TEON® THERAPEUTICS

Inventing New Hope

Targeting untapped metabolic signaling pathways to restore antitumor immunity

2022

Confidentiality and Forward-Looking Statements

These materials contain confidential information and are being provided to you with the express understanding that, without Teon Therapeutics, Inc.'s (the "Company") prior express written permission, you may not release these materials or any portion thereof or discuss the information contained therein or make reproductions of or use such materials for any purpose other than evaluating a potential investment in the Company.

Please note that an investment in the Company is speculative and involves a high degree of risk. Because of this risk, you should invest your funds only if you are an accredited investor, you are prepared to bear the economic risk of your investment for an indefinite period of time and you are able to withstand a total loss of your investment. These materials do not constitute an offer to sell to, or a solicitation of an offer to buy from, anyone in any state or jurisdiction in which such an offer or solicitation is not authorized.

This presentation contains forward-looking statements that involve risks and uncertainties. These forward-looking statements are neither historical facts nor assurances or guarantees of future performance. Instead, they are based on our current expectations about future events that we believe may affect our financial condition, results of operations, business strategy, and financial needs. All statements, other than statements of historical facts, contained in this presentation are forward-looking statements. Without limitation, any statements preceded or followed by or that include the words "future," "should," "would," "could" "expect," "positioned," "anticipate," "aim," "believe," "likely," "potential," "estimate," "may," "will," "intend," "plan," "projected," "target," "strategy" "continue." or words, phrases, or terms of similar substance or the negative thereof, are intended to identify forward-looking statements, although not all forward-looking statements contain these words. Forward-looking statements involve risks, uncertainties, assumptions and other factors, some of which are beyond our control, which may cause our actual results, performance or achievements to be materially different from those expressed or implied by the forward-looking statements. These factors include the ability of our clinical trials to demonstrate acceptable safety and efficacy of our product candidates, and other positive results; the timing, progress and results of clinical trials for our product candidates, including the timing of initiation and completion of studies or trials and related preparatory work, the period during which the results of the trials will become available, and our research and development programs; the timing, scope and likelihood of regulatory filings, NDA submissions and approvals; our ability to obtain marketing approvals of our product candidates and to meet existing or future regulatory standards or comply with post-approval requirements; our expectations regarding the potential advantages of our product candidates over existing therapies; the impact of any pandemic on our business, clinical development programs and operations, including actions taken by us, other businesses, and governments to address or mitigate any pandemic; our ability to identify and/or develop additional product candidates, product candidates for other indications or technologies with significant commercial potential that are consistent with our commercial objectives; our ability to finance, develop, acquire and advance additional product candidates into, and successfully complete, clinical trials; the initiation, timing, progress and results of our preclinical studies, clinical trials and research and development programs; challenges in accurately predicting the potential market opportunity and patient populations for our product candidates, including TT-702, TT-816, TT-083, TT-373 (UDT2) and TT-X (UDT3) if approved for commercial use; the commercialization and market acceptance of our product candidates; and the implementation of our business model and strategic plans for our business and product candidates, and overall global economic and business conditions impacting our business, including the strength of the oncology and related markets; demand, competition and pricing pressures in the markets we serve; volatility in currency exchange rates; risks associated with operating or engaging with foreign businesses; the impact of material cost and other inflation; our ability to comply with laws and regulations; the impact of changes in laws, regulations and administrative policy, including those that limit U.S. tax benefits or impact trade agreements and tariffs; and the outcome of litigation and governmental proceedings. Accordingly, readers are cautioned not to rely on these forward-looking statements and no representations or warranties (expressed or implied) are made about the accuracy of any such forward-looking statements. All forward-looking statements speak only as of the date of this presentation and, except as required by applicable law, we do not plan to update or revise any information contained herein.

Teon's Mission: Inventing New Hope For Cancer Patients

- The 5-year mortality rate varies between 50-90% across solid tumors[1]
- IO drugs only work in 12% of solid cancer patients[2]

Demonstrated Expertise

- World-class drug design, biology and chemistry of G-protein coupled receptors (GPCRs)
- Average of 20+ years experience with Biotech and Big Pharma

Impeccable Science Addressing Untapped Pathways

- Targeting unexploited metabolic pathways with initial focus on GPCRs
- First- and best-in-class small molecules protected by strong IP

Impactful Value Drivers

- Lead Molecule Milestones: TT-702 Ph1 complete (Q1 2023) and TT-816 IND accepted by FDA (Q2 2022) and FPI planned (Q3 2022)

Demonstrated Expertise: Executive Team

Serge Messerlian, MSc, MBA

Chief Executive Officer

- 20+ years track-record: specialty biologics, biosimilars & cell therapies
- Recent President Oncology, J&J; President Actelion, J&J

janssen Baxalta Baxter

Jim Liu, PhD

VP, Chemistry

- Previous Principal Scientist/Director of Medicinal Chemistry at Amgen
- 20 years R&D experience in GPCRs and kinase small molecule drugs

AMGEN Tularik

Lina Yao, MD, PhD

Chief Scientific Officer

- Previous Senior Director of Biology at Gilead
- 20+ years R&D in oncology, inflammation and fibrosis diseases; and GPCR research

GILEAD CV THERAPEUTICS™ UCSF

Elfatih Elzein, PhD

VP, Chemistry & Early Development

- Previous Principal Scientist/Director of Medicinal Chemistry at Gilead
- 20+ years R&D experience in oncology, CV & metabolic diseases
- Primary inventor of Lexiscan® (only adenosine receptor drug approved)

GILEAD CV THERAPEUTICS™

Robert Sikorski, MD, PhD

Acting Chief Medical Officer

- Former CMO at FivePrime; Sr Dir Global Oncology Clin Dev MedImmune and Amgen
- 20 years clinical research experience

AMGEN FivePrime MedImmune

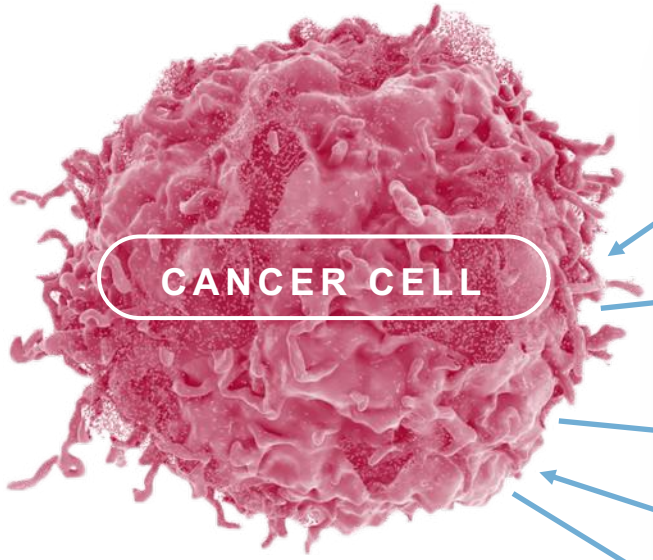
Peter Fan, PhD

VP, Biology

- Previous Principal Scientist/Director of Biology at Gilead
- 20+ year GPCR research experience; 15+ years biotech experience in oncology, inflammation and metabolic disease

