GCC19CAR-T Therapy for Refractory Colorectal Cancer

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Dana-Farber Cancer Institute

Disclosures

- Advisory board/consulting
  - Agenus
  - AstraZeneca

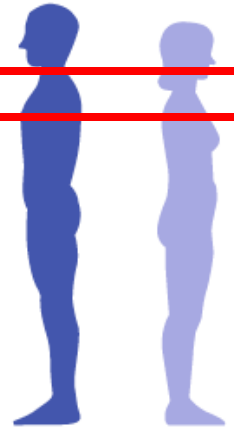
Agenda

- Background on colorectal cancer.
- Conventional immunotherapy in colorectal cancer.
- Limitations of CAR-T cell therapy solid tumors.
- GCC19CART/CARAPIA-1 phase I results.

Colorectal Cancer: The Scope of the Problem

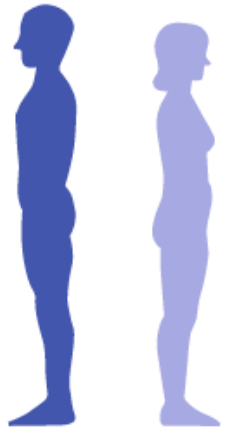
Estimated New Cases

Male			Female		
Prostate	313,780	30%	Breast	316,950	32%
Lung & bronchus	110,680	11%	Lung & bronchus	115,970	12%
Colon & rectum	82,460	8%	Colon & rectum	71,810	7%
Urinary bladder	65,080	6%	Uterine corpus	69,120	7%
Melanoma of the skin	60,550	6%	Melanoma of the skin	44,410	4%
Kidney & renal pelvis	52,410	5%	Non-Hodgkin lymphoma	35,210	4%
Non-Hodgkin lymphoma	45,140	4%	Pancreas	32,490	3%
Oral cavity & pharynx	42,500	4%	Thyroid	31,350	3%
Leukemia	38,720	4%	Kidney & renal pelvis	28,570	3%
Pancreas	34,950	3%	Leukemia	28,170	3%
All sites	**1,053,250**		**All sites**	**988,660**	



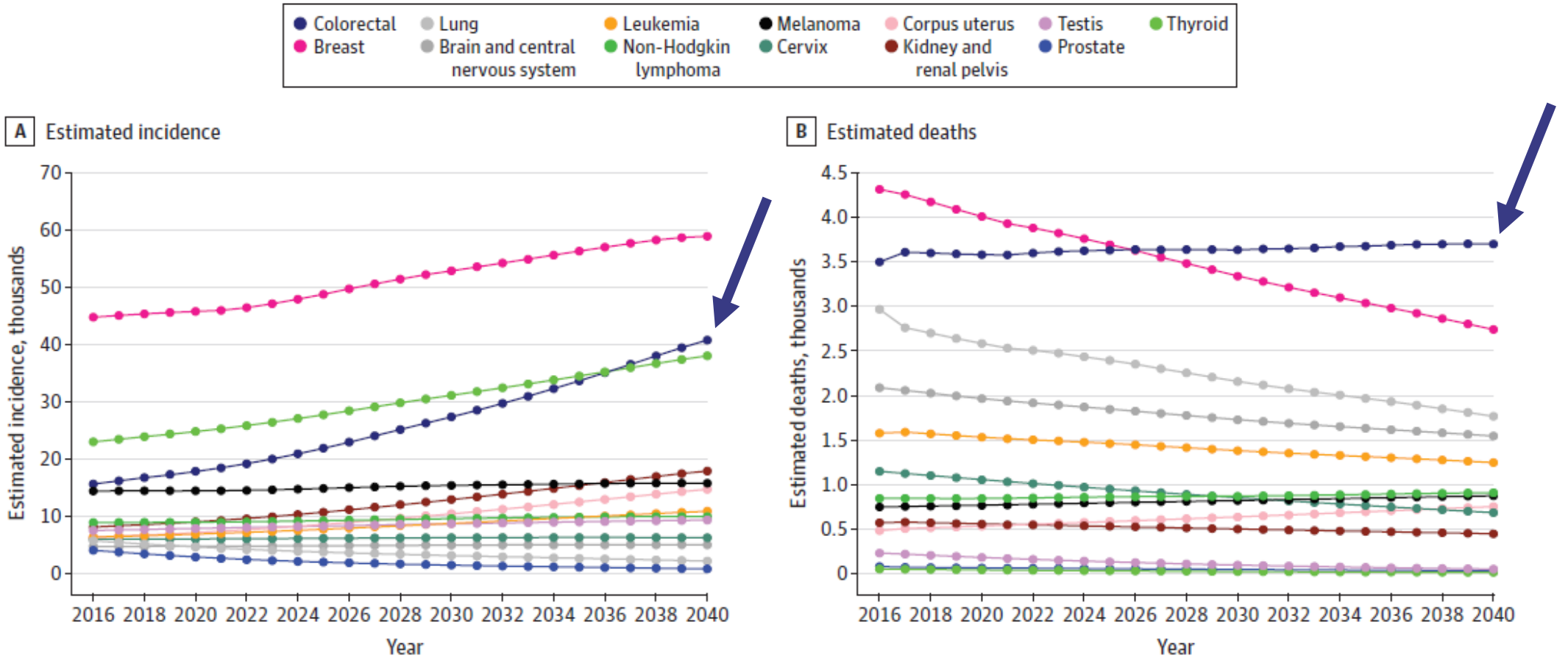
Estimated Deaths

Male			Female		
Lung & bronchus	64,190	20%	Lung & bronchus	60,540	21%
Prostate	35,770	11%	Breast	42,170	14%
Colon & rectum	28,900	9%	Pancreas	24,930	8%
Pancreas	27,050	8%	Colon & rectum	24,000	8%
Liver & intrahepatic bile duct	19,250	6%	Uterine corpus	13,860	5%
Leukemia	13,500	4%	Ovary	12,730	4%
Esophagus	12,940	4%	Liver & intrahepatic bile duct	10,840	4%
Urinary bladder	12,640	4%	Leukemia	10,040	3%
Non-Hodgkin lymphoma	11,060	3%	Non-Hodgkin lymphoma	8,330	3%
Brain & other nervous system	10,170	3%	Brain & other nervous system	8,160	3%
All sites	**323,900**		**All sites**	**294,220**	



https://www.facs.org/-/media/files/quality-programs/cancer/ncdb/cancer-cases-reported-to-the-ncdb-by-tumor-type-and-ajcc-stage.ashx

Colorectal Cancer is a Leading Cause of Cancer Mortality in Adults Under Age 50



Rahib L, et al. *JAMA Network Open* 2021; 4(4).

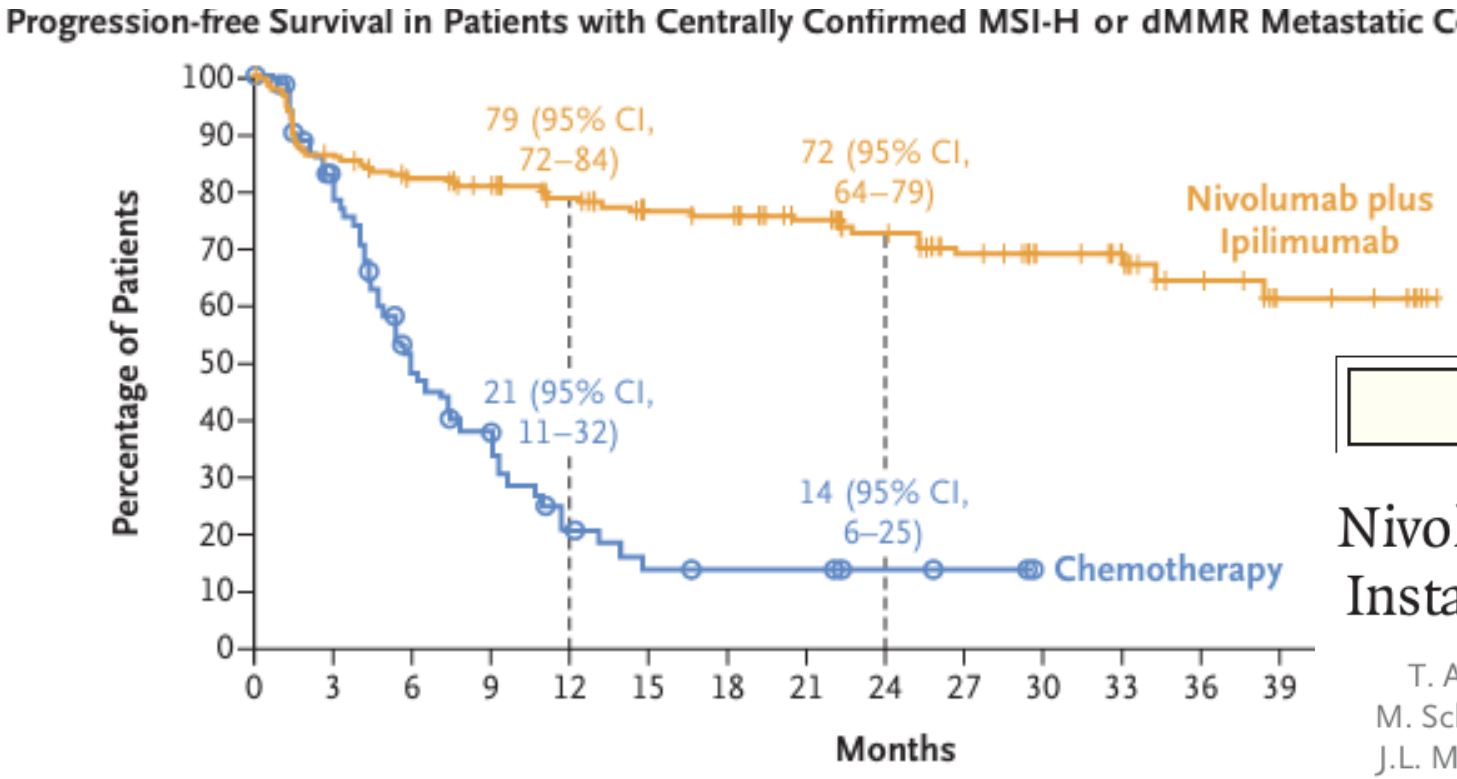
Immunotherapy Paradigms

- Pro-inflammatory drugs
  - IL-2, interferon
- Non-targeted immunotherapy
  - PD1/L1 inhibitors, CTLA4 inhibitors
- Targeted immunotherapy
  - BiTEs, TILs, TCR-T cells, CAR-T cells

Non-targeted Immunotherapy

- Targets normal immune cell, so not specific to any particular cancer cell type.
- Requires a tumor or microenvironment that permits an effective immune response.
- Checkpoint inhibitors are the main treatments here.
  - Generally, moderately effective to ineffective
  - May cause diseases of impaired tolerance, pan- “itis”
  - Limited efficacy in “cold” tumors

Checkpoint Inhibitors in Advanced MMRD Colorectal Cancer



Only 3-5% of advanced colorectal cancer patients have MMRD cancers

ORIGINAL ARTICLE

Nivolumab plus Ipilimumab in Microsatellite-Instability–High Metastatic Colorectal Cancer

T. André, E. Elez, E. Van Cutsem, L.H. Jensen, J. Bennouna, G. Mendez, M. Schenker, C. de la Fouchardiere, M.L. Limon, T. Yoshino, J. Li, H.-J. Lenz, J.L. Manzano Mozo, G. Tortora, R. Garcia-Carbonero, L. Dahan, M. Chalabi, R. Joshi, E. Goekkurt, M.I. Braghiroli, T. Cil, E. Cela, T. Chen, M. Lei, M. Dixon, S. Abdullaev, and S. Lonardi, for the CheckMate 8HW Investigators*

