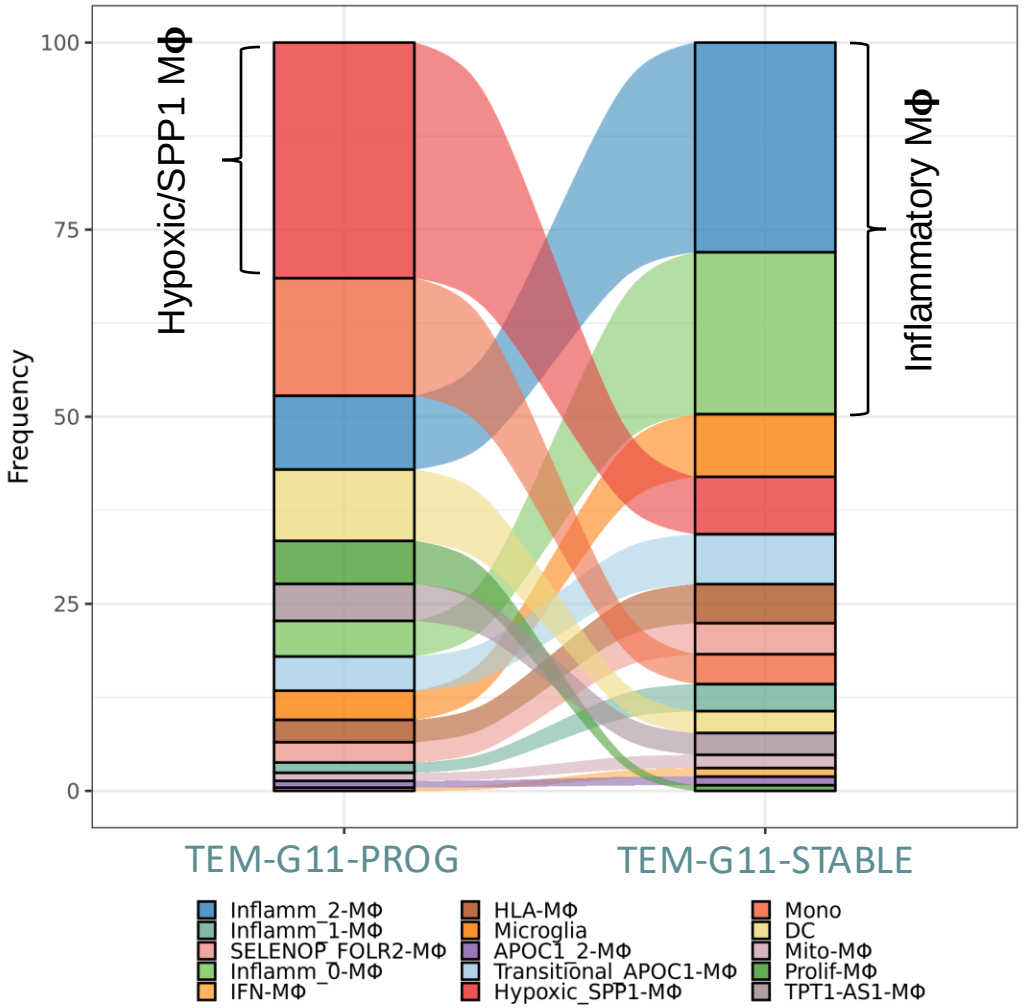
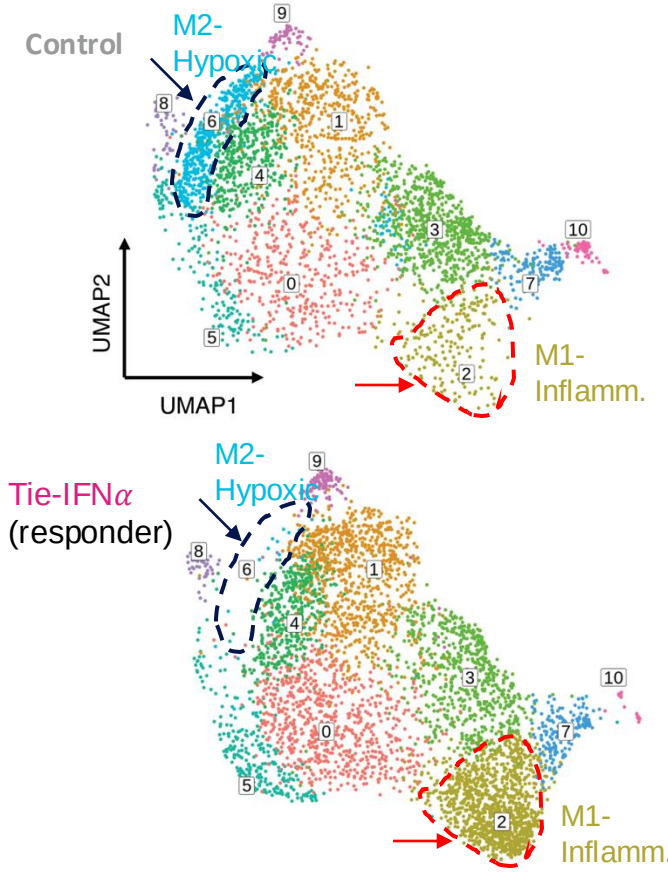
# Pre-clinical and clinical data suggest TME reprogramming induced by the pro inflammatory state created by Temferon




- Stable tumor: preponderance of inflammatory MΦ;
- Progressing tumor: hypoxic SPP1 MΦ as most frequent subtype;
- Shift in MΦ subtypes upon IFNa exposure similar to mouse model.

Myeloid GBM TME in the mouse model




Birocchi et al Sci Transl Med, *2022*



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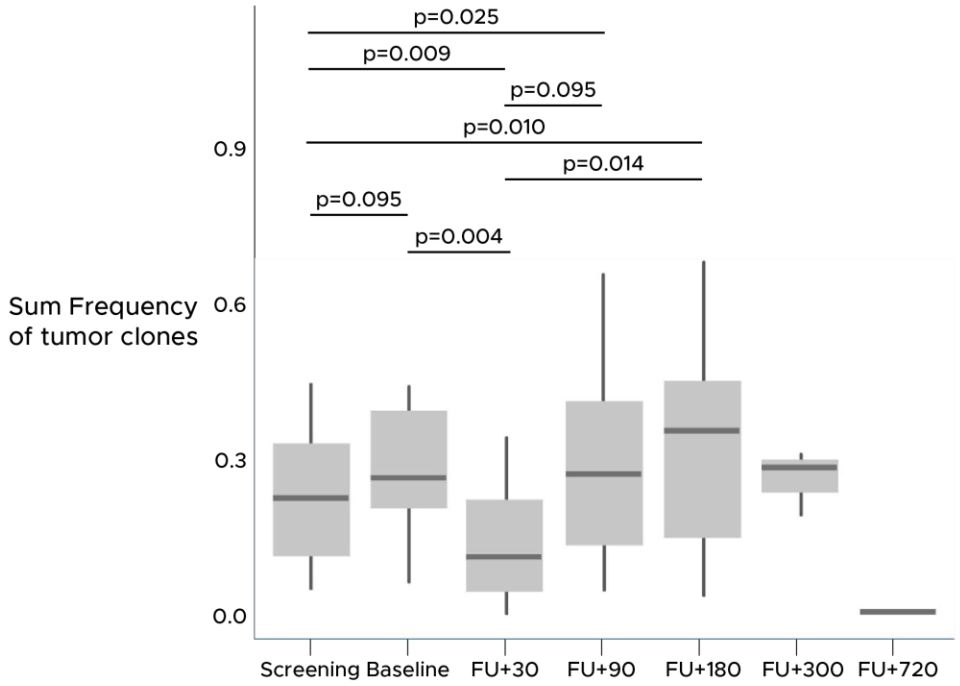
# Temferon may break tolerance allowing intra tumor infiltration of T Cell Clones

