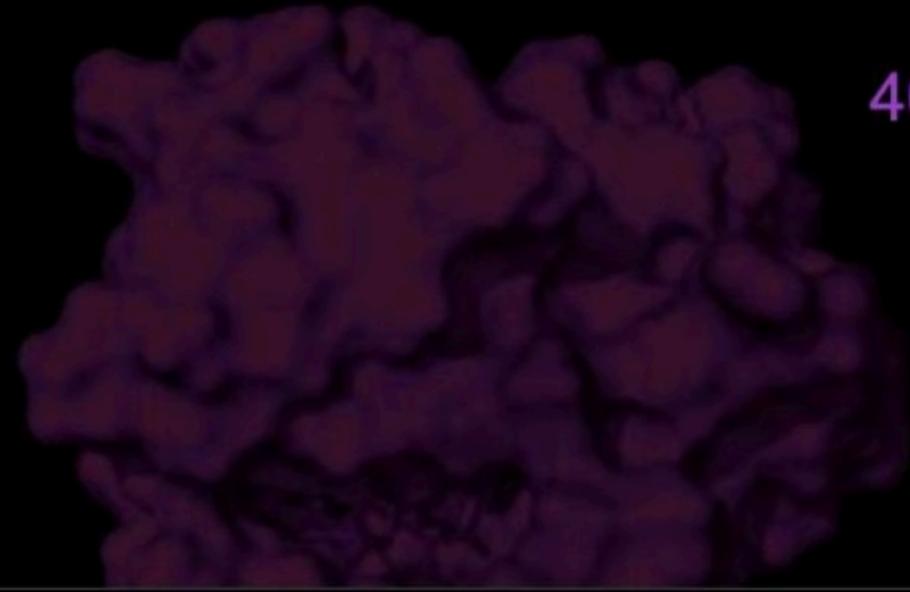


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Unlocking the power of T cells
against oncogenic driver mutations