Andre T. N Engl J Med 2024;391:2014-2026

Checkpoint Inhibitors in Advanced MSS Colorectal Cancer

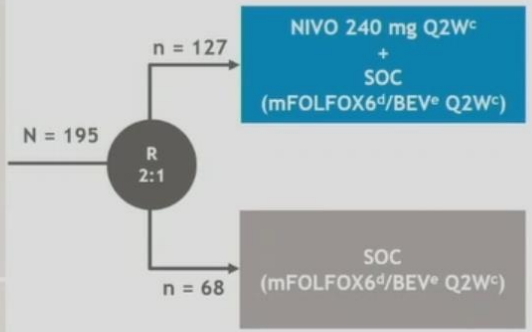
CheckMate 9X8

CheckMate 9X8 study design

- CheckMate 9X8 is a randomized, open-label phase 2/3 study[a]

Key eligibility criteria
- Histologically confirmed mCRC not amenable to curative resection
- No prior chemotherapy for mCRC
- No prior treatment with checkpoint inhibitors[b]
- ECOG PS 0-1

Stratification factors
- Tumor sidedness (left, right, transverse, or unknown)
- Prior oxaliplatin-based chemotherapy (yes or no)

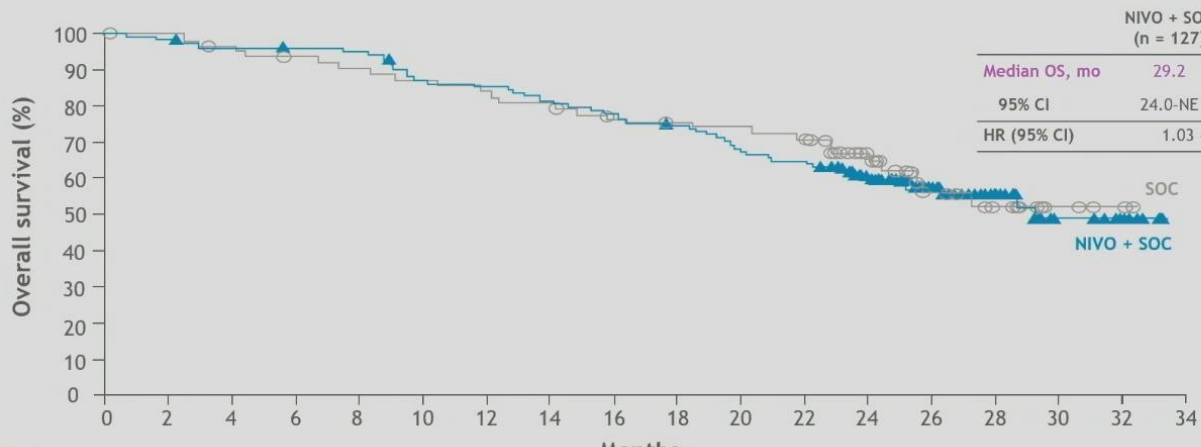


- At data cutoff (February 1, 2021), the minimum follow-up was 21.5 months[f]

[a]ClinicalTrials.gov. NCT03414983; [b]No prior treatment with an anti-PD-1, anti-PD-L1, anti-PD-L2, or anti-CTLA-4 antibody, or any other antibody or drug pathways; [c]Until disease progression, unacceptable toxicity, withdrawal of consent, or end of study; NIVO treatment for ≤ 24 months; [d]Oxaliplatin, 85 m standards; fluorouracil, bolus 400 mg/m², followed by 1200 mg/m² continuous infusion on day 1 (or 15) and day 2 (or 16), or 2400 mg/m² continuous inf 16) per local standards; [e]Bevacizumab, 5 mg/kg; [f]Time from randomization of the last patient to clinical data cutoff.

CheckMate 9X8

Overall survival



	NIVO + SOC (n = 127)	SOC (n = 68)
Median OS, mo	29.2	NR
95% CI	24.0-NE	24.4-NE
HR (95% CI)	1.03 (0.64-1.66)	

No. at risk																		
NIVO + SOC	127	123	119	118	117	106	104	99	93	90	82	78	60	39	23	10	6	0
SOC	68	64	61	58	56	54	52	49	46	44	43	41	28	17	12	5	3	0

- Minimum follow-up was 21.5 months; longer follow-up is warranted

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Next Generation Checkpoint Inhibitors in Colorectal Cancer

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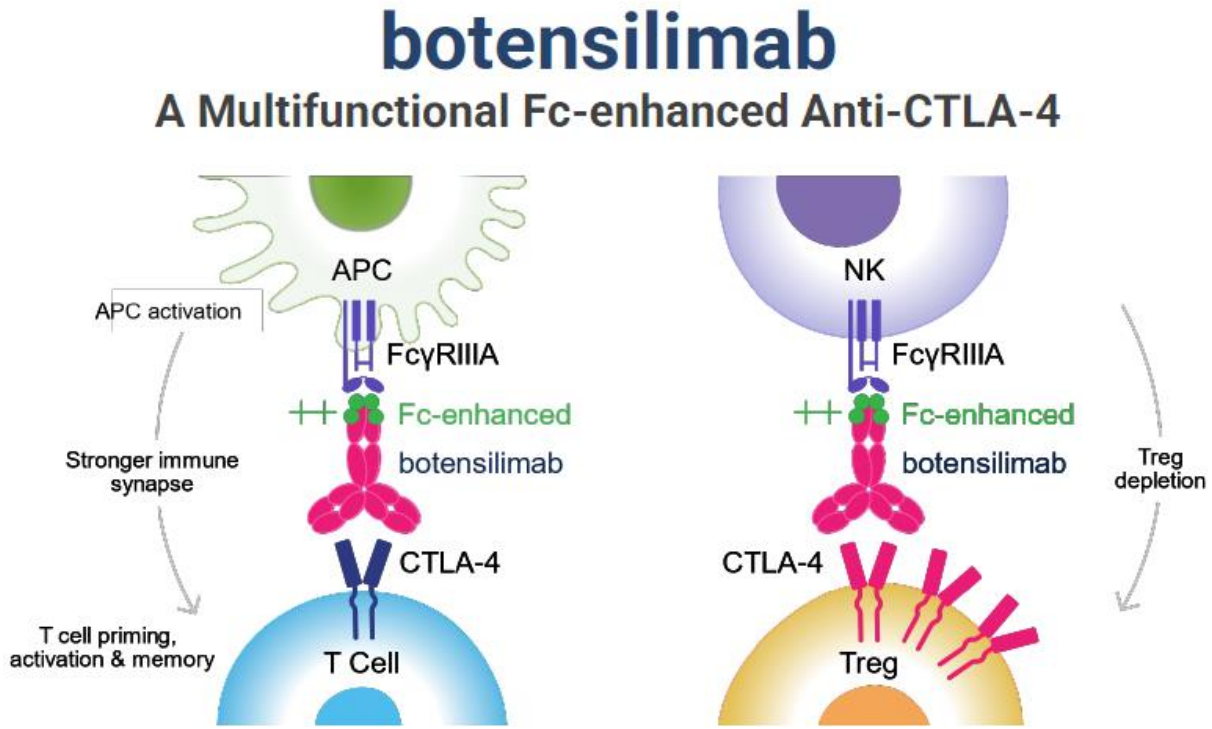
Article | Open access | Published: 13 June 2024

Botensilimab plus balstilimab in relapsed/refractory microsatellite stable metastatic colorectal cancer: a phase 1 trial

Andrea J. Bullock, Benjamin L. Schlechter, Marwan G. Fakih, Apostolia M. Tsimberidou, Joseph E. Grossman, Michael S. Gordon, Breelyn A. Wilky, Agustin Pimentel, Daruka Mahadevan, Ani S. Balmanoukian, Rachel E. Sanborn, Gary K. Schwartz, Ghassan K. Abou-Alfa, Neil H. Segal, Bruno Bockorny, Justin C. Moser, Sunil Sharma, Jaymin M. Patel, Wei Wu, Dhan Chand, Katherine Rosenthal, Gabriel Mednick, Chloe Delepine, Tyler J. Curiel, ... Anthony B. El-Khoueiry + Show authors

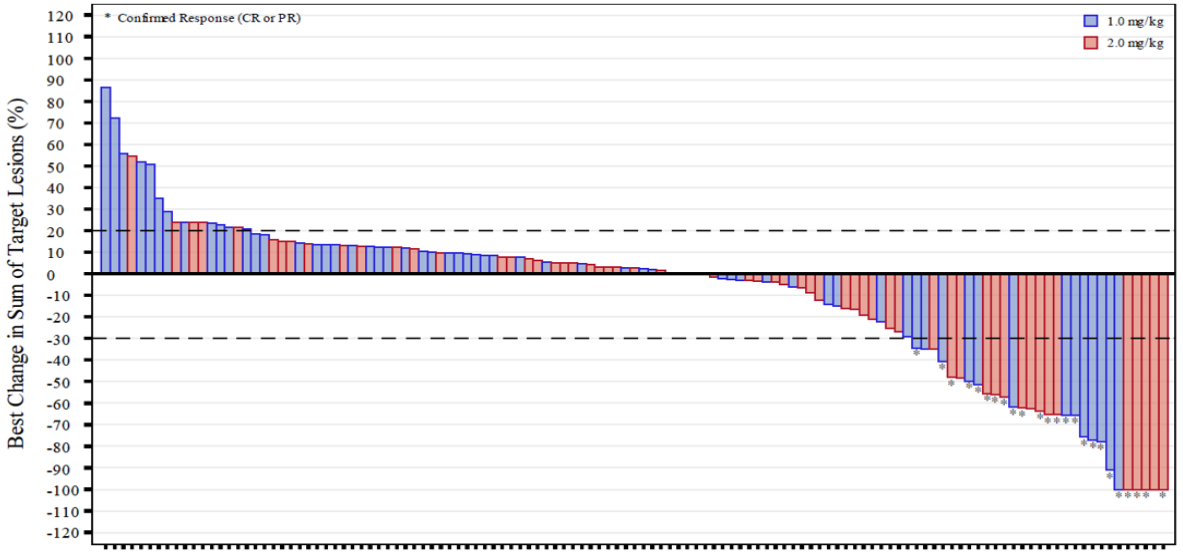
Nature Medicine **30**, 2558–2567 (2024) | Cite this article



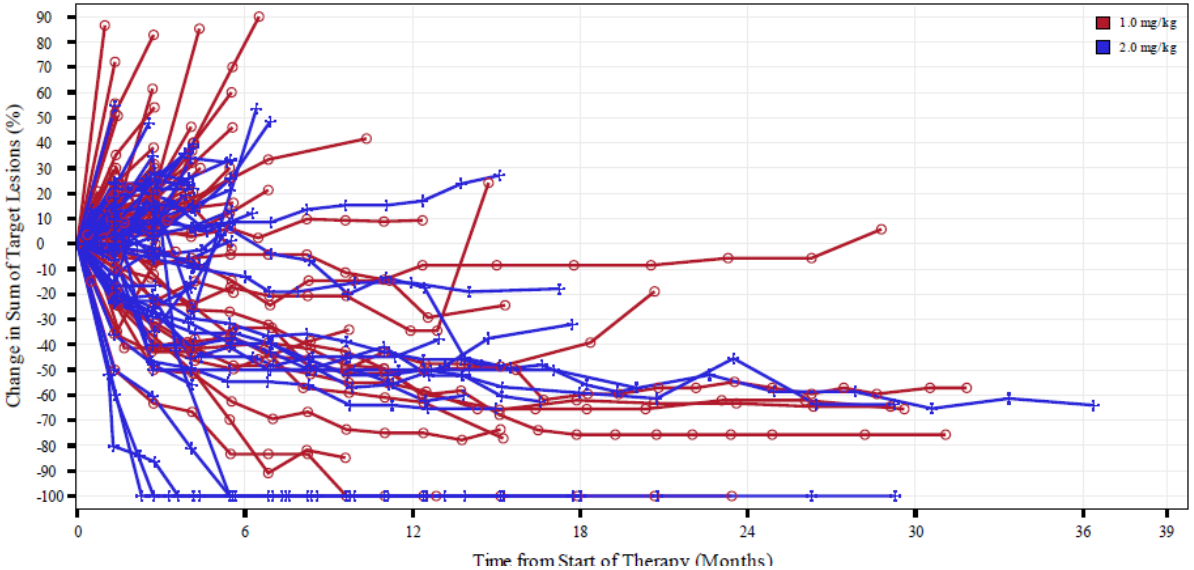
- Botensilimab is a potent CTLA4 mAb.
- CTLA4 is present on Tregs and MDSCs in the tumor microenvironment.
- The modified Fc portion may improve engagement of APCs.
- Importantly, the modified Fc receptor on botensilimab recruits NK cells.
- These modifications may result in clearance of Tregs and MDSCs with recruitment of NK cells to the tumor microenvironment.

