Allogeneic CD70-Targeted Chimeric Antigen Receptor T-Cell Therapy for Advanced Renal Cell Carcinoma: Results From the Phase I TRAVERSE Trial

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ABSTRACT

PURPOSE Treatment options for clear cell renal cell carcinoma (ccRCC) refractory to immune checkpoint inhibitors (ICIs) and targeted therapy are needed. ALLO-316 is an allogeneic chimeric antigen receptor (CAR) T-cell product that targets CD70-expressing ccRCC and resists immune rejection by targeting and eliminating patients' $CD70^+$ alloreactive T cells.

METHODS In the phase Ia/b TRAVERSE trial (ClinicalTrials.gov identifier: NCT04696731), patients with advanced ccRCC resistant to ICIs and vascular endothelial growth factor receptor–targeted therapy received lymphodepletion and ALLO-316 following a modified 3 + 3 design. Phase Ia evaluated ALLO-316 dose and fludarabine/cyclophosphamide (FC)–based lymphodepletion with/without the anti-CD52 antibody, ALLO-647. Additional patients were enrolled in phase Ib to confirm the expansion regimen identified in phase Ia. Primary end points were dose-limiting toxicities (DLTs) and adverse events (AEs).

RESULTS Fifty-one patients were enrolled (phase Ib, n = 23). Patients received a median of four prior lines of therapy. The median follow-up was 28.8 months. DLTs occurred in two patients who received ALLO-647 (grade 3 autoimmune hepatitis; grade 5 cardiogenic shock). Grade ≥3 cytokine release syndrome, immune effector cell–associated neurotoxicity syndrome, and immune effector cell–associated hemophagocytic lymphohistiocytosis-like syndrome occurred in 2.0%, 0%, and 6.0% of patients, respectively. Most grade ≥3 AEs were hematologic (neutropenia 62.0%; white blood cell count decreased 54.0%; anemia 36.0%). In phase Ib, patients received FC and 80×10^6 CAR T cells. The objective response rate was 17.4% overall, 25.0% in phase Ib patients and 31.3% in phase Ib patients with CD70 tumor proportion score ≥50%.

CONCLUSION ALLO-316 had manageable safety and encouraging antitumor activity in CD70-positive ccRCC. The TRAVERSE trial demonstrates proof of concept for the role of allogeneic CAR T-cell therapy in solid tumors.

ACCOMPANYING CONTENT

Data Sharing Statement

Data Supplement

Protocol

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INTRODUCTION

First-line treatment for advanced clear cell renal cell carcinoma (ccRCC) is immune checkpoint inhibitors (ICIs) with or without vascular endothelial growth factor receptor (VEGFR) tyrosine kinase inhibitors (TKIs).[1,2] However, most patients experience disease progression.[3] Treatments for ccRCC after ICIs include VEGFR TKIs or hypoxia-inducible factor 2α (HIF-2α) inhibitors such as belzutifan, but response rates are generally <25%.[1,4-6]

CD70 is an immunoregulatory molecule of the tumor necrosis factor family, normally restricted to activated T and B cells and dendritic and thymic stromal cells.[7] As the ligand for CD27—expressed predominantly on T cells—CD70-CD27 engagement serves as a driver of T-cell expansion, survival, and differentiation.[7] Aberrant CD70 expression is observed across multiple cancers, where it promotes immune evasion and tumor progression.[8] Approximately 80% of RCCs express CD70, with the highest seen in ccRCC.[9-11] CD70 expression is associated with poorer outcomes with ICIs.[12,13]

CONTEXT

Key Objective

Is ALLO-316, a CD70-targeted allogeneic chimeric antigen receptor (CAR) T-cell product, safe and effective for the treatment of advanced clear cell renal cell carcinoma (ccRCC)?

Knowledge Generated

The results of the TRAVERSE study showed that ALLO-316 had toxicity that was manageable with tailored therapy and showed encouraging antitumor activity in patients with heavily pretreated ccRCC and high CD70 expression.