40th Annual J.P. Morgan Healthcare Conference
January 2022



Forward-Looking Statements

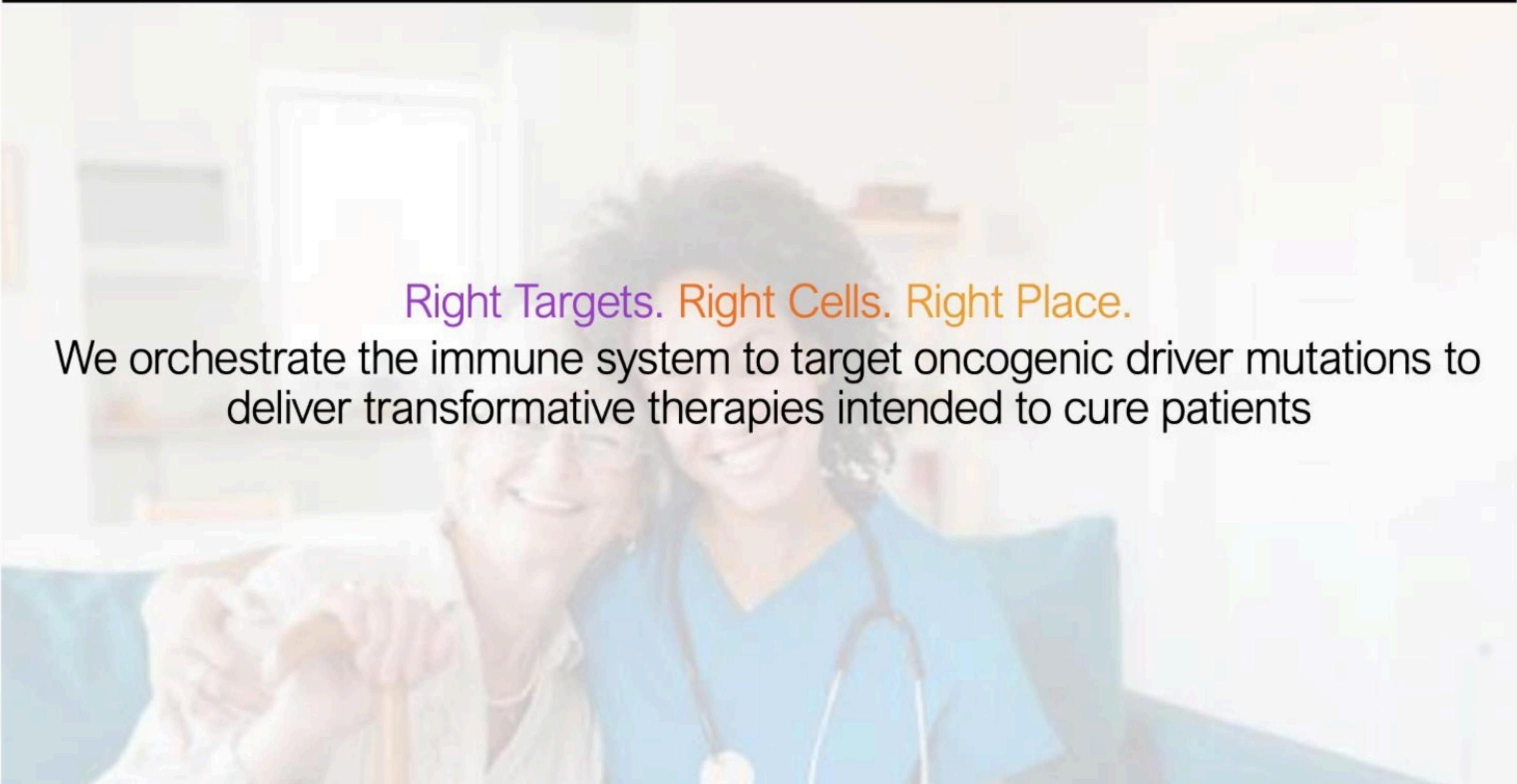
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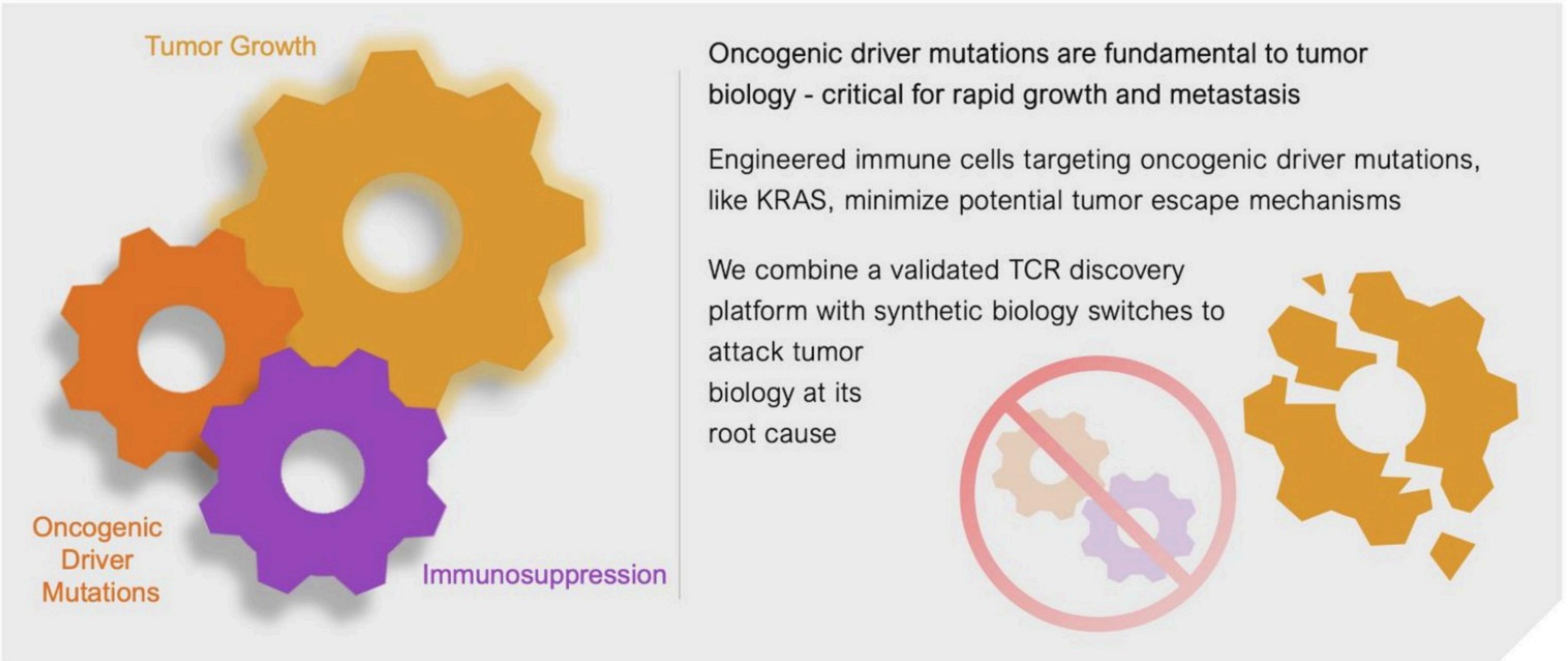
This presentation also includes statistical and other industry and market data that we obtained from industry publications and research, surveys and studies conducted by third parties as well as our own estimates of potential market opportunities. All of the market data used in this presentation involves a number of assumptions and limitations, and you are cautioned not to give undue weight to such data. Industry publications and third party research, surveys and studies generally indicate that their information has been obtained from sources believed to be reliable, although they do not guarantee the accuracy or completeness of such information. Our estimates of the potential market opportunities for our product candidates include several key assumptions based on our industry knowledge, industry publications, third-party research and other surveys, which may be based on a small sample size and may fail to accurately reflect market opportunities. While we believe that our internal assumptions are reasonable, no independent source has verified such assumptions.

Right Targets. Right Cells. Right Place.

We orchestrate the immune system to target oncogenic driver mutations to deliver transformative therapies intended to cure patients



Tumor growth powered by oncogenic driver mutations and immune cell dysfunction



Tumor Growth

Oncogenic Driver Mutations

Immunosuppression

Oncogenic driver mutations are fundamental to tumor biology - critical for rapid growth and metastasis

Engineered immune cells targeting oncogenic driver mutations, like KRAS, minimize potential tumor escape mechanisms

We combine a validated TCR discovery platform with synthetic biology switches to attack tumor biology at its root cause

Engineering T cells with the potential to cure hard-to-treat solid tumors

Right Targets

- Solid tumors are dependent on oncogene driver mutations like KRAS
- Driver mutations are present in every cancer cell, not in healthy tissues, and cannot be easily lost

