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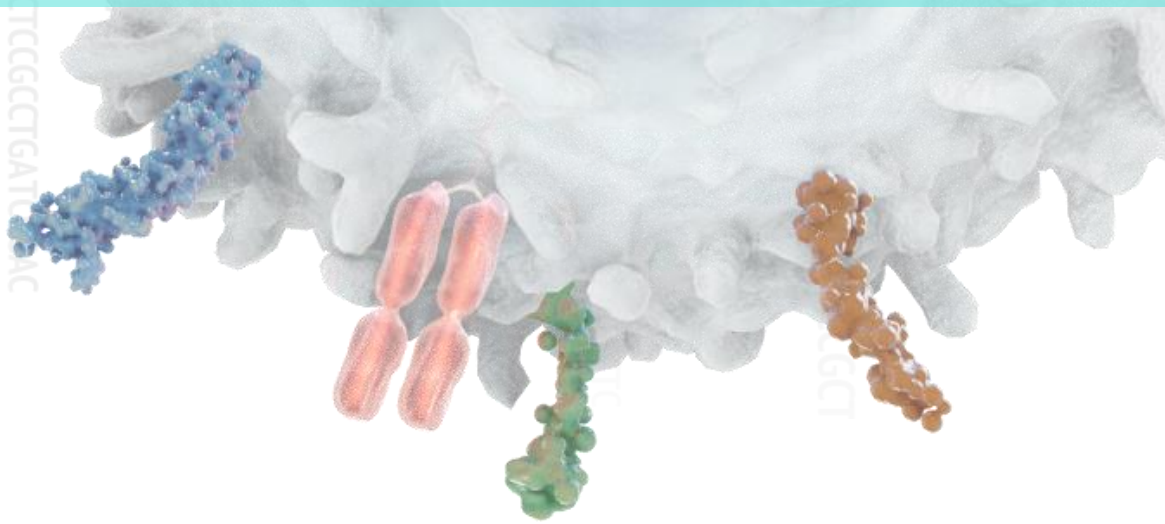

DNAM-1 Axis Virtual Investor Event with Drew Pardoll

23 May 2023

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This presentation contains “forward-looking statements” within the meaning of the Securities Act of 1933 and the Securities Exchange Act of 1934, as amended, and the safe-harbor provisions of the Private Securities Litigation Reform Act of 1995. Forward-looking statements can be identified by the use of terminology such as “will,” “may,” “expects,” “anticipates,” “believes,” “potential,” “plan,” “goal,” “estimate,” “likely,” “should,” and “intends,” and similar expressions that are intended to identify forward-looking statements, although not all forward-looking statements contain these identifying words. These forward-looking statements involve known and unknown risks and uncertainties that may cause the actual results, performance or achievements of Compugen to be materially different from any future results, performance or achievements expressed or implied by such forward-looking statements, including statements regarding the timing and success of our clinical trials, enrollment of patients, type of clinical trials, presentation of data and our cash position and expenditures. Among these risks: The global COVID-19 pandemic may negatively impact the global economy and may also adversely affect Compugen’s business; clinical development involves a lengthy and expensive process, with an uncertain outcome and Compugen may encounter substantial delays or even an inability to begin clinical trials for any specific product, or may not be able to conduct or complete its trials on the timelines it expects; Compugen relies and expects to continue to rely on third parties to conduct its clinical trials and these third parties may not successfully carry out their contractual duties, comply with regulatory requirements or meet expected deadlines, and Compugen may experience significant delays in the conduct of its clinical trials as well as significant increased expenditures; Compugen’s business model is substantially dependent on entering into collaboration agreements with third parties and Compugen may not be successful in generating adequate revenues or commercializing aspects of its business model; Compugen’s approach to the discovery of therapeutic products is based on its proprietary computational target discovery infrastructure, which is unproven clinically; and Compugen does not know whether it will be able to discover and develop additional potential product candidates or products of commercial value. These and other factors, including the ability to finance the Company, are more fully discussed in the "Risk Factors" section of Compugen’s most recent Annual Report on Form 20-F as filed with the Securities and Exchange Commission (“SEC”) as well as other documents that may be subsequently filed by Compugen from time to time with the SEC. In addition, any forward-looking statements represent Compugen’s views only as of the date of this presentation and should not be relied upon as representing its views as of any subsequent date. Compugen does not assume any obligation to update any forward-looking statements unless required by law. Certain studies and data presented herein have been conducted for us by other entities as indicated where relevant. Intellectual property, including patents, copyrights or trade secret displayed in this presentation, whether registered or unregistered, are the intellectual property rights of Compugen. Compugen's name and logo and other Compugen product names, slogans and logos referenced in this presentation are trademarks of Compugen Ltd. and/or its subsidiary, registered in the U.S.A., EU member states and Israel.

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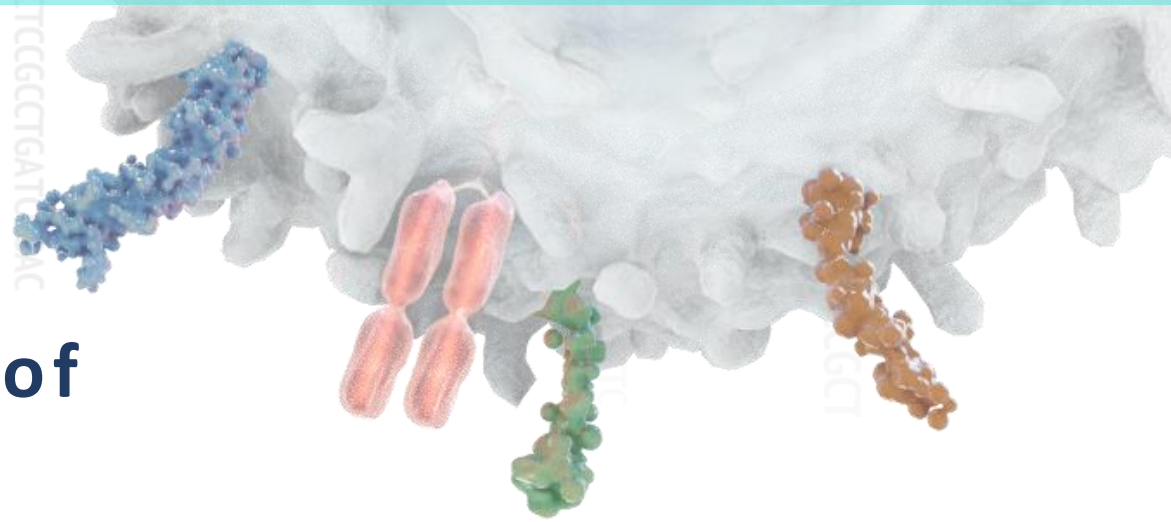

From Code to Cure: Compugen advances the next generation of cancer immunotherapies targeting the DNAM-1 axis