Relevance *(A. Necchi)*

This study expands our knowledge on salvage therapeutic options for patients with advanced ccRCC that is refractory to standard therapies. In a field dominated by the use of tyrosine kinase inhibitors, the use of allogeneic CAR T-cell therapy provides a feasible move beyond the use of immune-checkpoint inhibitors, and tumor biomarker selection may enable a salvage therapy personalization in this field.*

*Relevance section written by *JCO* Associate Editor Andrea Necchi, MD.

Other than immune cells, CD70 has limited expression in normal tissues, making it an attractive target for chimeric antigen receptor (CAR) T-cell therapy.[7,14-16]

CAR T-cell therapy has been transformative in hematologic malignancies; however, its use in solid tumors has been limited by challenges with trafficking, infiltration, immunoediting, and immunosuppressive tumor microenvironments.[17-19] Patient-specific manufacturing of autologous CAR T cells also imposes limitations on access. Use of healthy donor-derived CAR T cells would help address this, but has been limited by premature allorejection.

ALLO-316 is an off-the-shelf, human leukocyte antigen (HLA)–unmatched, allogeneic CAR T-cell product designed to treat CD70-positive cancers. Product features include elimination of T-cell receptor expression via *TRAC* disruption and knockout of CD52. These modifications, performed using TALEN gene-editing technology, prevent graft-versus-host disease (GVHD) and enable concomitant use of an anti-CD52 antibody, ALLO-647, during lymphodepletion (Data Supplement, Fig S1, online only).[20] ALLO-316 was designed to target both CD70-expressing tumor cells and alloreactive T cells that could suppress CAR T-cell expansion and persistence. Preclinical development showed potent antitumor activity in RCC cells and xenograft models.[20] TRAVERSE (ClinicalTrials.gov identifier: NCT04696731) was designed to evaluate the safety and efficacy of ALLO-316 in advanced or metastatic ccRCC.

METHODS

Study Design

TRAVERSE is a first-in-human, open-label, multicenter, phase Ia/b study. Phase Ia comprised a modified 3 + 3 dose escalation to establish the phase Ib regimen. TRAVERSE was conducted in accordance with Good Clinical Practice guidelines and the principles derived from the Declaration of Helsinki and was approved by appropriate institutional review boards and regulatory agencies. All patients provided written informed consent.

Study Population

Eligibility criteria for phase Ia and Ib included age ≥18 to <75 years, histologically confirmed advanced or metastatic ccRCC, ≥1 measurable lesion per RECIST v1.1, and an Eastern Cooperative Oncology Group (ECOG) performance status of 0 or 1. Patients had received an ICI and VEGFR-targeted therapy and experienced disease progression or discontinued because of toxicity. The study opened without a requirement for CD70 expression. After 19 patients were enrolled, the protocol was amended to mandate a CD70 tumor proportion score (TPS) of ≥1% in a fresh biopsy. A further amendment after one additional patient was enrolled allowed use of archival samples (Supplementary Methods). HLA matching between patients and donors was not required. Full eligibility criteria are detailed in the protocol (Data Supplement).

Study Procedures

The design and manufacturing methods for ALLO-316 have been published.[20] Anti-CD70 single-chain fragment variable–based allogeneic CAR T cells were produced via lentiviral vector transduction of healthy donor T cells with elimination of the T-cell receptor and CD52 by TALEN-based gene editing. Cis masking of the CAR receptor by CD70 provided protection from CAR T-cell–mediated fratricide.[20] In phase Ia, patients were assigned to four ALLO-316 dose levels, escalating from 40×10^6 to 80×10^6, 120×10^6, and 240×10^6