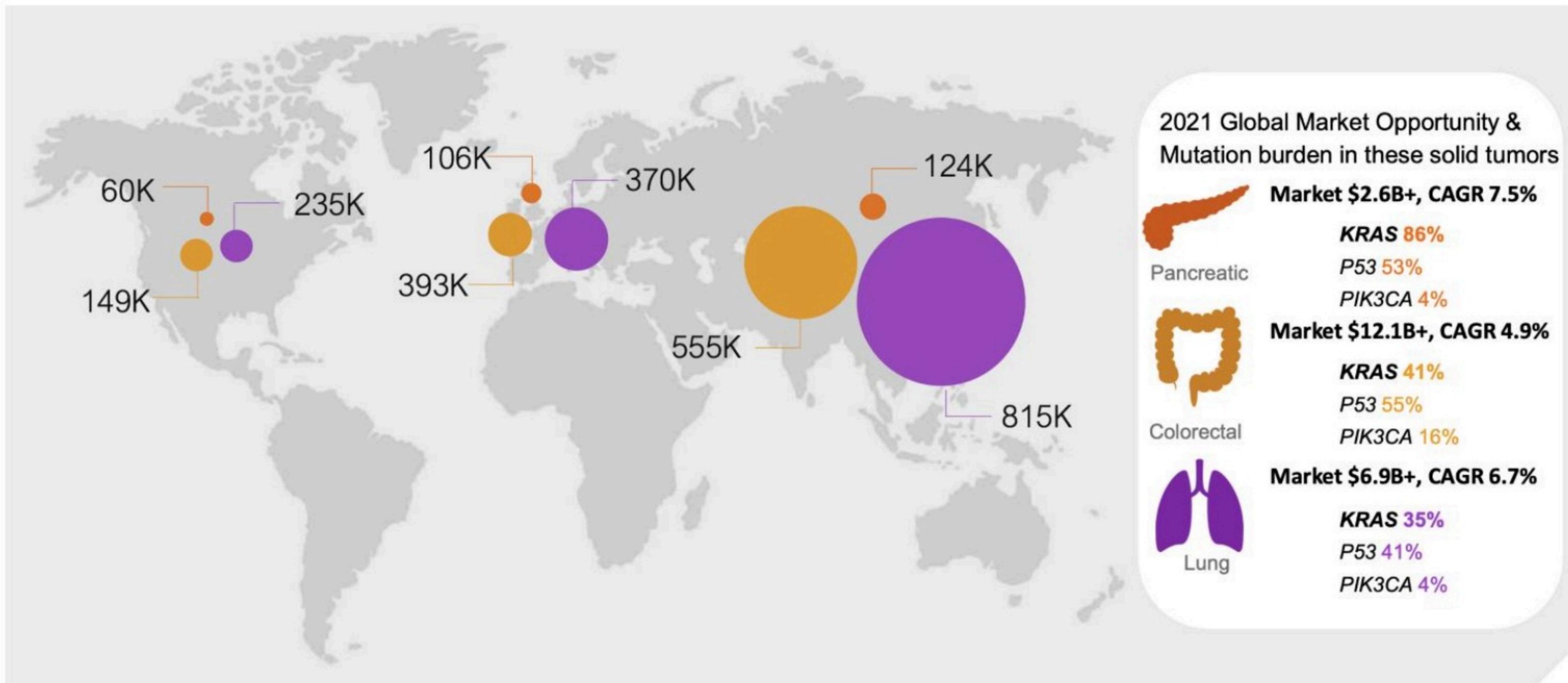
Right Cells

- Coordinate a CD4⁺ and CD8⁺ T cell response to sustain anti-tumor activity/response
- Select immune cells for stemness and central memory phenotype
- Manufacture to maintain cell naïveté and reduce terminal differentiation

Right Place

- Direct T cells to traffic into the solid tumor bed using proprietary TCRs
- Prevent deletion of T cells by converting Fas/FasL death signal to 41BB survival signal
- Convert immunosuppressive tumor microenvironment (TME) signals into stimulatory cues using additional synthetic biology switches