Efficacy of Botensilimab with Balstilimab

Best overall response without active liver metastases



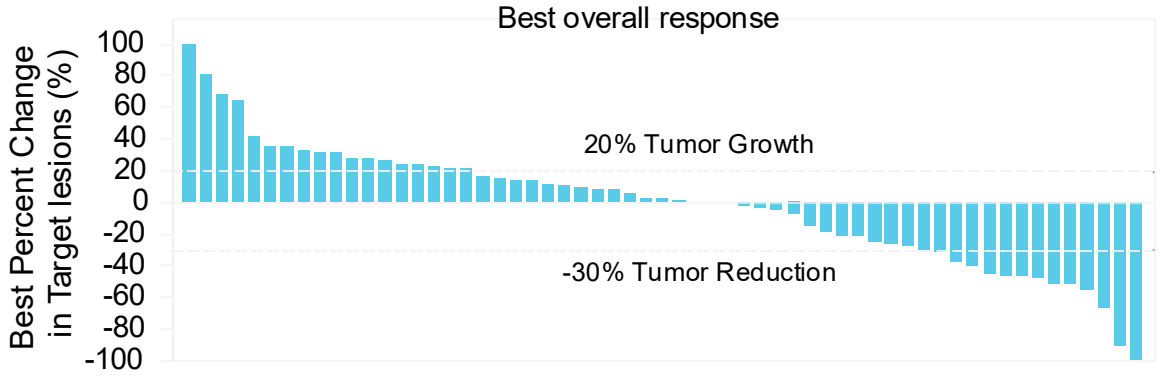
Response over time in patients without active liver metastases



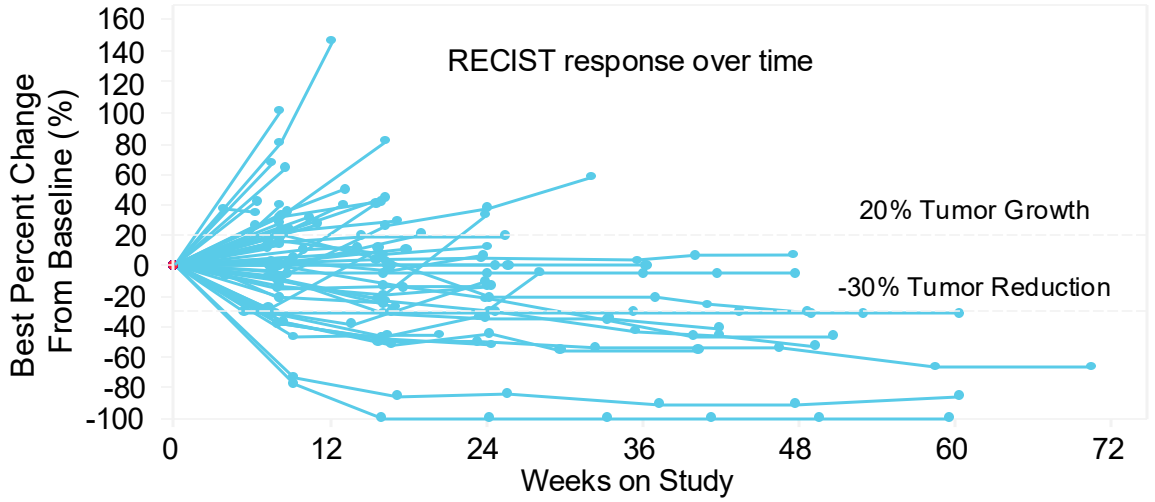
- This is a heavily treated patient population with exposure to prior 5-FU, oxaliplatin, irinotecan, bevacizumab, and guidelines-based monoclonal antibody therapy (EGFR, HER2, BRAF).
- 37 of 123 patients were also previously treated with the refractory regimens trifluridine/tipirical, reforafenib, and/or fruquintinib.

Randomized Phase 2 Trial of Botensilimab plus Balstilimab in Refractory CRC without Active Liver Metastases

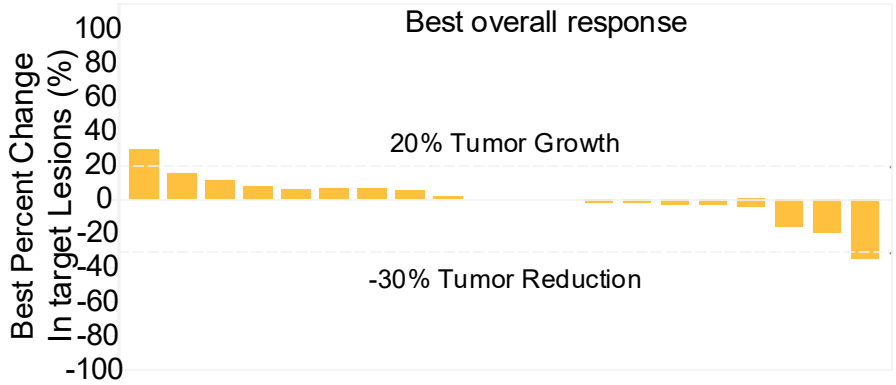
BOT 75 / BAL

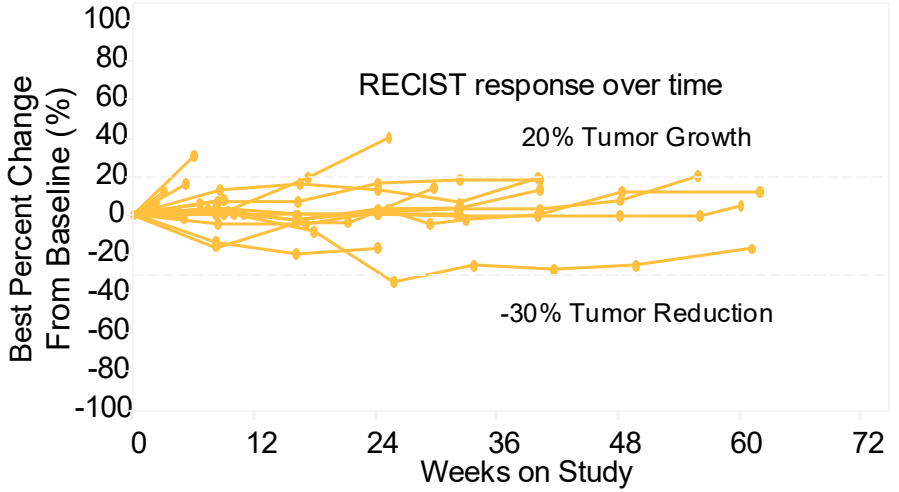


19% ORR 55% DCR



Trifluridine/Tipiracil (n=13) or Regorafenib (n=8)





Across investigational arms, **14/20 (70%)** of responses are ongoing

No objective responses in the chemotherapy arm

Checkpoint Immunotherapy in MSS Colorectal Cancer

- Approved checkpoint inhibitors are largely ineffective in MSS CRC, the most common type of CRC.
- Next generation CTLA4 antibodies such as botensilimab appear to be active in patients without active liver metastases.
- In refractory colorectal cancer, responses to chemotherapy are poor.
  - Objective response rates are below 10%
  - Progression free survival is generally less than 2 months
- Alternative approaches are needed to bring effective immunotherapy to patients with colorectal cancer, in particular those with active liver metastases (~80% of patients).

Immunotherapy Paradigms

- Pro-inflammatory drugs
  - IL-2, interferon
- Non-targeted immunotherapy
  - PD1/L1 inhibitors, CTLA4 inhibitors
- **Targeted immunotherapy**
  - BiTEs, TILs, TCR-T cells, **CAR-T cells**

Limitations of CAR-T Cell Therapies in Solid Tumors

- Targeting.
  - Requires a cancer specific target or a target that is sequestered from T cells
- Expansion.
  - For CD19 CAR-T cell therapies, circulating CAR-T cells rapidly expand due to target engagement
  - For desmoplastic and hypovascular tumors, CAR-T cell expansion may be limited
- Persistence.
  - Even when CAR-T cells expand, they may exhaust or simply die off due to lack of tonic signaling from on-target effect
- Coupled CAR-T cell therapy.
  - A second target that is highly expressed outside of the tumor may overcome these limitations in expansion and persistence of CAR-T cells in solid cancers

Target Selection in CAR Therapy

original article

See page 666

Case Report of a Serious Adverse Event Following the Administration of T Cells Transduced With a Chimeric Antigen Receptor Recognizing *ERBB2*

Richard A Morgan[1], James C Yang[1], Mio Kitano[1], Mark E Dudley[1], Carolyn M Laurencot[1] and Steven A Rosenberg[1]

[1]*Surgery Branch, National Cancer Institute, National Institutes of Health, Bethesda, Maryland, USA*

In an attempt to treat cancer patients with *ERBB2* overexpressing tumors, we developed a chimeric antigen receptor (CAR) based on the widely used humanized monoclonal antibody (mAb) Trastuzumab (Herceptin). An optimized CAR vector containing CD28, 4-1BB, and CD3ζ signaling moieties was assembled in a γ-retroviral vector and used to transduce autologous peripheral blood lymphocytes (PBLs) from a patient with colon cancer metastatic to the lungs and liver, refractory to

binding. *ERBB2* overexpression/amplification occurs in ~15–25% of human breast cancer patients, and is associated with more aggressive disease.[4] A proportion of other human cancers are also associated with *ERBB2* gene amplification and protein overexpression; including cancers of the colon, ovary, stomach, kidney, melanoma, and others.[5–7] Investigation of agents that target the *ERBB2* protein led to the development of Trastuzumab (Herceptin), a humanized monoclonal antibody (mAb) that binds to the extracellular domain of the receptor.[8] Trastuzumab has been shown

sion the patient experienced respiratory distress, and displayed a dramatic pulmonary infiltrate on chest X-ray. She was intubated and despite intensive medical intervention the patient died 5 days after treatment. Serum samples after cell infusion showed marked increases in interferon-γ (IFN-γ), granulocyte macrophage-colony stimulating factor (GM-CSF), tumor necrosis factor-α (TNF-α), interleukin-6 (IL-6), and IL-10, consistent with a cytokine storm. We speculate that the large number of administered cells localized to the lung immediately following infusion and were triggered to release cytokine by the recognition of low levels of ERBB2 on lung epithelial cells.