Eran Ophir, Ph.D., SVP Research and Drug Discovery

Focus on three novel antibody therapeutics

Discovered by Compugen's pioneering computational discovery engine

COM701
Potential 1st in-class anti-PVRIG antibody

- Monotherapy activity
- COM701 mediated anti-tumor activity in SOC relapsed or refractory patients typically not responding to immunotherapy

COM902
Potential best-in-class anti-TIGIT antibody

- Favorable safety, tolerability and pharmacokinetics
- Phase 1 Disease Control Rate 50%
- Prevents depletion of $CD8^+$ T cells and potential associated risks

COM503
Novel antibody approach to harness cytokine biology

- Fully human, IgG4 high affinity (<1pM KD) antibody
- Releases IL-18 from pre-formed IL18:IL18-BP complex
- Potential to enhance T and NK cell activation in the TME and not in periphery

Rich pipeline with multiple partnering opportunities

TME: Tumor Microenvironment

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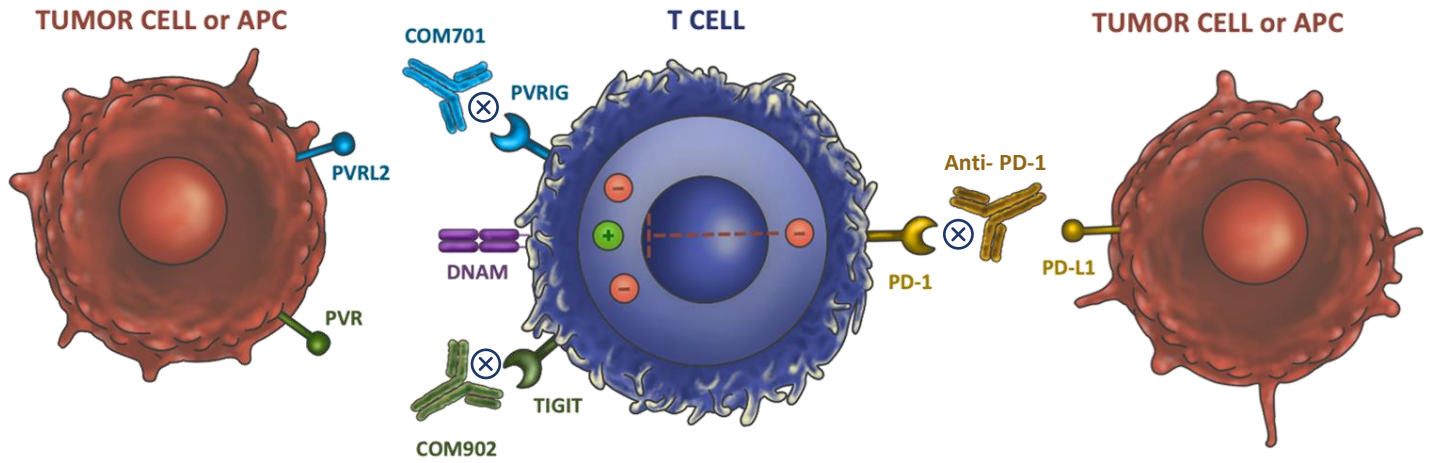
DNAM-1 axis potential game changer in fight against cancer

Blocking PVRIG may be the missing piece when checkpoint inhibitors fail



Support combination approach to overcome immunotherapy resistance

- PVRIG and TIGIT discovered by Compugen's discovery platform
- DNAM axis – two parallel and complementary inhibitory pathways (PVRIG & TIGIT)
- Potential synergy in blocking PVRIG, TIGIT and PD-1 pathways
- Blocking PVRIG is potentially unique in generating new waves of T cells to infiltrate the TME
- PVRL2 broadly expressed in PD-L1 high and low tumors

TME: Tumor microenvironment

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PVRIG and TIGIT – complementary but distinct pathways

PVRIG preferentially binds PVRL2

TIGIT preferentially binds PVR

Differentially expressed in tumor types

PVRL2 expression is more dominant than PVR on certain tumor types, including breast, endometrial and ovarian

Differentially expressed on immune cell types

TIGIT and PVRIG are both expressed on T and NK cells

PVRIG more dominant on Tscm

TIGIT is highly expressed on Tregs relative to PVRIG

Differentially expressed in the tumor microenvironment

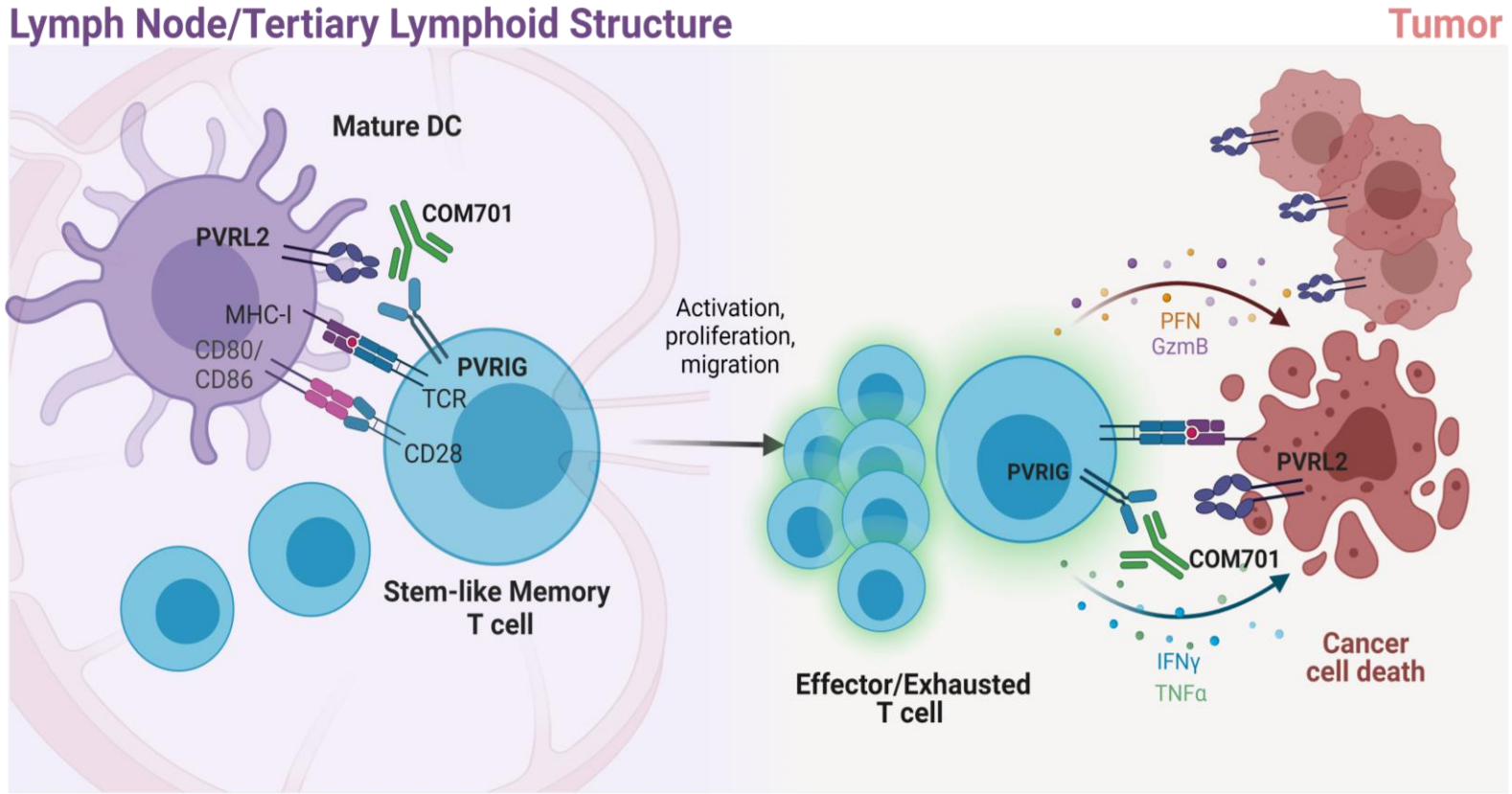
PVRL2 has higher expression on some myeloid lineage cells, **particularly DC subsets**