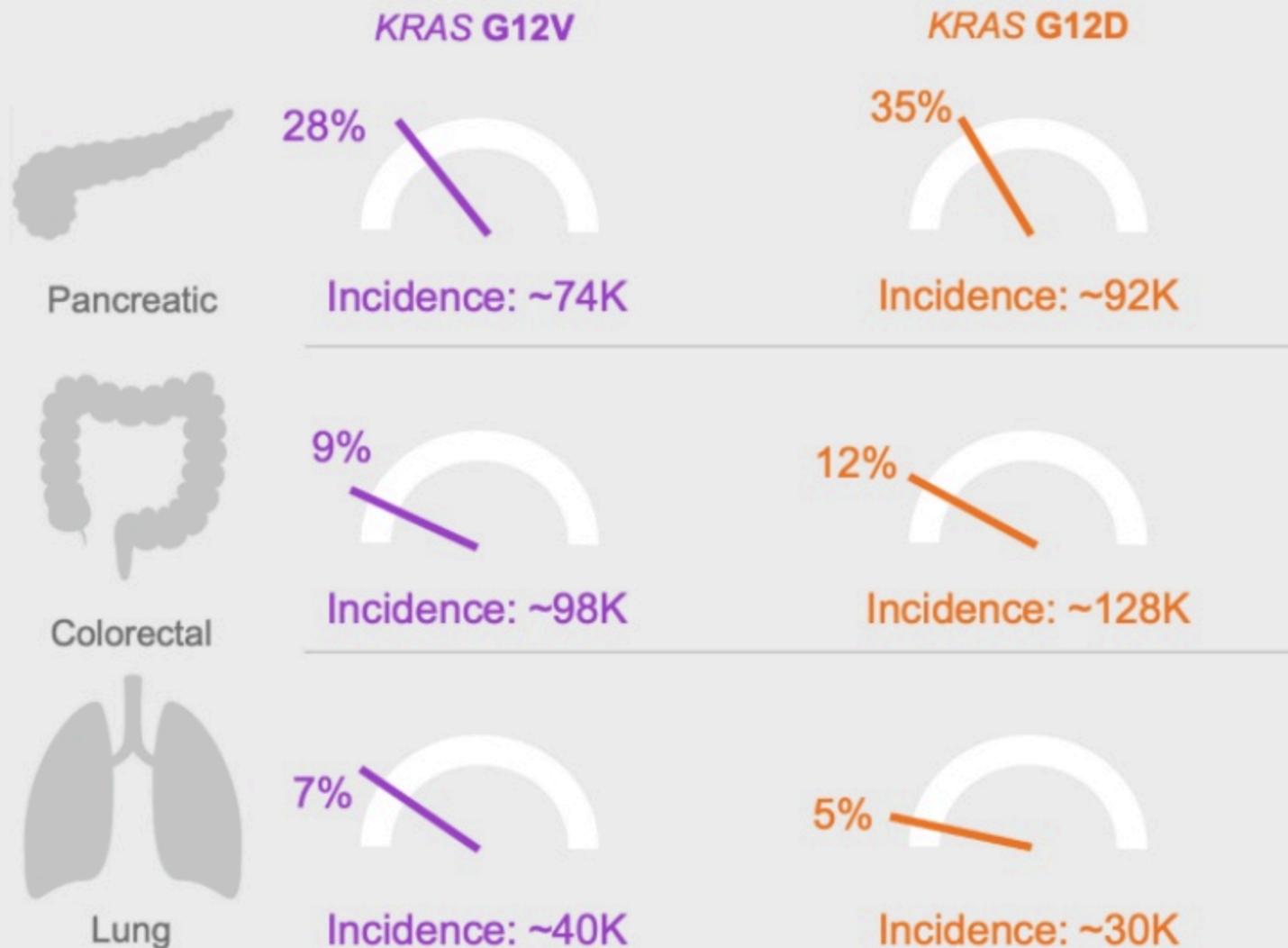
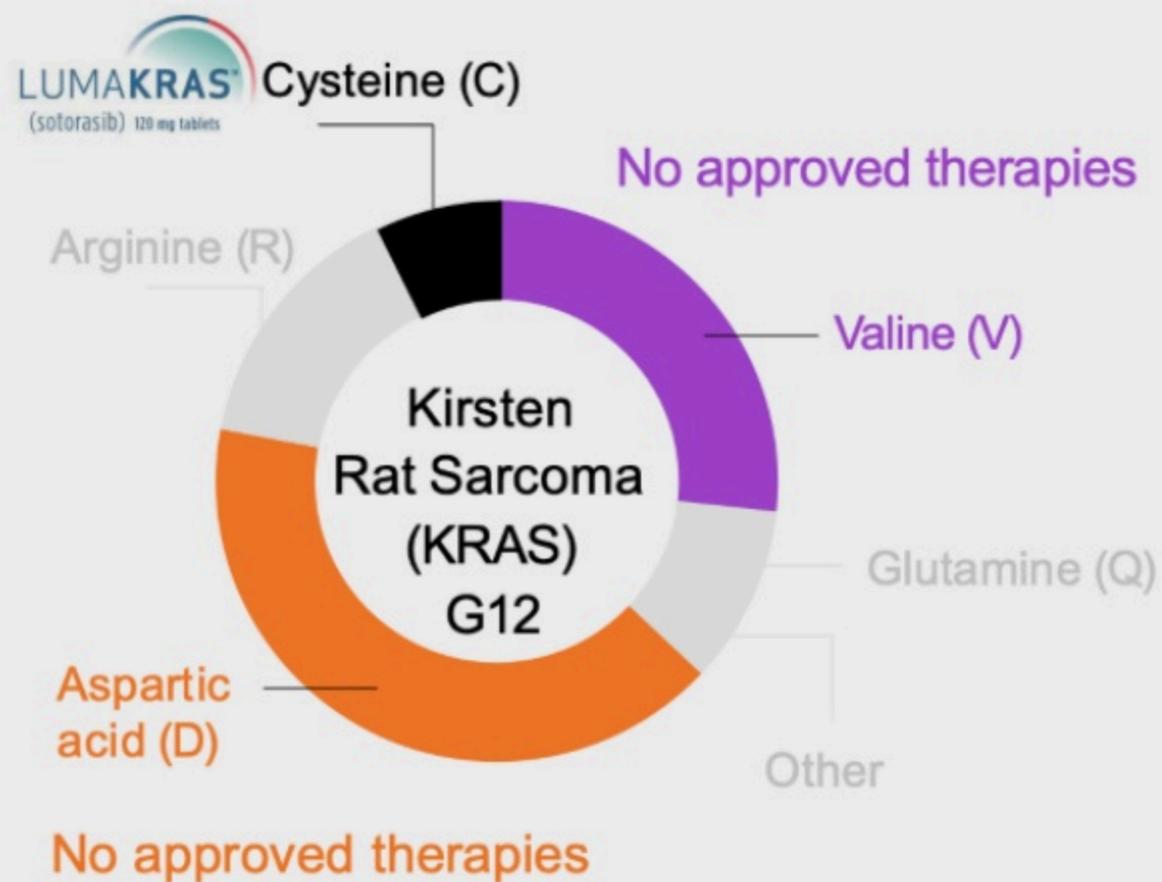
Solid tumors dependent on oncogenic drivers represent a large global market