Received 14 January 2010; accepted 22 January 2010; published online 23 February 2010. doi:10.1038/mt.2010.24

Targeting, Expansion, and Persistence

- HER2 expression on normal tissue is accessible to T-cells, which confers a greater risk of on-target off-tumor toxicity.
- Claudin18.2 is sequestered in the intracellular tight junction, so may be a safer target.

nature medicine

ARTICLES

https://doi.org/10.1038/s41591-022-01800-8

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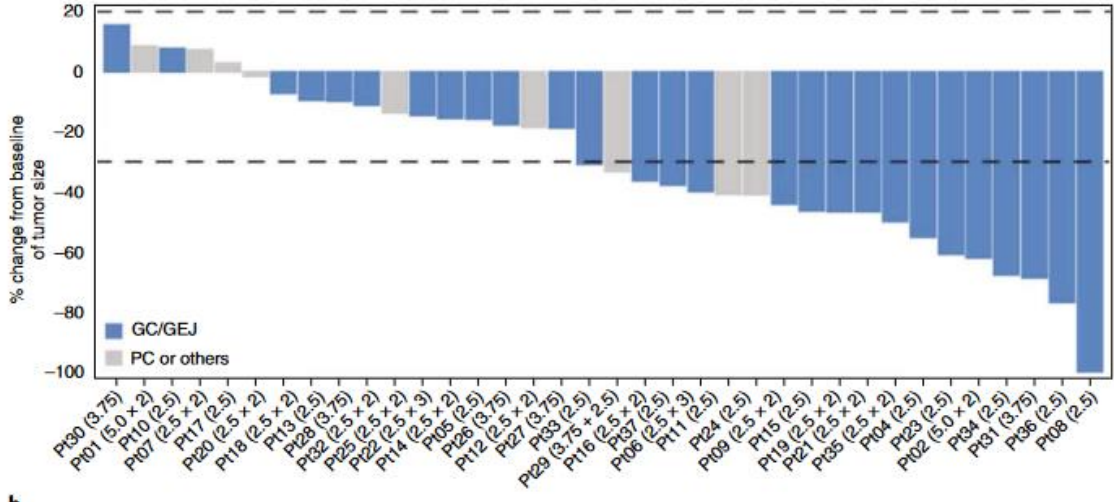
OPEN

Claudin18.2-specific CAR T cells in gastrointestinal cancers: phase 1 trial interim results

Changsong Qi[1,5], Jifang Gong[1,5], Jian Li[1,5], Dan Liu[2], Yanru Qin[3], Sai Ge[1], Miao Zhang[2], Zhi Peng[1], Jun Zhou[1], Yanshuo Cao[1], Xiaotian Zhang[1], Zhihao Lu[1], Ming Lu[1], Jiajia Yuan[1], Zhenghang Wang[1], Yakun Wang[2], Xiaohui Peng[4], Huiping Gao[4], Zhen Liu[4], Huamao Wang[4], Daijing Yuan[4], Jun Xiao[4], Hong Ma[4], Wei Wang[4], Zonghai Li[4] and Lin Shen[1]✉

Despite success in hematologic malignancies, the treatment landscape of chimeric antigen receptor (CAR) T cell therapy for solid tumors remains limited. Claudin18.2 (CLDN18.2)-redirected CAR T cells showed promising efficacy against gastric cancer (GC) in a preclinical study. Here we report the interim analysis results of an ongoing, open-label, single-arm, phase 1 clinical trial of CLDN18.2-targeted CAR T cells (CT041) in patients with previously treated, CLDN18.2-positive digestive system cancers (NCT03874897). The primary objective was safety after CT041 infusion; secondary objectives included CT041 efficacy, pharmacokinetics and immunogenicity. We treated 37 patients with one of three CT041 doses: 2.5×10^8, 3.75×10^8 or 5.0×10^8 cells. All patients experienced a grade 3 or higher hematologic toxicity. Grade 1 or 2 cytokine release syndrome (CRS) occurred in 94.6% of patients. No grade 3 or higher CRS or neurotoxicities, treatment-related deaths or dose-limiting toxicities were reported. The overall response rate (ORR) and disease control rate (DCR) reached 48.6% and 73.0%, respectively. The 6-month duration of response rate was 44.8%. In patients with GC, the ORR and DCR reached 57.1% and 75.0%, respectively, and the 6-month overall survival rate was 81.2%. These initial results suggest that CT041 has promising efficacy with an acceptable safety profile in patients with heavily pretreated, CLDN18.2-positive digestive system cancers, particularly in those with GC.

Targeting, Expansion, and Persistence



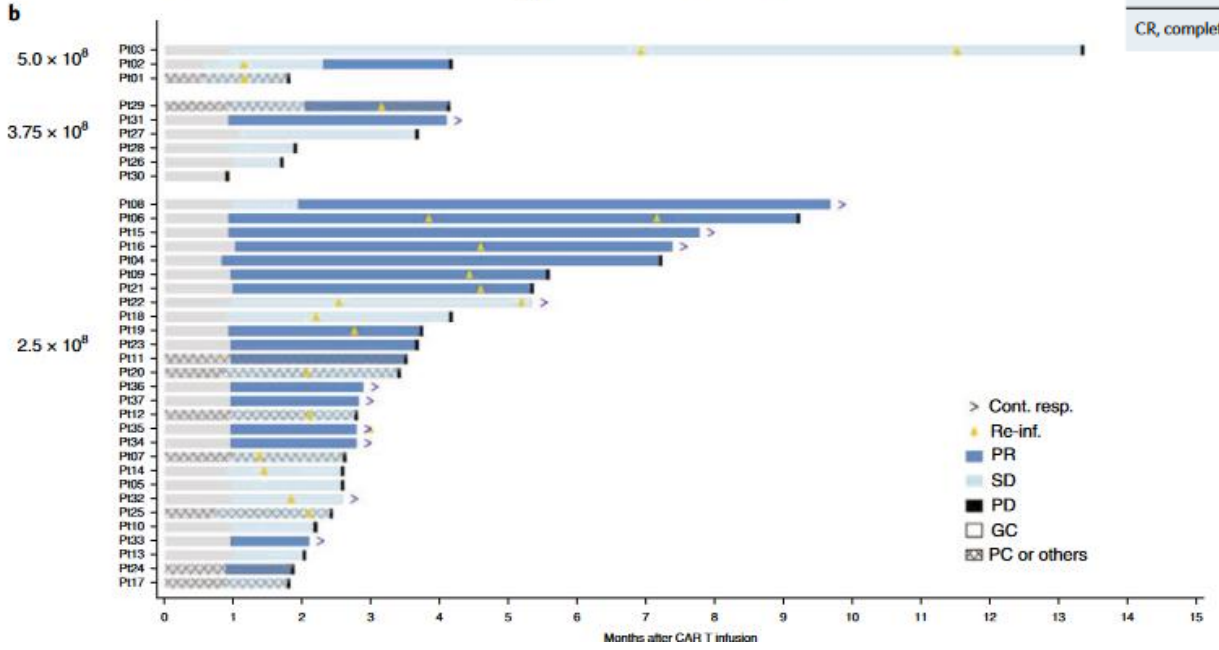


Table 3 | Efficacy evaluation according to tumor type*

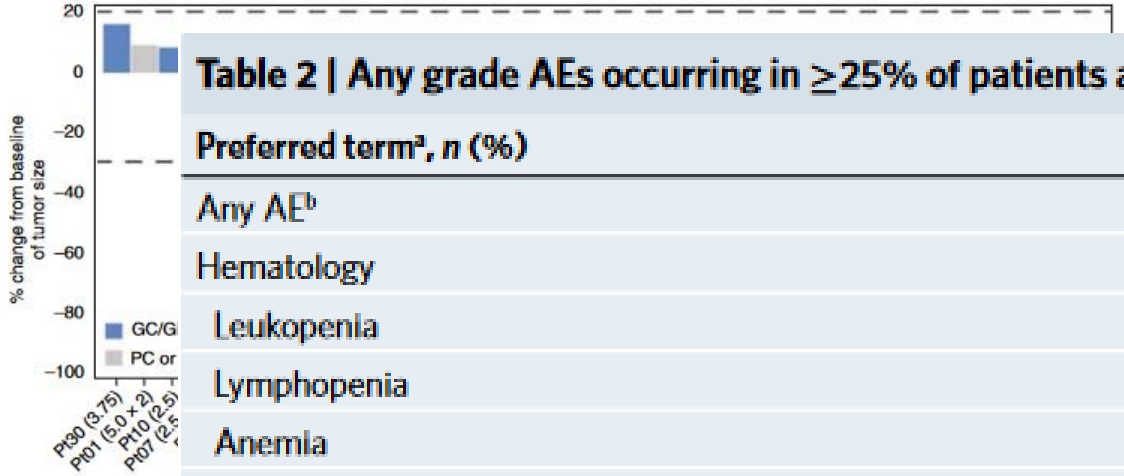
Variable	Gastric *n* = 28	Other *n* = 9	All *n* = 37
Best overall response			
CR, *n* (%)	0	0	0
PR, *n* (%)	16 (57.1)	2 (22.2)	18 (48.6)
SD, *n* (%)	5 (17.9)	4 (44.4)	9 (24.3)
PD, *n* (%)	7 (25.0)	3 (33.3)	10 (27.0)
ORR, *n* (%) [95% CI]	16 (57.1) [37.2, 75.5]	2 (22.2) [2.8, 60.0]	18 (48.6) [31.9, 65.6]
DCR, *n* (%) [95% CI]	21 (75.0) [55.1, 89.3]	6 (66.7) [29.9, 92.5]	27 (73.0) [55.9, 86.2]
mPFS (months) [95% CI]	4.2 [3.7, 9.2]	2.6 [1.8, 3.5]	3.7 [2.6, 5.4]
6-month OS rate (%) [95% CI]	81.2 [60.3, 91.8]	77.8 [36.5, 93.9]	80.1 [62.5, 90.0]
6-month DOR rate (%) [95% CI]	53.3 [20.7, 77.8]	NA	44.8 [17.3, 69.3]

CR, complete response; NA, not applicable. *Tumor response was confirmed based on investigator assessment according to RECIST version 1.1.

Concerns about toxicity, cell amplification, and duration of response.

However, this is a promising approach with a well validated target.

Targeting, Expansion, and Persistence



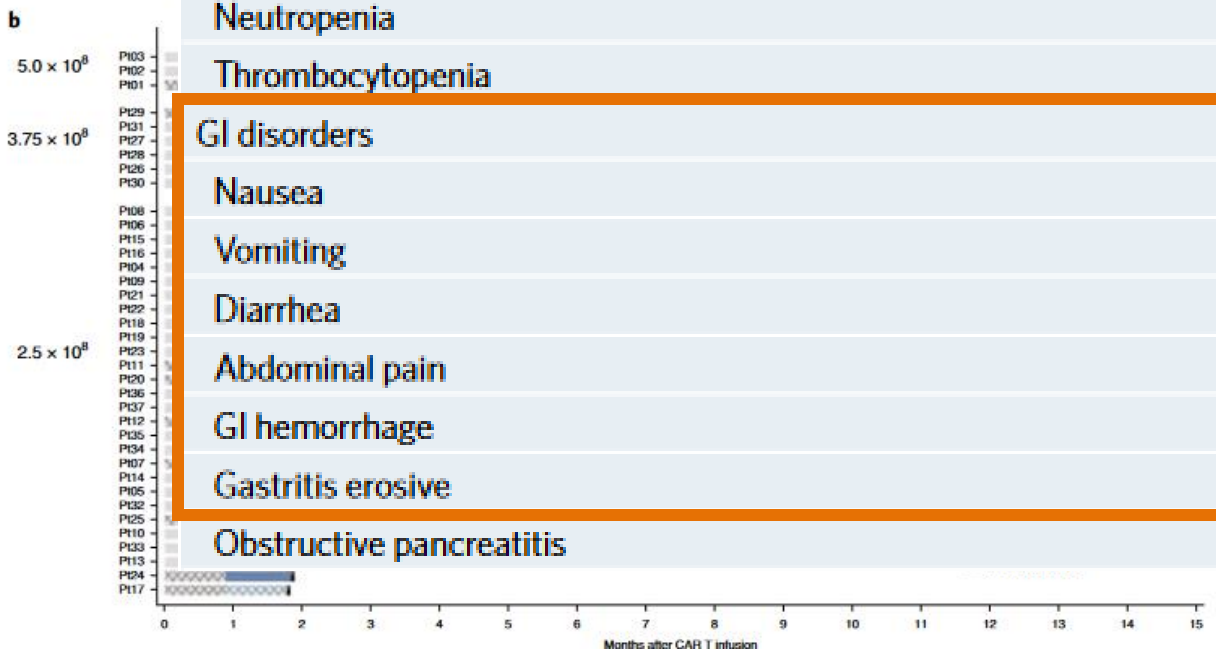
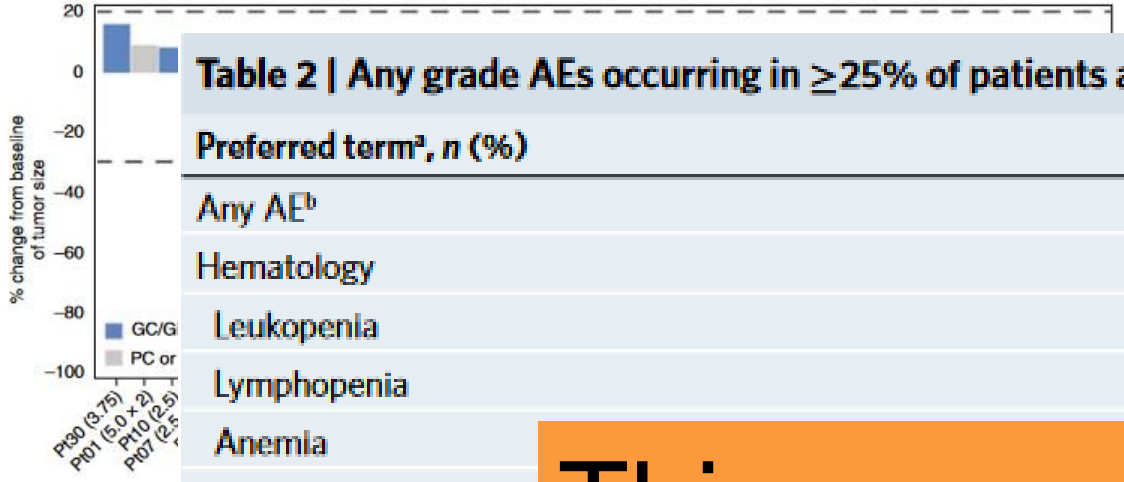