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

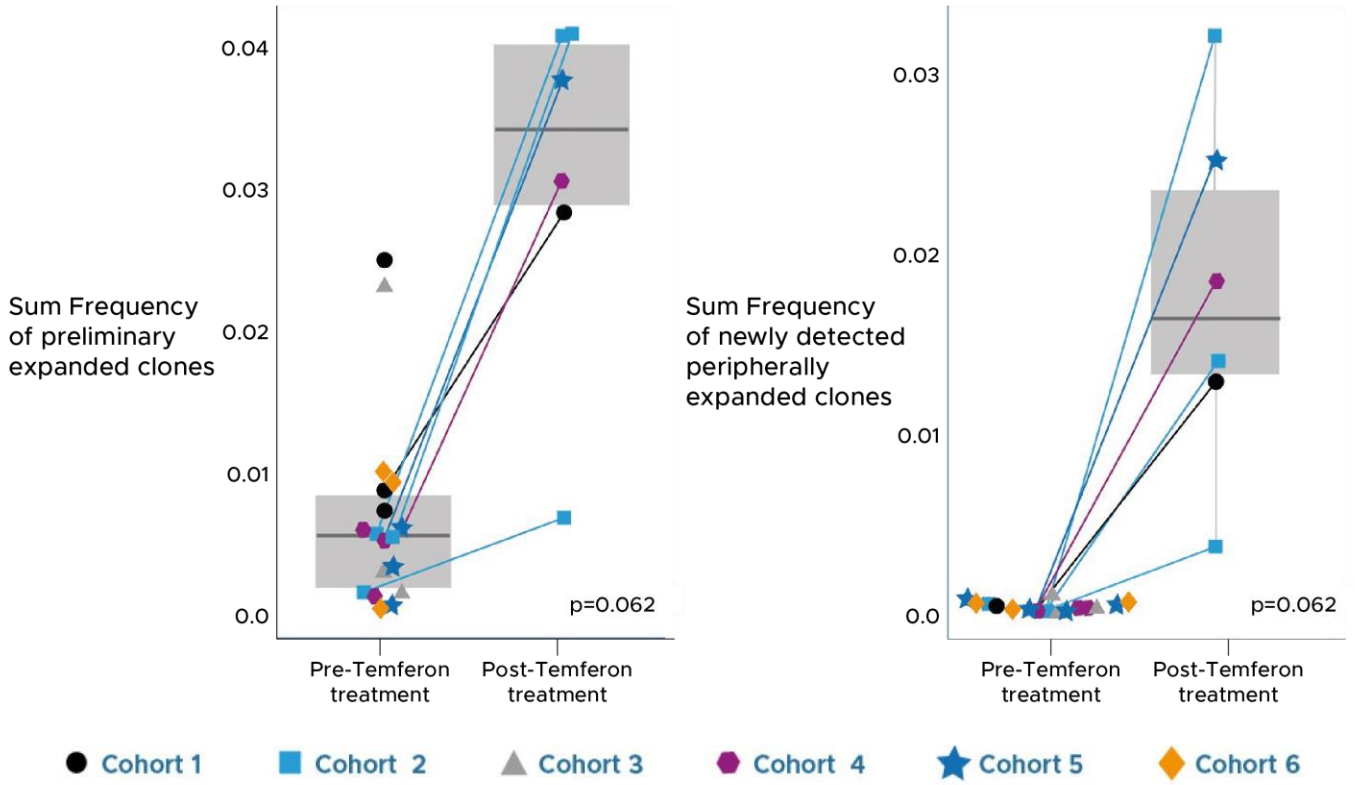
- Contraction at day +30 in the blood (ASCT conditioning);
- Rebound after 3-6 months.




## EXPANDED PERIPHERAL T CELL CLONES ARE INCREASED AT 2nd SURGERY

Pre-existing peripheral T cells clones

Newly-detected peripheral T cells clones



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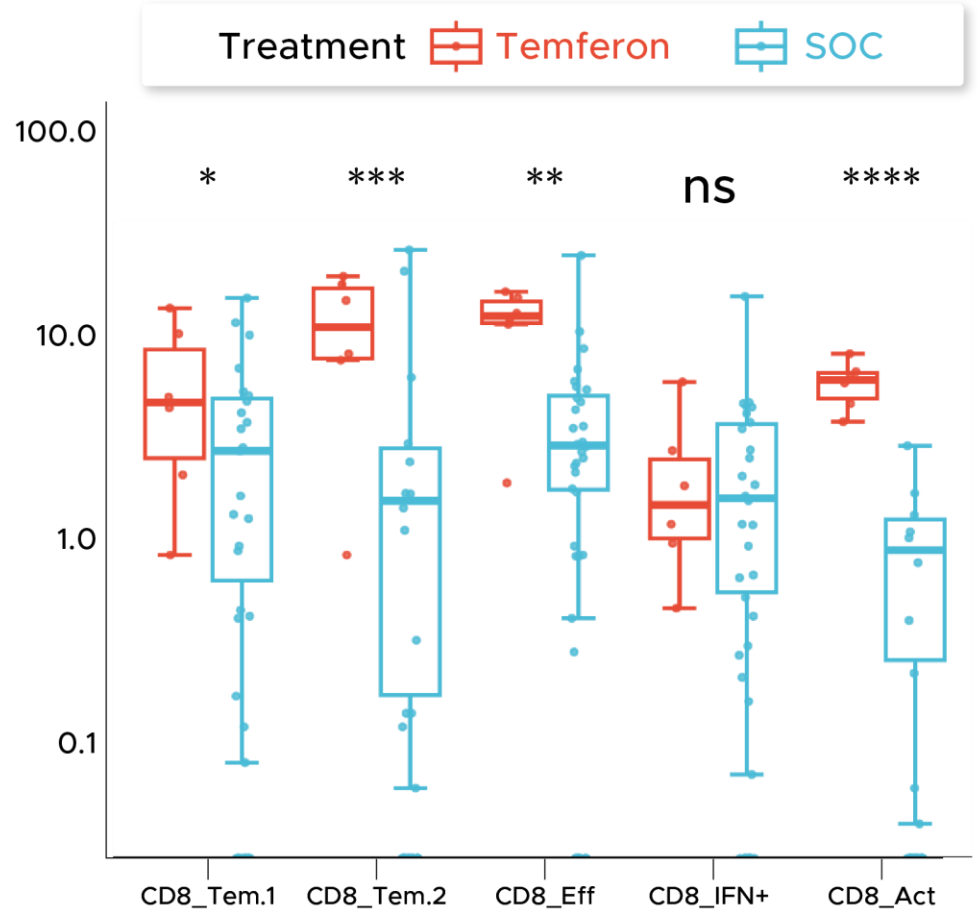
# Initial evidence of increased CD8 effector cells in the TME of Temferon treated patients