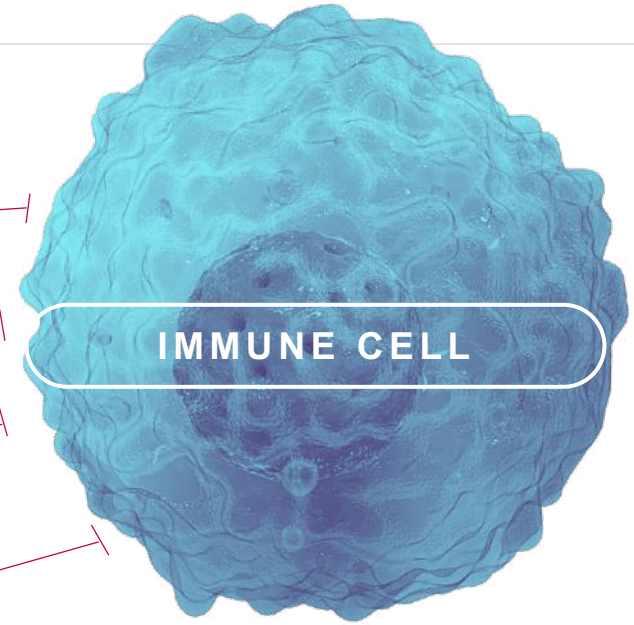
GILEAD CV THERAPEUTICS™

Targeting Unexploited Metabolic Pathways With a Focus on GPCRs



Tumor Metabolic Environment

- **Nutrient Competition**
  - Glucose
- **Metabolites**
  - Lipids, Adenosine
- **Low pH**
- **Hypoxia**
- **Oxidative Stress**



- T effector cells ↓
- Treg cells ↑
- Myeloid-derived suppressor cell ↑
- DC and NK cells ↓

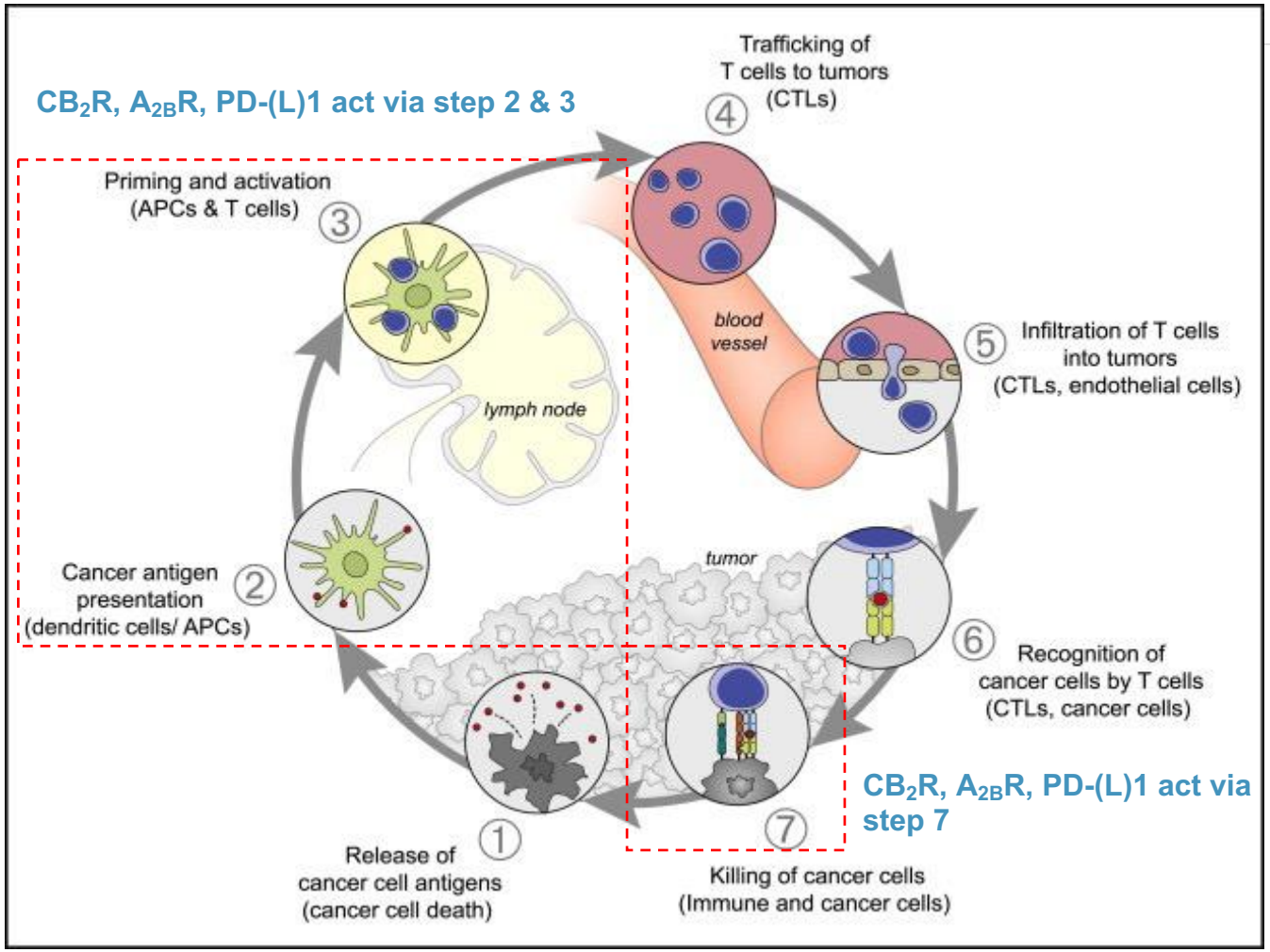
Our focus is developing highly selective and potent small molecule antagonists and inhibitors in glucose, lipid and adenosine pathways directly halting tumor cell growth and reversing immune cell suppression

Impeccable Science: First- and Best-In-Class

Candidate	First-in-Class or Best-in-Class	Indication(s)	Discovery	Pre-clinical	IND	Phase 1	Partners
TT-702 *A_{2B} Receptor Antagonist*	✓	mCRPC, TNBC, MSI					CANCER RESEARCH UK
TT-816 *CB_2 Receptor Antagonist (Checkpoint Inhibitor)*	✓	NSCLC, RCC, Ovarian					Proprietary
TT-373 *UDT_2 Metabolic Transport Inhibitor*	✓	Undisclosed					Proprietary
TT-X *UDT_3 Metabolic Enzyme Inhibitor*	✓	Undisclosed					Proprietary

UDT = undisclosed target

Like PD-(L)-1, CB_2R and $A_{2B}R$ Play A Critical Role in the Cancer-Immunity Cycle