CAR T cells in a single dose. Before receiving ALLO-316, patients underwent lymphodepletion with fludarabine 30 mg/m²/d plus cyclophosphamide 300 or 500 mg/m²/d (FC), or fludarabine 30 mg/m²/d plus cyclophosphamide 300 or 500 mg/m²/d plus ALLO-647 10 mg/d (FCA) once daily for 3 days on days −5, −4, and −3. On day 0, ALLO-316 was administered as a single intravenous infusion. In phase Ib, patients received FC and ALLO-316 at 80×10^6 CAR T cells in a single dose. Patients were hospitalized after ALLO-316 infusion for ≥5 days and were discharged once acute toxicities had resolved. Supportive therapy was allowed to manage adverse events (AEs). On-treatment biopsies and paired blood sampling were performed when feasible to evaluate infiltrating CAR T cells and endogenous immune cells.

Assessments

AEs were collected for 3 months after ALLO-316 or until initiation of new anticancer therapy. AEs of special interest (AESIs) and serious AEs were collected for 60 months or until disease progression or initiation of new anticancer therapy. AESIs were defined prospectively and included grade ≥3 cytokine release syndrome (CRS), immune effector cell–associated neurotoxicity syndrome (ICANS), or infection; or any-grade immune effector cell–associated hemophagocytic lymphohistiocytosis-like syndrome (IEC-HS), GVHD, autoimmune disorder, or secondary malignancy. CRS and ICANS were managed per institutional guidelines. Guidance on diagnosis, grading, and management of IEC-HS was developed during phase Ia and was in place for phase Ib (Data Supplement, Table S1). Tumor response was assessed by computed tomography or magnetic resonance imaging.

Biomarkers

Tumor CD70 expression was assessed at Ventana Medical Systems (Roche Tissue Diagnostics, Tuscon, AZ; Supplementary Methods; Data Supplement, Fig S2). TPS was defined as the percentage of tumor cells with membranous staining for CD70. Cases with TPS ≥1% were considered CD70-positive. Tumors with CD70 TPS ≥50% and <50% were arbitrarily referred to as CD70-high and CD70-low, respectively. Optional tumor aspirates or biopsy samples were collected after treatment to assess ALLO-316 infiltration (Supplementary Methods).

End Points

The primary end point was safety, including dose-limiting toxicities (DLTs) during phase Ia. AEs were graded using National Cancer Institute Common Terminology Criteria for Adverse Events v5.0 and coded per the Medical Dictionary for Regulatory Activities (MedDRA). The severity of CRS, ICANS, GVHD, and IEC-HS was assessed according to modified grading criteria from published standards.[21-23] Secondary end points included overall response rate (ORR) and duration of response (DOR) per RECIST v1.1 by the investigator, overall survival (OS), and CAR T-cell expansion kinetics and immunogenicity.

Statistics

Safety was analyzed in patients who received ≥1 dose of any study treatment (safety analysis set). Efficacy was analyzed in patients who received ALLO-316 (modified intention-to-treat population). Descriptive summaries were reported for all end points. ORR was summarized with proportions. DOR and OS were estimated using the Kaplan-Meier method. Vector copy number, absolute lymphocyte count, absolute neutrophil count, white blood cell count, cytokines, and flow cytometry data were analyzed using R (version 4.4). When applicable, group comparisons used the Wilcoxon rank-sum test with Benjamini-Hochberg adjustment (false discovery rate <0.05).

RESULTS

Patients

Between March 15, 2021, and February 13, 2025, 51 patients were enrolled across 10 centers (data cutoff: November 3, 2025). The safety analysis set included 50 patients; 46 received ALLO-316 (modified intention-to-treat population; Fig 1). At data cutoff, 37 patients had discontinued the study (Data Supplement, Table S2). The median follow-up was 28.8 months (range, 8.5-55.4). Patients had received a median of four prior lines of therapy (Table 1). Forty-two patients (84.0%) had CD70-positive tumors (33 [66.0%] CD70-high [TPS ≥ 50%]), including 40 patients with tumor response information (Data Supplement, Fig S3). The Data Supplement (Table S3) summarizes baseline tumor burden and metastatic sites.