PVRIG blockade may enhance Tscm activation by DCs in lymph nodes and TLS, potentially leading to T cell expansion and infiltration into cold tumors making them more sensitive to anti-PD-1 and anti-TIGIT

Tscm: Early differentiated stem like memory T cells; DC: Dendritic Cells; TLS: Tertiary Lymphoid Structures

Strong biological rationale for targeting PVRIG in cold tumors

PVRIG+ Tscm interaction with PVRL2 + Dendritic Cells hypothesis



COM701 may sensitize cold tumors to PD-1 and TIGIT

Tscm: Early differentiated stem like memory T cells

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COM701 mediated clinical benefit in relapsed or refractory patients typically not responding to immunotherapy

- Monotherapy and in combination with anti-PD-1 ± anti-TIGIT
- ICI refractory/experienced
- Less inflamed tumors

PATIENTS TYPICALLY NOT RESPONDING TO IMMUNOTHERAPY		TREATMENT	BEST RESPONSE	REFERENCE
Platinum resistant ovarian cancer	3 prior lines, immune desert based on biopsy	COM701	PR >18 months	ASCO 2021
	7 prior lines, refractory to nivolumab	COM701 + nivolumab	PR 4 months	ESMO IO 2022
	6 prior lines		PR >4 months*	
	Median 4 prior lines	COM701 + nivolumab + BMS-986207	PR >9 months*	
			PR >9 months*	
			PR >9 months*	
			PR >4 months*	SITC 2022
MSS CRC with liver metastases	Immune desert based on biopsy	COM701 + nivolumab	PR >11 months	
	Median 4 prior lines		PR >4 months	ESMO IO 2022
ICI experienced anal SCC	Progression on nivolumab		CR >22 months	ASCO 2021
ICI experienced NSCLC	Median 6 prior lines, ≥ 2 prior lines with ICI	COM701 ± nivolumab	SD in 4/6 patients	ESMO IO 2022

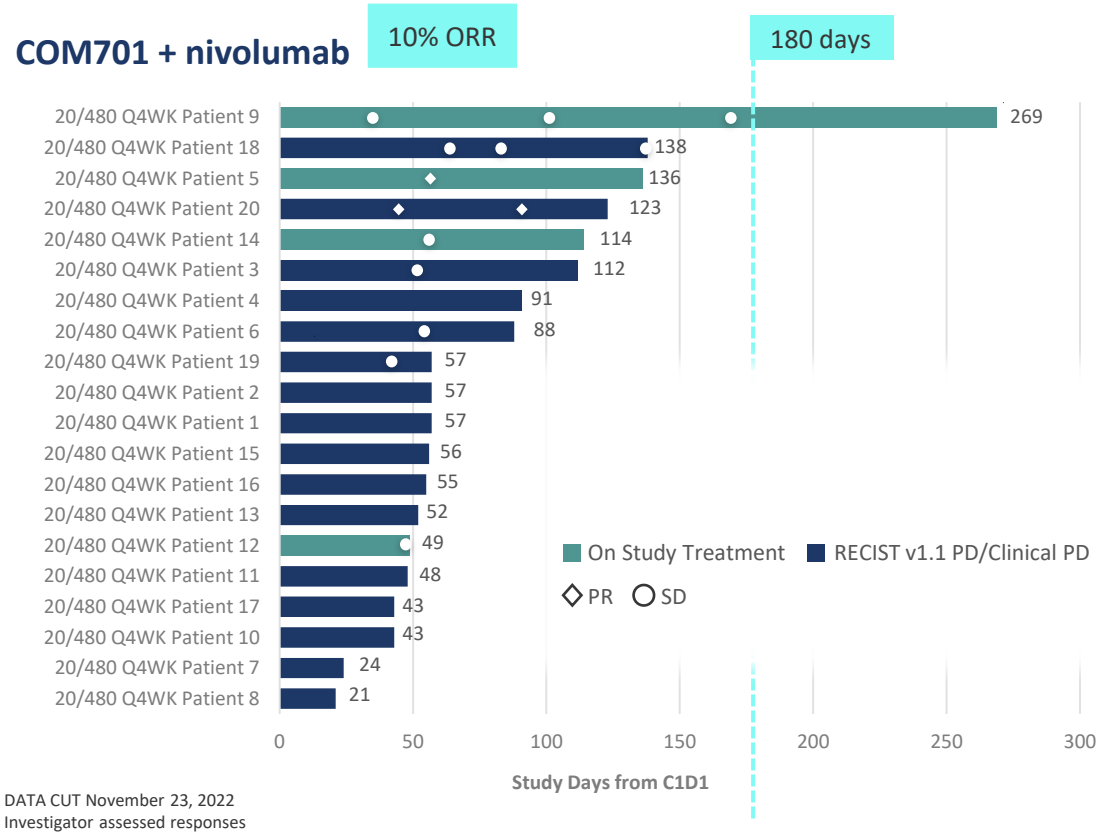
Favorable safety and tolerability profile as monotherapy and in combination