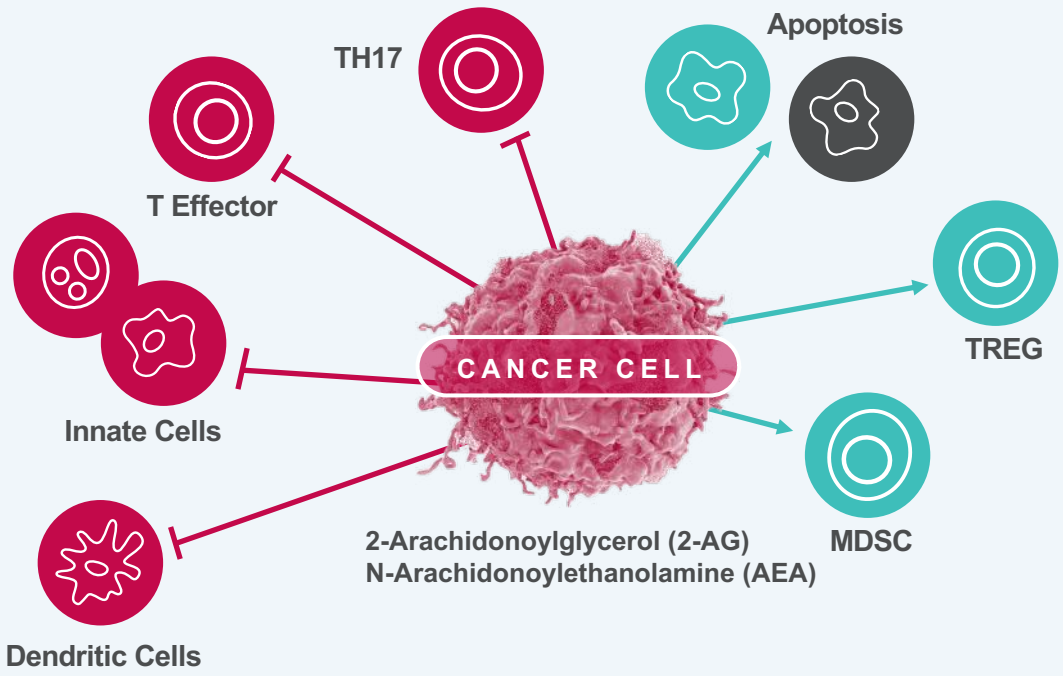
Chen & Mellman, Immunity 2013

- DCs present captured antigens to T cells (step 2), resulting in the priming and activation of effector T cell responses against the cancer-specific antigens (step 3). The nature of the immune response is determined at this stage, where TT-702, TT-816 and anti-PD-(L)1 can enhance DCs functions and promote effector T cell proliferation.
- Tumor killing effector T cells (step 7) in the TME can be suppressed by adenosine, endocannabinoids and PD-L1. $A_{2A}R$ antagonists, TT-816 and anti-PD-(L)1 can prevent T cell suppression directly, and TT-702 can do so indirectly by inhibiting Tregs and MDSCs.
- Like anti-PD-(L)1, TT-702 and TT-816 act on both step 2-3 and 7, which is critical for IO agents to show efficacy.

TT-816

First-in-Class Cannabinoid Antagonist acting as an Immune Checkpoint Inhibitor

CB_2R Activation Promotes Cancer Cell Growth and Inhibits Antitumor Immunity



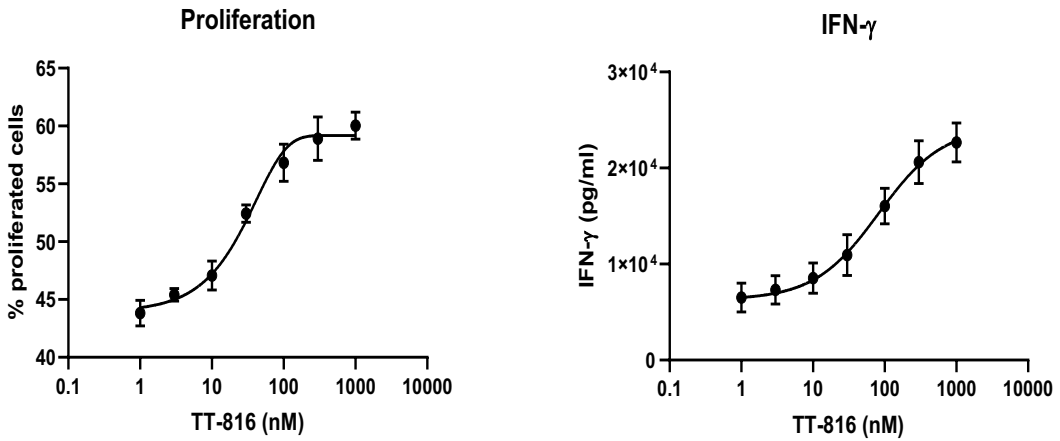
CB_2R Rationale

- Immune cells express high levels of CB_2R[1] and produce endocannabinoids thereby forming an "immune endocannabinoid system"[2]
- CB_2R suppresses innate and adaptive antitumor immune responses[3,4]
  - lowers NK cell number and inhibits NK cell function
  - inhibit DCs maturation, function, IFN-γ responses and DC-mediated T cell proliferation and activation
  - induces T cell apoptosis, suppresses T cell activity and cytokine release
- Elevated CB_2R expression and levels of endogenous ligands in the TME is associated with worse overall survival[5-9] and aggressiveness of cancer[10,11]
- Cannabis use during immune checkpoint inhibitor treatment is associated with lower response rate and worse overall survival[12-14]

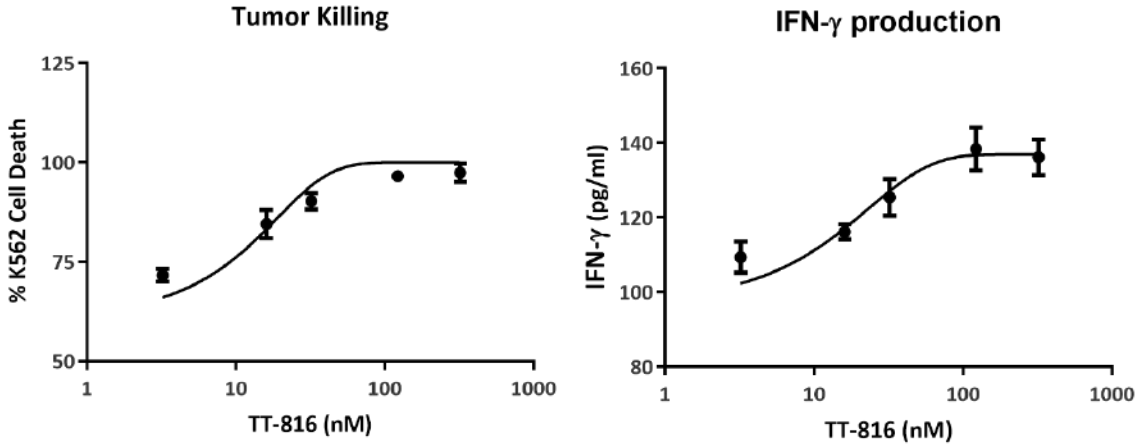
- TT-816 has unique properties: oral small molecule that enhances innate and adaptive immunity
- IND accepted by FDA Q2 2022 and FPI on track for Q3 2022