Attacking KRAS: the most prevalent oncogenic driver mutation in solid tumors

Relative frequency of G12 mutations

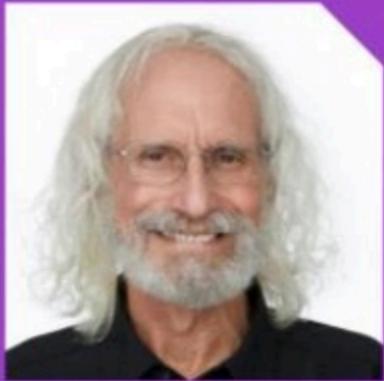
Incidence of G12V and G12D in certain adenocarcinomas



Exceptional Science Leaders and Experienced Team



Fred Hutch co-founders among the most prolific in the industry for generating T cell clinical programs



Phil Greenberg, MD



Aude Chapuis, MD



Tom Schmitt, PhD

nature medicine

T cell receptor gene therapy targeting WT1 prevents acute myeloid leukemia relapse post-transplant

Aude G. Chapuis, Daniel N. Egan, Merav Bar, Thomas M. Schmitt, Megan S. McAfee, Kelly G. Paulson, Valentin Voillet, Raphael Gottardo, Gunnar B. Ragnarsson, Marie Bleakley, Cecilia C. Yeung, Petri Muhlihauser, Hieu N. Nguyen, Lara A. Kropp, Luca Castelli, Felecia Wagener, Daniel Hunter, Marcus Lindberg, Kristen Cohen, Aaron Seese, M. Juliana McElrath, Natalie Duerkopp, Ted A. Gooley & Philip D. Greenberg 

Cancer Cell

T Cells Engineered against a Native Antigen Can Surmount Immunologic and Physical Barriers to Treat Pancreatic Ductal Adenocarcinoma

Ingunn M. Stromnes,^{1,2} Thomas M. Schmitt,¹ Ayaka Hulbert,¹ J. Scott Brockenbrough,¹ Hieu N. Nguyen,¹ Carlos Cuevas,⁴ Ashley M. Dotson,¹ Xiaoxia Tan,³ Jennifer L. Hotes,¹ Philip D. Greenberg,^{1,3,5,6,*} and Sunil R. Hingorani^{1,2,5,6,*}