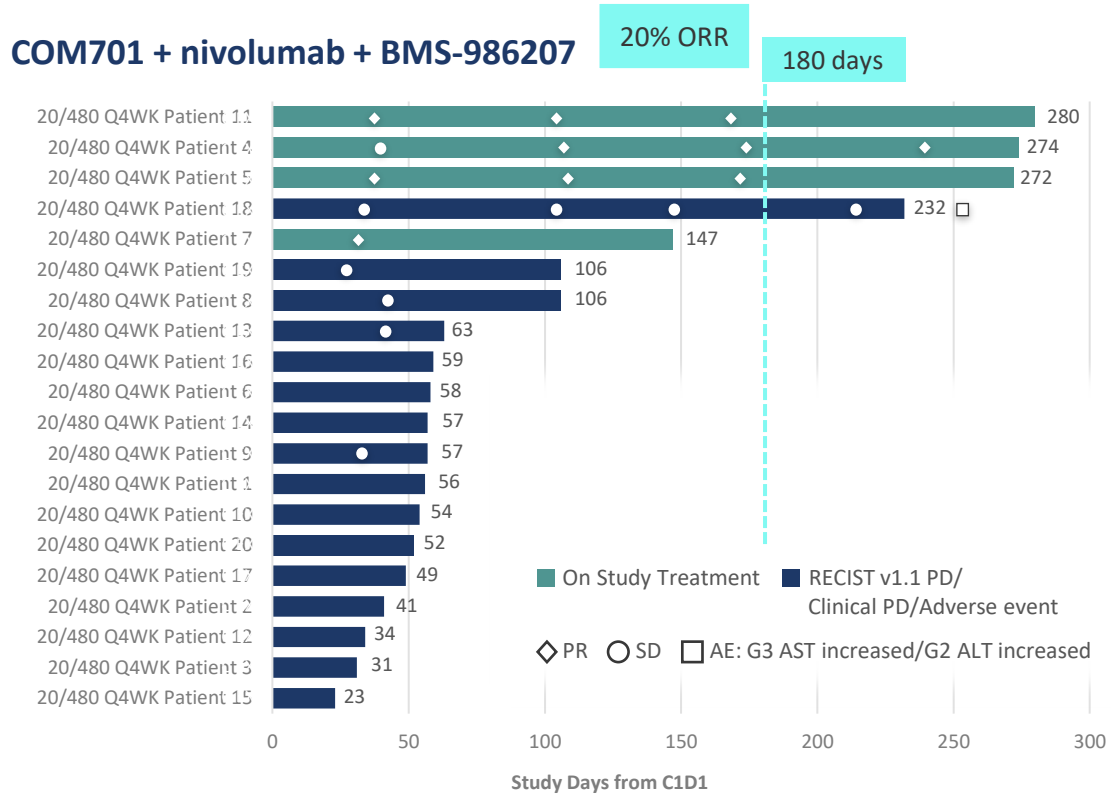
*On study treatment at the time of reporting
BMS-986207 (Bristol Myers Squibb, anti-TIGIT)

PROC patients treated with COM701 + nivolumab ± BMS-986207– swimmer plots

Increase in response rate & duration suggest greater benefit with triplet

Platinum-Resistant Standard of Care: Single agent chemo (ORR ~ 12%, mPFS ~3-4 months, mOS ~ 13 months, with significant toxicity)[1,2]





DATA CUT November 23, 2022
Investigator assessed responses

Other investigational immune check point inhibitors: anti-PD-1 ± anti-TIGIT (ORR <10% in all-comers and 0% in PD-L1 <1)[3,4,5]

Yeku O, et al, ESMO-IO 2022 Modified; Moroney J, et al, ESMO-IO 2022 Modified
1. Pujade-Lauraine et al JCI 2014, 2. Secord et al JCO 2007, 3. Matulonis et al ASCO 2022, 4. Holmes et al JCO ASCO 2022, 5. Perets et al ACCR 2022

Dual:
COM701 20 mg/kg
Nivolumab 480mg Q4W

Triplet:
Patent 7: PR confirmed outside of data cut date
BMS-986207 480mg IV Q4W, COM701 20 mg/kg IV Q4W, nivolumab 480mg IV Q4W
BMS-986207 (Bristol Myer's Squibb, anti-TIGIT).

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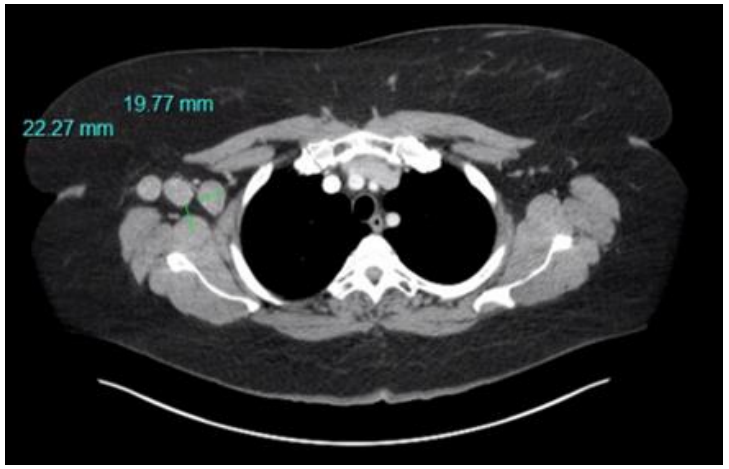
Clinical Vignette – COM701+nivolumab partial response in nivolumab refractory PROC patient

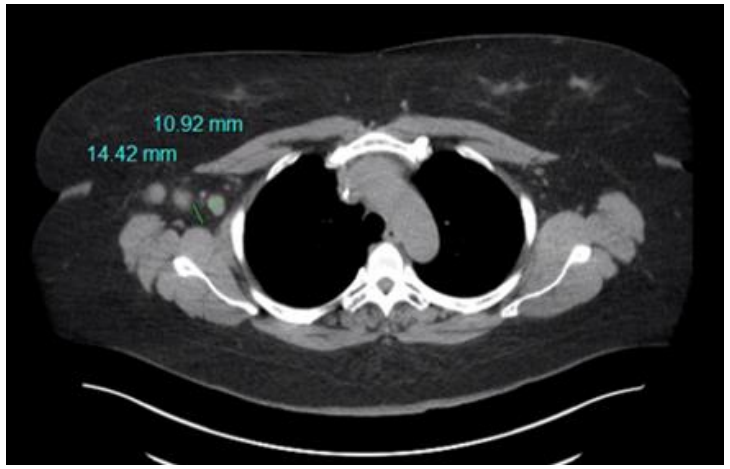
Received 7 prior lines of therapy including progressed disease on nivolumab

53-year-old female with high grade serous adenocarcinoma (HGSC) platinum resistant ovarian cancer