Table 2 | Any grade AEs occurring in ≥25% of patients and all grade 3 or 4 AEs

Preferred term[a], *n* (%)	Grade 3 or 4	Any	All *n* = 37
Any AE[b]	37 (100)	37 (100)	0
Hematology	37 (100)	37(100)	18 (48.6)
Leukopenia	31 (83.8)	37 (100)	9 (24.3)
Lymphopenia	37 (100)	37 (100)	10 (27.0)
Anemia	15 (40.5)	36 (97.3)	18 (48.6) [31.9, 65.6]
Neutropenia	25 (67.6)	34 (91.9)	27 (73.0) [55.9, 86.2]
Thrombocytopenia	6 (16.2)	22 (59.5)	3.7 [2.6, 5.4]
GI disorders	4 (10.8)	31 (83.8)	80.1 [62.5, 90.0]
Nausea	0	18 (48.6)	44.8 [17.3, 69.3]
Vomiting	0	13 (35.1)	
Diarrhea	0	10 (27.0)	
Abdominal pain	1 (2.7)	9 (24.3)	
GI hemorrhage	1 (2.7)	2 (5.4)	
Gastritis erosive	1 (2.7)	1 (2.7)	
Obstructive pancreatitis	1 (2.7)	1 (2.7)	

Targeting, Expansion, and Persistence



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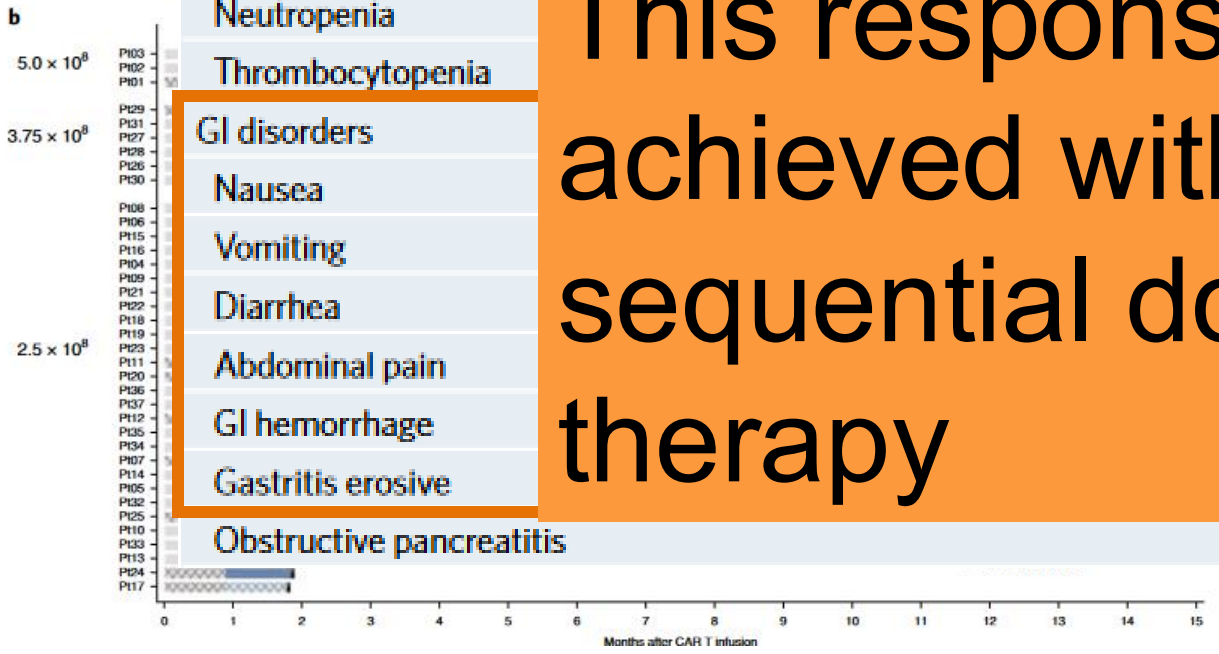
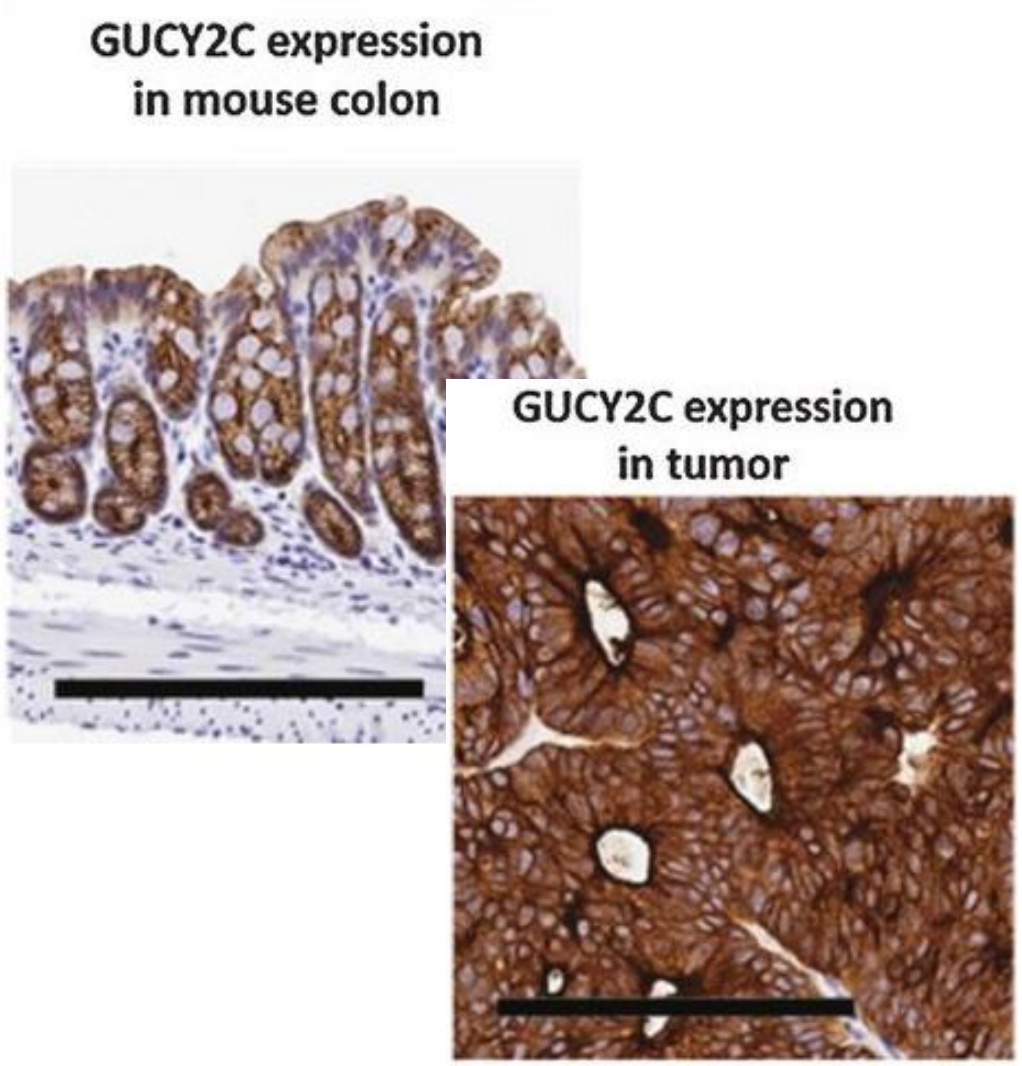
Lymphopenia	37 (100)	37 (100)	10 (27.0)
Anemia			18 (48.6) [31.9, 65.6]
Neutropenia			27 (73.0) [55.9, 86.2]
Thrombocytopenia			3.7 [2.6, 5.4]
GI disorders			80.1 [62.5, 90.0]
Nausea			44.8 [17.3, 69.3]
Vomiting			
Diarrhea			
Abdominal pain			
GI hemorrhage			
Gastritis erosive			
Obstructive pancreatitis	1 (2.7)	1 (2.7)	



This response rate was achieved with up to 3 sequential doses of CAR therapy

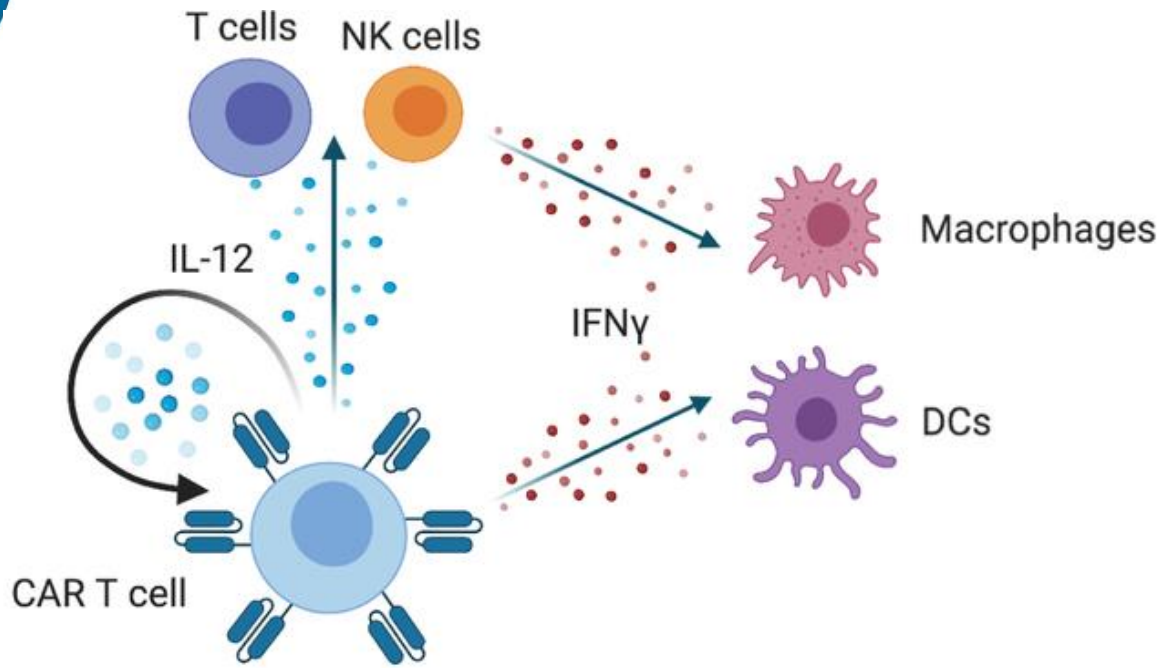
Targeting: Guanylate Cyclase C (GCC) in Colorectal Cancer



- GCC is a common marker of normal colon and colon cancer.
- Because GCC is a luminal marker, it's expression may be sequestered from the immune system.
- Aberrant expression is common on colorectal cancer.
- Biomarker is not yet validated.
- Over 95% of cancers were positive in this trial.

