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FROM CODE TO CURE®

Corporate Overview
For investors and analysts



November 2026

Safe Harbor Statement

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: 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.

Our Vision

Transforming patient lives by developing first-in-class therapeutics based on Compugen's AI/ML-powered computational target discovery platform Unigen™

From Code to Cure®



Focus on novel immuno-oncology antibodies

Discovered by Compugen's computational discovery platform-Unigen™

Rich clinical pipeline with validating partnerships >$1bn in milestones PLUS royalties

Fully-owned Clinical Assets

COM701
Potential 1st in-class
Anti-PVRIG antibody

COM902
Fc-reduced anti-TIGIT antibody

Phase 3 AstraZeneca program

Rilvegostomig
AZ est. **>$5bn PYR[1]**
anti-PD1/TIGIT bispecific Ab,
TIGIT component derived
from COM902

AstraZeneca

Phase 1 licensed to Gilead

GS-0321 (previously COM503)[2]
Potential first-in-class
Anti-IL-18BP antibody

GILEAD

Early-stage pipeline

Multiple assets
in research undisclosed

Cash balance $117.5 M
as of September 30, 2026

Expected cash runway into
2029 to support operations

COM701 MAIA-ovarian interim analysis projected by Q1 2027
Phase 3 ARTEMIDE-Biliary01 readout by AZ in H2 2027
Advancement of GS-0321 Ph 1 trial | Advancement early-stage pipeline

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1. PYR: AstraZeneca 's guidance from 2024 Investor Day for non-risk adjusted peak year revenue for rilvegostomig inclusive of all indications and excludes non-registrational trials
2. Compugen responsible for preclinical development and first- in-human Phase 1 trial evaluating the safety and tolerability of GS-0321. Thereafter, Gilead will have sole right to develop and commercialize GS-0321.

Immuno-oncology pipeline focus on novel antibodies

Programs (mechanism of action)	Stage and Indication	Sponsor/ Partner
MAIA-ovarian COM701 maintenance therapy (anti-PVRIG antibody)	**Adaptive platform trial:** relapsed platinum sensitive ovarian cancer, sub-trial 1 randomized vs. placebo	compugen FROM CODE TO CURE®
Rilvegostomig monotherapy and combination trials (anti-PD1/TIGIT antibody) TIGIT component derived from COM902	**12** Phase 3 across multiple indications	AstraZeneca
	Phase 3 readout from ARTEMIDE-Biliary01 anticipated in **H2 2027**	
GS-0321 (anti-IL18BP antibody)	Phase 1: solid tumors	GILEAD
Early-stage pipeline (potential first-in-class drugs/new mechanism of actions)	Undisclosed	

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CT.gov ; AstraZeneca Clinical Trial appendix
GS-0321: Compugen responsible for preclinical development and the first- in-human Phase 1 trial evaluating the safety and tolerability of GS-0321. Thereafter Gilead will have the sole right to develop and commercialize GS-0321.

Rilvegostomig broad AstraZeneca development strategy as IO backbone for future combinations

Rilvegostomig broad Phase 3 development program

Potential reflect >$5Bn PYR target*

	Study name	Indication	Rilvegostomig monotherapy or in combination	Data anticipated
Lung	ARTEMIDE-Lung02	squamous NSCLC 1L PD-L1 ≥1%	rilvegostomig + chemo vs pembro + chemo	>2027
	ARTEMIDE-Lung03	non-sq NSCLC 1L PD-L1 ≥1%	rilvegostomig + chemo vs pembro + chemo	>2027
	ARTEMIDE-Lung04	metastatic NSCLC 1L PD-L1 ≥50%	mono vs pembro	>2027
	TROPION-Lung10	non-sq NSCLC 1L PD-L1 ≥50% w/o actionable genomic alterations	rilvegostomig ± TROP2 ADC (Datroway) vs pembro	>2027
Gastrointestinal	ARTEMIDE-Biliary02	1L advanced BTC	rilvegostomig + chemo vs durvalumab + chemo	>2027
	ARTEMIDE-Biliary01	adjuvant BTC	rilvegostomig + chemo vs chemo	**H2 2027**
	ARTEMIDE-Gastric01	1L gastric cancer HER2+	rilvegostomig + Enhertu + chemo vs rilvegostomig + Herceptin + chemo vs pembro + Herceptin + chemo	>2027
	ARTEMIDE-HCC01	1L HCC	rilvegostomig + anti-VEGF (Avastin) ± anti-CTLA4 (Imjudo) vs Avastin + atezo	>2027
	DESTINY-BTC01	1L HER2+ BTC	Enhertu ± rilvegostomig vs chemo + durvalumab	>2027
	CLARITY-Gastric 02	1L CLDN18.2+ HER2-, gastric & gastroesophageal junction	Sonesitatug vedotin + rilvegostomig/Nivo + chemo vs Nivo + chemo	>2027
Other	DESTINY- Endometrial01	stg III/IV/recurrent endometrial cancer	rilvegostomig + Enhertu vs pembro + Enhertu vs pembro + chemo	>2027
	TROPION-Urothelial04	high-risk muscle invasive urothelial carcinoma	rilvegostomig + Datroway vs Datroway vs nivo or durva or pembro + EV	>2027

12 phase 3 trials across multiple indications

Trials announced by AstraZeneca- CT.gov; AstraZeneca Clinical Trial Appendix., not comprehensive, does not include investigator-initiated studies

*PYR: AstraZeneca 's guidance from 2024 Investor Day for non-risk adjusted peak year revenue for rilvegostomig inclusive of all indications and excludes non-registrational trials

Rilvegostomig: Significant value creation potential

Rilvegostomig
AZ est. **>$5bn PYR[1]**
PD-1/TIGIT bispecific Ab

- **AZ dual checkpoint inhibitor bispecific:** to replace anti-PD-(L)1's and be the backbone for future combinations
- **AZ 12 pivotal trials**: novel combinations across NSCLC, GI, Endometrial, Urothelial Cancers
- **Promising Phase 1/2 data presented**: ARTEMIDE-01, GEMINI Gastric, TROPION-Lung04, GEMINI-Hepatobiliary TROPION-PanTumor 03, Destiny-Gastric 03

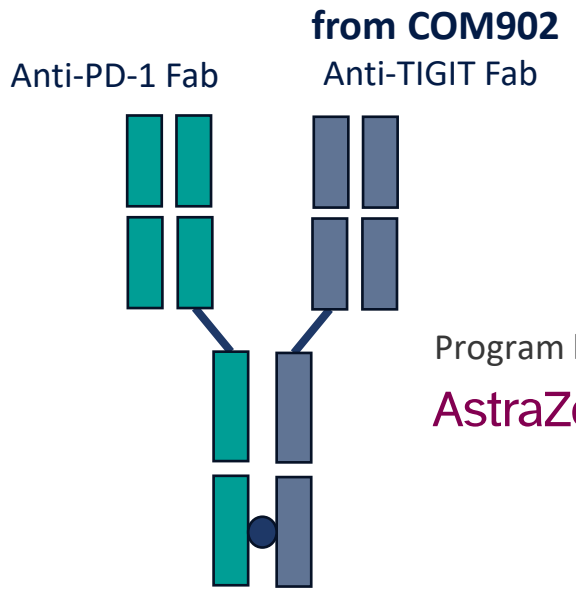
Compugen eligible to receive up to **$195m** in additional milestone payments & tiered royalties up to mid-single digit

- **$105M received** (upfront payments, milestones, monetization of small royalty portion)
- AstraZeneca has rights to develop TIGIT bispecifics
- Compugen retains rights to PVRIG (PVRL2)/TIGIT bispecifics

1. PYR: AstraZeneca 's guidance from 2024 Investor Day for non-risk adjusted peak year revenue for rilvegostomig inclusive of all indications and excludes non-registrational trials

Not all anti-TIGIT antibodies are created equally

Antibody format matters



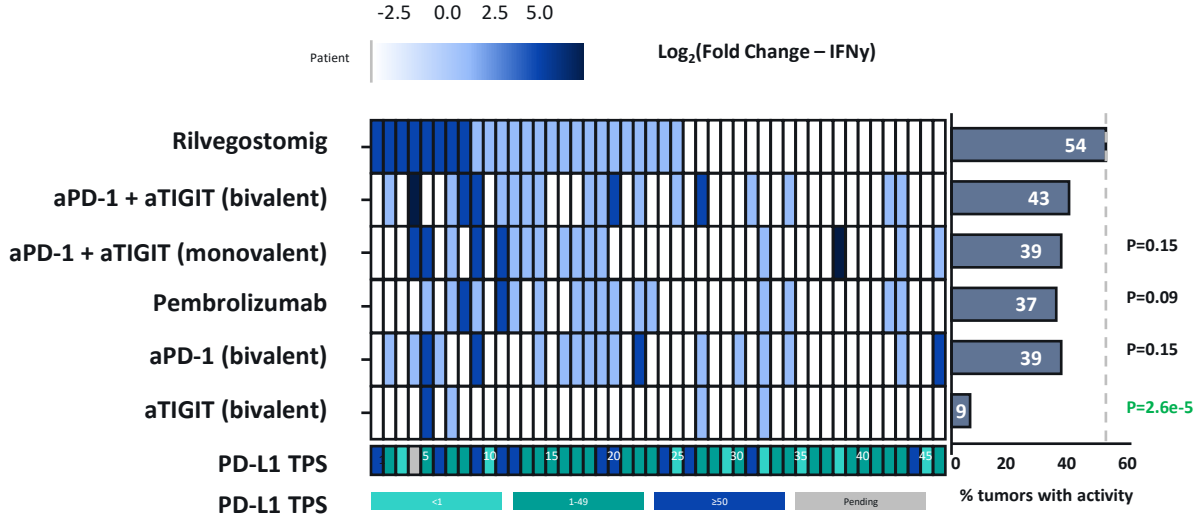
Program by: AstraZeneca

(PD-1/TIGIT)

Rilvegostomig (IgG1-mut Fc reduced)

Rilvegostomig*, an Fc-reduced, monovalent, bispecific humanized IgG1 monoclonal antibody targeting PD-1 and TIGIT. The anti-TIGIT component of rilvegostomig is derived from **COM902 developed by Compugen**

- **Bispecific approach differentiated:** coordinated inhibition of PD-1/TIGIT induces greater ex vivo activity than its components[1]



- **Fc attenuated** triple -mutant IgG1 potentially maintaining and enhancing immune responses, and **conferring improved safety** [1]
- **Fc-reduced functionality important for efficacy** - unmodified or enhanced Fc-functionality appears to be detrimental to rilvegostomig anti-tumor activity in NSCLC explants[1]

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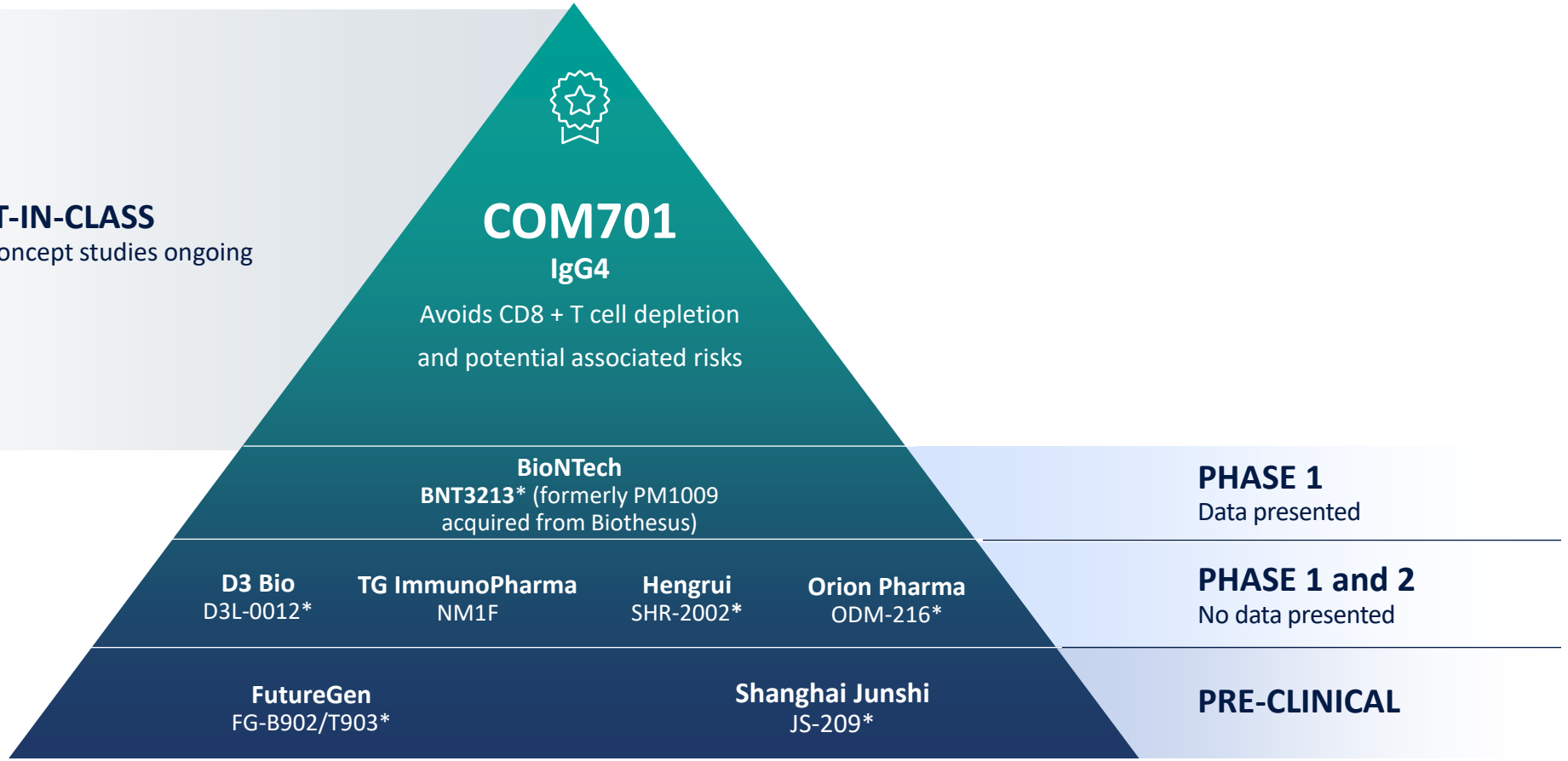
*Compugen is eligible for milestone and up to mid single digit tiered royalty payments