Pre-treatment

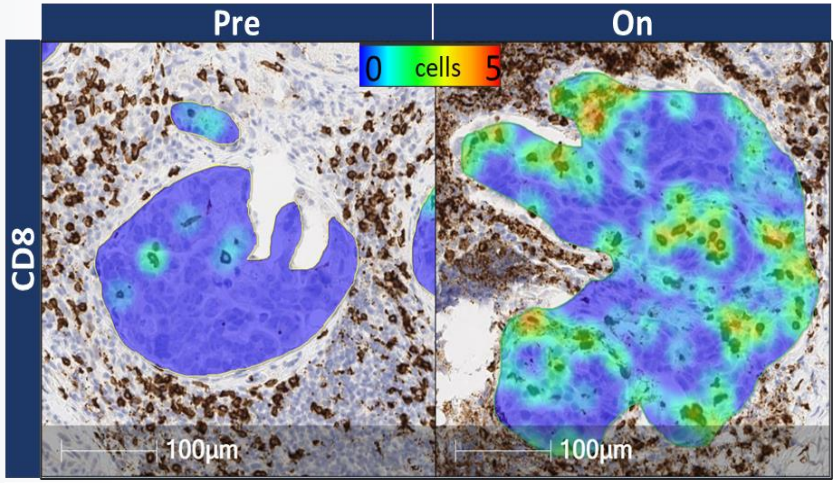


On-treatment



7 prior lines of therapy include chemo, bevacizumab, nivolumab, lucitanib (TKI), niraparib (PARPi)

Increased CD8⁺ T cell infiltration On-treatment



	Pre	On
% CD8 positive in tumor area	15.7%	25.7%
Average CD8 density (CD8/µm²)	8.5 x 10^{-4}	16 x 10^{-4}

Modified from ASCO June 2021 Presentation
Yeku e al, ESMO-IO 2022 Modified

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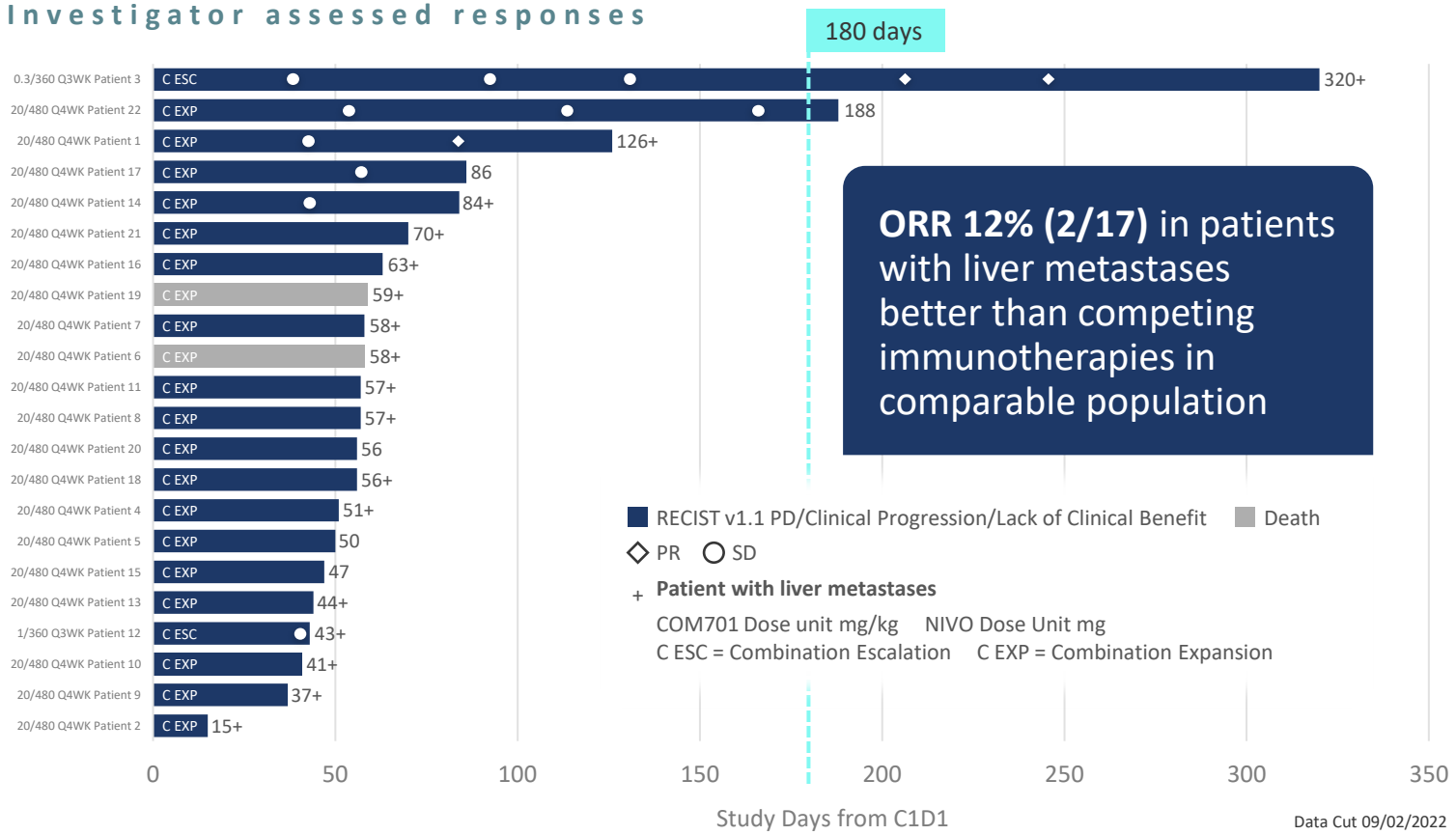
COM701 +nivolumab in MSS CRC cohort expansion Swimmer plot

MSS CRC Standard of Care 3L+: Regorafenib/Lonsurf (ORR ~ 1-2%, MPFS ~2 months, OS ~ 6-7months, with significant toxicity) [1,2]



~70% of patients with 3L+ MSS-CRC patients have metastases to the liver

- Presence of liver metastases correlates with lack of response to PD-1/PD-L1 inhibition 0% ORR [n=54][3]
- Nivolumab + regorafenib **0% ORR** [n=47][4]
- Balstilimab [anti-PD-1] + botensilimab [anti-CTLA-4] **0% ORR** [n=17][5]

Overman M et al, SITC 2022, Modified
1. Van Cutsem,, 2012, ASCO; 2. Mayer et al. N Eng J Med. 2015;372:1909-1919; 3. Wang et al, JAMA 2021, 4. Fakih et al , 2021, ASCO, 5. Bullock AJ et al, 2022 ESMO GI