**The Temferon group suggests more activated, effector and effector memory CD8 T cells**

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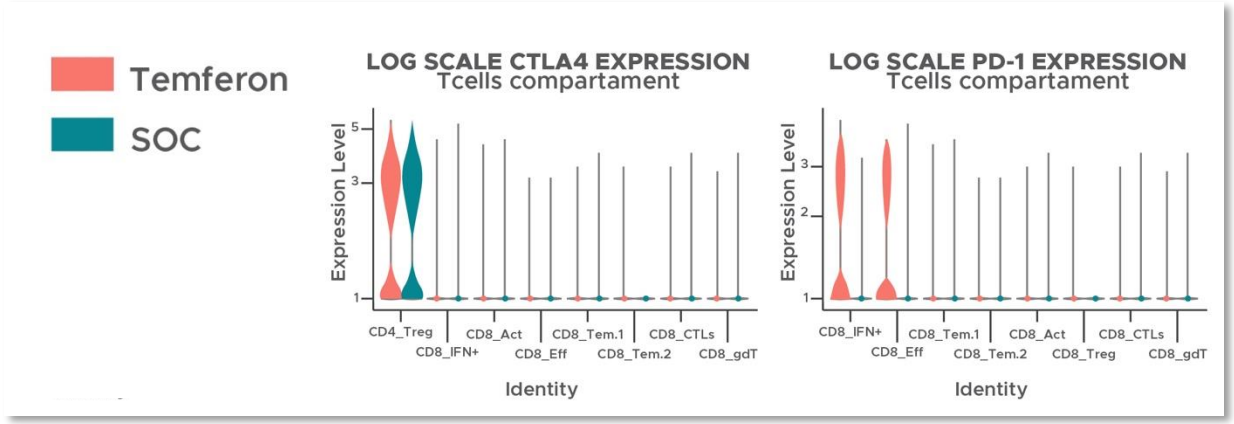
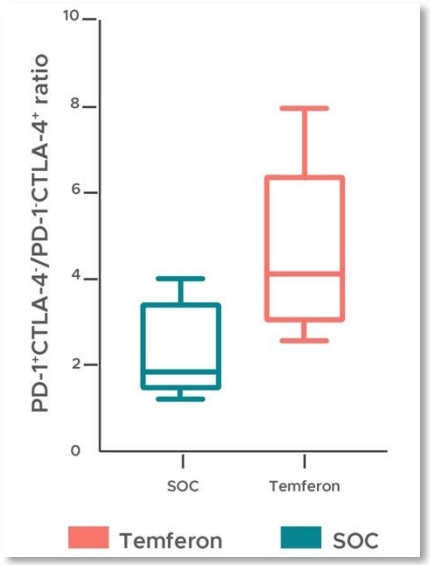
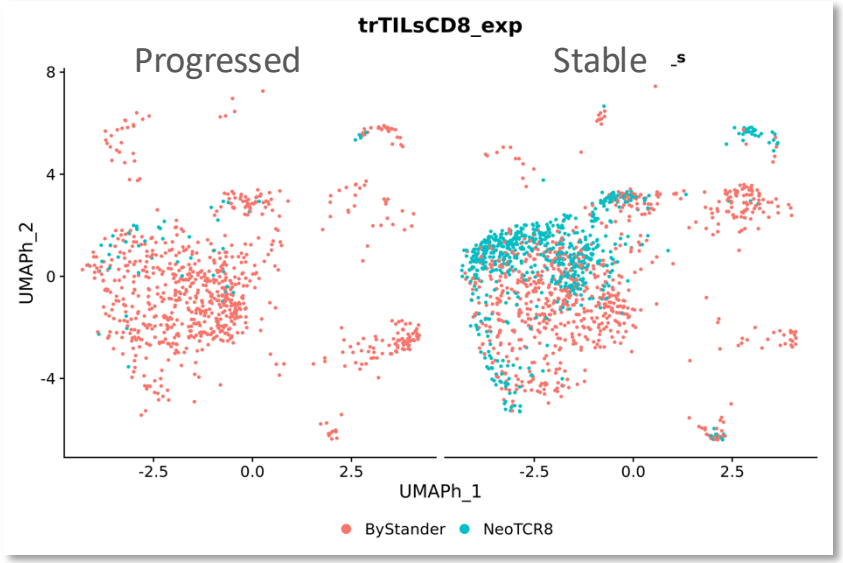
# High expression of PD-1 in CD8+ effector cells of Temferon treated patients




- Higher expression of PD-1 in CD8-Effector and CD8-IFNα populations, in Temferon;
- Temferon treated patients exert a 2-fold enriched PD1+ vs CTLA4+ median ratio;
- Strong enrichment of PD1+ CD8 TILs tumor reactive T-cells, not shared with peripheral blood clones, in stable lesion compared to progressed one, mostly characterized by putatively by-standers and shared with PB CD8-CTLs.



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# Temferon Cell Therapy for Cancer

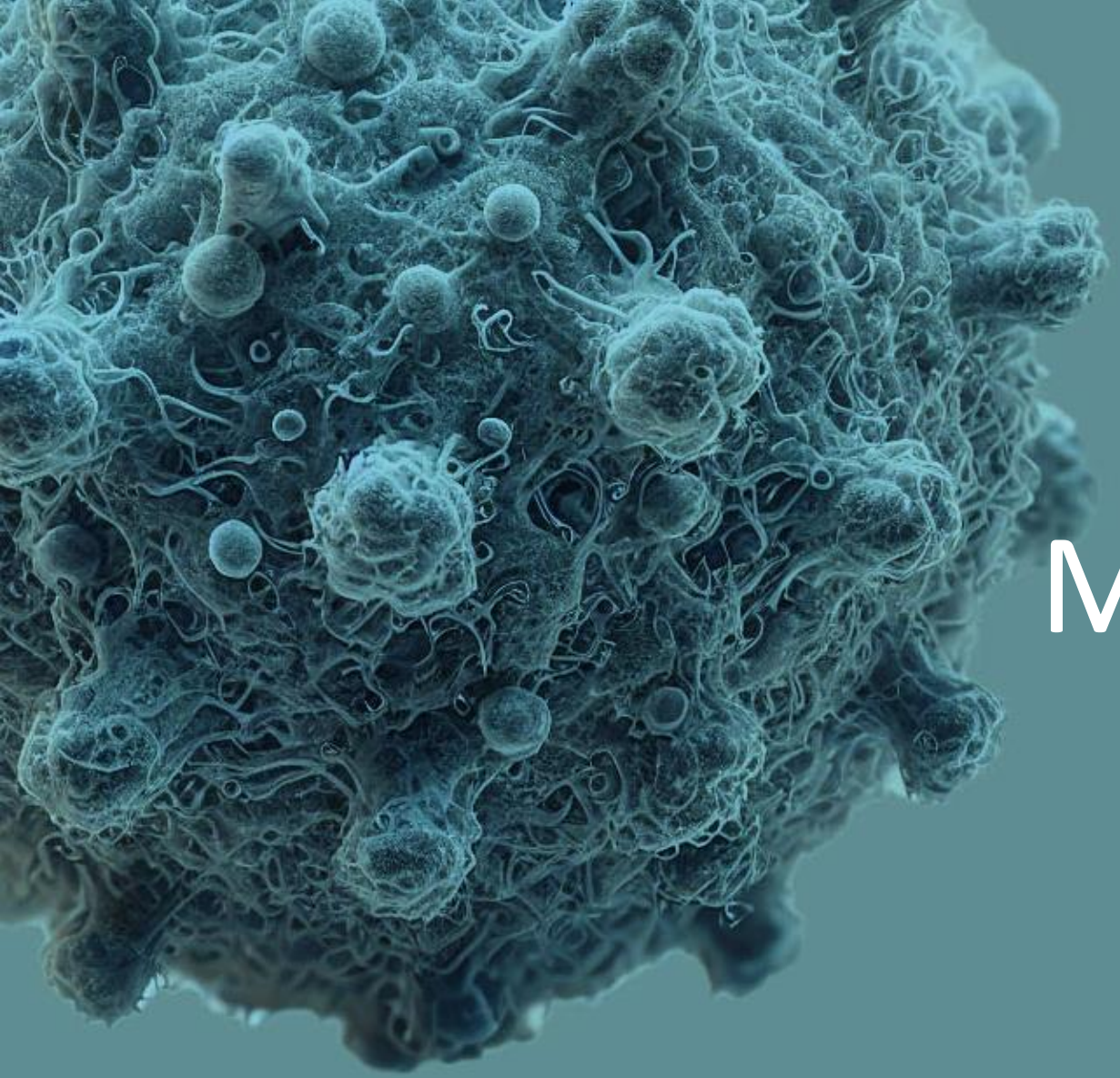
## Feasibility, safety and tolerability

- **Successful engraftment** of Temferon, consistently and durably above pre-defined threshold (VCN >0.07) in DL3 patients;
- Temferon well tolerated, no dose limiting toxicities. Expected **very low systemic exposure to IFNα;**
- **Rapid hematologic recovery** observed in all patients;
- Adverse events compatible with autologous stem cell transplantation and glioblastoma.