Advanced over a dozen T cell therapeutic candidates



Undisclosed industry partners

Guided by a world class SAB specialized in T cell biology and immunology

Scientific Advisory Board



Phil Greenberg, MD
Scientific Co-Founder



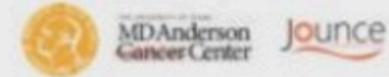
Aude Chapuis, MD
Scientific Co-Founder



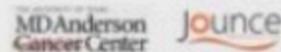
Tom Schmitt, PhD
Scientific Co-Founder



Jim Allison, PhD



Pam Sharma, MD, PhD



Rafi Ahmed, PhD



David Kranz, PhD



Susan Kaech, PhD



Experienced management poised to advance multiple T cell therapies

C-Suite



Jak Knowles, MD
CEO



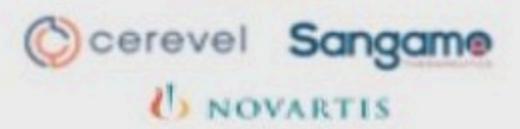
Loïc Vincent, PhD
CSO



Kim Nguyen, PhD
CTO



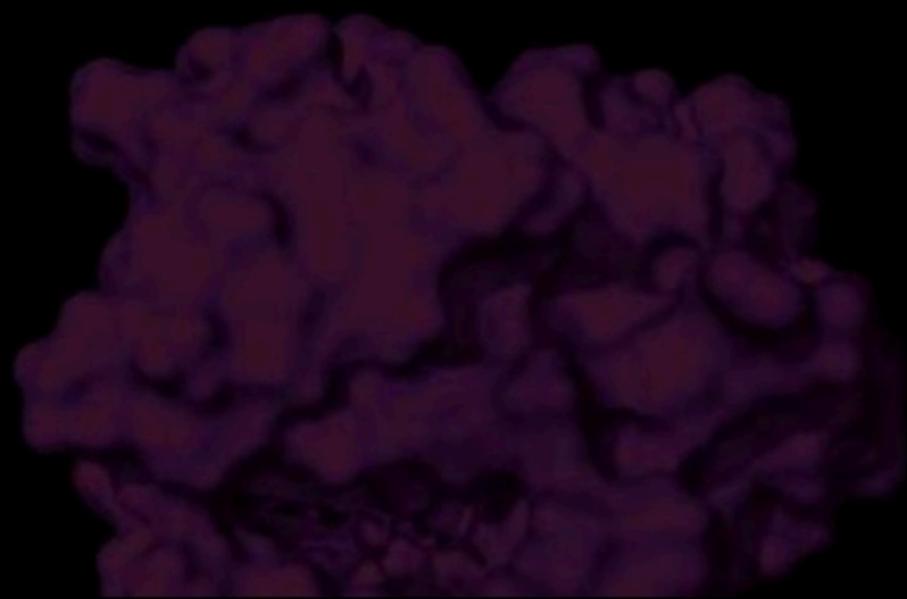
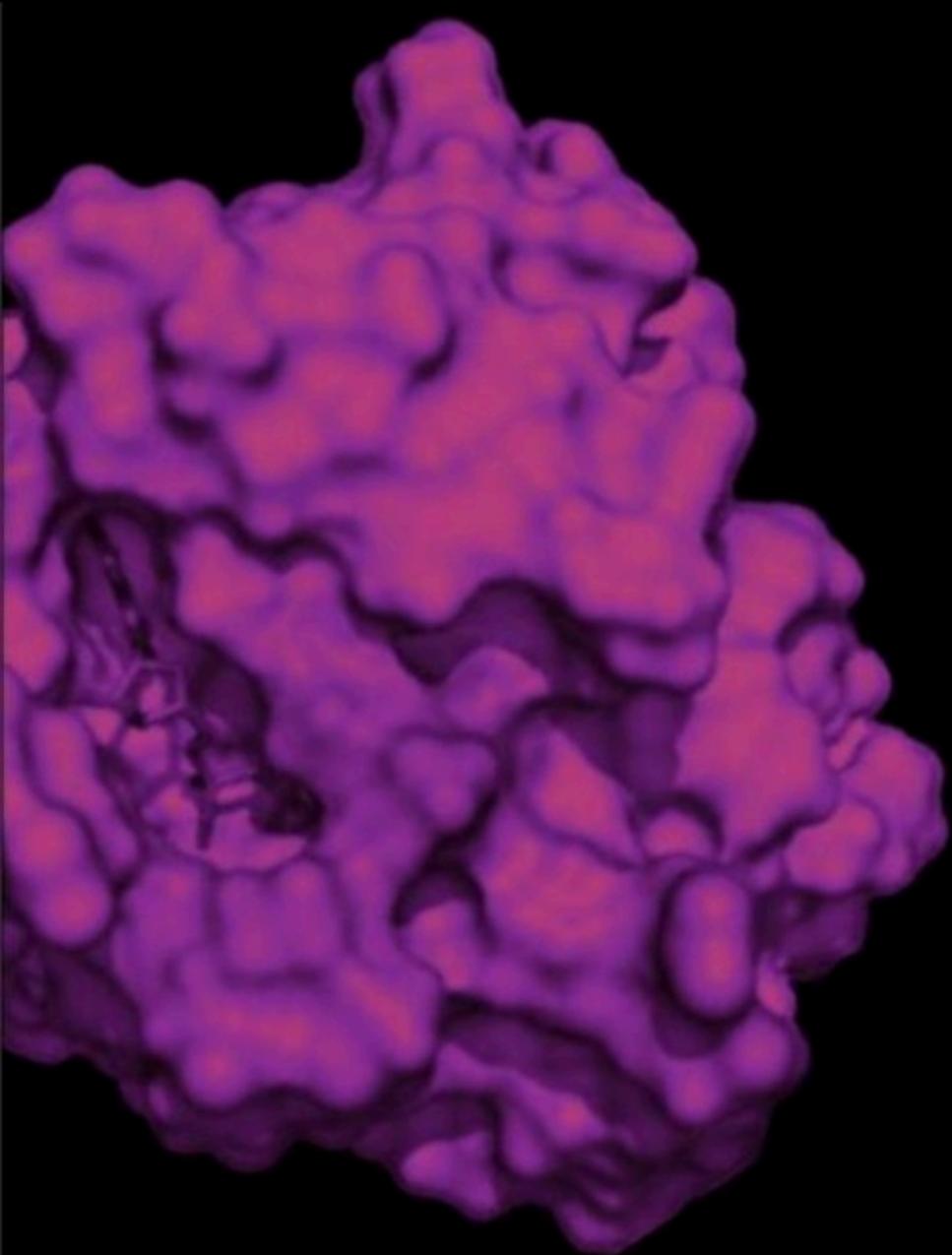
Kathy Yi, MBA
COO/CFO