1. https://www.astrazeneca.com/content/dam/az/Investor_Relations/events/AZN-2025-ESMO-brochure.pdf

COM701 - potential first-in-class anti-PVRIG antibody

COM701 potential 1st in class anti-PVRIG antibody

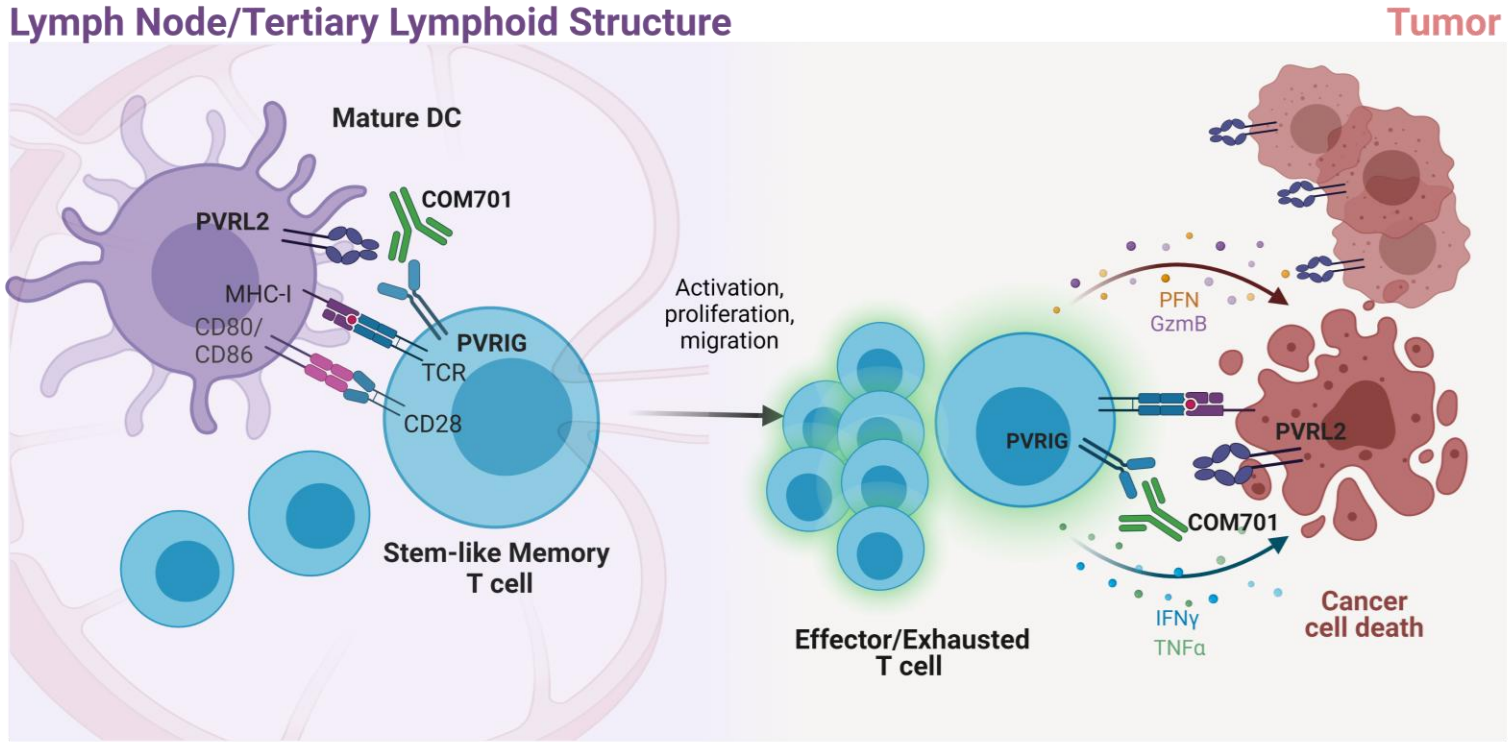
COM701 POTENTIAL FIRST-IN-CLASS
Phase 1 data presented; proof-of-concept studies ongoing



*Bi-specifics
The preclinical list of players is not exhaustive

Strong biological rationale to target PVRIG in ovarian cancer

- High **PVRIG pathway** expression levels in **ovarian cancer**
- **PVRIG pathway** differentially expressed on early differentiated stem like memory T cells and dendritic cells compared to other immune checkpoints
- PVRIG blockade with **COM701** alters the tumor microenvironment including in less inflamed tumors **like ovarian cancer**



Alteber et al, Cancer Immunology Research, 2024

COM701 showed clinical benefit in patients with hard-to-treat platinum resistant ovarian cancer

Typically, not responding to immunotherapy[1-3]

Patient characteristics

- Platinum resistant ovarian cancer
- Heavily pretreated
- Some patients failed ADCs
- Across PD-L1 tumor expression level

Encouraging durable responses as monotherapy and in combination

Single agent COM701 activity[4-6]
PR >18 months in immune desert patient
Immune modulation of the TME

Triple combination blocking PVRIG, TIGIT, PD-1[7-9]
ORR 17-20%* DCR 45-46% (n=44)
mPFS in patients with CB 10.5 months
Responses across PD-L1 levels, supportive of COM701 mediated effect

Safety and tolerability profile in combination consistent with anti-PD-(L)1s[7-8]

- Most common adverse events grade 1/2 fatigue, diarrhea, nausea
- Grade ≥3 treatment related adverse events 20%
- No grade 4/5 treatment related adverse events
- Treatment related discontinuations 4.4%

1. Matulonis et al, ASCO 2022,
2. Holmes et al, JCO ASCO 2022,
3. Perets et al, AACR 2022
4. Ophir E et al, SITC 2022
5. Alteber E et al, Cancer Immunology Research 2024
6. Vaena et al, ASCO 2021
7. Gaillard S et al, SITC 2023
8. Yeku et al, SITC 2024
9. Yeku et al, ESMO 2025

PR: Partial Response; CR: Complete Response; ORR: Overall Response Rate; DCR: Disease Control Rate;

ADCs: Antibody Drug Conjugates
TME: Tumor Microenvironment
CB: Clinical Benefit
*anti-PD-1 ± anti-TIGIT (ORR <10% all-comers, 0% PD-L1 <1)[1-3]

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Strong clinical and biological rationale to advance COM701 development to platinum sensitive ovarian cancer

- **High PVRIG pathway expression** levels in ovarian cancer and PVRIG unique biology differentiated from other checkpoints supporting activity in less inflamed tumors[1]
- **COM701** showed **clinical benefit** in hard-to-treat platinum resistant ovarian cancer patients including PD-L1 low expressing tumors[1-6]
- Platinum sensitive ovarian cancer patients are less heavily pre-treated, less immune compromised[7-8]
- Platinum based chemotherapy may reduce disease burden and sensitize tumors **to COM701's unique mechanism of action**[9]
- COM701 is **well tolerated and associated with durable responses** especially important in a maintenance setting[1-6]

Platinum sensitive ovarian cancer represents a less competitive landscape than platinum resistant ovarian cancer

1. Alteber et al, Cancer Immunology Research, 2024
2. Vaena et al, ASCO 2021
3. Ophir E et al, SITC 2022
4. Gaillard S et al, SITC 2023
5. Yeku et al, SITC 2024
6. Yeku et al, ESMO 2025
7. Hanker LC, et al, Ann Oncol. 2012;23(10):2605-12.
8. González-Martín et al, Ann Oncol . 2023
9. Lanickova et al, Clin Cancer Res; 2025 Oct;34(10):833-848

MAIA-ovarian: adaptive trial with maintenance COM701 therapy in relapsed platinum sensitive ovarian cancer

Key eligibility criteria

Relapsed platinum sensitive ovarian cancer

Complete or partial response following platinum-based chemotherapy

Maintenance therapy post bevacizumab and/or PARP inhibitor or not a candidate for bevacizumab and/or PARP inhibitor

Presence of liver metastases excluded

Sub-trial 1
Single agent activity of COM701
versus placebo (standard of care)

Adaptive trial n= 60
2:1 Randomization

Sub-trial 2 and beyond
COM701 combination studies e.g.
anti-PD-1/TIGIT or bevacizumab or PARPi or others

Endpoints

Primary: Efficacy- median progression free survival

Secondary: Safety

Exploratory: Biomarkers

Timelines: sub-trial 1

Trial initiation: Q2 2025

First patient dosed: July 2025

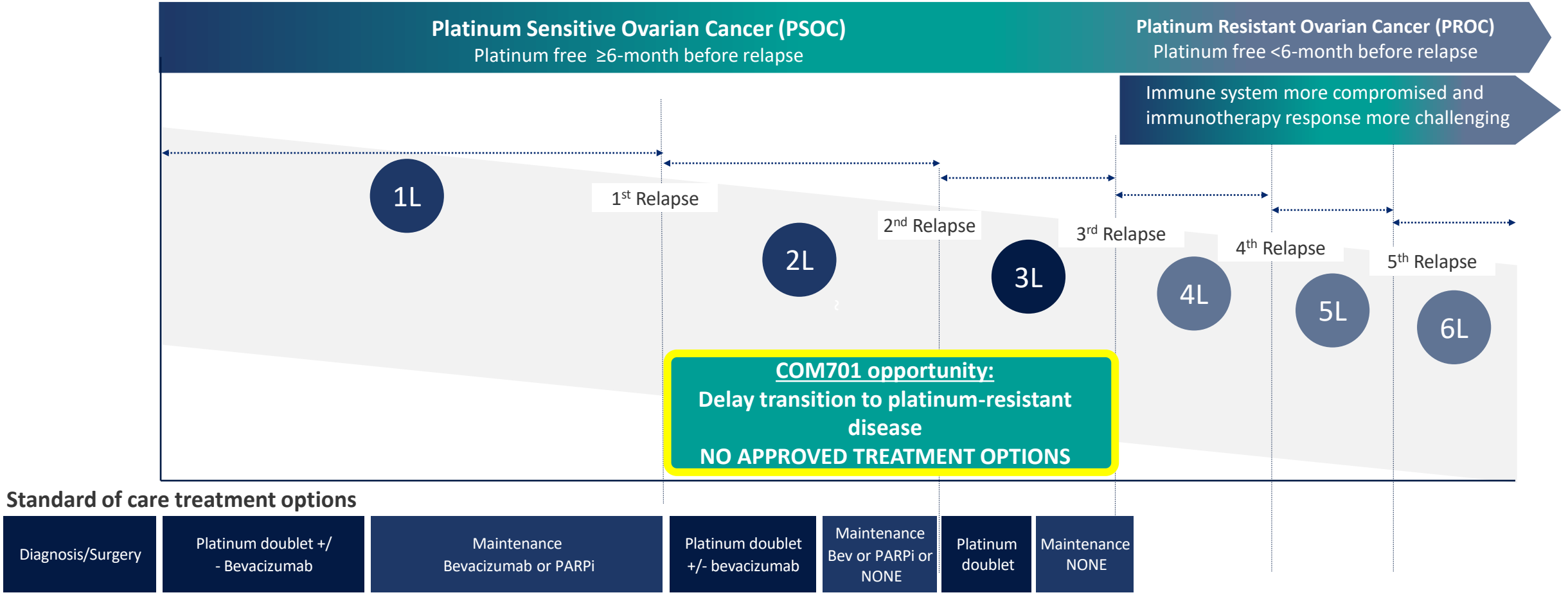
Projected Interim analysis: Q1 2027

Evaluation of COM701 single agent activity, combinations and contribution of effects, represents a regulatory and commercial opportunity

https://clinicaltrials.gov/study/NCT06888921?cond=COM701&rank=1

Advancing COM701 to address unmet need in PSOC

Building on data generated in PROC



Potential benefits of COM701:

- ✓ Longer time before progression
- ✓ Recovery break from chemotherapy
- ✓ Delay transition to PROC
- ✓ Preserve additional treatment options