Phase Ia Dose-Finding Phase

Thirty-four patients were enrolled in phase Ia (Table 1); 20 received FC, and 14 received FCA. Thirty-two patients received ALLO-316. All patients experienced ≥1 treatment-emergent AE (TEAE; grade ≥3, n = 30 [88.2%]; Table 2; Data Supplement, Table S4). FCA did not lead to worse neutropenia or slower white blood cell recovery relative to FC; however, lymphocytes were comparatively reduced through day 90 (Data Supplement, Fig S4). Two DLTs occurred with FCA once daily for 3 days at the 80×10^6 CAR T cells in a single dose. Grade three autoimmune hepatitis resolved with treatment in a patient who enrolled shortly after progressing on pembrolizumab/axitinib. Grade 5 cardiogenic shock occurred in a patient who developed hyperinflammation 21 days after ALLO-316 that rapidly progressed to vasopressor-refractory cardiovascular collapse despite tocilizumab and methylprednisolone; autopsy revealed structural cardiac abnormalities, potentially limiting cardiac reserve in the context of systemic inflammation. An emergent safety review was conducted, and enrollment in the FCA cohorts was halted.

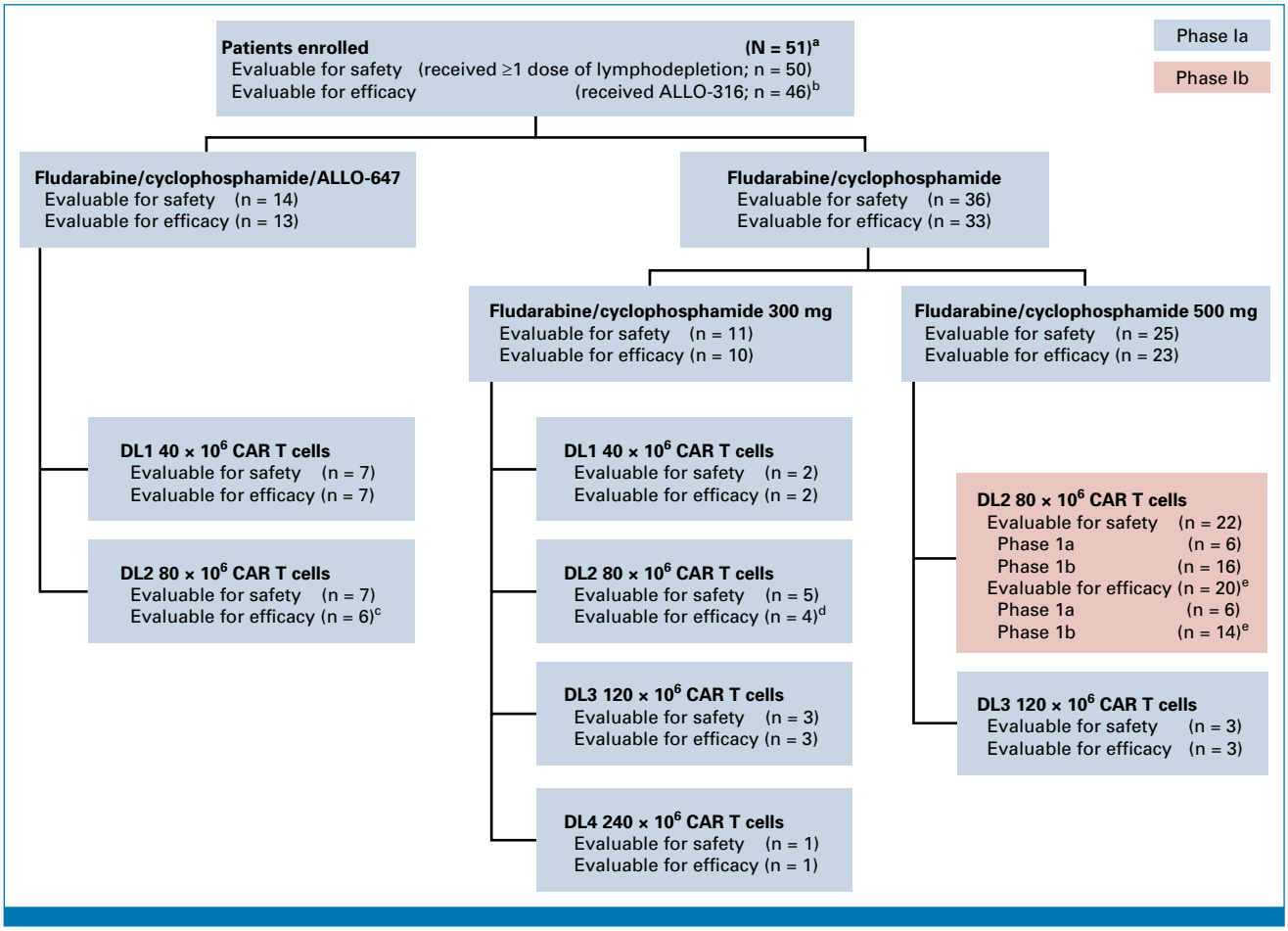


FIG 1. CONSORT diagram. [a]One patient developed central nervous system metastasis before receiving any study drug and was unable to proceed with treatment. [b]Four patients underwent lymphodepletion but did not receive treatment with ALLO-316. [c]One patient started lymphodepletion and then did not meet eligibility for ALLO-316 administration because of elevated transaminases. [d]One patient started lymphodepletion and then withdrew consent after they were found to be COVID-19–positive. [e]Two patients included in the phase Ib safety analysis population received lymphodepletion but did not receive ALLO-316. One patient had altered mental status immediately after completion of lymphodepletion; brain MRI confirmed metastases