## Immune activation

- **Temferon** progeny found **inside the GBM tumor** at second surgery (n=6 out of 7 patients);
- **Local IFNα release inside the tumor**;
- Genetic reprogramming of tumor-associated myeloid cells to awaken anti-cancer immunity;
- Temferon **TME reprogramming** may favor PD-1 activity.

## Immune activation data supports immuno-oncology combination approaches



# Metastatic Renal Cell Carcinoma

- Phase 1/2a Program

# Temferon 2nd solid tumor indication mRCC

## RATIONALE

Strong historical evidence of IFNα and I/O responsiveness supporting potential Temferon efficacy.

Targeted IFNα delivery as an innovative and clinically relevant approach.

- Until 2005, systemic administration of IFNα and IL-2 were standard of care treatment for mRCC;
- IFNα 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 IFNα use limited in the past by the systemic toxicities.

FERRING PHARMACEUTICALS

ADSTILADRIN (nadofaragene firadenovec-vncg)

BCG-unresponsive non-muscle invasive bladder;

- CR in 51% of patients with *in situ* carcinoma by three months[2] .

## SOLUTION

Temferon has been designed to attenuate systemic toxicity associated with IFNα and to achieve reprogramming of the TME.

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

[1] Coppin et al. 2004; [2] Adstiladrin USPI 2023



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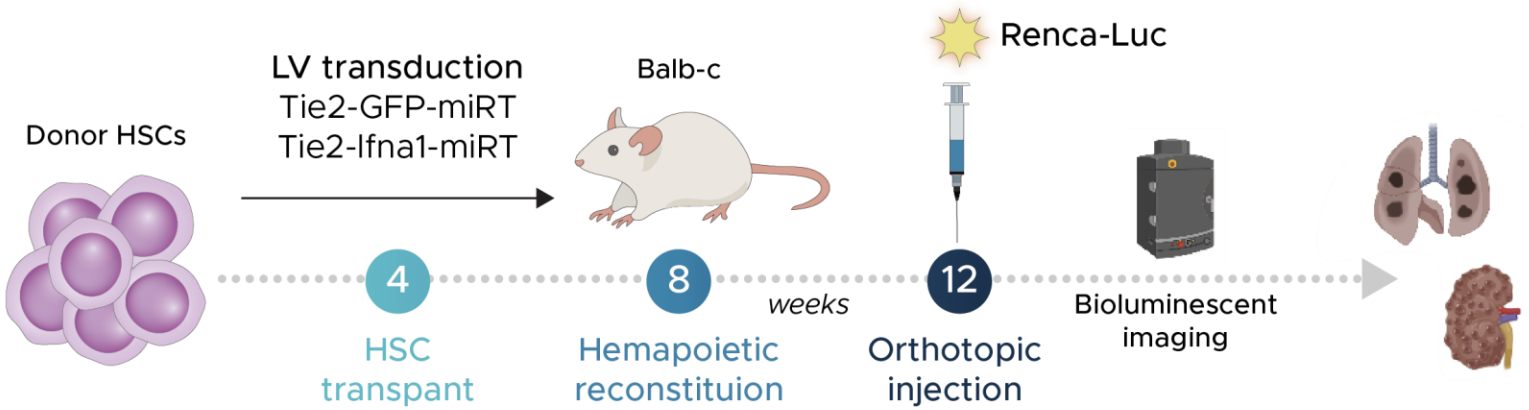
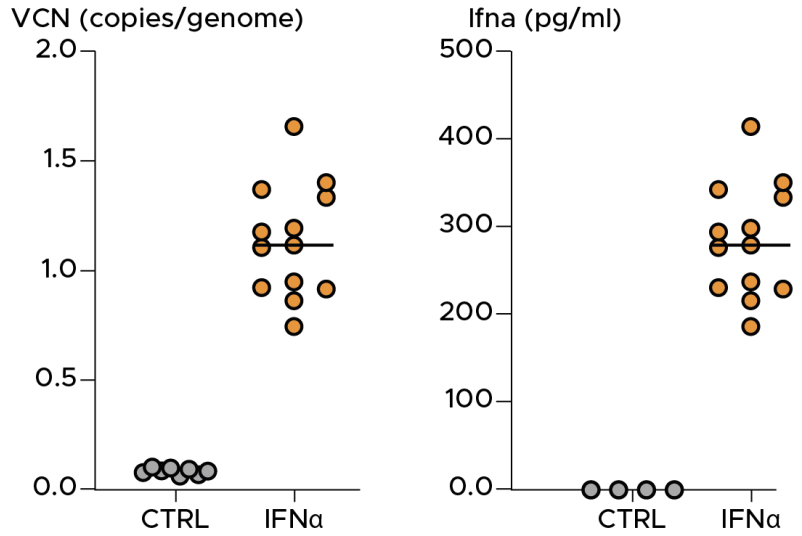
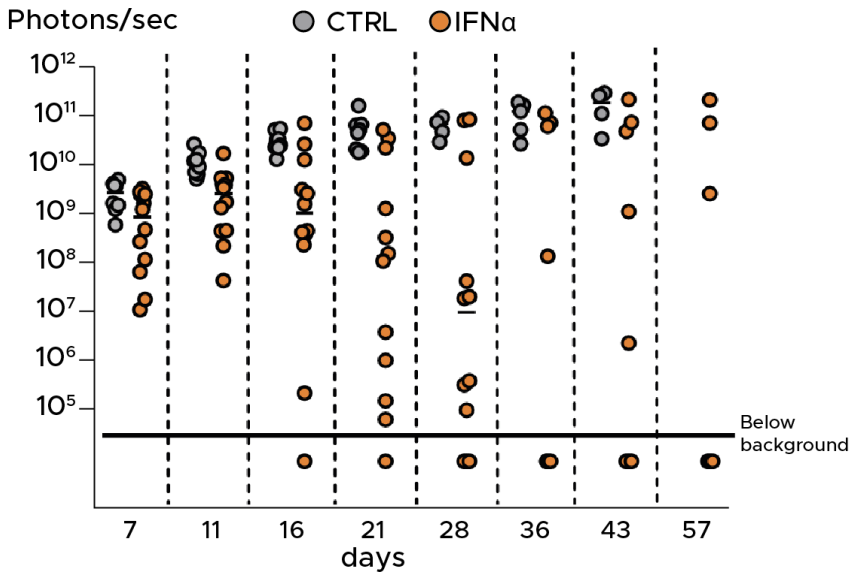
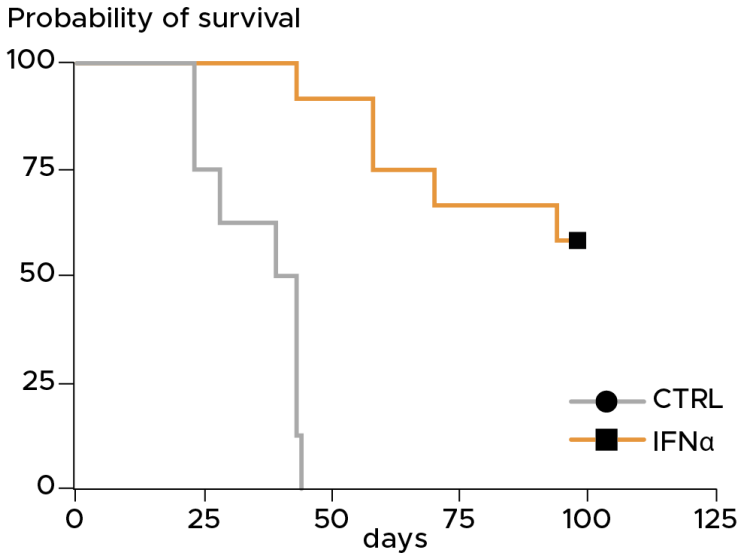
# Testing IFNα GT in mRCC mouse model (ongoing work)




At FU+57 92% (11/12) and at FU 125 58% (7/12) of IFNα GT treated mice are alive.