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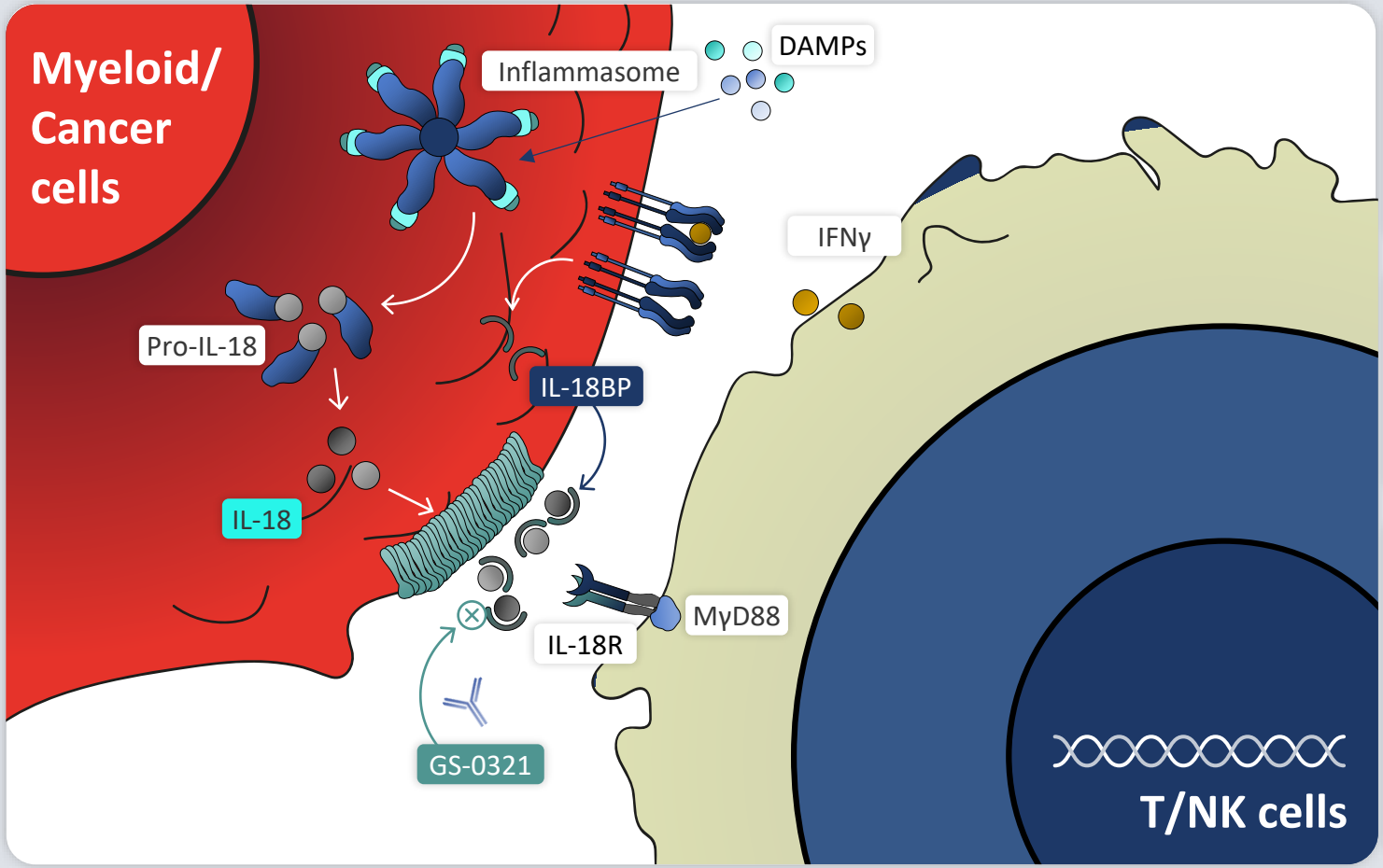
GS-0321 (previously COM503) – potential first-in-class anti-IL18BP antibody

GS-0321: Compugen identified potential dominant immunosuppression mechanism and antibody therapeutic

Interleukin-18 binding protein, an endogenous inhibitor of interleukin-18



IL-18 immune stimulatory cytokine upregulated in tumor microenvironment

IL-18BP blocks IL-18 activity

GS-0321 potential first-in-class high affinity antibody releases IL-18 to enhance T and NK cell activation in the tumor

DAMPs- Damage-associated molecular patterns . GS-0321 is licensed to Gilead, Compugen responsible for ongoing preclinical and future Phase 1 development, Thereafter, Gilead will have sole right to develop and commercialize GS-0321

GS-0321 differentiated approach to harness cytokine biology

License agreement with GILEAD Creating Possible

Key Benefits

- Highlights potential of GS-0321 **differentiated** approach to harness cytokine biology to treat cancer
- Reflects **quality of our computational** capabilities & ability to **advance** discoveries into drugs candidates
- **Strengthens our balance sheet**
- **Advances our vision** to bring potential 1st in class therapies to patients

up to $848M deal value

Signed December 2023

$60 million upfront and **$30 million** milestone payment on **achieving IND clearance, received***

Up to **additional $758M** in additional development, regulatory and commercialization future milestone payment

Single-digit to low double-digit tiered royalties on WW future net sales

Compugen responsible for preclinical and ongoing **Ph** 1 trial **initiated** in Q4 2024. Thereafter, Gilead will have sole right to develop and commercialize GS-0321

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GILEAD Creating Possible

* less 15% tax withheld at source

GS-0321: advantages of a differentiated approach

	GS-0321 anti-IL-18 binding protein	Recombinant cytokines
Novelty	Novel antibody approach	Multiple challenges
	Anti-IL-18BP releases endogenous IL-18 in the TME	Systemic administration of a recombinant protein
Pharmacokinetics	✓ Slow elimination	✗ Requires modification/engineering to overcome pharmacokinetic limitations
Immunogenicity	✓ Potentially low risk, human IgG antibody	✗ Modified/engineered recombinant cytokine, increases risk
Therapeutic window	✓ Immune modulation selectively targets TME, potential for better tolerance	✗ Systemic immune modulation, potential for unmanageable side effects

GS-0321 first-in-human global Phase 1 trial in patients with advanced solid tumors

Key eligibility criteria

Advanced recurrent or metastatic solid tumor malignancy

First in human dose escalation dose expansion trial

Part 1: dose escalation
- GS-0321 monotherapy and in combination with zimberelimab* (anti-PD-1)

Part 2: dose expansion
- GS-0321 monotherapy and in combination with zimberelimab*

Endpoints

Primary: safety and identify recommended dose for expansion

Secondary: characterize GS-0321 pharmacology

Exploratory: preliminary anti-tumor activity

Trial initiated Q4 2024

First patient dosed January 2025

Trial in progress poster presented at SITC 2025

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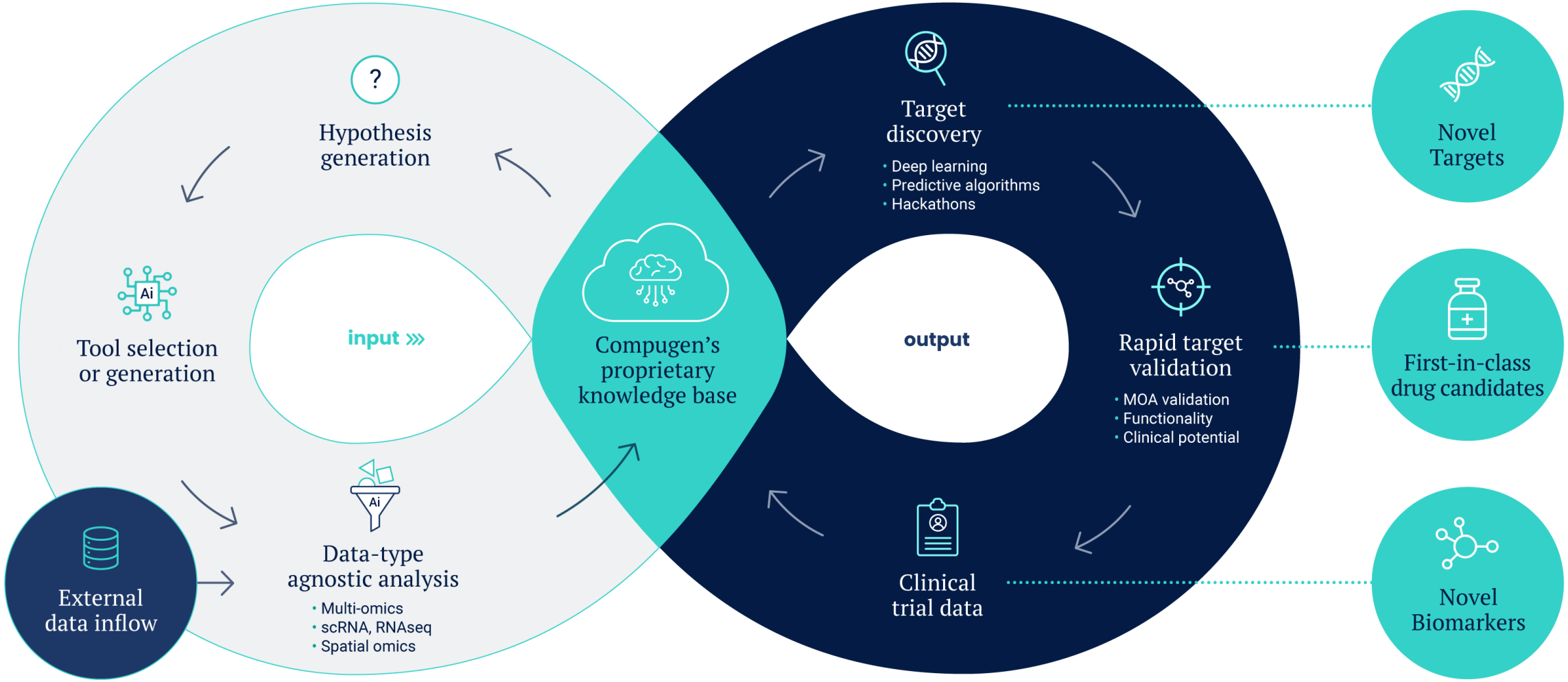
GILEAD Creating Possible

Clinicaltrials.gov identifier NCT06759649
*zimberelimab, Gilead's anti-PD-1

Differentiated by integrating pioneering computational capabilities with immuno-oncology drug development

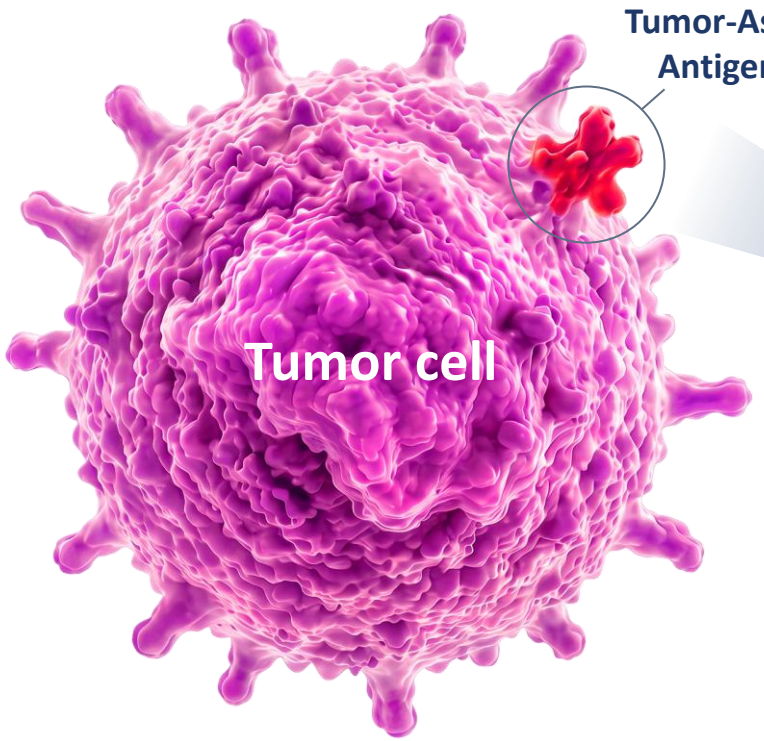
unigen™ by compugen

Flexible-loop AI/ML-powered validated computational platform for target discovery & development



Expanding Compugen's target discovery into tumor-associated antigens (TAAs)

TAAs provide the tumor address for targeted therapies, directing immune activity or drug delivery toward cancer cells





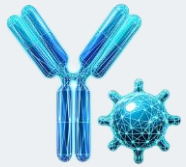
T-cell Engagers (TCEs)

Bring T cells into direct contact with tumor cells, triggering anti-tumor activity.



Targeted Immune Activation

Engage other immune cells or activate immune pathways (e.g., cytokines, co-stimulation) through tumor-selective targets.



Additional Targeted Modalities

TAA-directed therapies beyond immune engagers, including antibody-drug conjugates (ADCs).

Large unmet need in targeted therapies- novel selective TAAs

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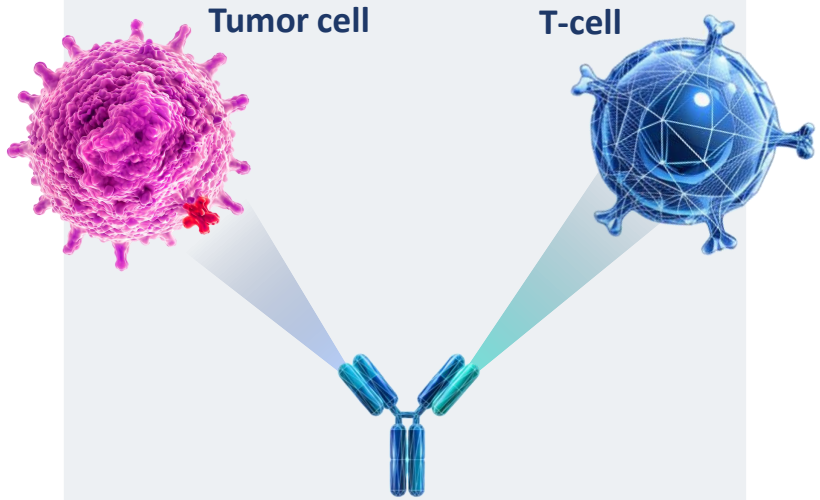
T-cell Engagers create an immediate need for the right tumor targets

A Powerful Modality

TCEs trigger potent anti-tumor activity, with growing potential in solid tumors

TAAs Direct the Activity

TCEs rely on TAAs to direct T-cell activity toward tumor cells, making target selection critical to efficacy and safety



T-cell Engager (TCE)