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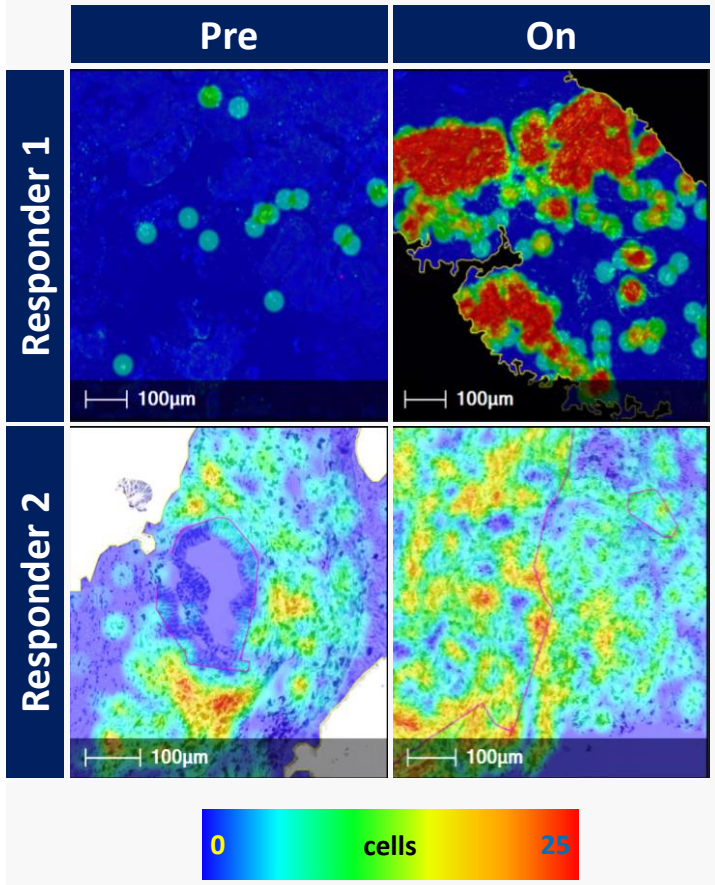
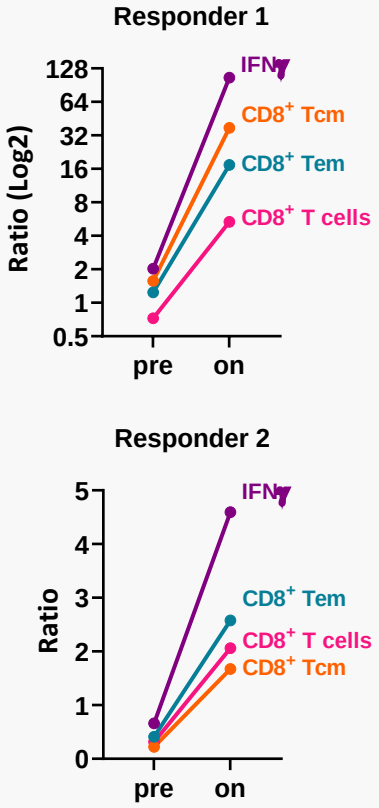
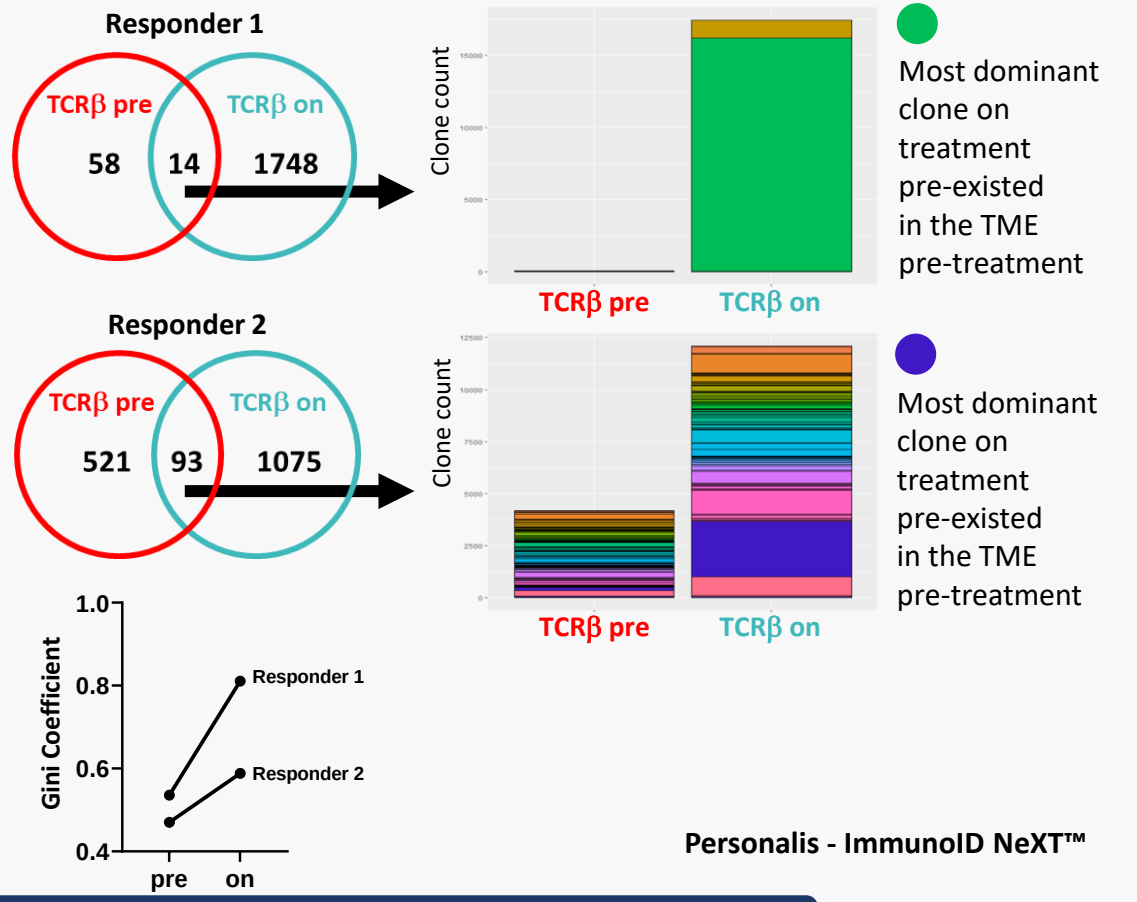
Extensive TME modulation in MSS CRC patients partially responding to COM701+ nivolumab

Increased CD8 infiltration and immune modulation





Increased TCR clonality and clonal expansion



Infiltration of new clones as well as expansion of pre-existing clones in responding patients

Overman M J et al, SITC 2022; Ophir E et al, SITC 2022, Modified

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Compugen's immuno-oncology pipeline

Executing a unique combination approach

PROGRAM (TARGET)	INDICATION	SPONSOR	PHASE	RECENT DEVELOPMENTS & 2023 EXPECTED MILESTONES
COM701+ COM902 + pembrolizumab (PVRIG, TIGIT, PD-1)	Metastatic Microsatellite Stable Colorectal Cancer	compugen	Drug Discovery / IND enabling / 1 / POC / 2	Q1 2023 – First patient dosed Complete enrollment by end of 2023 Report initial findings by end of 2023
COM701+ COM902 + pembrolizumab (PVRIG, TIGIT, PD-1)	Platinum Resistant Ovarian Cancer	compugen	Drug Discovery / IND enabling / 1 / POC / 2	On track to dose first patient Q2 2023 Complete 50% enrollment by end of 2023 Report initial findings by end of 2023
COM503 (IL-18 binding protein)	Solid Tumors		Drug Discovery / IND enabling / 1 / POC / 2	Q2 2023 – Presented pre-clinical data CIMT
Early-Stage Programs	Undisclosed		Drug Discovery / IND enabling / 1 / POC / 2	
rilvegostomig derived from COM902 (PD-1/TIGIT)	ARTEMIDE-01 – Advanced/Metastatic NSCLC	AstraZeneca	Drug Discovery / IND enabling / 1 / POC / 2	AstraZeneca expect to initiate a Phase 3 study in 2023
	TROPION-Lung04 – Advanced/Metastatic NSCLC			
	Locally Advanced or Metastatic Gastric Cancer			
	MAGELLAN – Previously Untreated NSCLC			