Preliminary Data - experiment currently ongoing

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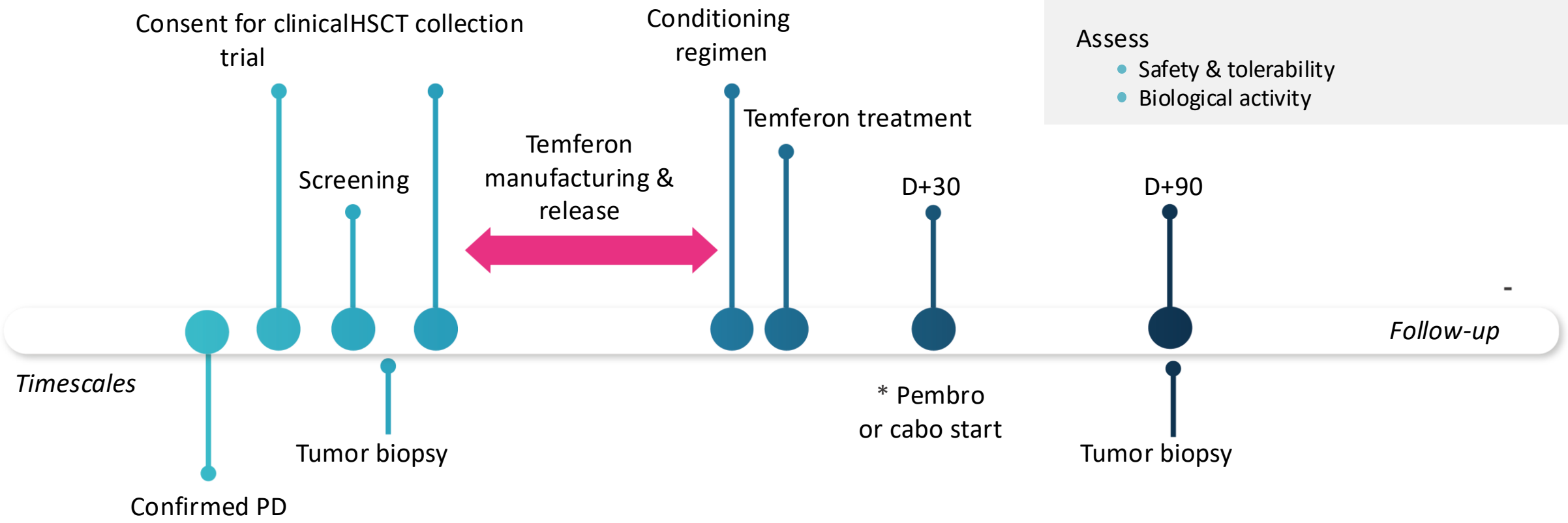
# TEM-GU Study Design

**Study design**

- Open label, single-centre phase 1/2a study
- Temferon dosing 4x106 cells/kg, 12 Patients
- Patients 18-70 years old

Assess

- Safety & tolerability
- Biological activity




*Patients will start to receive pembrolizumab providing they have not received ICI in the six months prior to entry into the study. Patients allocated to pembrolizumab will receive pembrolizumab 400mg IV every six weeks commencing at D+30.



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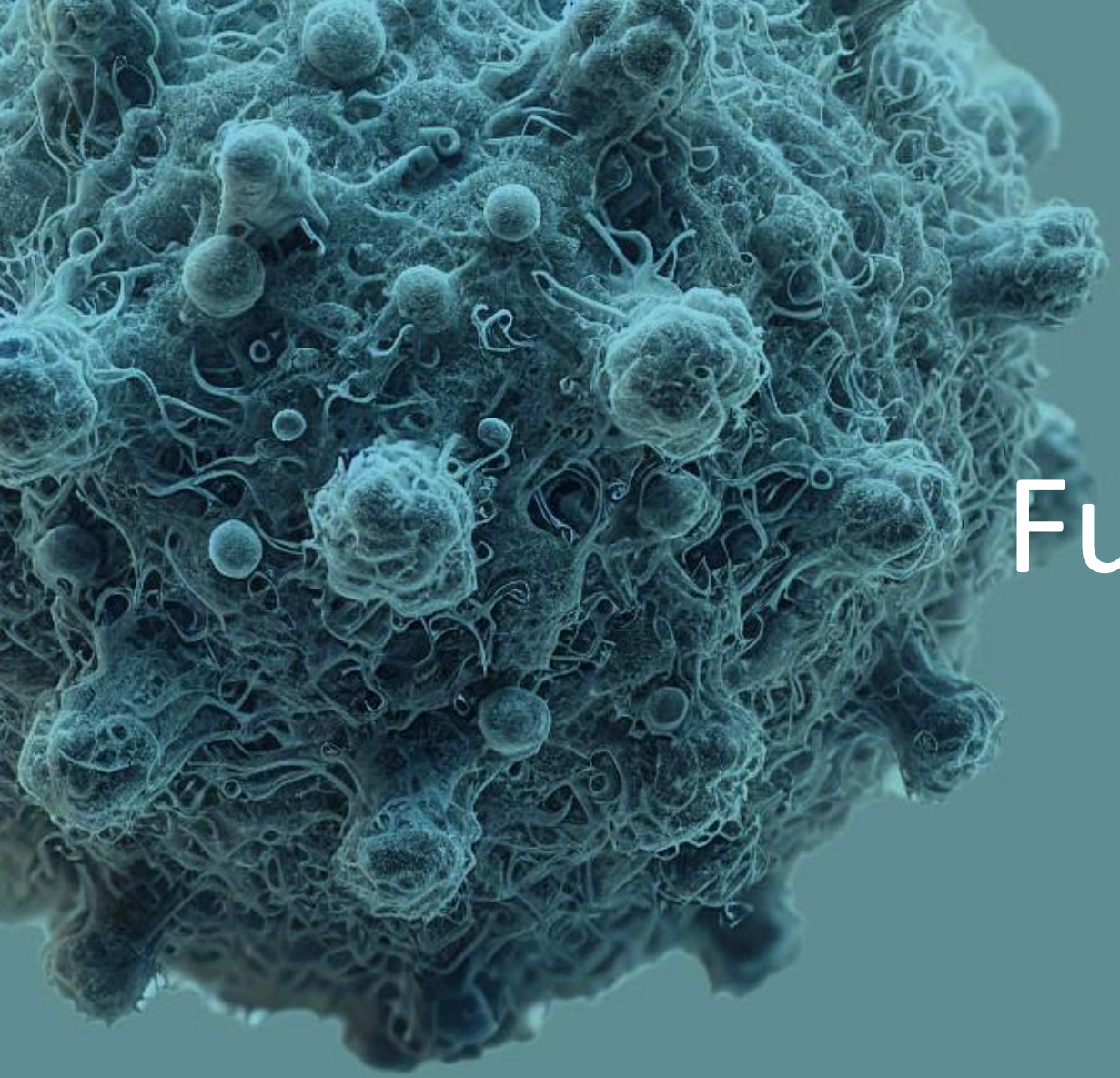