Growing Market Opportunity

>$18–20B

projected market by 2035*

Few TAAs Have the Right Profile

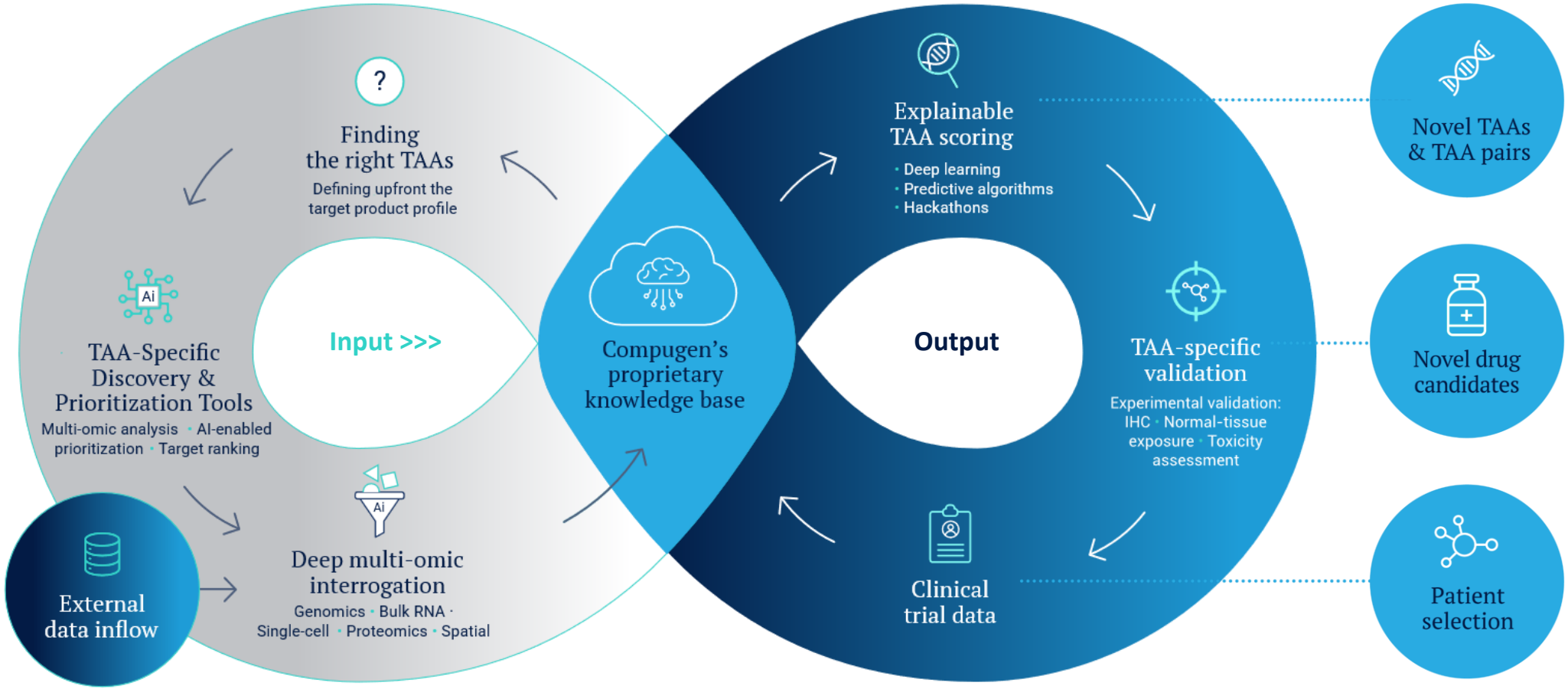
The right TAAs combine tumor specificity, low normal-tissue exposure, and high tumor cell coverage

*Source: Custommarketinsights report T-cell engager market size

AI-Powered Discovery Engine, Built for TAAs

unigenX™ by compugen

Compugen's validated AI-powered discovery engine, continuously connecting computational prediction, biological validation and clinical learning



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Unique multi-omics approach

unigenX™ by compugen

Search Broadly

A rich biological landscape for discovery

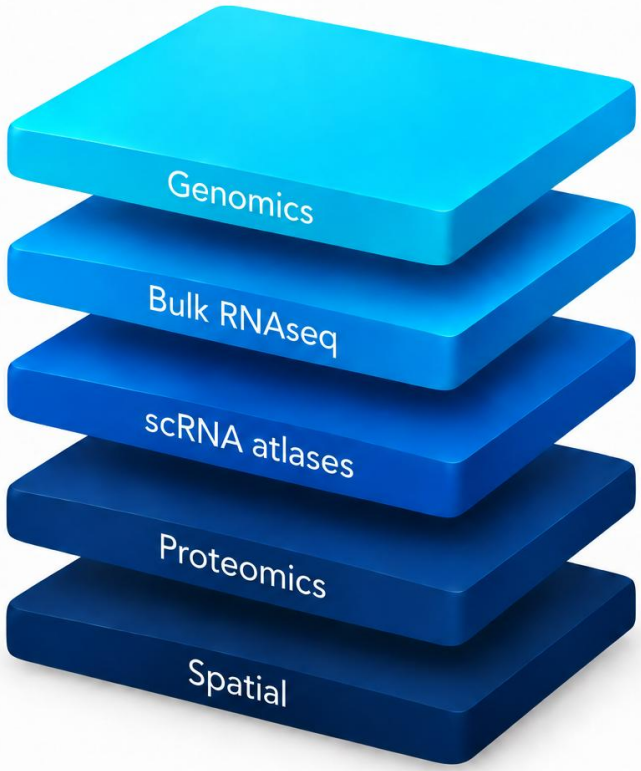
> 100M
Single cells

> 50K
Samples across >1000 IO datasets

>3,700
Tumor & normal proteomic samples

21
Cancer subtypes

42
Normal-tissue scRNA atlas spanning >150 cell types



Understand Deeply

A multidimensional target profile

- Tumor specificity
- Patient population coverage
- Intratumoral heterogeneity
- Safety – Normal tissue exposure
- Therapeutic index

Versatile discovery platform

Adapting AI-driven discovery and analysis to each biological challenge

Novel target discovery

Uncovering novel targets with the goal of translating them into clinical-stage therapeutics

Experienced Team Leading Compugen

Diverse experienced leadership team

Management team



Eran Ophir, PhD

President and Chief Executive Officer



David Silberman

Chief Financial Officer



Michelle Mahler, MD

Chief Medical Officer



Pierre Ferre, PhD, Dr. Vet. Med.

Chief Operating Officer



Sharon Kredo-Russo, PhD

SVP, Research & Discovery



Zurit Levine, PhD

SVP, Business Development



Eran Ben Dor

General Counsel and Corporate Secretary



Dorit Amitay

VP, Human Resources

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Director

Gilead Halevy
Director

Kinneret Livnat Savitzky, PhD
Director

Eran Perry
Director

Sanford (Sandy) Zweifach
Director

Michele Holcomb, PhD
Director

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Strategic advisors

Industry veterans, renowned oncologists and immunologists

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Bristol Myers Squibb



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Strategic Advisor
Former CSO, EVP & Director,
Bristol Myers Squibb



Iain McInnes, FRCP, PhD



Anthony Tolcher, MD

Deep dive into COM701 data in platinum resistant ovarian cancer

COM701 clinical benefit across multiple tumors typically unresponsive to immunotherapy

TUMOR	TREATMENT	MED PRIOR LINES	BEST RESPONSE	DESCRIPTION	REFERENCE	IO BENCHMARK
Platinum resistant ovarian cancer	COM701	6 across indications	1/6 ORR (16.6%) 4/6 DCR (66%)	1 PR >18 months~* in immune desert TME	ASCO 2021	Pembro+ vibo: <10% ORR 0% ORR PD-L1 low 2 months mPFS [1-3]
	COM701 + nivolumab	6	2/20 ORR (10%) 9/20 DCR (45%)	1 PR in patient refractory to nivolumab	ESMO IO 2022	
	COM701 + nivolumab + BMS-986207	4	4/20 ORR (20%) 9/20 DCR (45%)	3 PR >16 months*	ESMO IO 2022 SITC 2023	
	COM701 +pembrolizumab +COM902	4	4/24 ORR (17%) 11/24 DCR (46%)	5 patients on treatment for >200 days	SITC 2024	
MSS CRC with liver metastases	COM701 + nivolumab	4	2/17 ORR (12%) 4/17 DCR (24%)	1 PR in patient with immune desert TME 1 PR >11 months	SITC 2022	ICI: 0% ORR
	COM701+ COM902+ pembrolizumab	3	1/15 ORR (7%) 6/15 DCR (40%)	1 PR > 9 months* in patient who had PD on chemo +bev (post data cut patient reassessed as non target liver lesion of uncertain etiology at baseline) 2 SD >7 months*	ASCO 2024	
ICI experienced NSCLC	COM701 ± nivolumab	6, ≥ 2 prior ICI	5/7 DCR (71%)	3 SD on COM701 monotherapy	ESMO IO 2022	Small study, no benchmark
Recurrent metastatic MSS endometrial cancer	COM701 + nivolumab + BMS-986207	2, 33% prior PD1x	2/9 ORR (22%) 4/9 DCR (44%)	1 PR in patient refractory to lenvatinib/pembro	ASCO 2023	ICI in IO naive: ~10% ORR[4-6]
Metastatic breast cancer	COM701 + nivolumab	5	2/17 ORR (12%) 5/17 DCR (30%)	1 CR > 21 months*, low immunogenic HER2 negative tumor	SITC 2023	no benchmark heterogenous population

BMS-986207 (Bristol Myers Squibb, anti-TIGIT); Pembro: pembrolizumab; Vibo: vibostolimab; ICI: Immune checkpoint inhibitor; bev: bevacizumab; PR: Partial Response; PD: Progressed Disease.
*ongoing at time of data cut-off, ~patient had primary peritoneal cancer.

1. Matulonis et al ASCO 2022, 2. Holmes et al JCO ASCO 2022, 3. Perets et al AACR 2022; 4 Oaknin et al J Immun of Cancer 2022; 5. Lheureux et al; J Immun of Cancer 2022; 6. Antill et al: J Immun of Cancer 2021.

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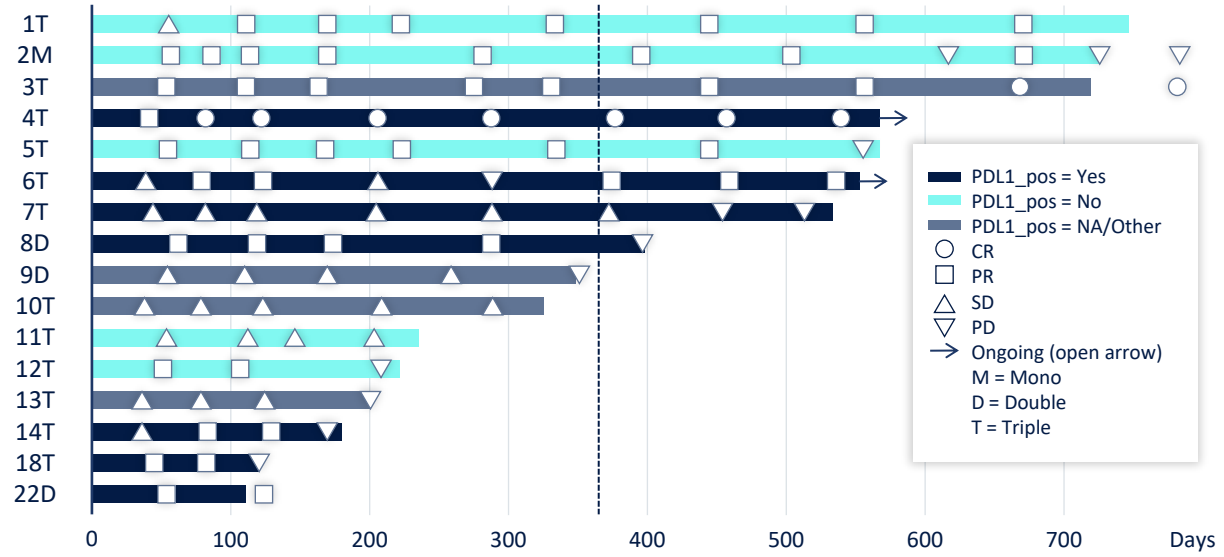
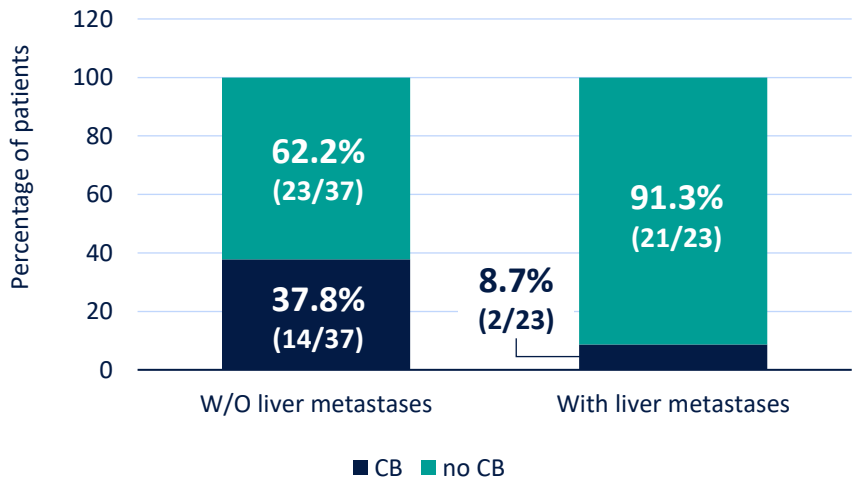
Characterizing PROC responding patients across Phase 1

Guided the design of ongoing MAIA-ovarian PSOC maintenance platform trial

Patients derived clinical benefit irrespective of PD-L1 status



Patients without liver metastases - more likely to derive clinical benefit



C1D1: Cycle 1 Day 1; CR: complete response; NA: not applicable; PD: progressive disease; POS: positive; PR: partial response; SD: stable disease.

CB: clinical benefit; W/O: without.