Selected programs, not comprehensive

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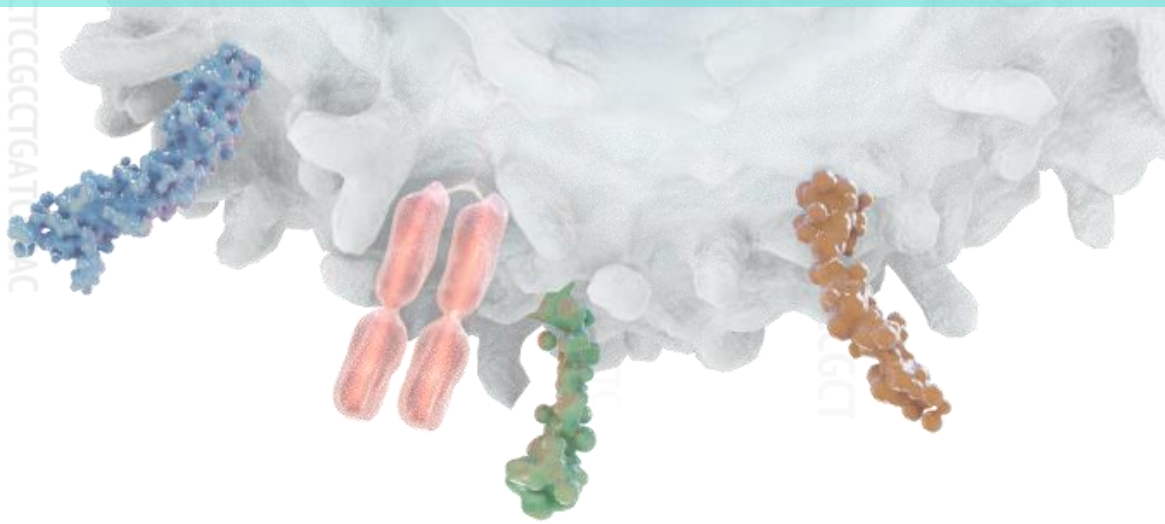


Discussant

Drew Pardoll, M.D., Ph.D. Professor of Oncology, Johns Hopkins University and Chairman of Compugen's Scientific Advisory Board

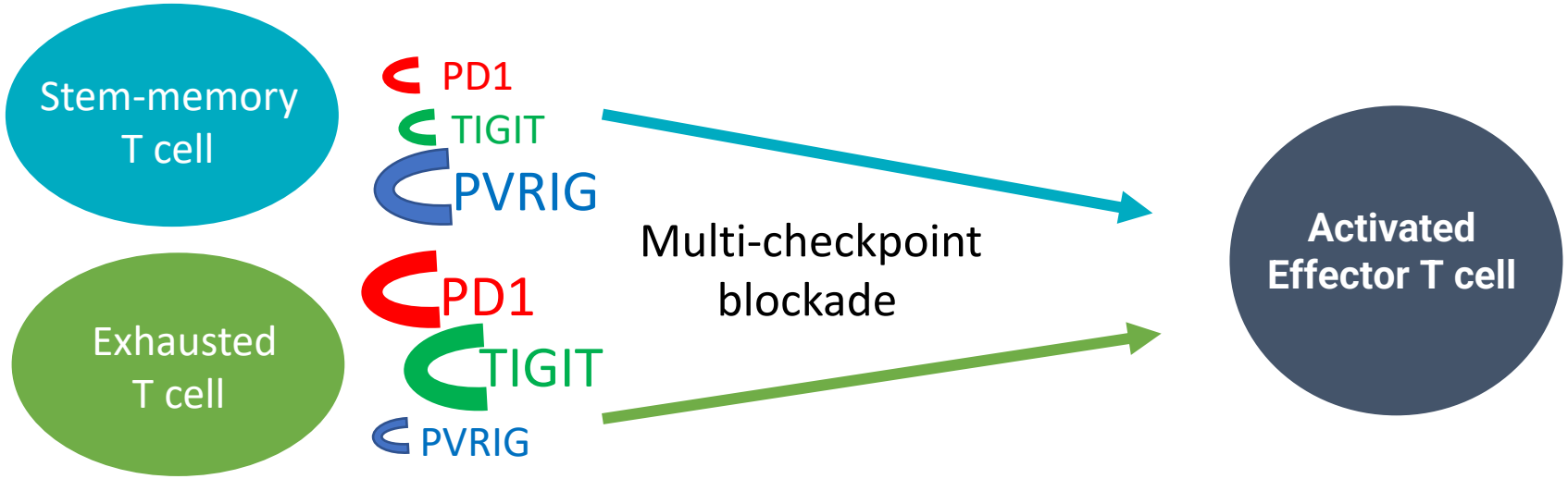
Disclosures

- Research support for PVRIG scientific research
- Compugen SAB chair
- No relevant IP
- No personal equity in Compugen

Why block PVRIG together with TIGIT & PD-1?

- PD-1 blockade foundational
- PVRIG and TIGIT are parallel pathways so blocking only one leaves the other checkpoint intact
- PVRIG and TIGIT are preferentially expressed on two populations of tumor-specific T cell that can give rise to activated effector cells
- Combination KO of PVRIG and TIGIT reproducibly results in additive or synergistic anti-tumor activity in multiple PD-1 non-responsive tumors
- PVRIG likely plays an even larger checkpoint role in humans than in mice: 1) Higher expression in human 2) Human PVRIG has developed a full ITIM 3) Higher expression of PVRIG ligand

2 key Teffector precursor populations are restrained differently by key checkpoints