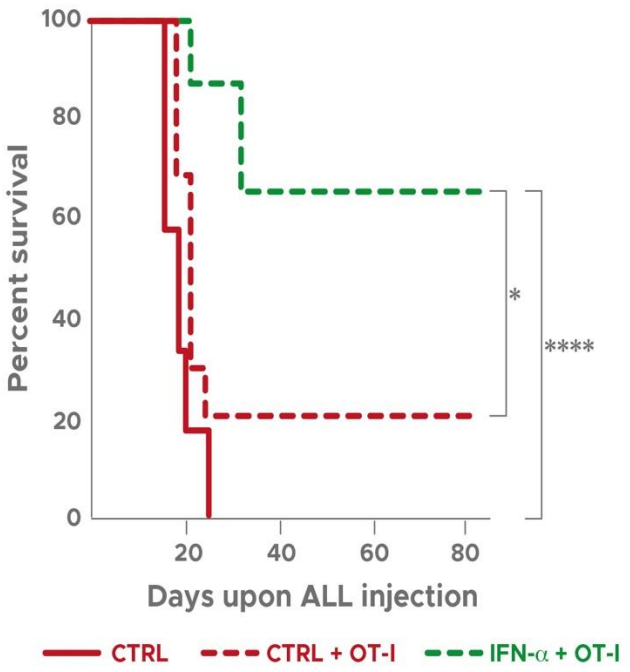
# Future Development & Summary

# Combination of Temferon with immuno oncology treatments may increase overall survival and tumor control

- Our research partner SR-TiGET has explored in preclinical models Temferon with CAR-T demonstrating:
  - **Improved survival** versus CAR-T alone;
  - Enhanced **durability of CAR-T** response ( i.e. Temferon reduces T cell exhaustion).
- Ongoing pre-clinical work, evaluation of CAR-T therapies administered in combination with Temferon in solid tumors.

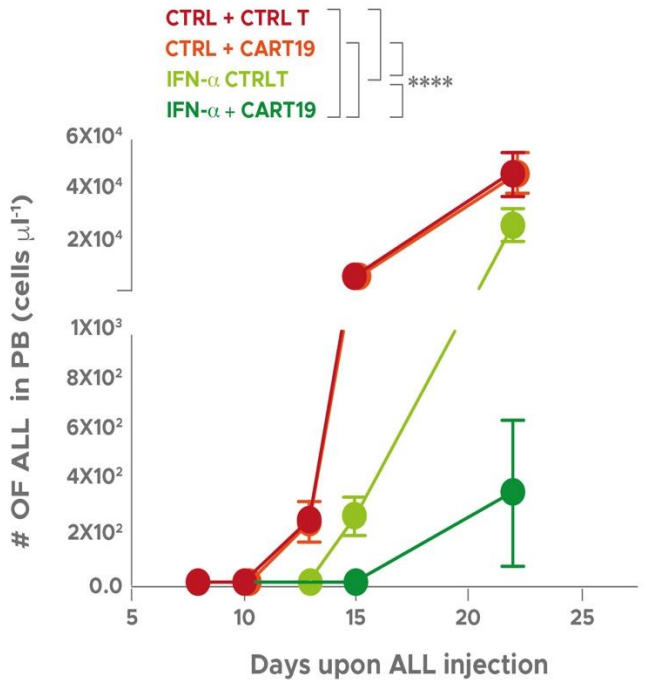
Temferon may enhance other I/O therapies

ADOPTIVE T CELLS TRANSFER




OT-I = Ovalbumin transgenic mice

CAR-T




Graphs have been faithfully reproduced by the original articles

Escobar et al., Nature Communication 2018



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# Anticipated Pipeline Development Milestones

## H1 2025

- TEM-GBM Phase 1 interim report completion.
- TEM-GU Phase 1 six patients expected to be treated at the end of the first half 2025 (+90 TME data and +30 TME data).

## H2 2025

- TEM-GBM expansion cohort dosing completion.
- TEM-GU 12 patients expected to be dosed by the end of 2025. Interim clinical data and immune activation data

Up to 27 Drug Products expected to be manufactured in 2025



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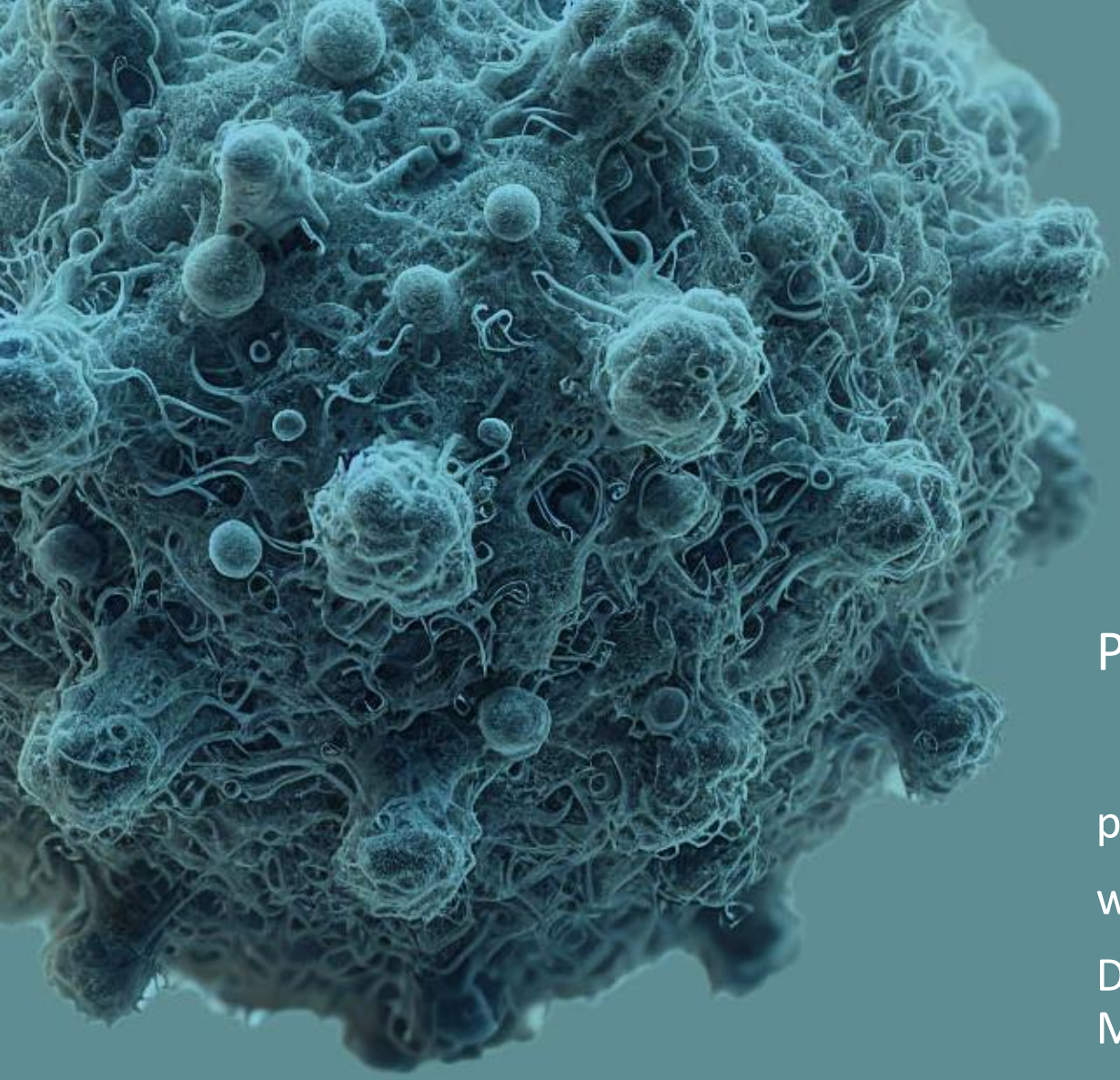
# Summary: Temferon Harnessing the Power of Stem Cells

- In 2023, the U.S. Food and Drug Administration (FDA) and the European Commission granted **Orphan Drug Designation to Temferon** for the treatment of glioblastoma multiforme.
- Temferon harnesses the power of stem cells while incorporating miRNA and a well characterized cytokine.
- **uMGMT GBM Phase 1 study enrollment completed:**
  - Demonstrated durability, targeted expression and no observed dose limiting toxicity;
  - Evidence of reprogramming of the tumor microenvironment to awaken anti-cancer immunity;
  - Immune activation data supports combination regimens with other immuno-oncology targets, potential to reduce T Cell exhaustion.
- **mRCC Phase 1/2a study; patient enrollment in Q4 2024. First patient dosed in Q1 2025:**
  - Reprogramming may favor PD-1 activity, strong rationale for immune checkpoint inhibitor combination.
- **Opportunities to expand Temferon pipeline programs:**
  - Agnostic efficacy designed to be suitable for a large number of solid tumors with high unmet need;
  - Ongoing preclinical work on immuno-oncology combinations including CAR-T across a broad range of solid tumors.