COM701 alone and in combination was well tolerated and demonstrated consistent, durable responses in heavily pre-treated PROC patients, particularly in patients without liver metastases, as well as patients who were PD-L1 negative

Data support advancing to a maintenance strategy in an earlier setting of ovarian cancer

Ongoing MAIA-ovarian platform trial is evaluating COM701 maintenance therapy in relapsed PSOC without liver metastases, for which there is a significant unmet need

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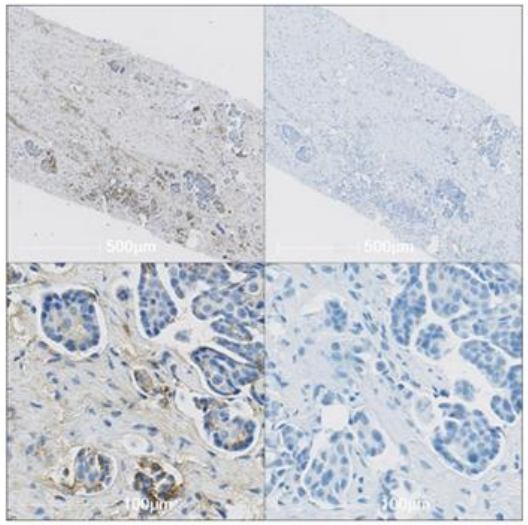
Yeku et.al. ESMO 2025

COM701 single agent activity: Partial response >18 months*

PD-L1 neg ovarian cancer patient supported by immune activation

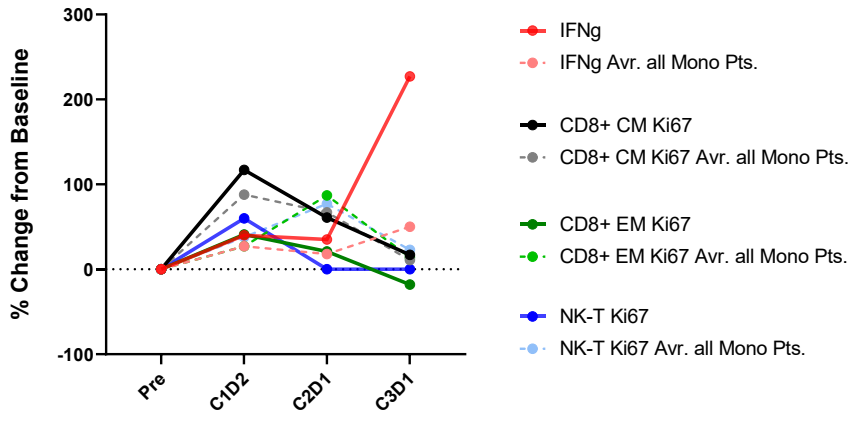
Patient received 3 prior lines of anti cancer therapy

PVRL2	PD-L1



- Pre- treatment archival biopsy (>1 year)
- **Negative PD-L1 staining**
- PVRL2 expression found on tumor and endothelial cells
- Immune “desert”: no immune cells detected in the biopsy

Increase in IFNγ induction and immune activation in peripheral blood



ASCO, June 2021, Vaena et al., Oral presentation

COM701 single agent immune modulation of the tumor microenvironment also demonstrated

Ophir E et al SITC 2022; Alteber E et al Cancer Immunology Research 2024

* This patient was on study treatment for 24 months i.e., she completed her treatment course allowed per protocol

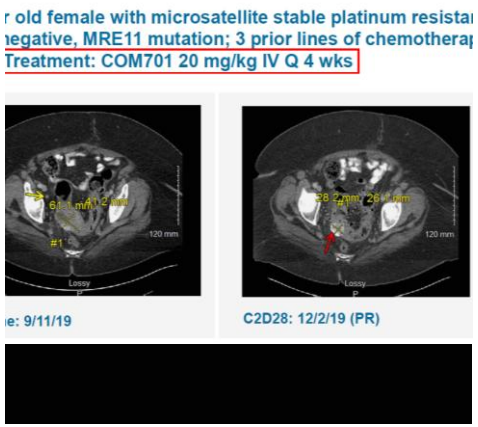
COM701 monotherapy: partial response in patient with PD-L1 negative ovarian ongoing treatment >18 months

63-year-old female with microsatellite stable platinum resistant primary peritoneal cancer

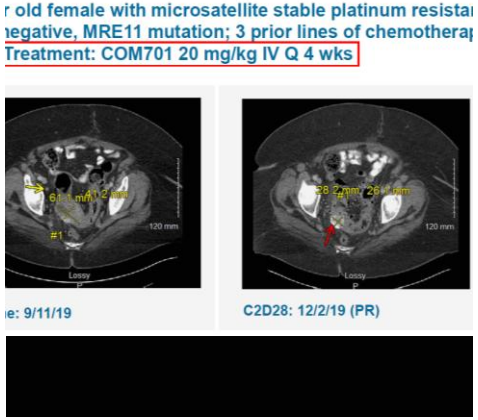
PDL1 negative, MRE11 mutation; 3 prior lines of chemotherapy

Study Treatment: COM701 20mg/kg IV Q 4 weeks

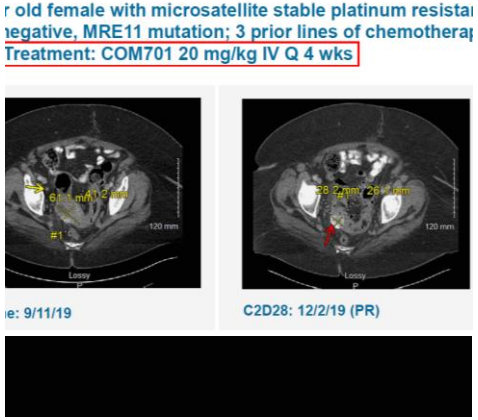
Baseline: 9/11/19



C2D28: 12/2/19 (PR)



C6D28: 3/23/20 (PR)



Had 3 prior lines of SOC treatment

- 1st line carboplatin/paclitaxel, SD (best response)
- Carboplatin/paclitaxel, PD (best response)
- Doxorubicin/Bevacizumab, PR (best response, d/c due to toxicity)
- Enrolled into mono dose escalation (COM701 20 mg/kg IV Q4 wks)

Consistent efficacy & safety with COM701 triplet combination

In platinum resistant ovarian cancer patients who have exhausted all options

Efficacy

Investigator assessed by RECIST v1.1	COM701+ nivolumab + BMS-986207[1] (N=20)	COM701+ pembrolizumab+ COM902[2] (N=24)
Overall Response rate % (CR+PR)	4 (20%)	4 (17%)
Disease control rate % (CR+PR+SD)	9 (45%)	11 (46%)
Best response (%)		
Complete response	–	1 (4%)
Partial response	4 (20%)	3 (13%)
Stable Disease	5 (25%)	7 (29%)
Progressive Disease/Clinical PD/lack of clinical benefit	11 (55%)	13 (54%)

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]

The safety and tolerability profile generally consistent with approved anti-PD-(L)1s

- Most common reported AEs grade 1/2 fatigue, diarrhea and nausea
- Grade ≥3 TRAEs 20%
- No grade 4/5 TRAEs
- Tx related discontinuations 4.4%
  - 1 patient in each study had grade 3 AE leading to drug discontinuation

Activation of the immune system

- Translational assessment of peripheral blood and on-treatment tumor biopsies showed a positive pharmacodynamic activation of the immune system

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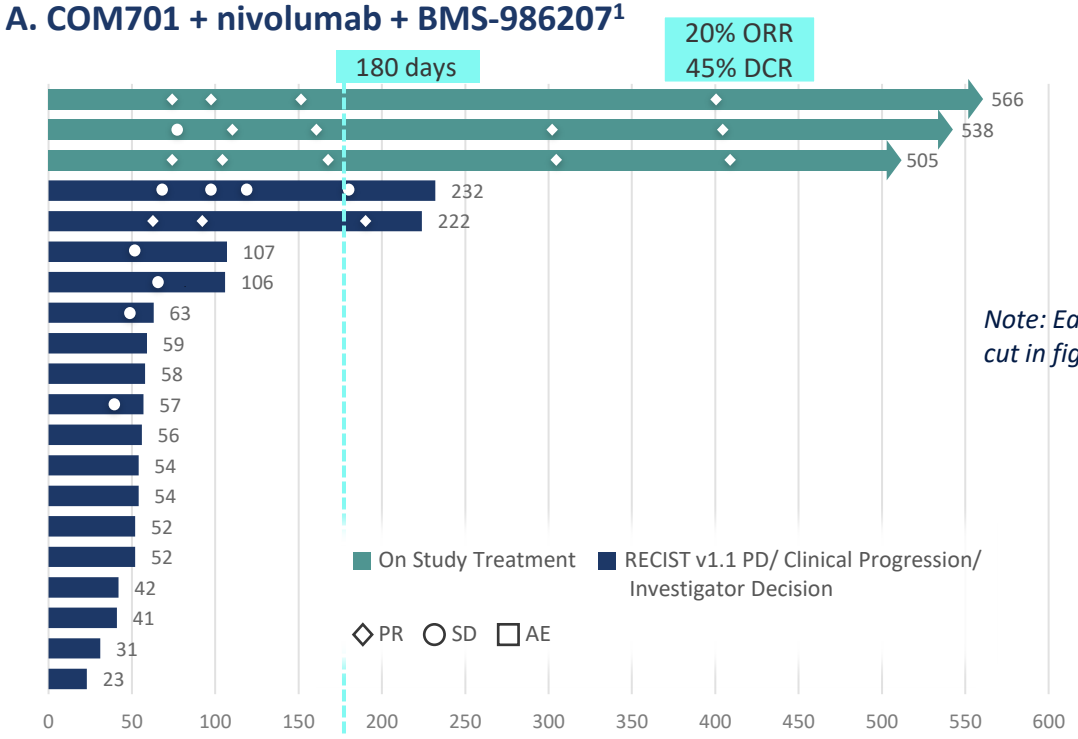
BMS-986207 (Bristol Myers Squibb, anti-TIGIT)
TRAE: Treatment related adverse event
AE: Adverse event

1. Moroney J, et al, ESMO IO 2022 Modified
2. Yeku O, et al, SITC 2024 Modified
3. Matulonis et al ASCO 2022,
4. Holmes et al JCO ASCO 2022,
5. Perets et al AACR 2022

Consistent durable responses demonstrated across studies

In platinum resistant ovarian cancer patients who have exhausted all options

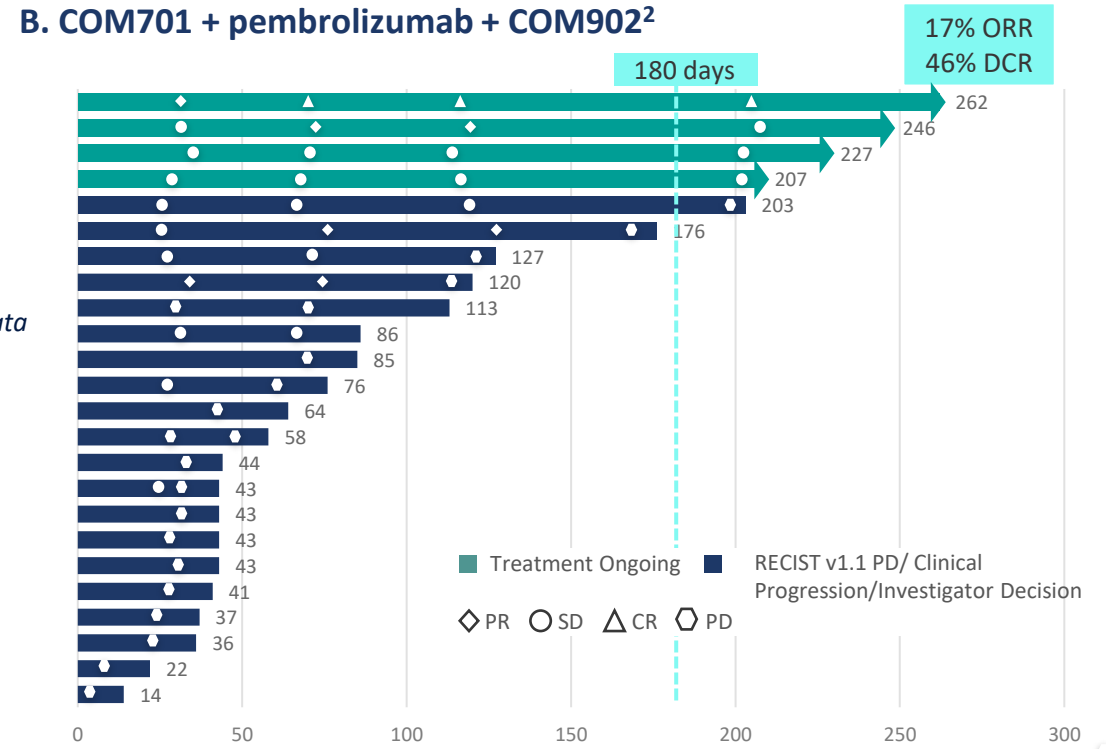
A. COM701 + nivolumab + BMS-986207[1]



Note: Earlier data cut in figure B

Data cut: September 5, 2023 investigator assessed responses

B. COM701 + pembrolizumab + COM902[2]



Data cut: August 29, 2024 investigator assessed responses

Data supports COM701 driven activity [1,2] versus historical data [5-7]

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

1. Gaillard S et al SITC 2023, modified
2. Yeku et al; SITC 2024 modified
3. Pujade-Lauraine et al JCI 2014,
4. Secord et al JCO 2007,
5. Matulonis et al ASCO 2022,
6. Holmes et al JCO ASCO 2022,
7. Perets et al AACR 2022

BMS-986207 (Bristol Myers Squibb, anti-TIGIT).