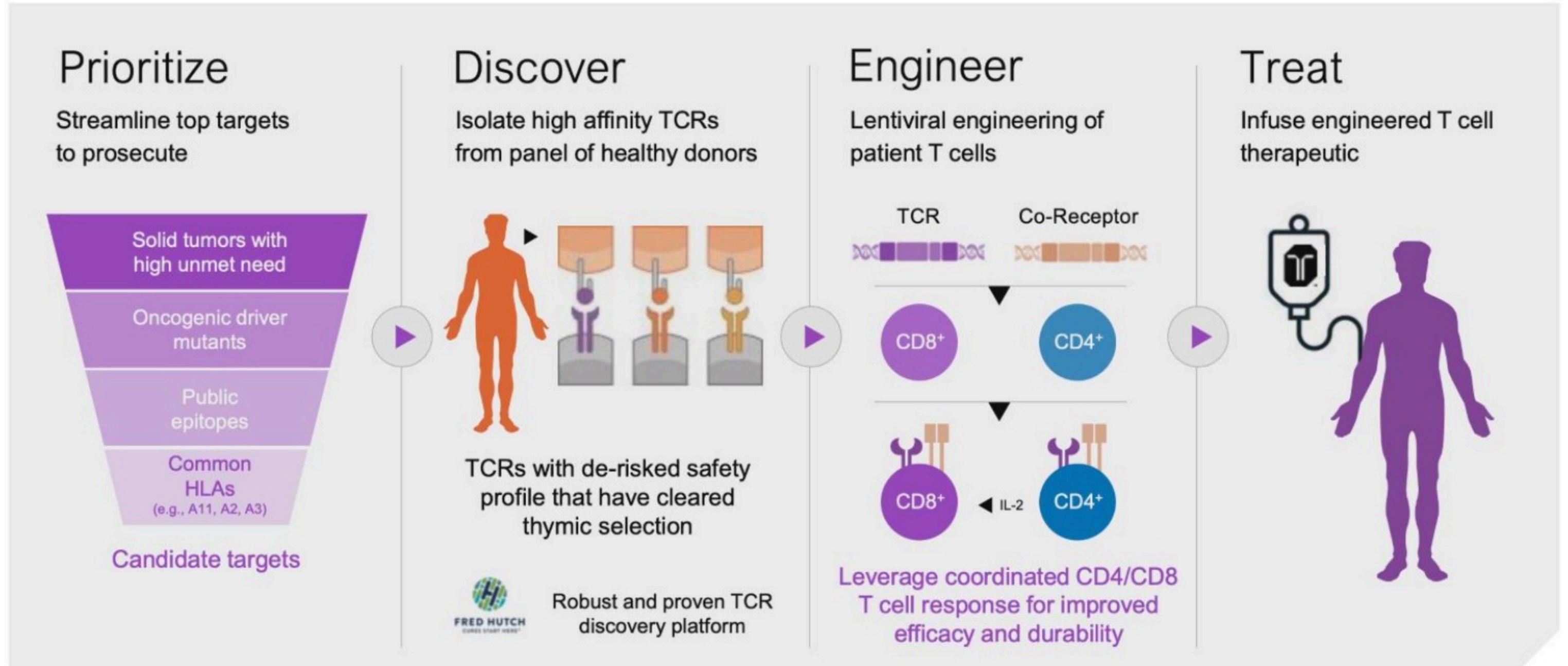
Seed Investors



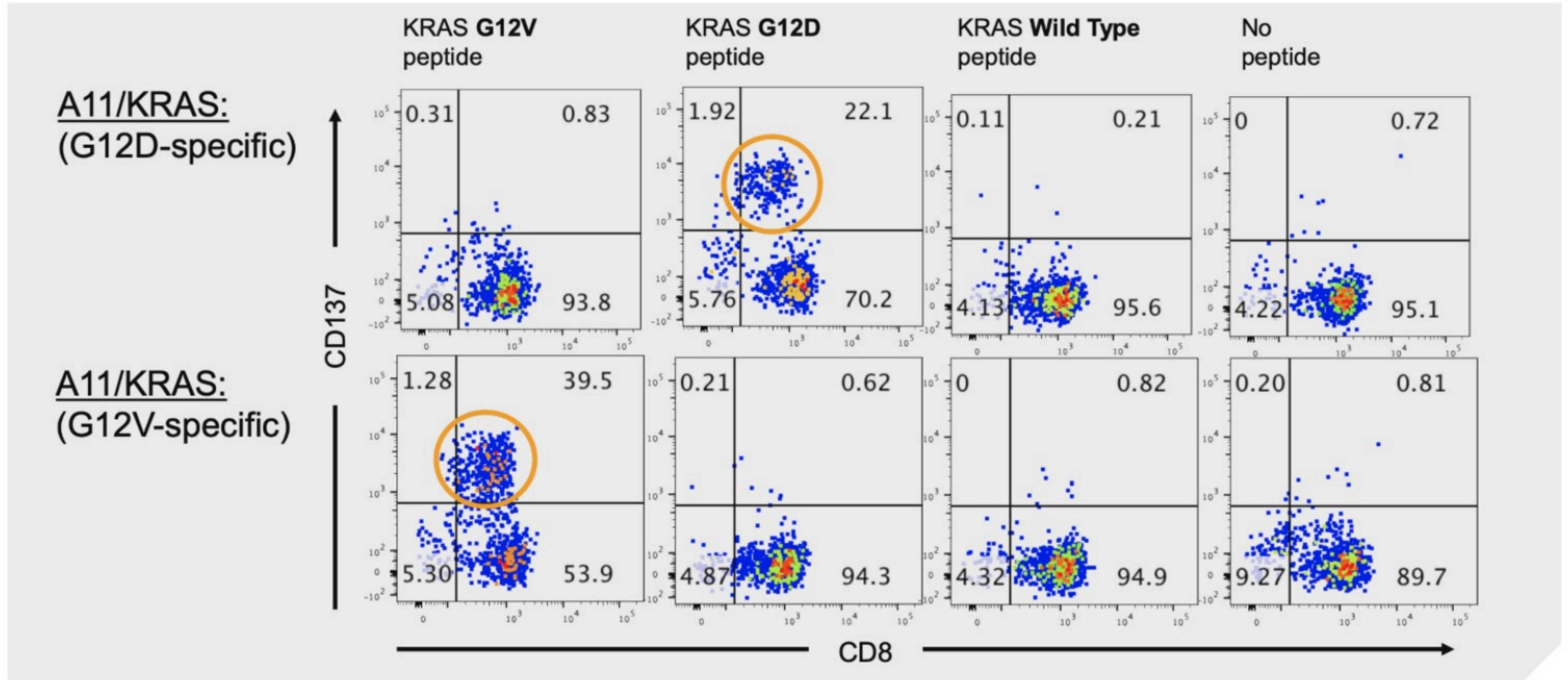
Solving Hard-to-Treat Solid Tumors



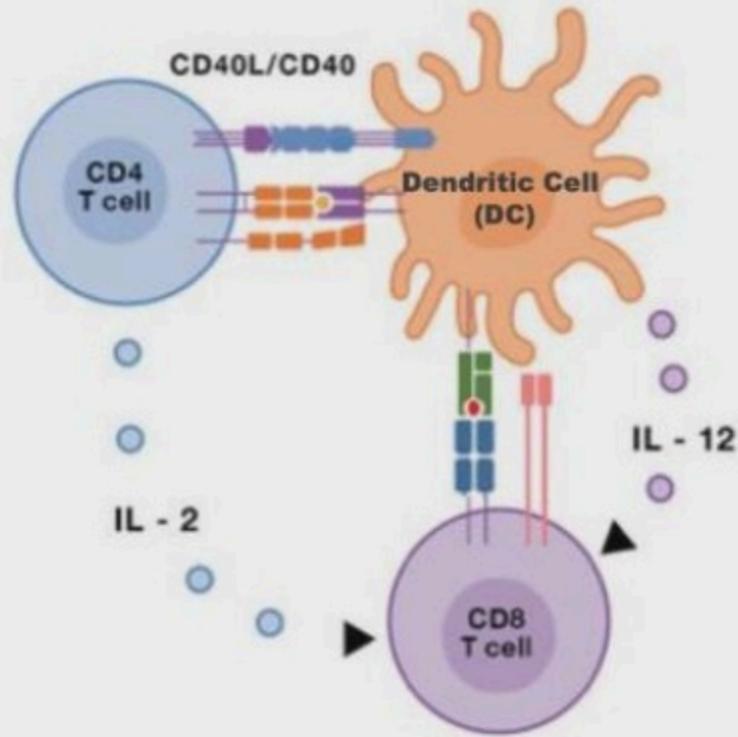
Affini-T's discovery platform has identified multiple TCR clinical candidates



KRAS G12V and G12D TCRs are highly selective and do not react to Wild Type peptide