TT-816 Enhances Both Human T and NK Cell Function

Impact on T Cell Function



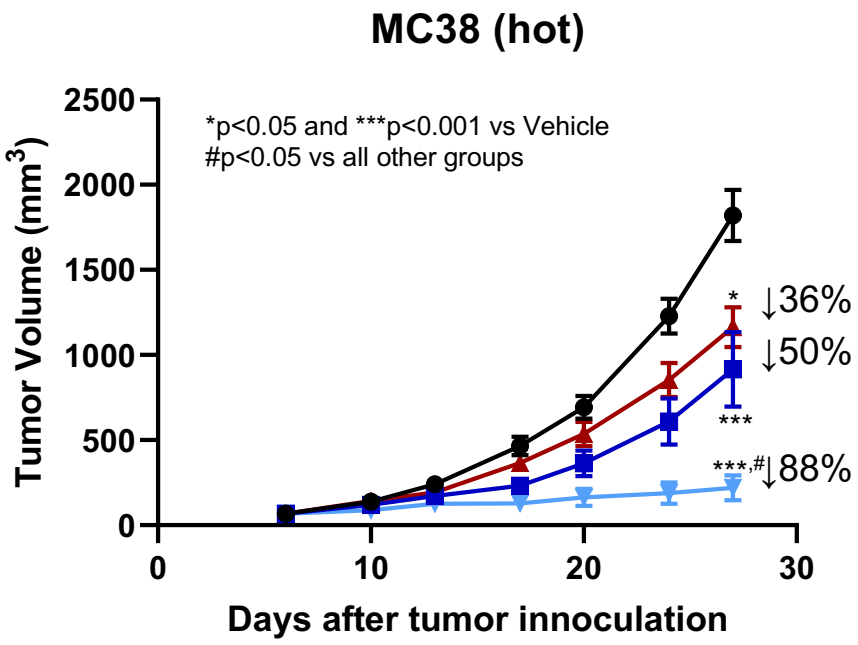
Impact on NK Cell Function

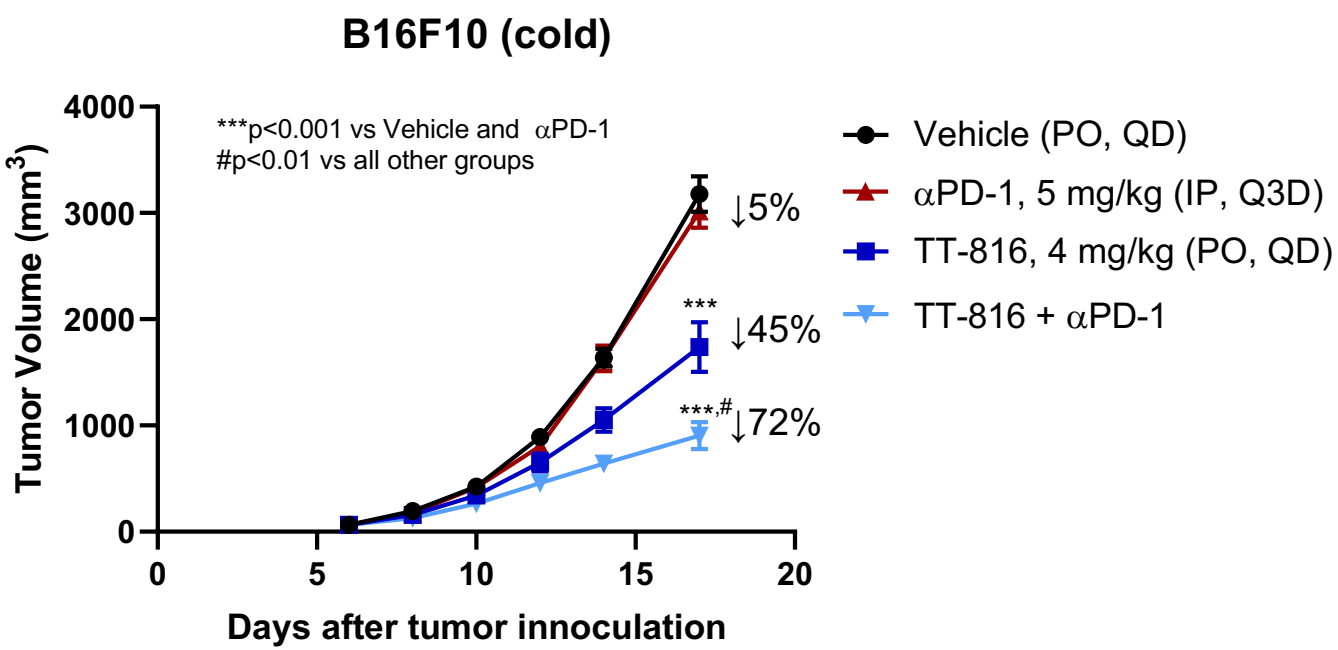


- TT-816 dose-dependently stimulates T cell proliferation and IFN-γ production
- TT-816 stimulates adaptive antitumor immunity

- TT-816 dose-dependently activates NK cells, increases IFN-γ production and promotes cancer cell killing
- TT-816 stimulates innate antitumor immunity

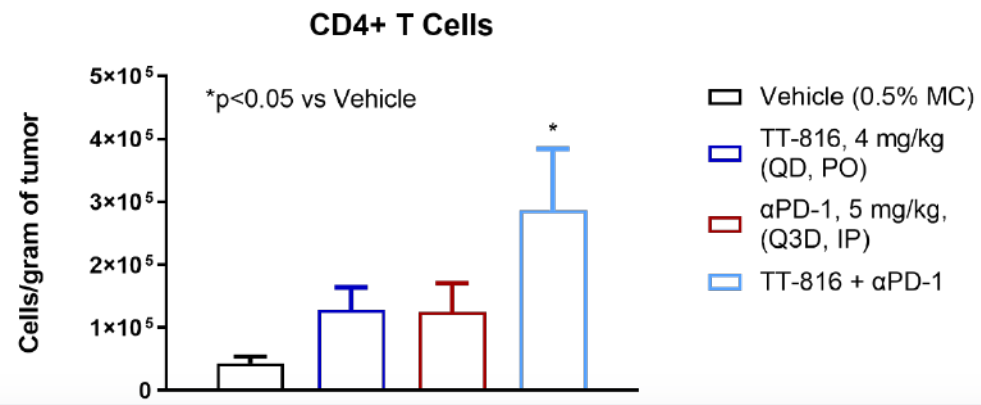
TT-816 Is Superior to Anti-PD-1 and Synergizes Anti-PD-1 Efficacy