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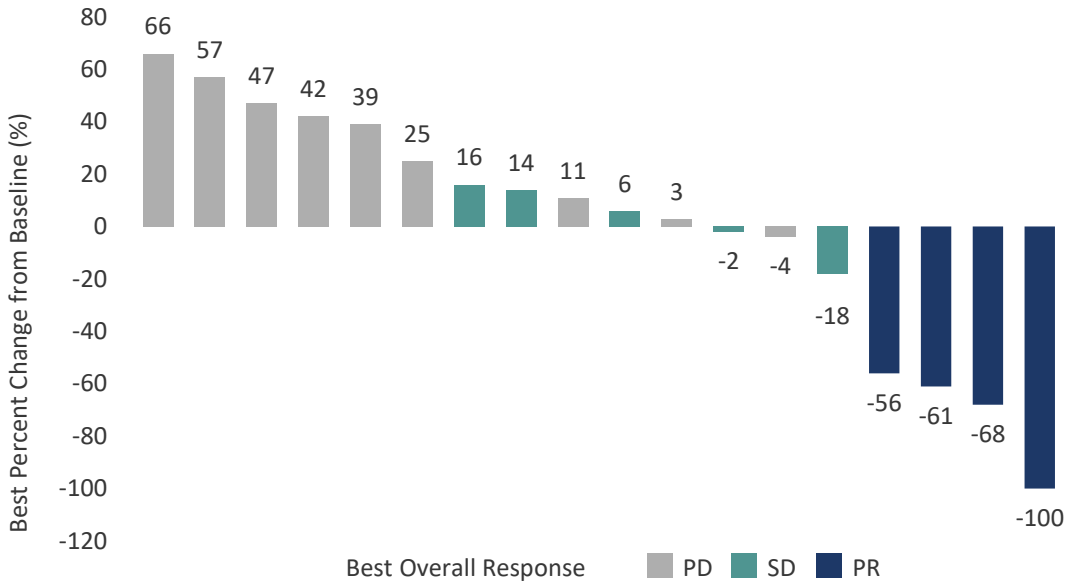
Consistent deep responses across studies

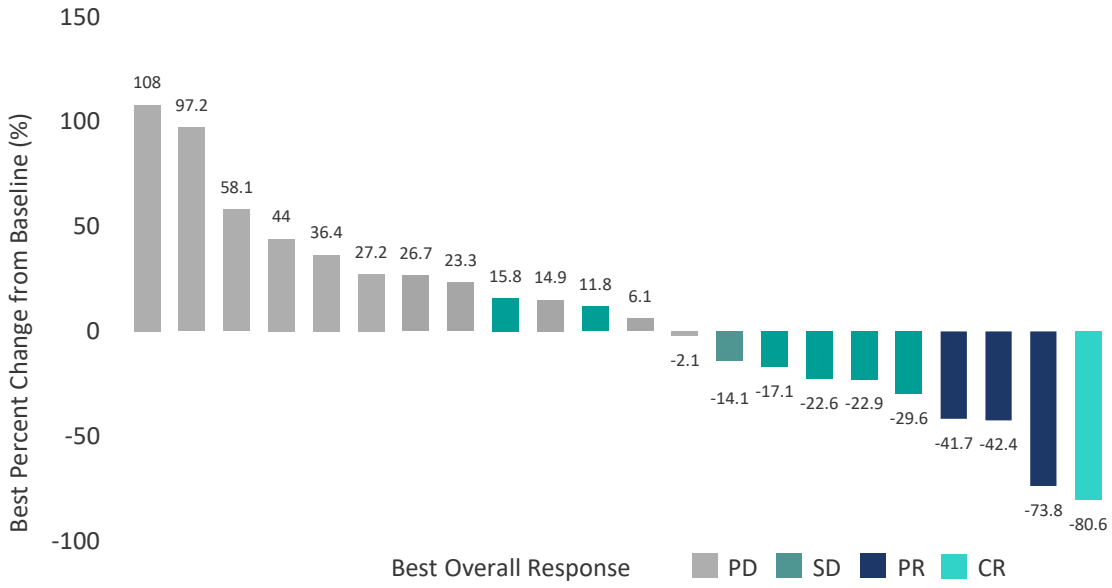
In platinum resistant ovarian cancer patients who have exhausted all options

COM701 + nivolumab + BMS-986207[1]



Data cut off date 5 September 2023
2 patients with PD (clinical evaluation) not included.

COM701 + pembrolizumab + COM902[2]



Data cut off date 29 August 2024

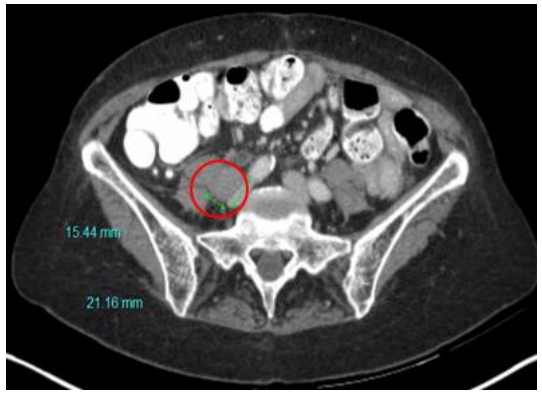
1. Gaillard, S. et al; SITC 2023 modified; 2. Yeku O, et al, SITC 2024, modified
BMS-986207 (Bristol Myers Squibb, anti-TIGIT)

Clinical Vignette – COM701 + pembrolizumab + COM902 complete response in PROC patient

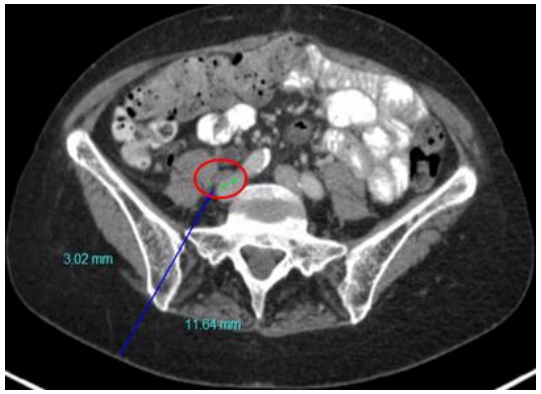
4 prior lines of therapy including chemotherapy, bevacizumab maintenance and an investigational agent (smac-mimetic) with SD best response prior to study entry

69-year-old female with high grade serous adenocarcinoma platinum resistant ovarian cancer

Right common iliac node 2



Baseline
15.44 x 21.16 mm



100 days from C1D1
3.02 x 11.64 mm

Right external iliac node 1



Baseline
20.6 x 23.36 mm



100 days from C1D1
4.27 x 4.89 mm

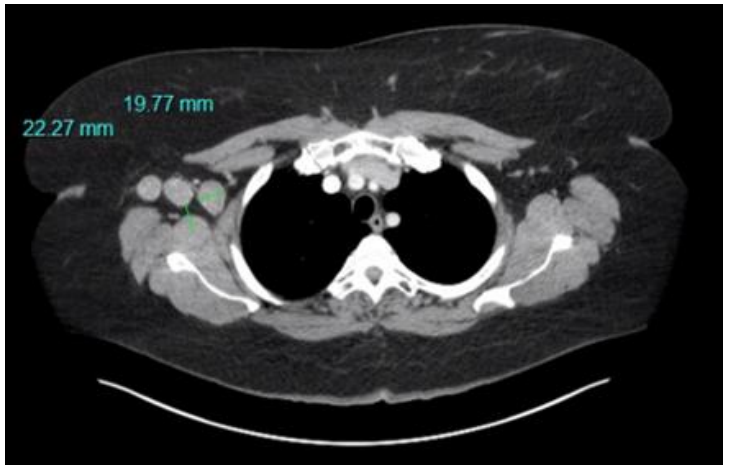
Yeku O et all SITC 2024, modified

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

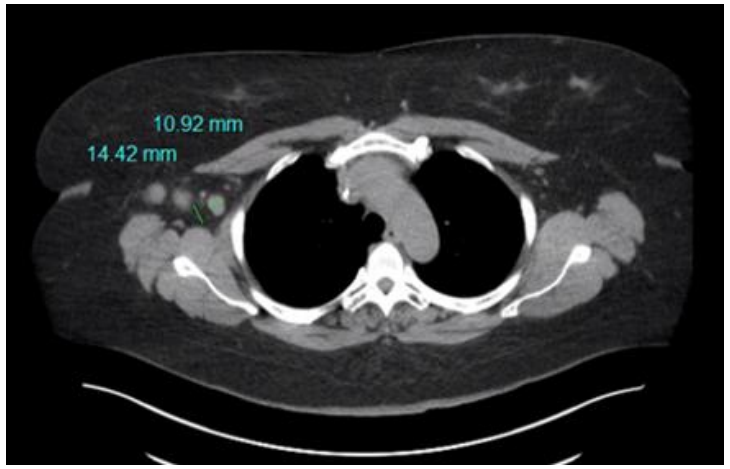
Received 7 prior lines of therapy including progressed disease on nivolumab

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

Pre-treatment

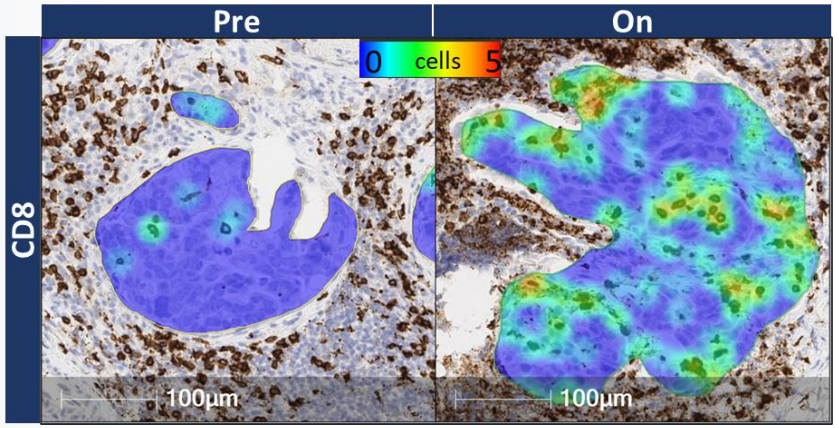


On-treatment



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/mm^2)	8.5 x 10^{-4}	16 x 10^{-4}

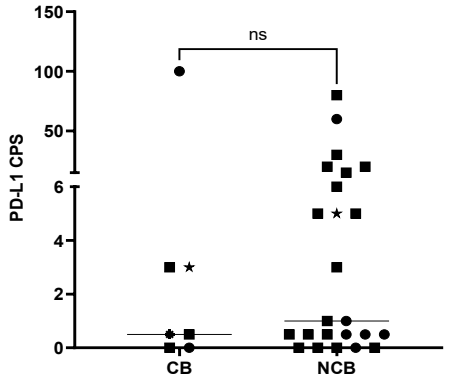
Modified from ASCO June 2021 Presentation
Yeku e al, ESMO-IO 2022 Modified

Clinical benefit and TME T cell clonal expansion independent of baseline inflammatory status

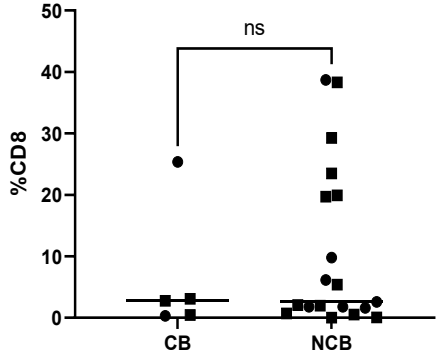
Platinum Resistant Ovarian Cancer Patients

Baseline inflammatory status

PD-L1 CPS



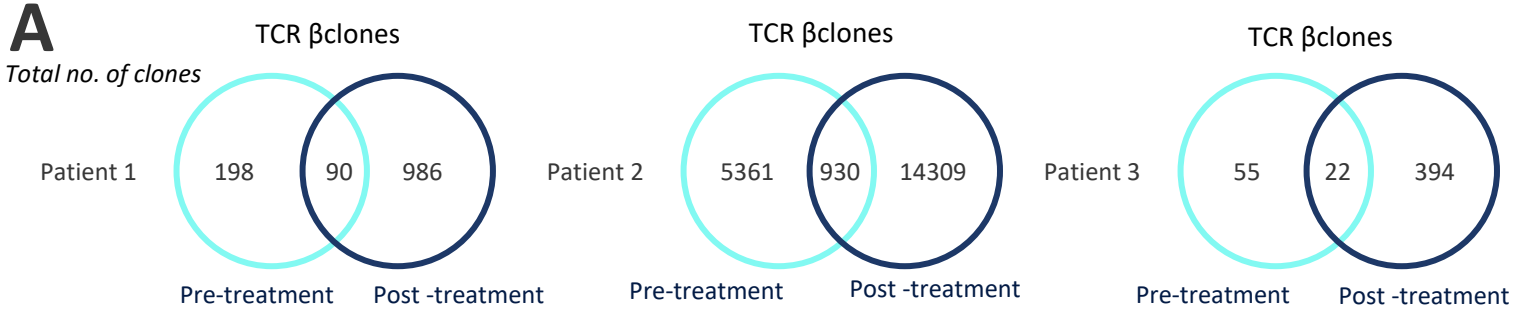
CD8 % positive



TME T cell clonal expansion in patients who derived clinical benefit from COM701 + nivolumab ± BMS-986207

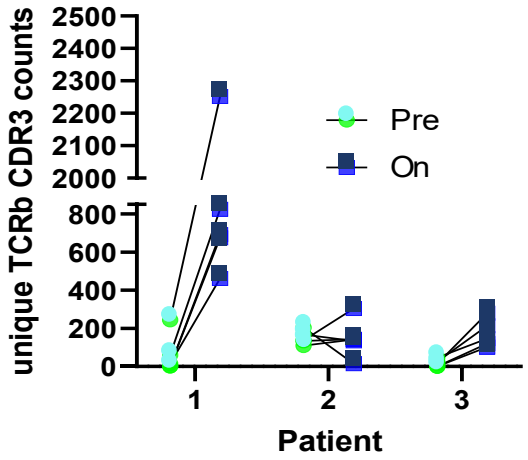
A

Total no. of clones



B

Top 5 clones



Definition: Clinical Benefit (CB)= CR+PR+SD>180 days on study; No Clinical Benefit (NCB) = PD +SD<180 days on study