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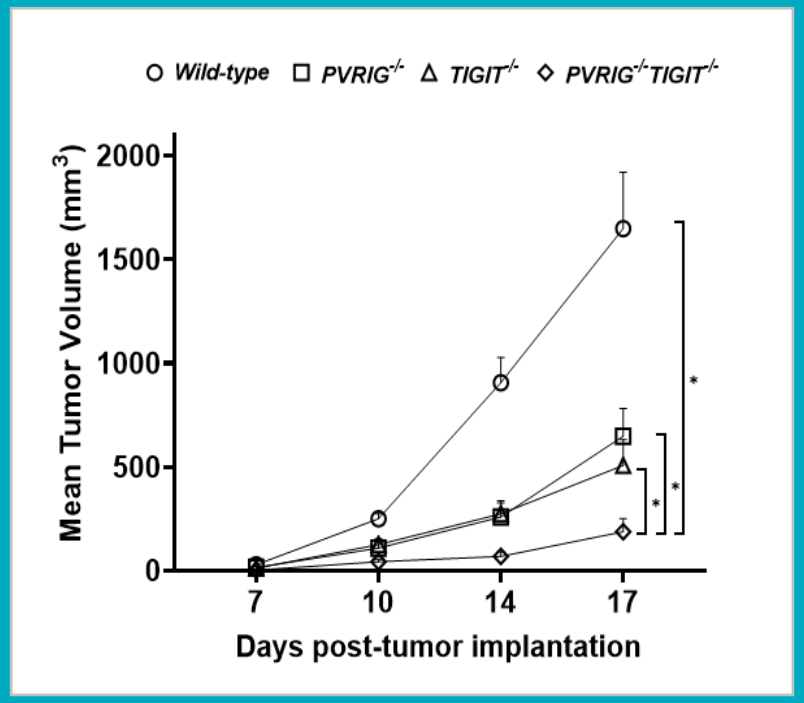
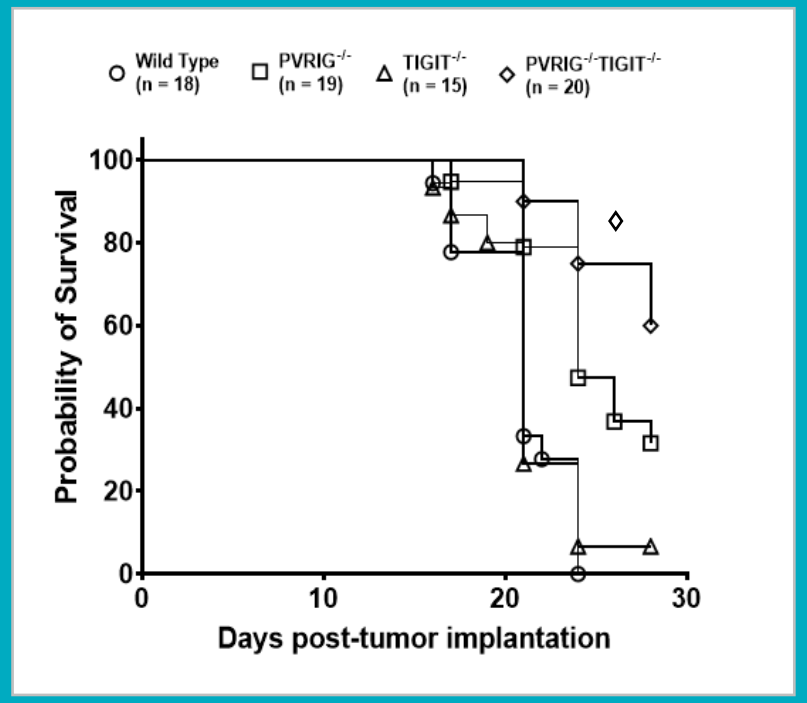
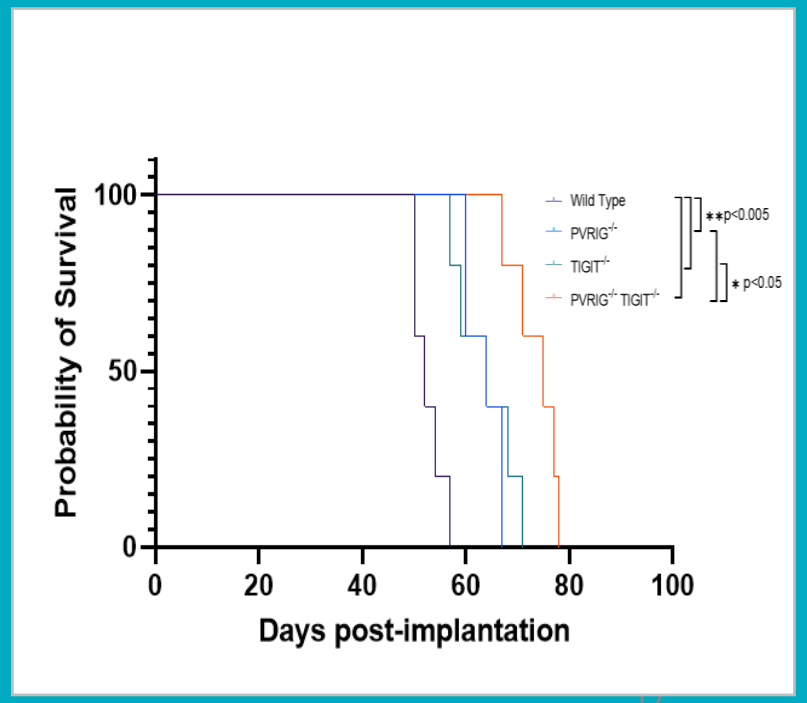




ID8-VEGF



Has TIGIT been a clinical failure?
Answer: We don't yet know

- Roche trial – PFS curves **not** reported and OS has not read out. Furthermore, endpoint not necessarily improvement over atezo alone. Endpoint **not** just a positive trial. They have to beat pembro alone in PD-L1>50% NSCLC
- Failure in pembro/chemo resistant NSCLC does not say anything about first line activity
- Lower response baseline with Arcus anti-PD-1 in NSCLC **does not** negate improvement relative to anti-PD-1 alone - could reflect either that their anti-PD-1 less active than pembro (just like atezo) or patient selection differences with Merck approval trials

Does anti-TIGIT need to be "Fc-active"?
Answer: Unclear but likely NOT

- Conflicting reports in mouse models – Kuchroo/Anderson reported synergy between anti-PD-L1 and Fc-inactive anti-TIGIT
- Anti-tumor activity seen clearly in TIGIT KO mice
- Requirement for Fc-active anti-CTLA4 in mice DOES NOT translate to human
- Fc-active anti-TIGIT may eliminate anti-tumor effector cells
- Scientific explanations for Fc activity are all handwaving

Ovarian Cancer/MSS CRC great opportunities

Ovarian Cancer

- PD-1/TIGIT/PVRIG triple prioritizes potency with 3 excellent mABs
- Ovarian cancer has a decent lymphocyte infiltrate but low response to anti-PD-1 (<10% ORR and 0% in PD-L1^{neg}) so bar is low
- High expression of PVRIG ligand in tumors
- Opportunities for identification of biomarkers for patient selection

MSS CRC

- Some activity with PD-1/PVRIG doublet in a disease with an ORR to PD-1 blockade of <4%
- High unmet medical need so accrual will be fast
- Possibility for on-treatment biopsy
- Opportunities for identification of biomarkers for patient selection