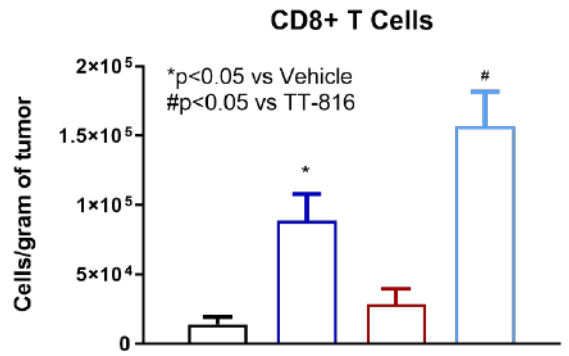


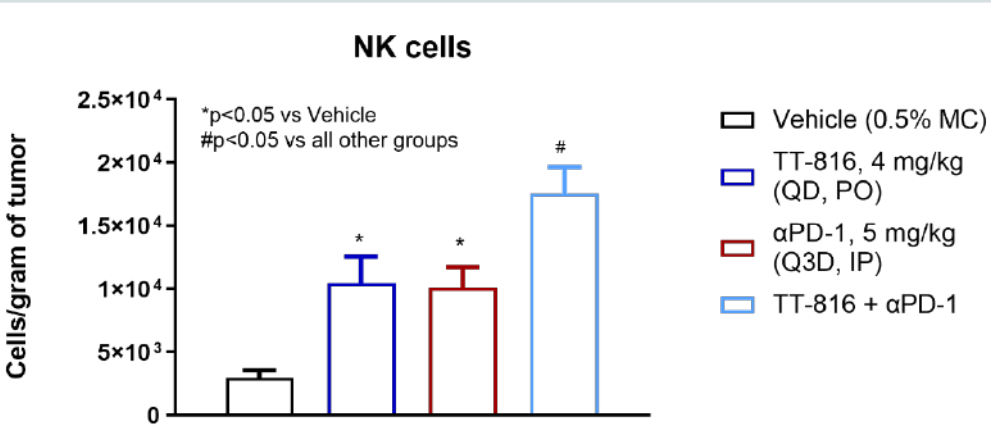


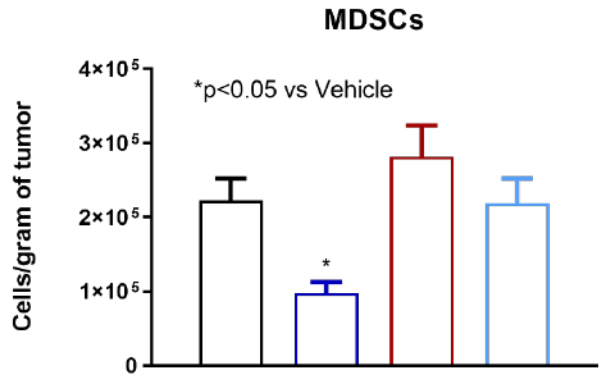
- TT-816 enhances the effect of anti-PD-1 in the hot tumor model
- TT-816 is superior to anti-PD-1 in cold tumor model and combination with anti-PD-1 synergistically inhibits cold tumor growth

TT-816 Increases T and NK Cell Infiltration and Reduces MDSCs







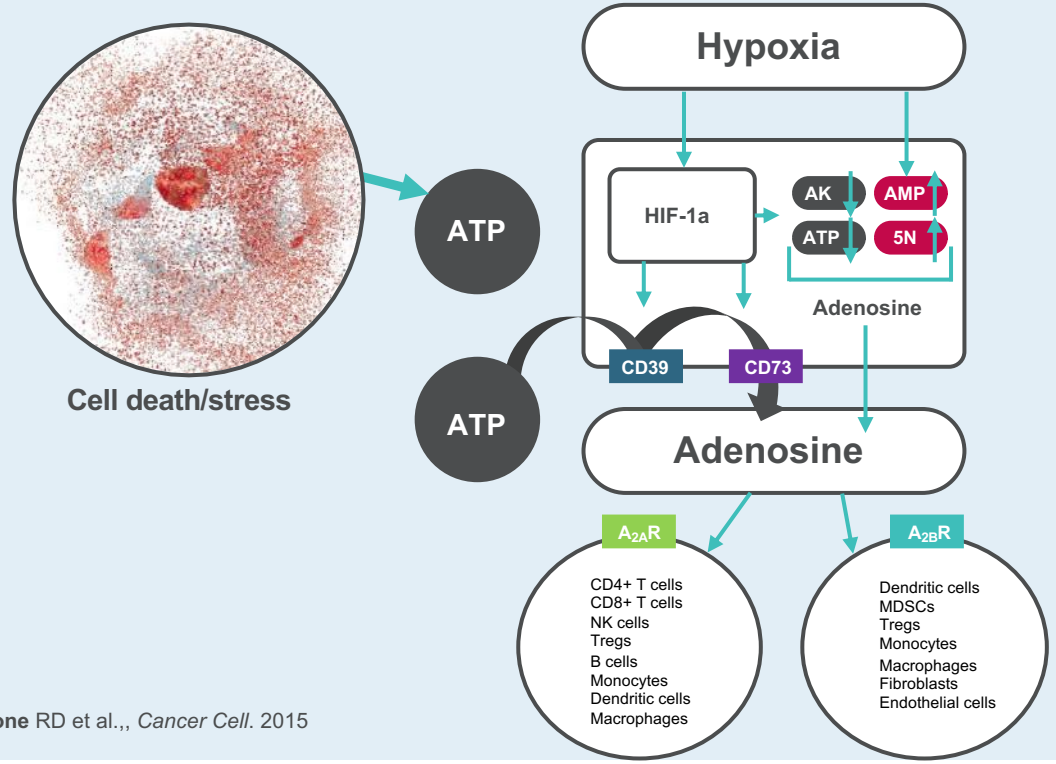


- TT-816 facilitates infiltration of CD4+ and CD8+ T cells, NK cells and reduces MDSCs
- TT-816 enhances the effect of anti-PD-1 on T cell and NK cell infiltration

TT-702

First-in-Class A_{2B} Selective Adenosine Receptor Antagonist

$A_{2B}R$ Expression And Activation Are Regulated by Hypoxia In the TME to Promote Tumor Cell Growth and Inhibit Antitumor Immunity