Cojocaru G, et al; SITC 2023, modified

COM701 safety profile may provide clinically relevant differentiation in earlier cancer settings

The safety and tolerability profile was generally consistent with approved anti-PD-(L)1s as monotherapy and in combination across tumors

COM701 ± anti-TIGIT ± anti-PD-1[1]

- Majority of TRAEs ≤ Grade 2
  - Most common reported AEs grade 1/2 fatigue, diarrhea and nausea
- Grade ≥3 TRAEs 9.5%
- No grade 4/5 TRAEs
- Low rate of treatment related discontinuations 4.9%

COM701 activity potentially occurs mostly in the tumor microenvironment due to unique PVRIG biology[2]

1. Data Cutoff 30 Oct 2024
2. Alteber et al, Cancer Immunology Research, 2024

TRAE: treatment related adverse event; AE: Adverse event

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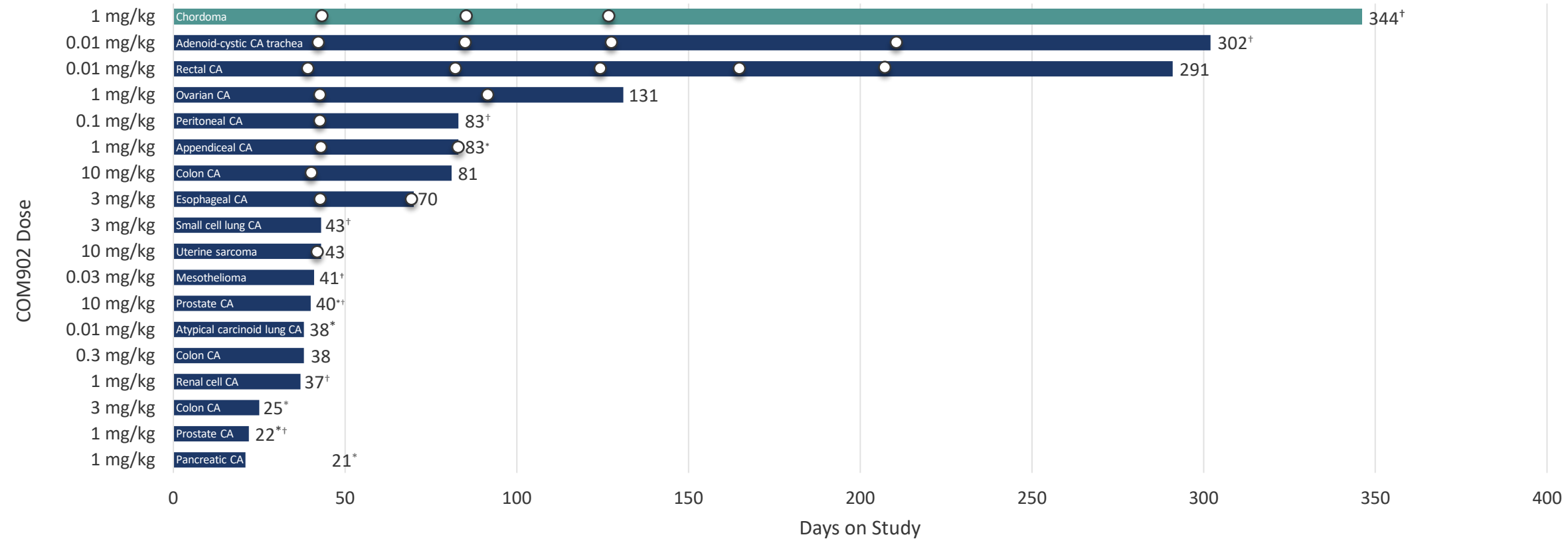
COM902- potential best-in-class anti-TIGIT antibody

COM902 monotherapy showed a 50% disease control rate

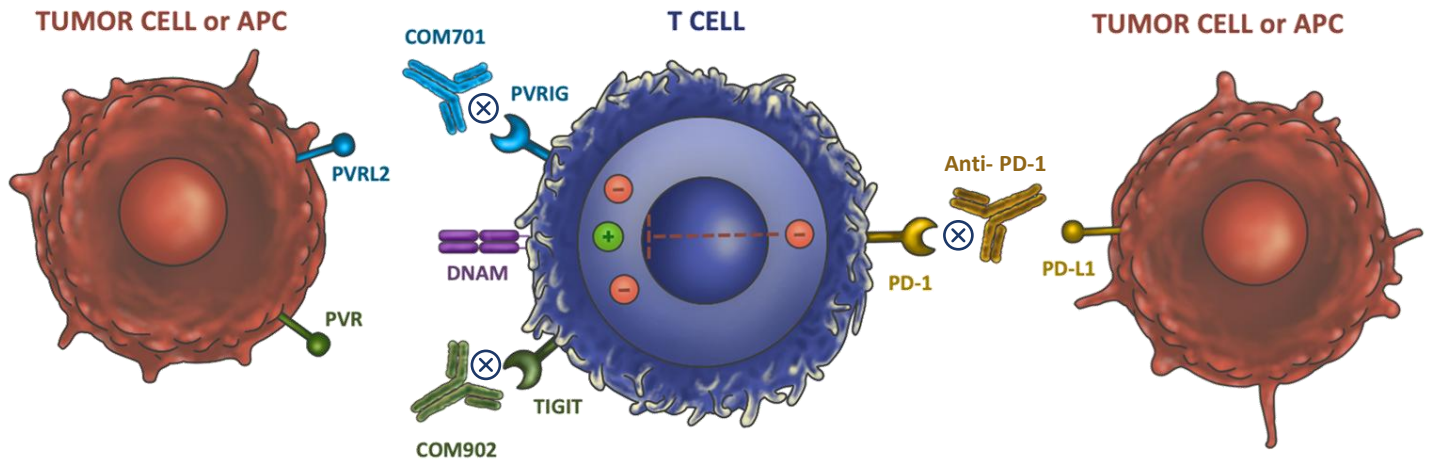


Data cut 03 September 2021

Dumbrava E E, et al, SITC 2021, Modified

DLT: Dose limiting toxicity PK: Pharmacokinetics ICI: Immune checkpoint inhibitor SD: Stable disease MTD: Maximum Tolerated Dose

Compugen differentiated in TIGIT space by unique properties of Fc reduced COM902 and clinical strategy



DNAM-1 axis potential game changer in fight against cancer

- One of only two clinical stage Fc-reduced anti-TIGIT monoclonal antibodies
- Combination of TIGIT with PD-1 blockade may be effective in PD-1 high tumors
- **DNAM-1 axis**: 2 parallel and complementary inhibitory pathways - TIGIT & PVRIG
- Blocking PVRIG with **COM701** may be disruptive in **less inflamed tumors** where TIGIT/PD-1 blockade alone is insufficient

Martinet & Smyth, 2015 (modified)

COM902 – well tolerated safety profile[1]

Favorable safety and well tolerated
as monotherapy and in combination

COM902 ± COM701± pembrolizumab

- Most commonly reported AEs were grade 1/2
- Grade ≥3 TRAEs 8.5%, fatigue – most common
- No grade 4/5 TRAEs
- Low treatment related discontinuations 4.2%
- IrAE 17% all ≤Gr 3 all in triplet
- IRR 5.3% all ≤ Gr 2

COM902 naturally Fc reduced antibody (IgG4 backbone) chosen with efficacy and safety in mind

Avoids depletion of effector T-cells required for efficacy[2]

Potential to avoid infusion related reactions that active Fc may trigger

Fc silent anti-TIGIT avoids peripheral Treg depletion that may lead to IrAEs [3]

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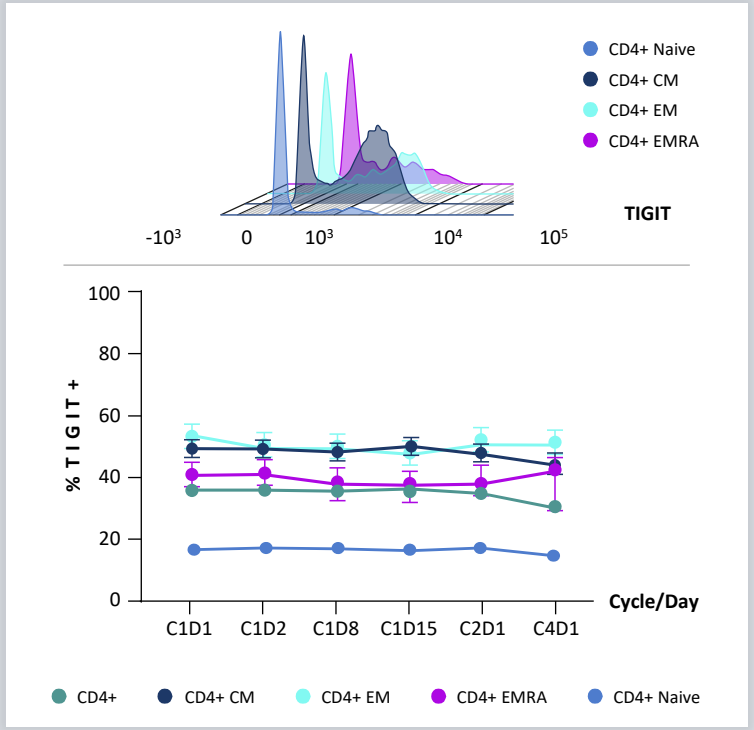
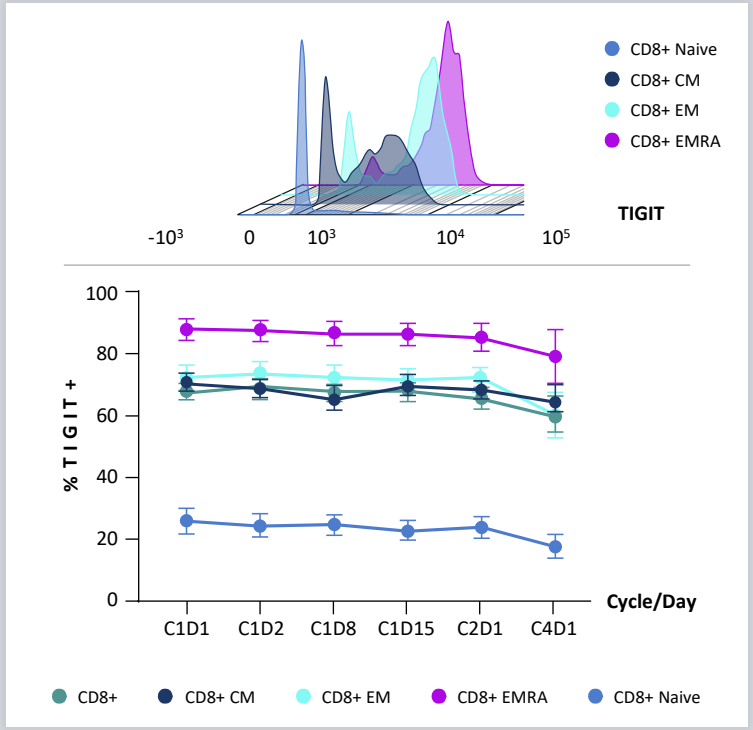
1. Internal data as of data cut Oct 2024
2. DumBrava E,E, et al, SITC 2021
3. Hansen et al; Cancer Immunol Immunother, 2021

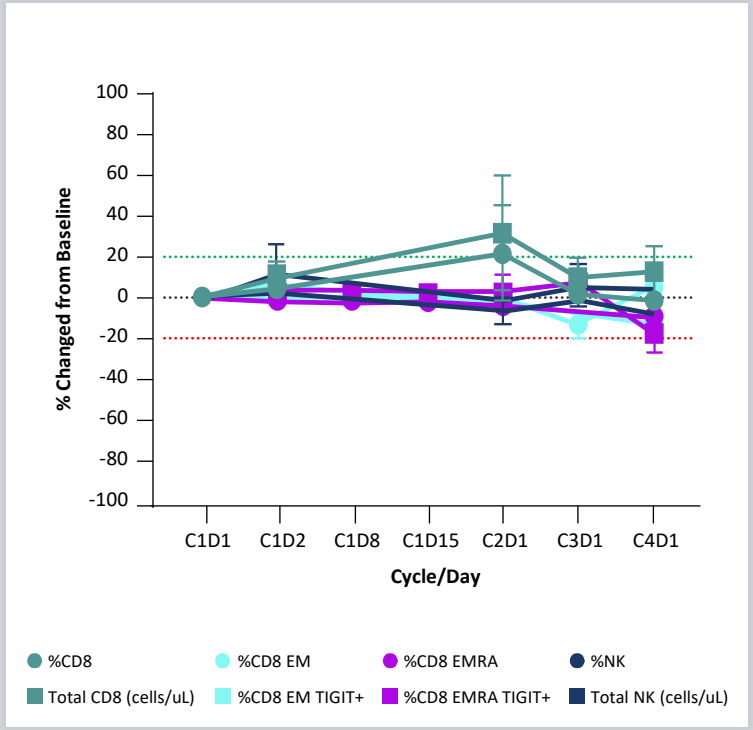
IRR – Infusion Related Reactions
IrAE – Immune related Adverse Event
TRAE: Treatment related adverse events

COM902 prevents depletion of major TIGIT+ expressing lymphocytes- NK, CD4 and CD8 T cells

Supporting rationale for selecting a high affinity anti-TIGIT antibody with an IgG4 backbone and low Fc effector function- COM902