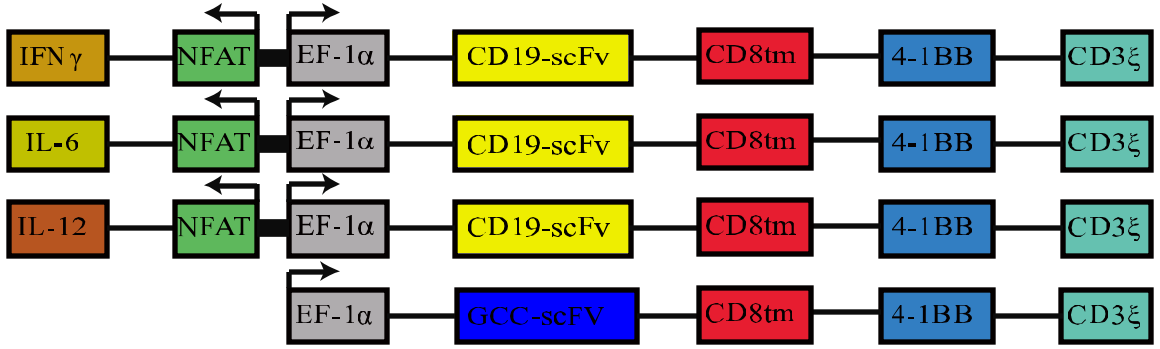
Clin Cancer Res. 2020 May 1;26(9):2188-2202

GCC19CAR-T cell design and function



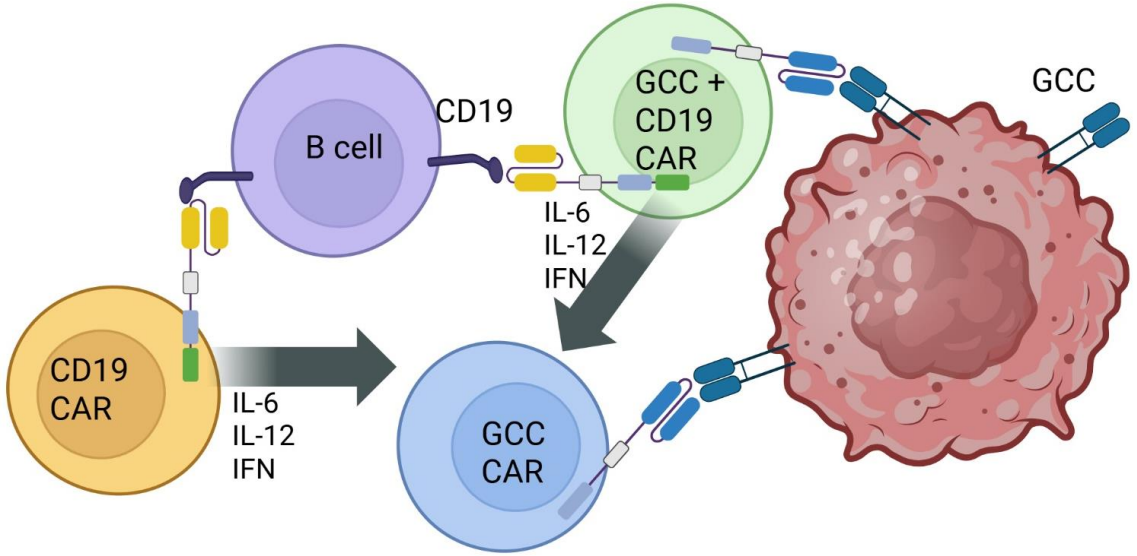
- Preclinical models show that IFN, IL-6, and IL-12 are critical to CAR-T cell function in desmoplastic tumors.
- CD19 is a well established CAR target and CD19+ve B cells regenerate in approximately 6 months following CAR therapy.
- CD19 can use used as “fuel” for CAR expansion and cytokine generation.

- Cytokine armoring may promote both local CAR-T cell activity as well as systemic expansion and activation.
- Importantly, recruitment of macrophages and APCs may generate a more pleomorphic immune response.



Schlechter BL, ESMO GI 2025
Bell M, Front. Immunol., 03 June 2021

GCC19CAR-T cell design and function



- Preclinical models show that IFN, IL-6, and IL-12 are critical to CAR-T cell function in desmoplastic tumors.
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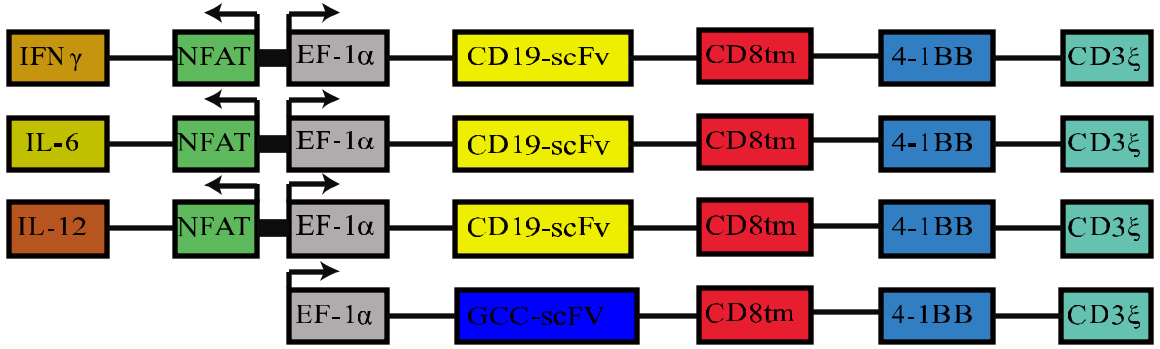

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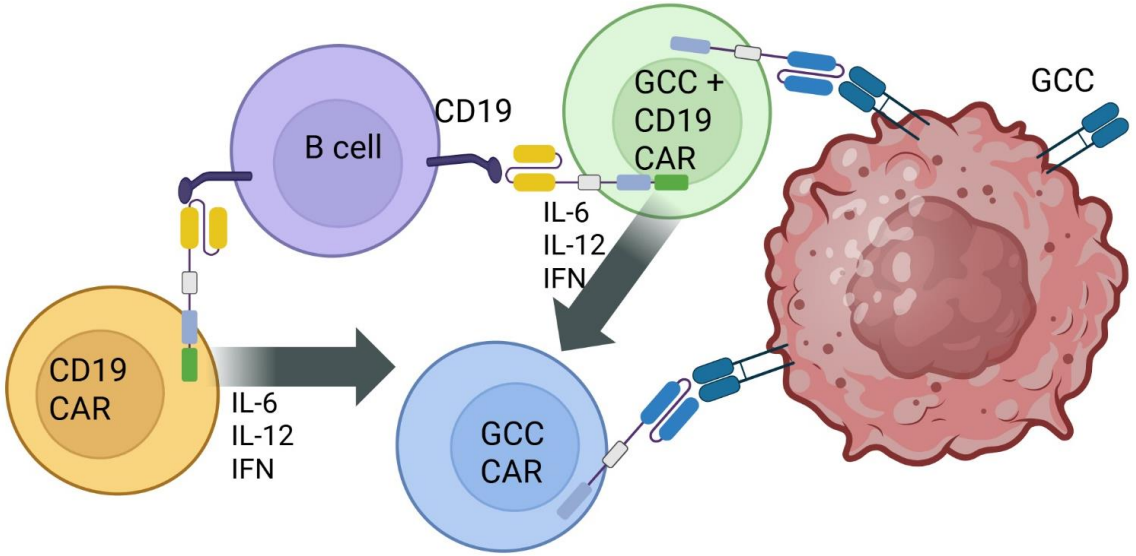
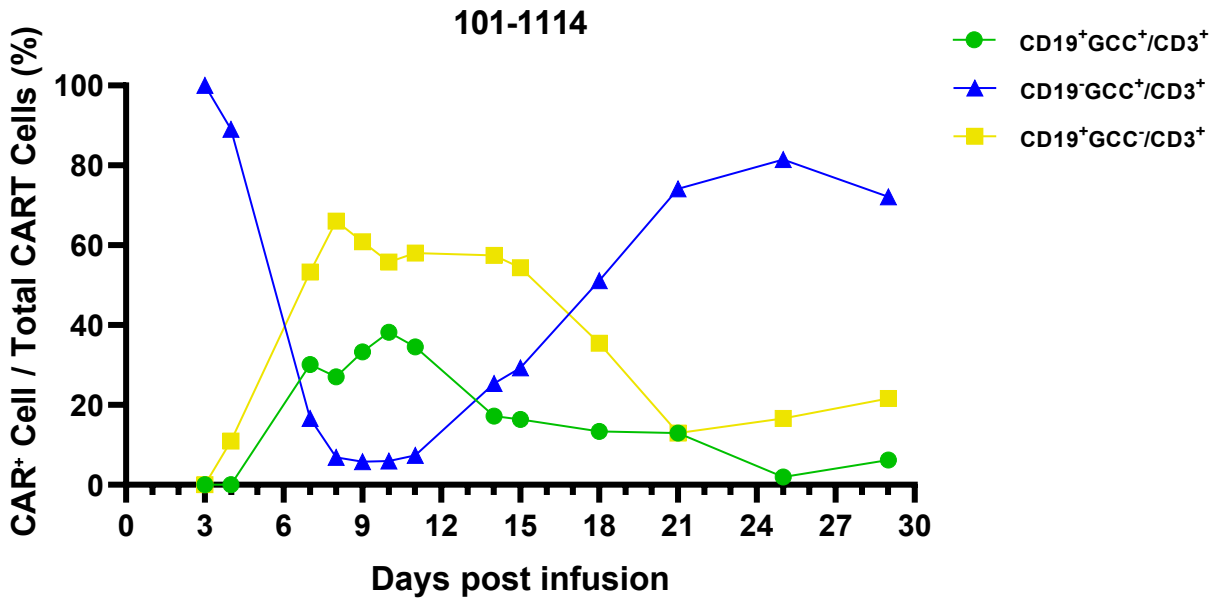
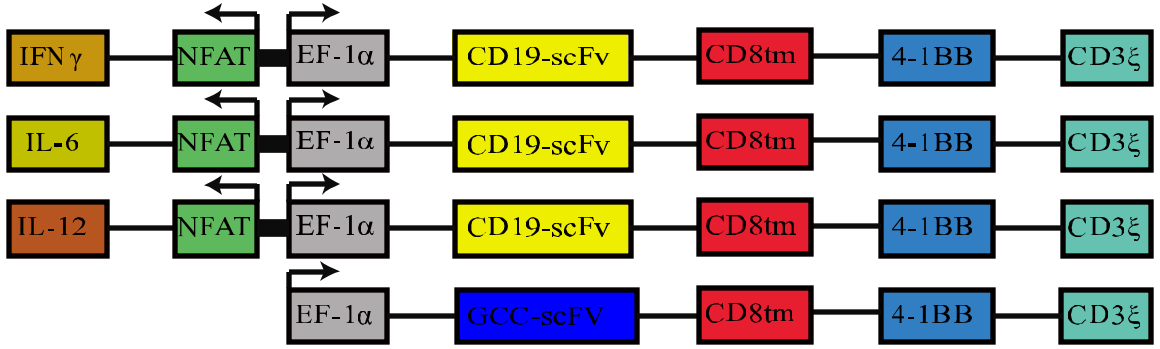




- Each collection is transfected with 4 separate vectors.
- The vast majority of the CAR-T cells are GCC CAR-T.
- A minority are CD19 CAR-T.
- A very small number are dual GCC+CD19 CAR-T cells.
- These dual targeting CAR-T cells may support efficacy.