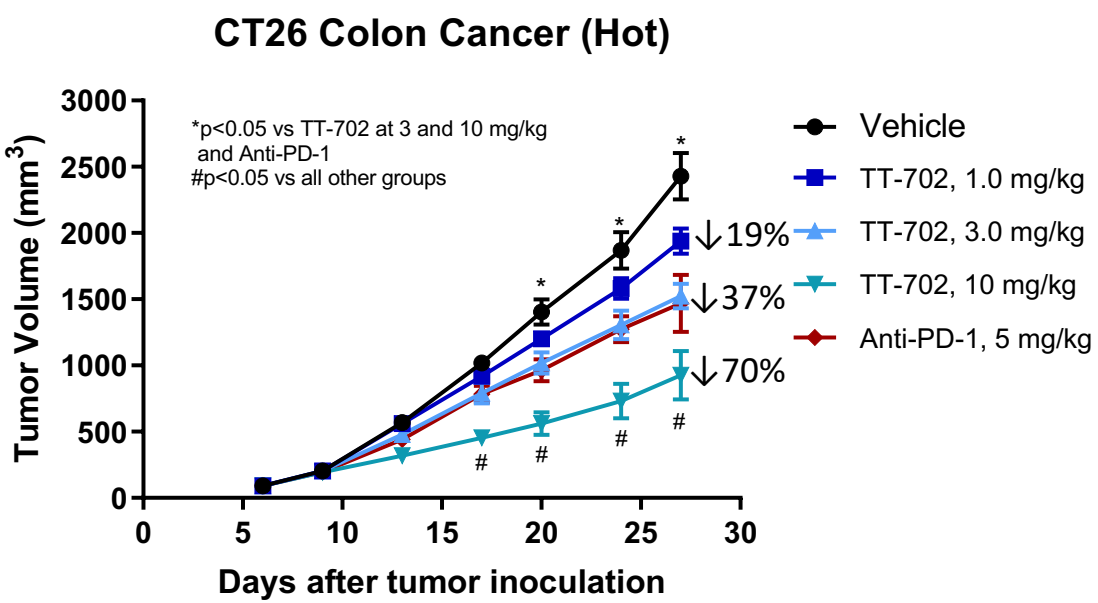
Leone RD et al.,, *Cancer Cell*. 2015

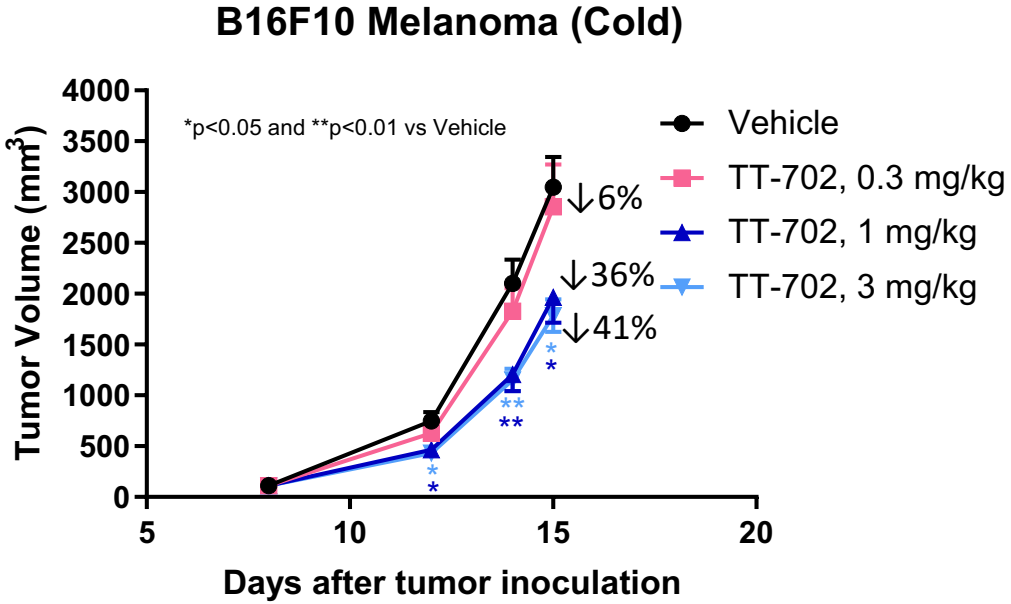
$A_{2B}R$ Rationale

- $A_{2B}R$ is highly expressed in many tumors and upregulated by hypoxia (Hif-1a)[1]. High $A_{2B}R$ expression is associated with worse survival in prostate[2,3], breast[4,5], and bladder cancer[6]
- $A_{2B}R$ inhibition or knockdown prevents cancer cell proliferation of prostate, breast, bladder, renal, liver, oral, colon cancers[7-14]
- $A_{2B}R$ blocking has reverse-immunosuppressive effect (via MDSCs, Tregs, DCs) and additional mechanism of anti-cancer cell proliferation, anti-angiogenesis, anti-fibrosis and anti-metastasis[1]
- Clinical evidence supports $A_{2B}R$ as a valid target as inhibition of either CD73 or CD39 reduces ~50% adenosine in the TME[15,16], which may be sufficient to inhibit the low affinity $A_{2B}R$ activation, but not affect the high affinity $A_{2A}R$[17]
- $A_{2A}R$ antagonist has limited efficacy in treating cancer such as NSCLC and mCRPC[18,19]

- Clinical and pre-clinical evidence support targeting $A_{2B}R$ is more effective approach to blocking adenosine effect
- Phase 1 trial for TT-702 on track with several patients safely dosed and Phase 2 start mid-2023

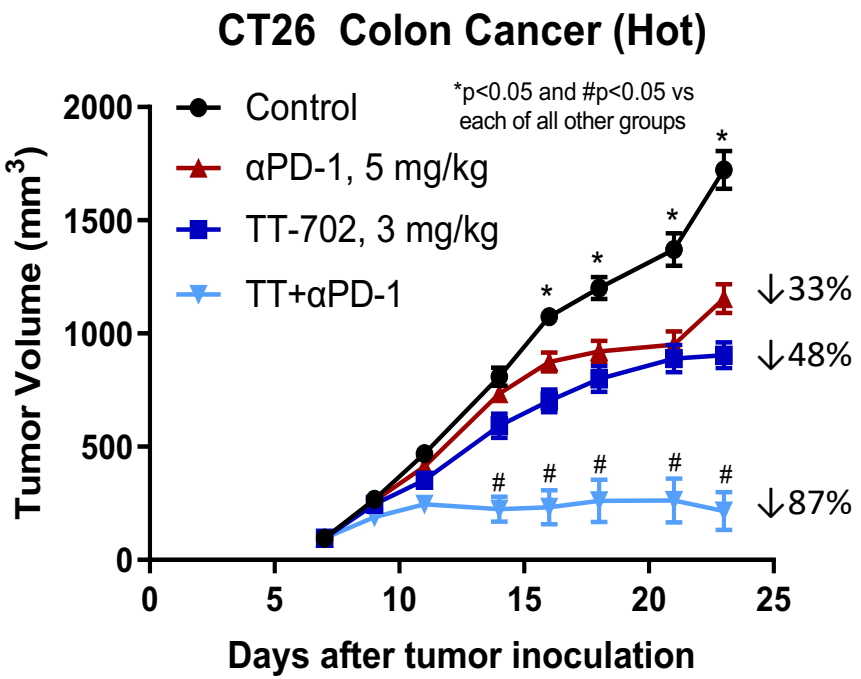
TT-702 Effective as Monotherapy in Hot and Cold Tumor Models