COM902 prevents CD8+ T Cell depletion and potential associated risks

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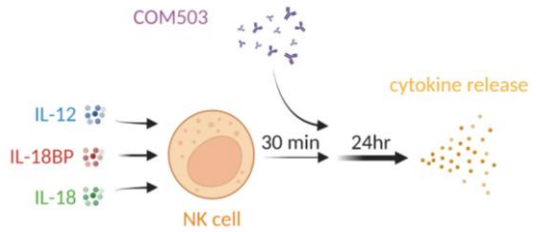
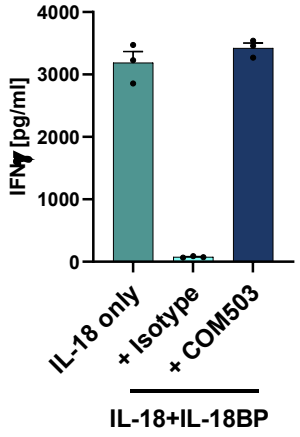
Dumbrava E,E, et al, SITC 2021, Modified

Deep dive into GS-0321 data

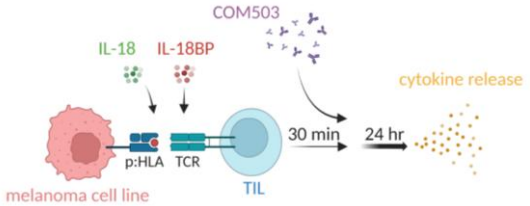
GS-0321, fully human, high affinity anti-IL-18BP antibody restores human TIL and NK cell activity in human assays

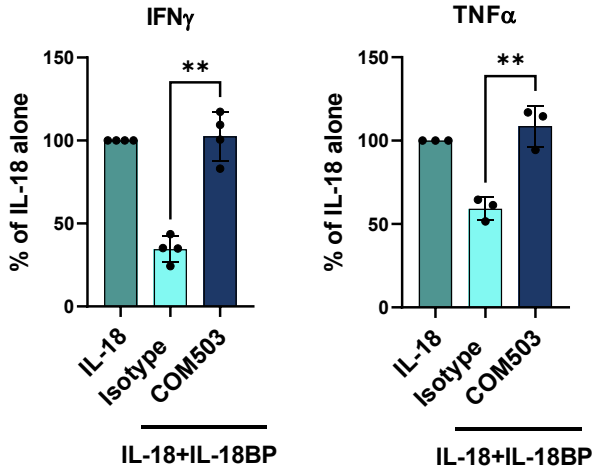
GS-0321 restored NK cell activity



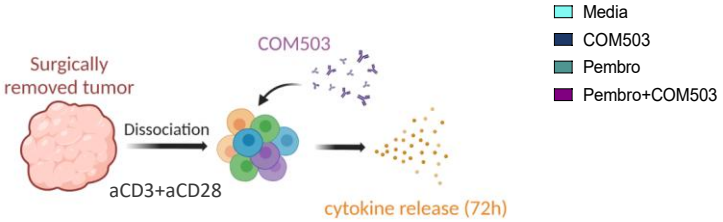


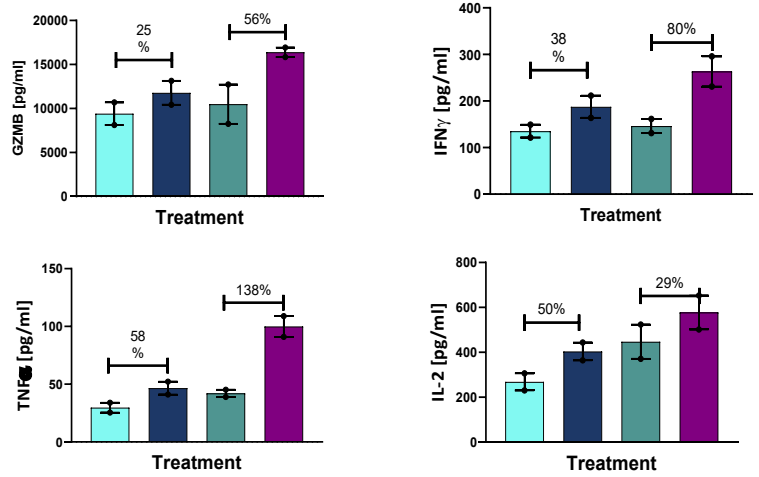
GS-0321 restored TILs activity





GS-0321 enhanced T cell activation in human dissociated tumor cells assay

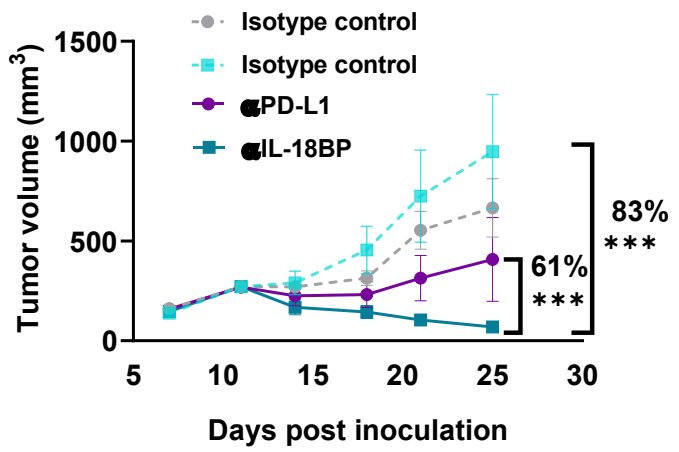




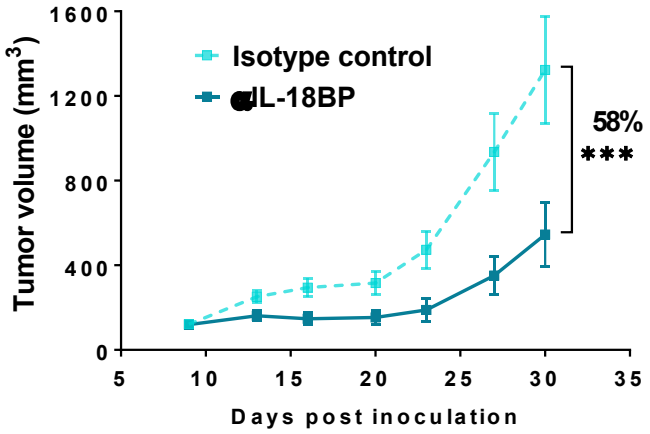
TILs- tumor infiltrating lymphocytes
Ferre P, presentation, SITC, 2023, modified

Anti-mouse IL-18BP antibody demonstrates monotherapy activity across murine syngeneic tumor models

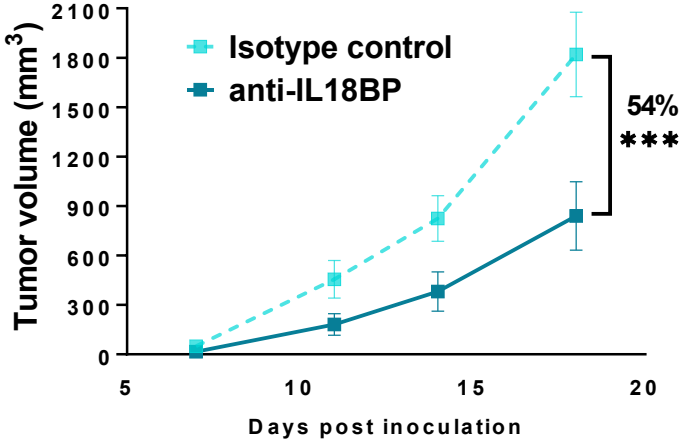
αIL-18BP Ab inhibits tumor growth in E0771 orthotopic mouse breast tumor model



αIL-18BP Ab inhibits tumor growth in MC38ova mouse CRC tumor model



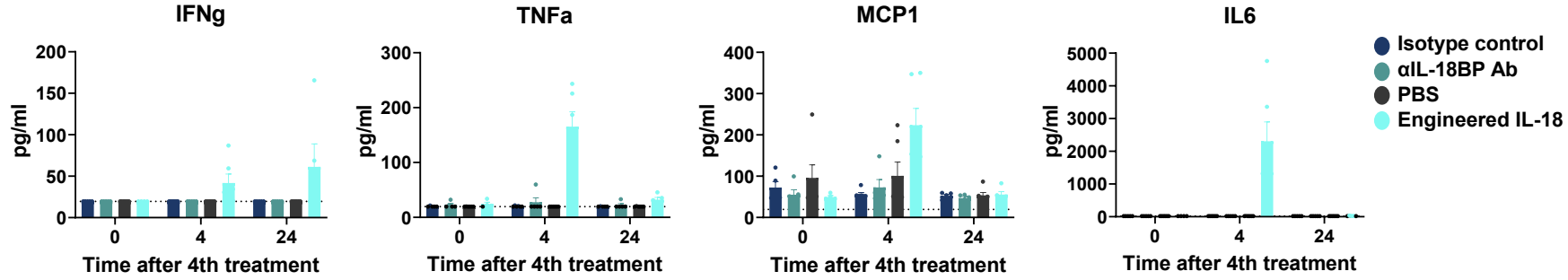
αIL-18BP Ab inhibits tumor growth in B16F10-hmgp100 mouse melanoma model



Ophir E, presentation, CIMT 2023, modified

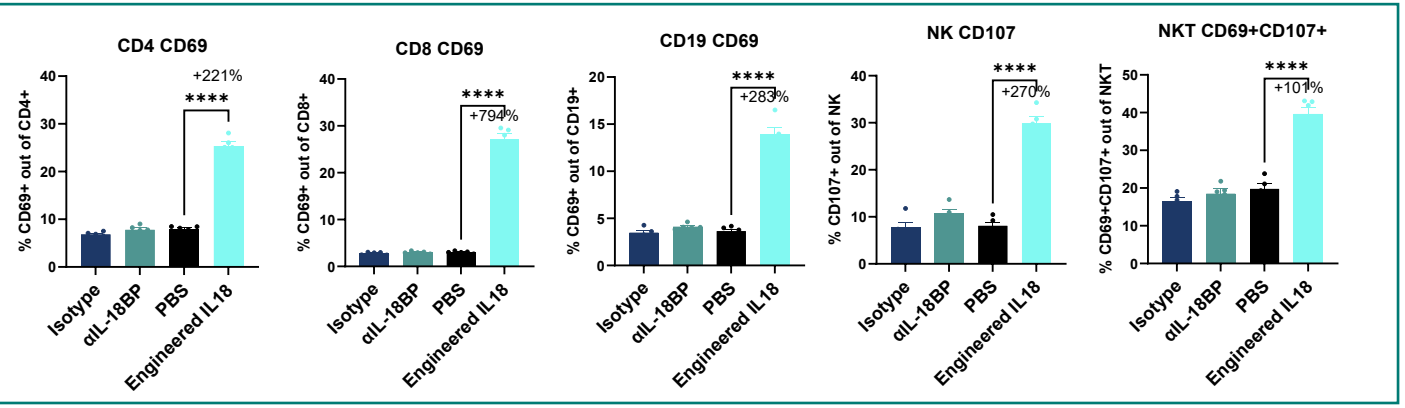
Anti-IL18BP Ab is expected to have a better therapeutic window than recombinant cytokines

Administration of anti-mouse IL-18BP Ab to mice did not affect serum cytokines in contrast to engineered mouse IL-18*

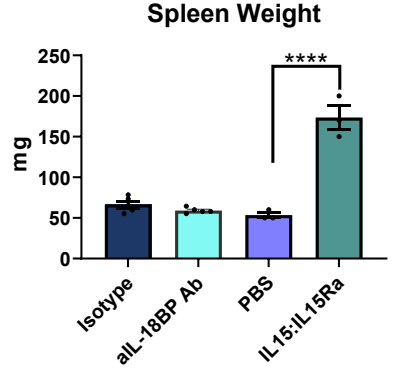


*Engineered IL-18 does not bind to IL18BP but retains its binding to IL-18R

Administration of anti-mouse IL-18BP Ab to mice did not affect lymphocytes activation in contrast to engineered mouse IL-18

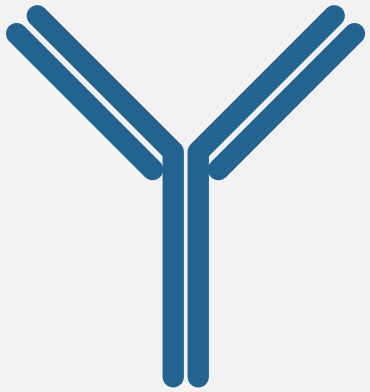


Administration anti-mouse IL-18BP Ab to mice did not result in splenomegaly in contrast to rIL-15:IL15Ra



Appendix

COM701 and COM902 distinguishing properties



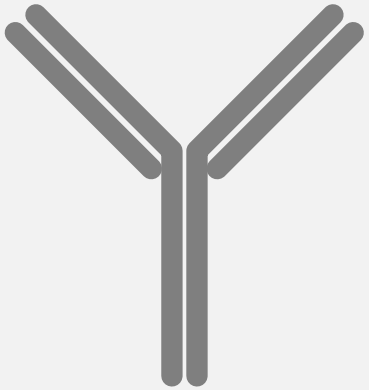
COM701

Antibody characteristics

- Humanized IgG4 antibody
- High affinity (2 pM KD by KinExA)
- Blocks PVRIG/PVRL2 binding interaction
- Enhances T and NK cell activation alone and in combination with TIGIT and PD1 blockade
- Favorable PK in patients: Linear PK and PVRIG receptor occupancy > 90% threshold over 21 days from 1 mg/kg

Strong IP position

- Internally discovered and developed, potentially first-in-class asset
- Issued and pending patents for PVRIG antibodies for use in cancer treatment, COM701 composition of matter, use and combinations worldwide



COM902

Antibody characteristics

- Favorable linear PK and very large TIGIT receptor occupancy
- Fully human anti-TIGIT antibody, cross-reactive with cynomolgus and mouse
- Femto-molar affinity to human TIGIT (626 fM KD by KinExA)
- IgG4 (S228P) isotype, reduced Fc function prevents depletion of effector cells providing optimal anti-tumor response

Strong IP position

- Issued and pending patents for COM902 composition of matter, use and combinations worldwide

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