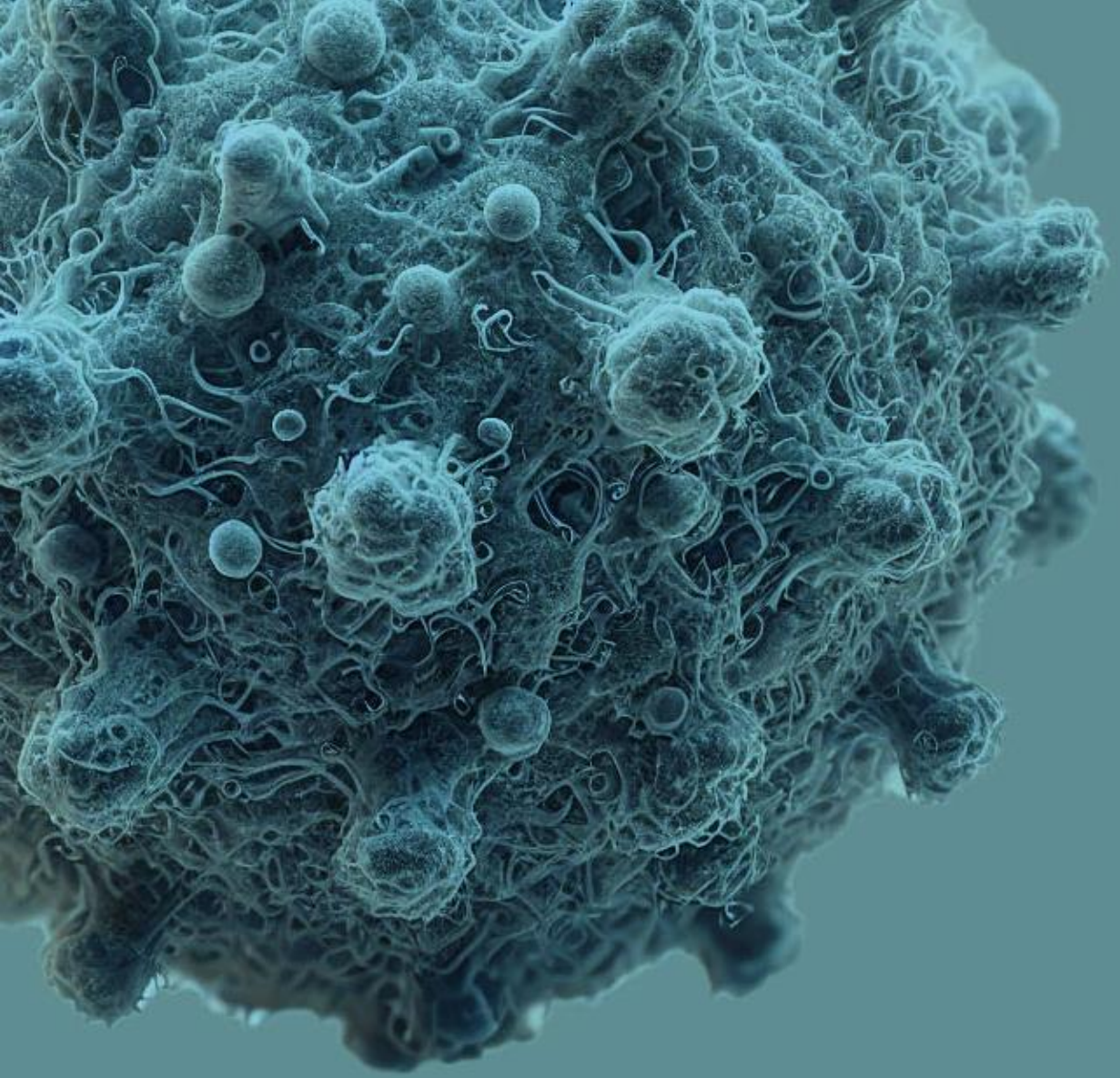
Pierluigi Paracchi, CEO

pierluigi.paracchi@genenta.com

www.genenta.com

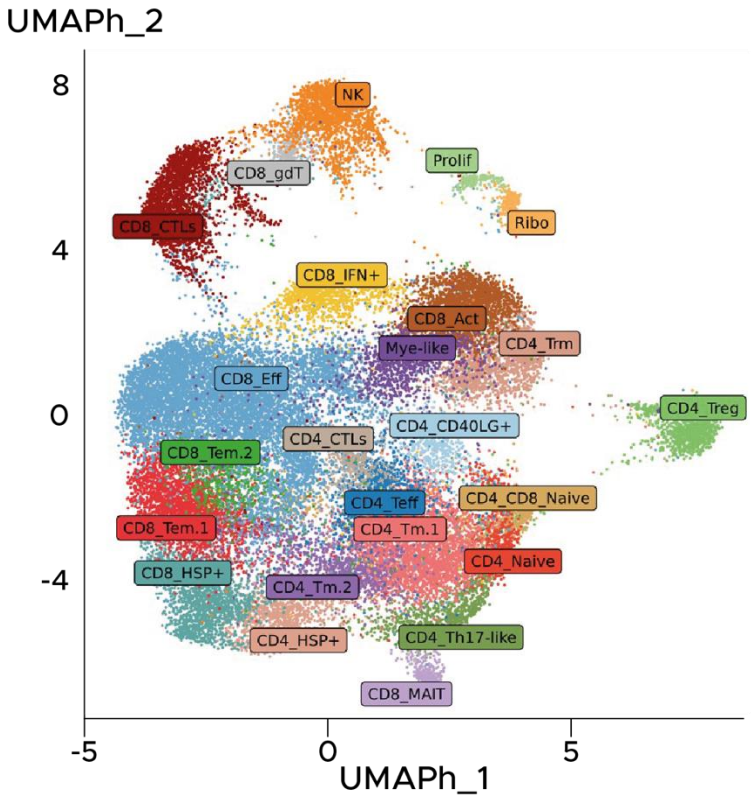
Dibit 1 - San Raffaele Hospital, via Olgettina 58 - 20132 Milano, Italy