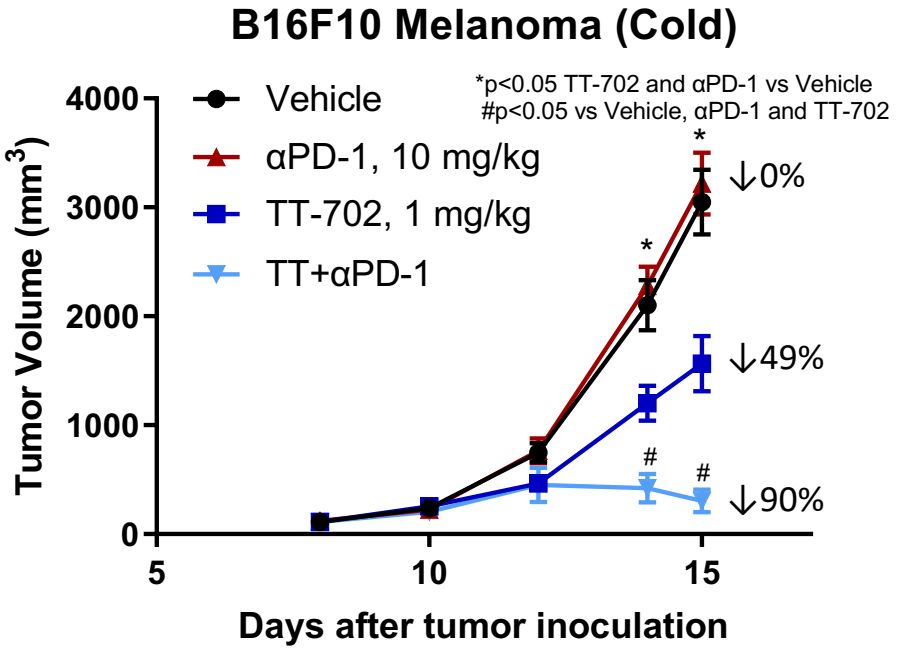




- TT-702 dose-dependently inhibits CT26 (hot) and B16F10 (cold) tumor growth

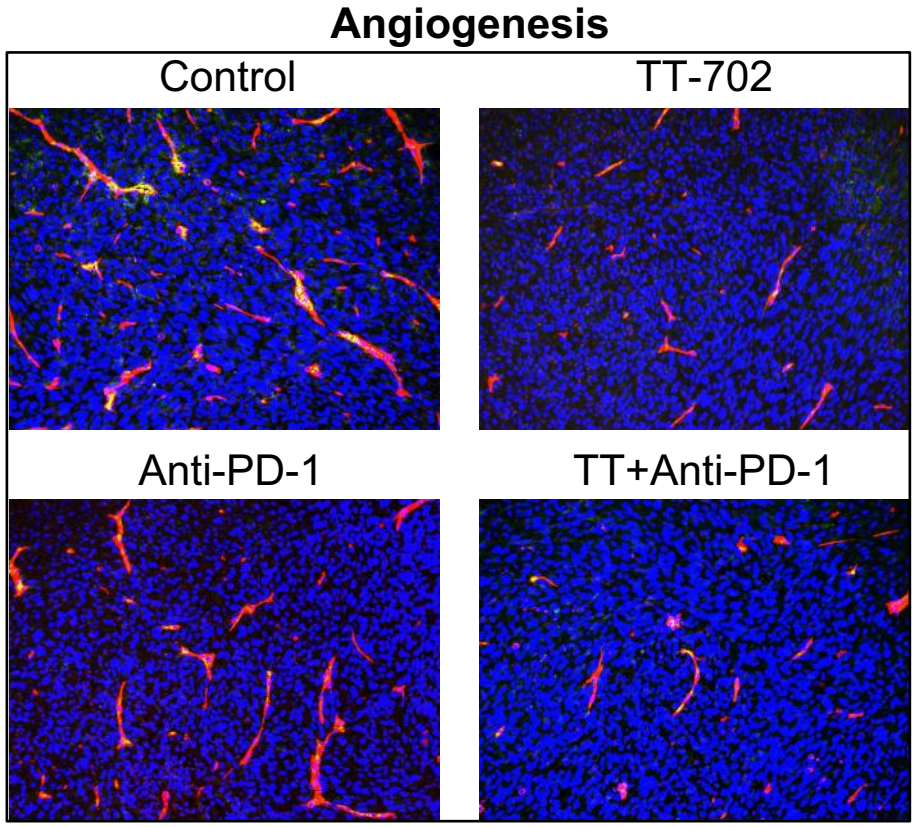
TT-702 Is Superior to Anti-PD-1 and Synergizes Anti-PD-1 Efficacy





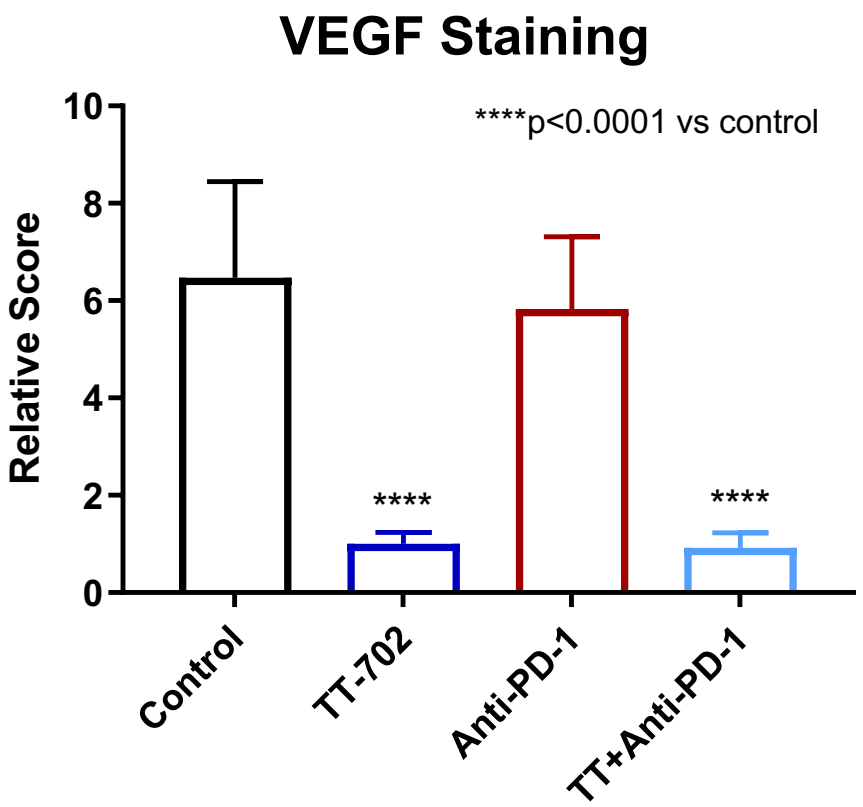
- TT-702 enhances the effect of anti-PD-1 in the hot tumor model
- TT-702 is superior to anti-PD-1 in cold tumor model and combination with anti-PD-1 synergistically inhibits cold tumor growth