GCC19CAR-T Patient Characteristics

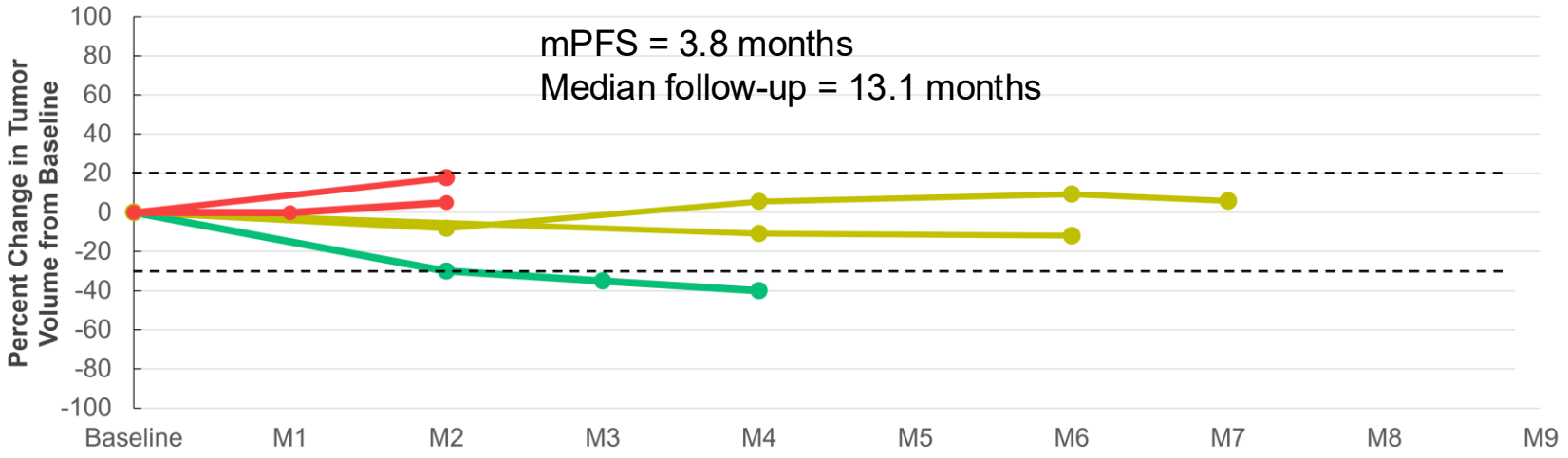
Characteristics	Dose level 1 N=5	Dose level 2 N=5	Overall N=10
Age (range)			
Median	43 (39-48)	53 (46-57)	48 (39-57)
Sex, n (%)			
Male	3 (60%)	3(60%)	6(60%)
Female	2 (40%)	2(40%)	4(40%)
Prior lines of therapy, n (range)			
Median	3 (2-6)	2 (2-5)	3 (2-6)
Origin of Disease, n (%)			
Left Colon	2 (40%)	2 (40%)	4 (40%)
Right Colon	0	0	0
Rectosigmoid	1 (20%)	1 (20%)	2 (20%)
Rectum	2 (40%)	2 (40%)	4 (40%)
Disease Sites, n (range)			
Median	2 (1-4)	3 (1-4)	2 (1-4)
Mutations, n (%)			
RAS mutated	4 (80%)	3 (60%)	7 (70%)

GCC19CAR-T Events and Outcomes

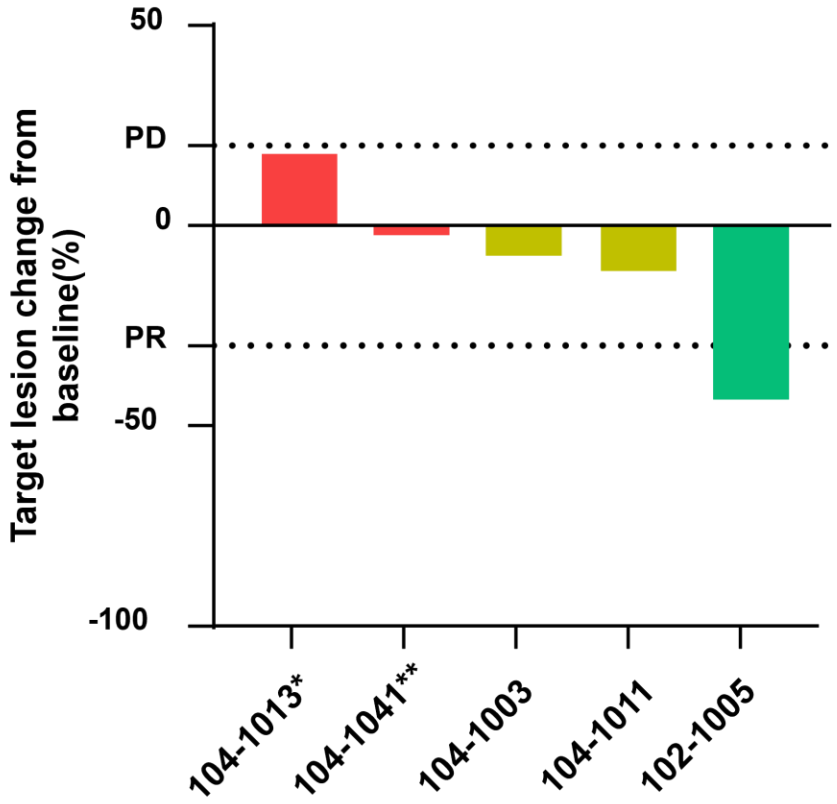
Characteristics	Dose level 1 N=5	Dose level 2 N=5	Overall N=10
Related Adverse Events, n (grades)			
CRS	5 (1-2)	5 (1-2)	10 (1-2)
ICANS	0	2 (1,3)	2 (1,3)
Diarrhea	4 (1-3)	5 (1-3)	9 (1-3)
Sepsis - related to on target colitis, immune suppression	0	**1 (5)**	**1 (5)**
Outcomes			
Median Follow-up, months	13.1	11.8	12.5
Median Progression Free Survival, months	3.8	7.8	3.8
Best Response, n (%)			
CR	0	1(20%)	1(10%)
PR	1 (20%)	3 (60%)	4(40%)
SD	2 (40%)	0	2 (20%)
PD	2 (40%)	1 (20%)	3 (30%)
ORR (CR + PR)[1]	**1 (20%)**	**4 (80%)**	**5 (50%)**
DCR (CR+PR+SD)	**3 (60%)**	**4 (80%)**	**7 (70%)**

GCC19CAR-T Responses, 1 x 10^6 cells/kg

Response over time by RECIST1.1



Best response by RECIST1.1



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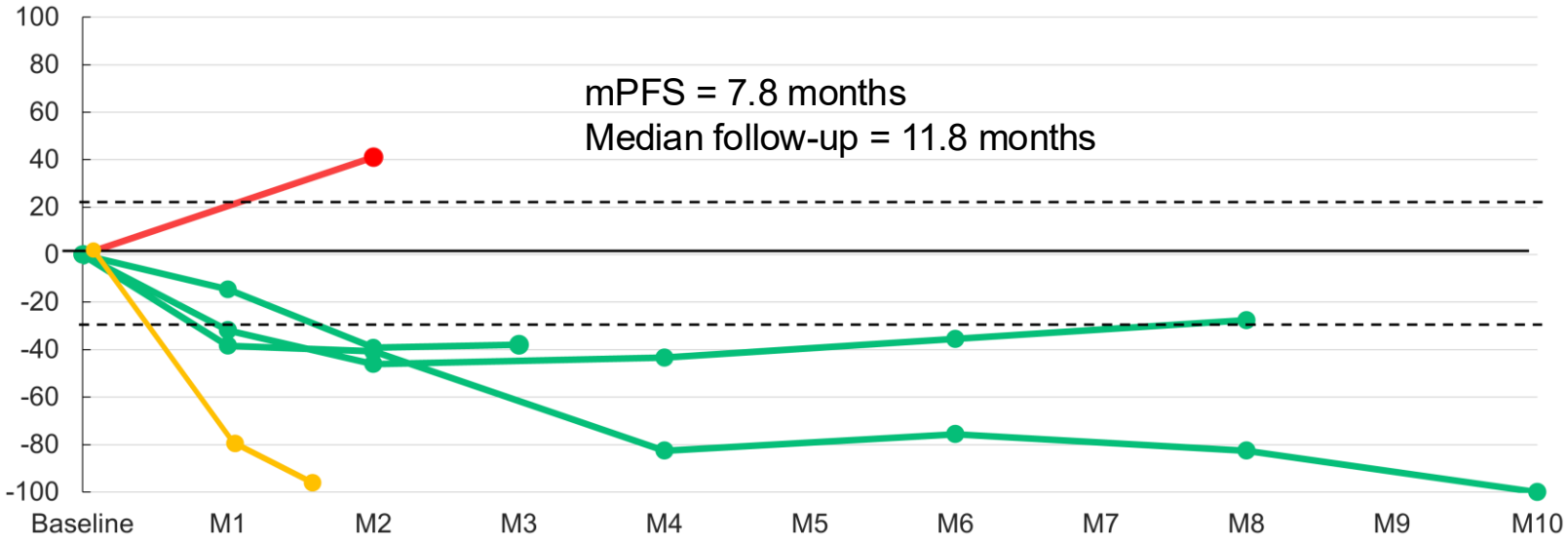
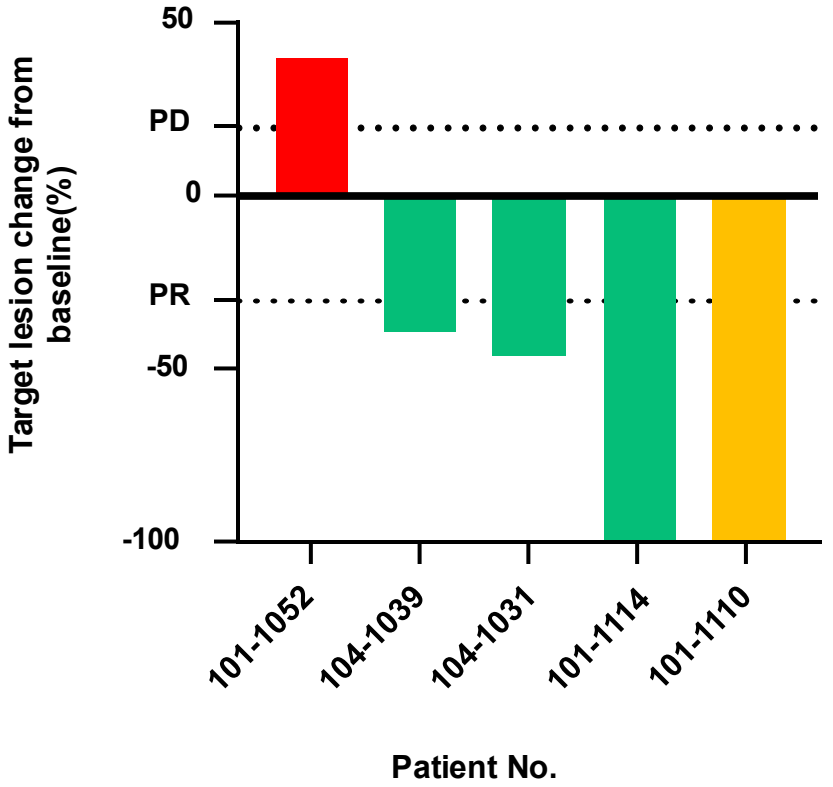
GCC19CAR-T Responses, 2 x 10^6 cells/kg