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

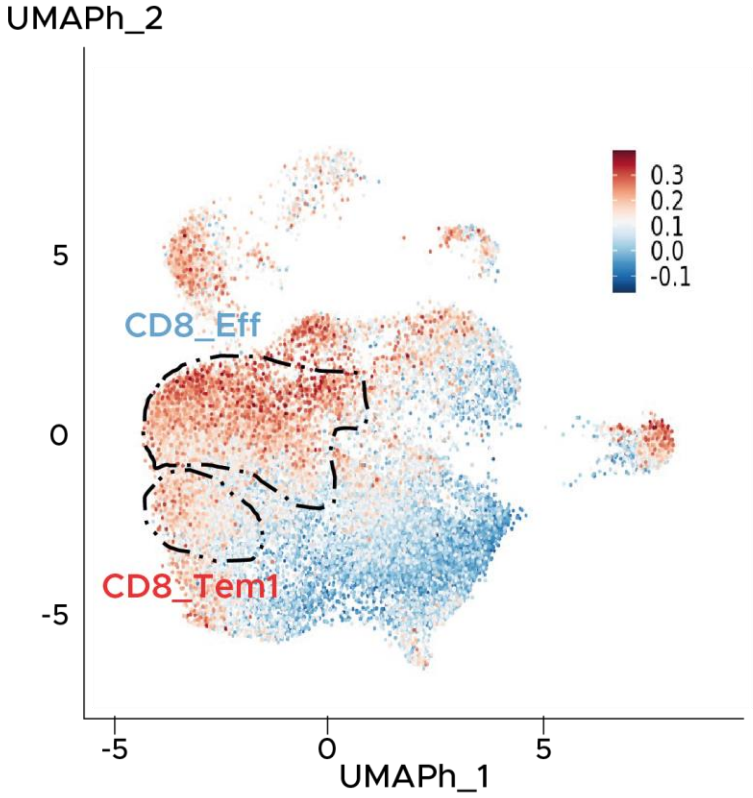


# Appendix

# CD8 subsets overrepresented in Temferon are putative tumor reactive clones with the NeoTCR signature



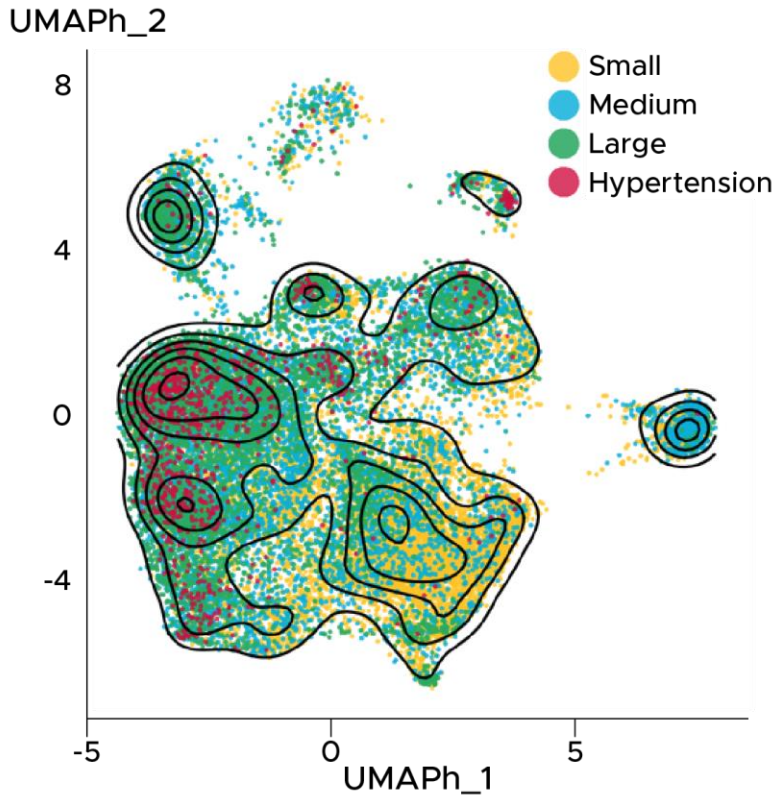

NeoTCR8 signature
(predicted tumor reactivity)




CD8 Eff = Effector

CD8_Tem1 = Effector Memory

Clonotype frequency
distribution




Clonotype abundance increases with T cell differentiation towards a CD8 effector state



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# Temferon agnostic efficacy designed to be suitable for treatment of large number of solid tumors[1]

| 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.



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# Intellectual Property

| Patent | Expiration |
| --- | --- |
| miRNA Regulated Vectors * | 4/30/2026 |
| Gene vector compromising miRNA | 5/26/2030 |
| Type 1 IFN Gene Therapy | 4/20/2038 |
| Combination Immunotherapy of Solid Tumors (provisional)** | TBD |
| Combination of Immunotherapy of Renal Cell Carcinoma (provisional)** | TBD |

* Later expiration for certain U.S. patents pursuant to patent term adjustment (35 U.S.C. §154(b))
** Provisional patents filed in April and September 2024 respectively

# Serious adverse events

| COHORT | CONDITIONING REGIMEN | NUMBER OF PATIENTS | NUMBER OF SAES | START DATE ≤90 DAYS AFTER TEMFERON | | START DATE >90 DAYS AFTER TEMFERON | |
|---|---|---|---|---|---|---|---|
| | | | | CTCAE ≤3 | CTCAE >3 | CTCAE ≤3 | CTCAE >3 |
| 1-4, 6 | BCNU + thiotepa | 15 | 22 | Pneumonia (x3), C.Diff infection, CMV infection, Seizure (x2), PE, ECOG deterioration, Anaemia, GGT increase[ǂ] | Graft failure, Febrile neutropenia (x2), Pneumonia*, Septic shock*, Respiratory failure* | Status epilepticus, Cerebral abscess, Seizure, Hemiparesis | Sudden death* |
| 5 | Busulfan + thiotepa | 3 | 6 | | Status epilepticus | | Hypoglycaemia, Thrombocytopenia, Myocardial fibrosis*, Cardiac thrombosis*, Pneumonia* |
| 7, 8 | Busulfan only | 6 | 3 | | | Asthenia (D+333) | Pneumonia (D+115) Pulmonary embolism (D+115) |

[ǂ] Reported as SUSAR

*Grade 5 SAEs (NB multiple SAEs listed as contributing to a single death)

4 patients died as a result of SAEs (1 patient each from Cohort 3 and 6, ≤60 days post Temferon; 1 patient Cohort 5 at D+122; 1 patient Cohort 1 at D+402)



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# 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 **Axòn 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, obtained his Ph.D. at Massachusetts Institute of Technology and received his 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 Biotetherapeutics**, 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 received his AB, with high honors and Phi Beta Kappa, from Princeton University and his M.D. from Columbia University Vagelos College of Physicians and Surgeons.
- 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.



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# Scientific Advisory Board

**LUIGI NALDINI**
Professor, M.D., Ph.D.,

Co-founder Genenta. Naldini is **Professor of Cell and Tissue Biology and of Gene and Cell Therapy at the San Raffaele University School of Medicine and Scientific Director of the San Raffaele Telethon Institute for Gene Therapy** (Milan, Italy). He has pioneered the development and the applications of lentiviral vectors for gene therapy and he has continued to investigate new strategies to overcome the major hurdles to safe and effective gene transfer, bringing about innovative solutions that are not only being translated into new therapeutic strategies for genetic disease and cancer, but have also allowed novel insights into hematopoietic stem cell function, induction of immunological tolerance, and tumor angiogenesis.

**BERNHARD GENTNER**
Professor, M.D., Ph.D.,

Co-founder Genenta. 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 the 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 MonitoringCenter**. 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.

**PATRICK Y. WEN**
Professor, M.D.

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



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# Financial Profile

| | |
|---|---|
| Cash & cash equivalents and marketable securities[1] | € 16.9 MM |
| Expected cash runway | Q4 of 2025 |
| Debt and warrants[2] | 0 |
| Number of shares outstanding[3] | € 18.3 MM |
| Average volume[3] | ~ 14K shares |

1 - As of June 30, 2024
2 - Except normal payables, accruals and underwriters' warrants
3 - As of October 29, 2024

# Stock Ownership Info

| | |
|---|---|
| Founders and Leadership | 28% |
| San Raffaele Hospital[4] | 10% |
| Institutions/Large FOs/Sovereign Funds | 19% |

4 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



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