TT-702 Inhibits Angiogenesis in MC38 model



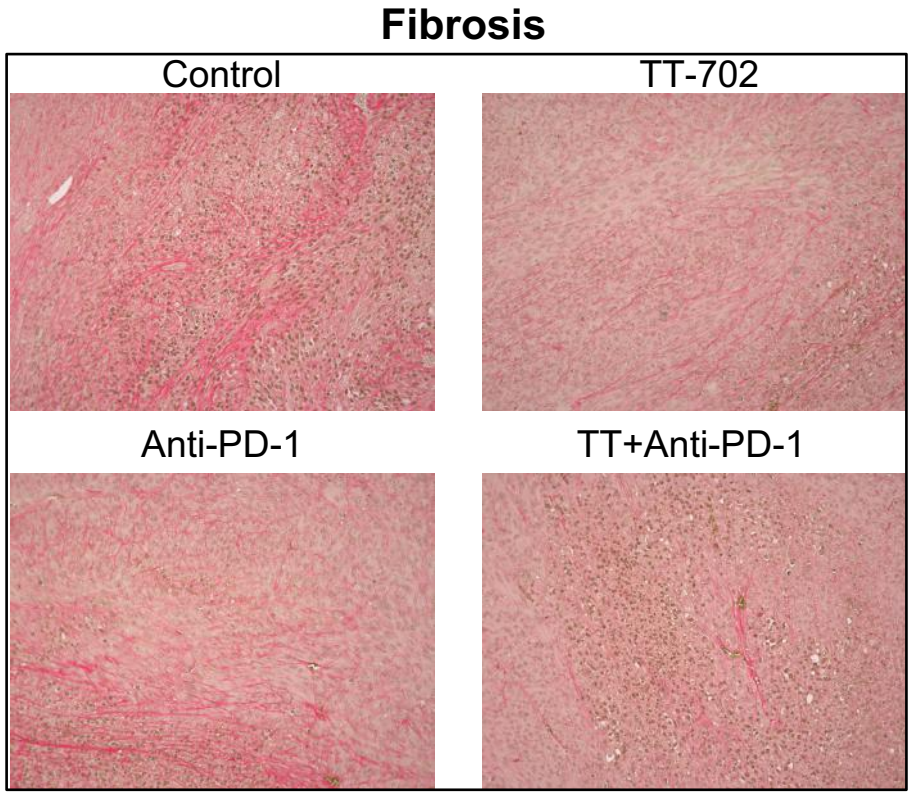
Blue: nucleus, Red: VEGF

↓ Angiogenesis = ↓ metastasis and tumor growth



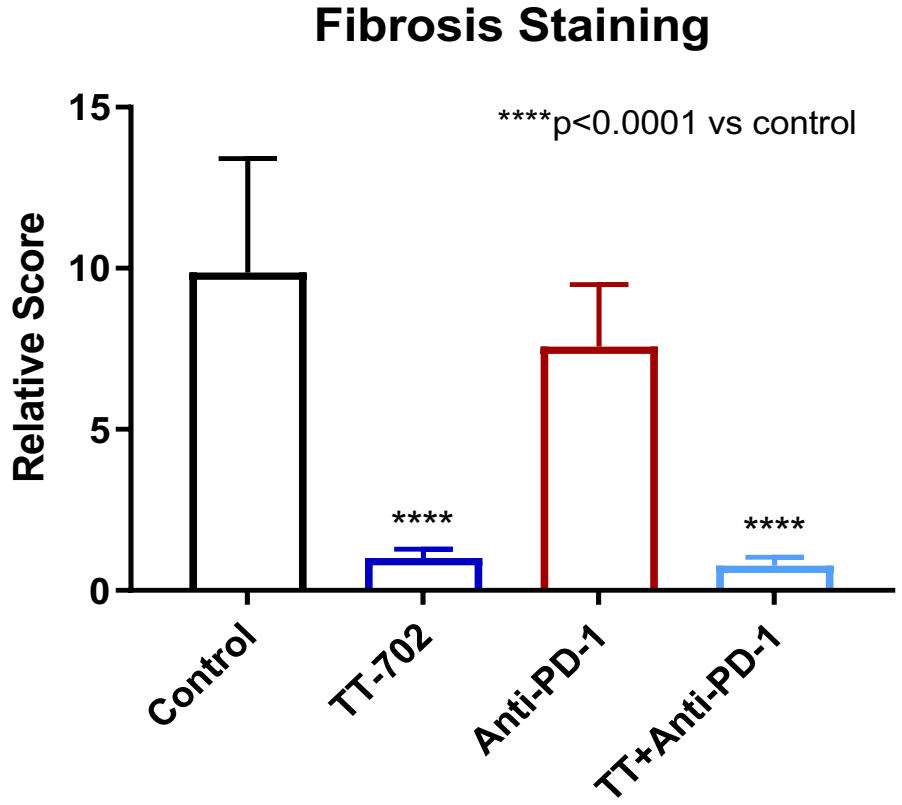
- TT-702 reduces VEGF production which could prevent angiogenesis, metastasis and tumor growth

TT-702 Inhibits Fibrosis in MC38 model



Red: Picrosirius Staining

↓ Fibrosis = ↓ barrier to T cell penetration



- TT-702 reduces fibrosis which could prevent tumor metastasis and enhance response to immunotherapy

References

Slide 3:
1. ZS Secondary Research
2. Haslam, et al. 2020

Slide 9:
1. Galiègue et al., 1995;
2. Kienzl et al., 2020;
3. Basu et al., 2011;
4. Chiurchiu et al., 2016;
5. Chen et al., 2015;
6. Martinez-Martinez et al., 2015;
7. Pérez-Gómez et al., 2015;
8. Tsoukalas et al., 2018;
9. Xu et al., 2019;
10. Fraguas-Sánchez et al., 2018;
11. Śledziński et al., 2018;
12. Bar-Sela et al., 2020;
13. Biednyet al., 2020;
14. Taha et al., 2019

Slide 14:
Graphic, left: Leone RD et al.,, *Cancer Cell*. 2015
1. Gao, Int J Mol Sci. 2019;
2. Mousavi, The prostate 2015;
3. DiRenzo, SITC 2019;
4. Mittal, Cancer Res, 2016;
5. Horigome, Oncotarget. 2018;
6. Zhou, Oncotarget 2017;
7. Wei, Purinergic Signaling 2013;
8. Vecchio, J Pharmacol Exp Ther 2016;
9. Zhou, Oncotarget 2017;
10. Mittal, Cancer Res 2016;
11. KWON, EXPERIMENTAL AND THERAPEUTIC MEDICINE 2019;
12. Kasama, BMC Cancer 2015;
13. Lan, PNAS 2018;
14. Yi, Journal of Cancer 2020;
15. Fernández, The Am J of Path 2013;
16. ORIC Pharmaceutics, AACR 2020;
17. Fredholm, Biochemical Pharmacology 2001;
18. Corvus, AACR 2017;
19. Lim, ASCO 2020;
20. 20. Fong, ASCO 2020