Response over time by RECIST1.1

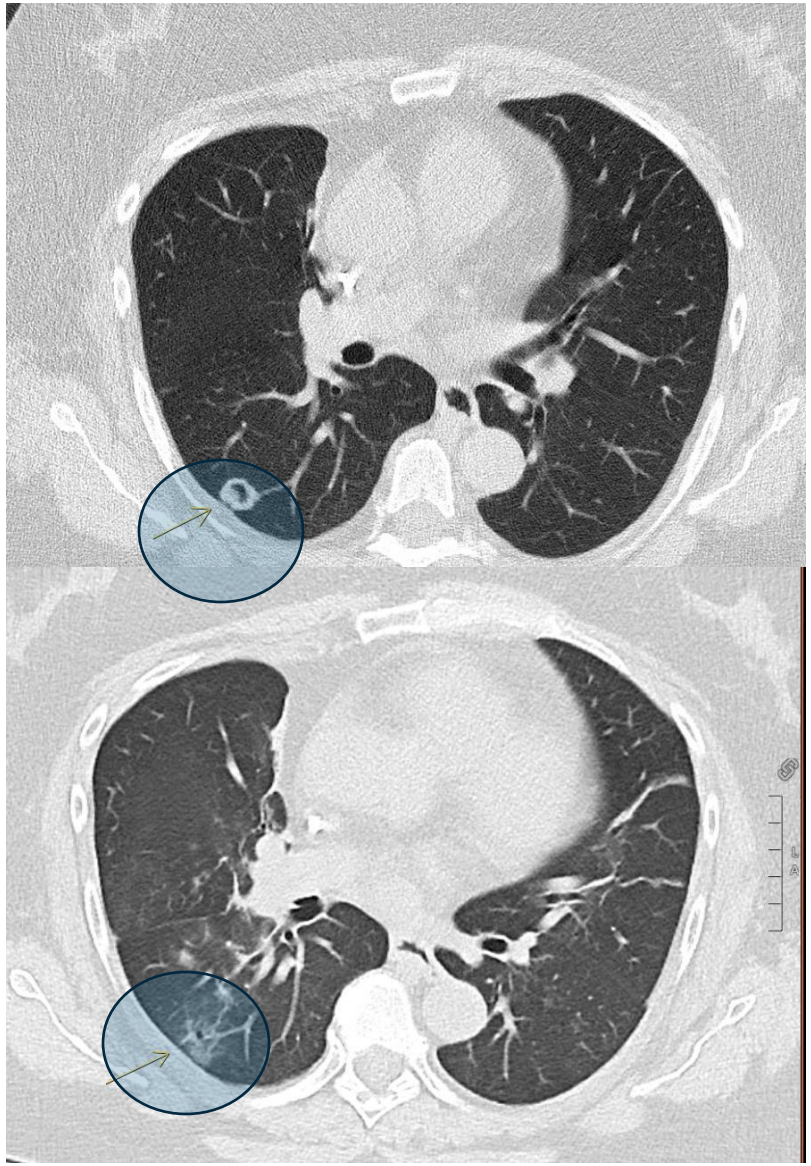


Best response by RECIST1.1



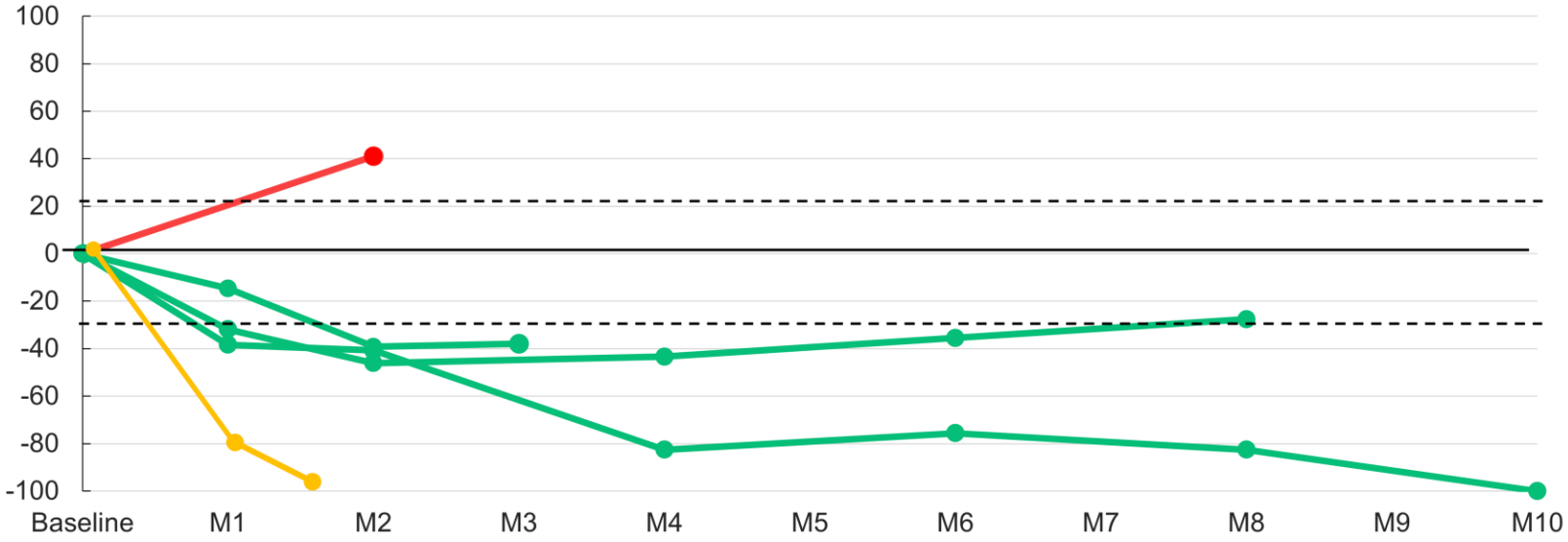
GCC19CAR-T Cell Patient with PR, 101-1114

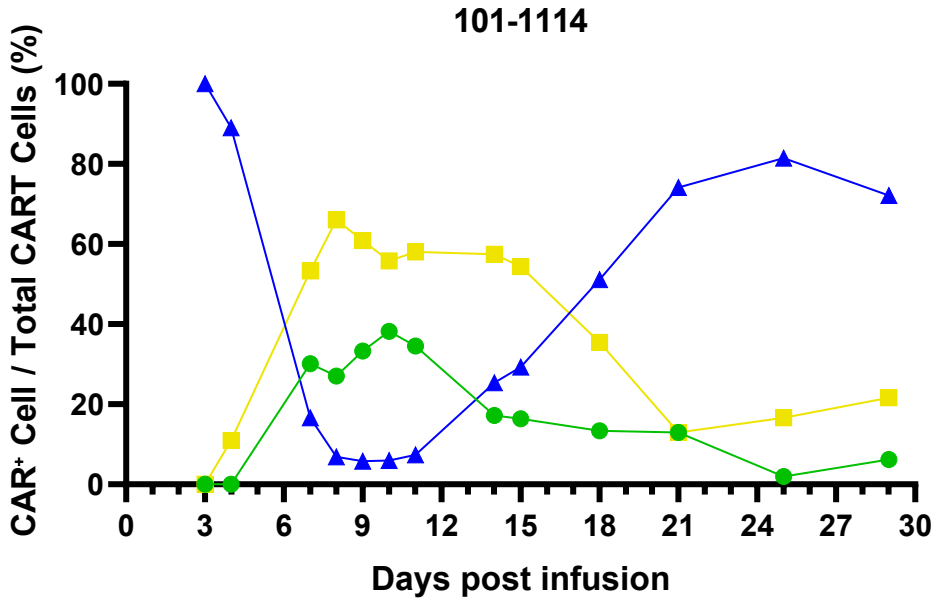
- 54F with metastatic colorectal cancer, MSS and *KRAS* G12D.
- Received FOLFOXIRI with bevacizumab followed by hepatectomy.
- Developed recurrent liver and lung metastases in less than six months from resection.
- Received subsequent FOLFIRI with bevacizumab, FOLFOX with bevacizumab, and Lonsurf with bevacizumab.
- Underwent GCC19CAR-T cell infusion of $2x10^6$ cell/kg with course complicated by persistent G1 CRS and G1 colitis.
- Discharged home on D+15.
- Partial response by RECIST1.1 at D+29.



GCC19CAR-T Cell Patient with PR, 101-1114

Response over time by RECIST1.1





GCC19CAR-T Cell Patient with PR, 101-1114

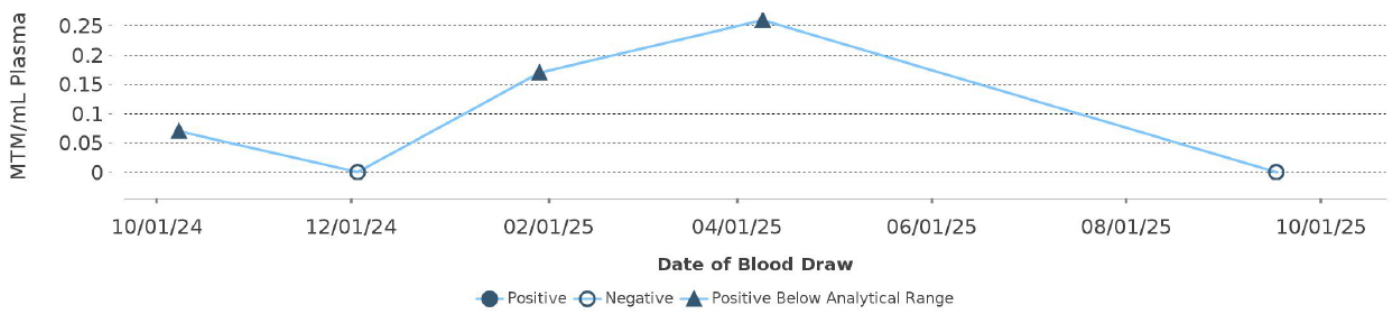
FINAL RESULTS SUMMARY

Signatera Negative

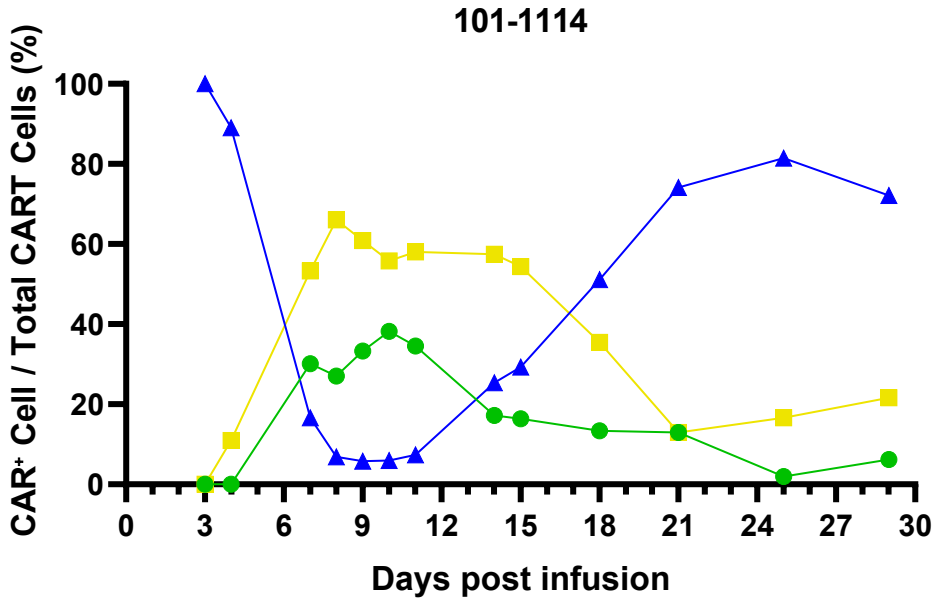
MTM/mL: Not Detected

Mean tumor molecules per mL is calculated based on the mean of ctDNA molecules detected per mL of the patient's plasma. See Limitations section below.

Historical Results



Date	Reported MTM/mL
Sep 17, 2025	0.00
Apr 09, 2025	0.26
Jan 29, 2025	0.17
Dec 03, 2024	0.00
Oct 08, 2024	0.07



On-Target Toxicity

- Solid tumors are deeply similar to normal tissue.
- Ultimately, on-target toxicity is the main driver of risk in solid tumor CAR therapy (CARvHD).
- Toxicity management strategies are key.
- Logic gating may be useful.

GCC19CAR-T Toxicity Management

- 101-1110 died of sepsis due to on-target colitis, CARvHD.
- Since that G5 SAE, patients received prophylactic vedolizumab starting D+3 followed by infliximab on D+5 with PO/PR budesonide.
- Grading of “diarrhea” may underestimate colitis –especially in colorectal cancer patients. Quantifying and qualifying stool appears to provider a better assessment of CARvHD.

GCC19CAR-T Cell Therapy Conclusions

- GCC is a promising target for CAR-T cell therapy in refractory colorectal cancer with moderate volume disease.
- Dual targeting with CD19 may overcome known limitations of CAR-T cell therapy in solid tumors by promoting expansion and persistence.
- Co-stimulatory cytokines may support expansion and efficacy.
- CRS and ICANs can be well managed with standard treatments.
- Enterocolitis management requires ongoing investigation to prevent mucosal injury and secondary infections.
- Efficacy is promising in a heavily pretreated patient population with moderate volume refractory colorectal cancer.

Thank you!

- Dr. Kimmie Ng
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- Dr. Sarah Nikiforrow
- Dr. Caron Jacobson
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- IEC Research Nurses and CRCs
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- Cell Manufacturing Core Facility
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- Our collaborators at ICT
- **Our patients and their families**