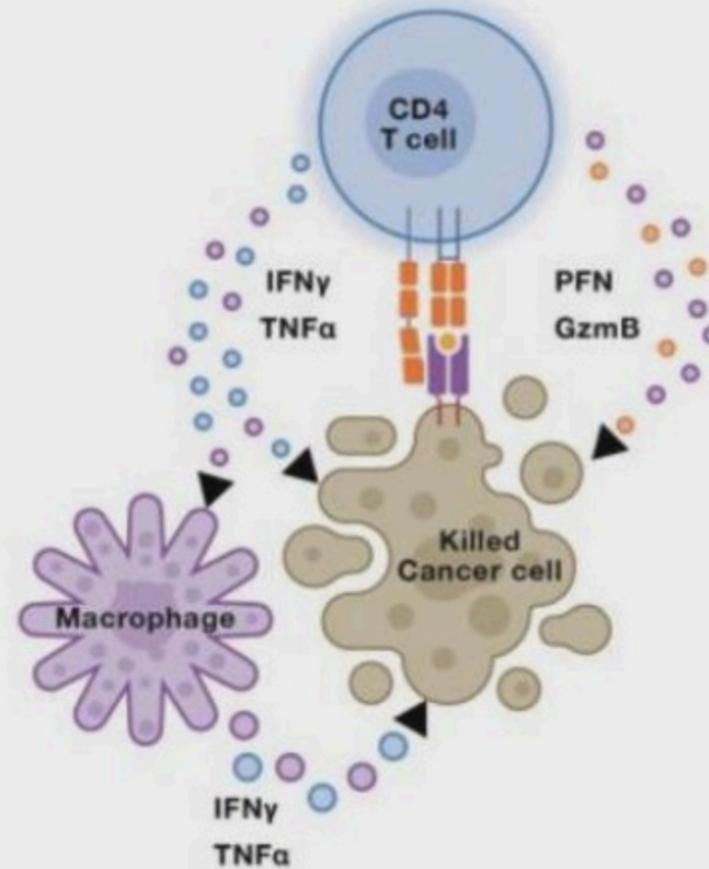


Fundamental immunology: CD4 T cells can orchestrate durable anti-tumor responses



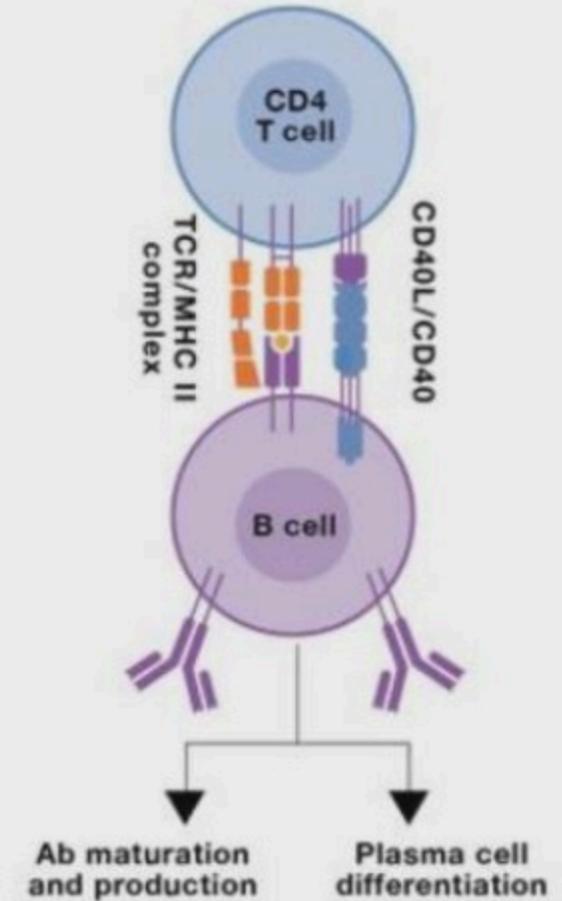
CD4 Helper

Promotes CD8 T cell function, proliferation and survival



CD4 Effector

Direct and indirect killing of tumor cells



CD4 Activator

Drives B cell maturation and antibody production