originally thought to be stable on prior imaging, and the patient was transitioned to hospice. One patient developed kidney and liver failure and did not meet criteria for dosing of ALLO-316; the patient subsequently transitioned to hospice. CAR, chimeric antigen receptor; DL, dose level; the lymphodepletion regimens described as follows were administered once daily for 3 days: fludarabine/cyclophosphamide 300 mg, fludarabine 30 mg/m^2, and cyclophosphamide 300 mg/m^2; fludarabine/cyclophosphamide 500 mg, fludarabine 30 mg/m^2, and cyclophosphamide 500 mg/m^2; fludarabine/cyclophosphamide/ALLO-647, fludarabine 30 mg/m^2, cyclophosphamide 300 or 500 mg/m^2, and ALLO-647 30 mg; all CAR T cells were administered in a single dose; MRI, magnetic resonance imaging.

Serious AEs occurred in 16 patients (80.0%) in the FC cohort and in 10 (71.4%) in the FCA cohort. Fatal treatment-related AEs occurred in three patients (8.8%), including the DLT of cardiogenic shock in the FCA cohort and grade 5 failure to thrive and grade 5 sepsis in the FC cohort (Supplementary Results). Although the grade 5 sepsis was not considered a DLT, as it occurred outside the DLT window, because of a preceding grade 2 IEC-HS event in this patient, enrollment was paused for review of emergent safety data with a focus on hyperinflammation consistent with IEC-HS. After consultation with external experts and the US Food and Drug Administration, the protocol was amended to include detailed diagnostic and grading criteria for IEC-HS and a grade-based treatment algorithm (Data Supplement, Table S1). Enrollment resumed at two dose levels lower, 80 × 10^6 CAR T cells in a single dose, with fludarabine 30 mg/m^2/d plus cyclophosphamide 500 mg/m^2/d (FC500) lymphodepletion once daily for 3 days.

A confirmed partial response (PR) occurred in five of 32 patients (ORR, 15.6% [95% CI, 5.3 to 32.8]; four in FC, one in FCA; Table 3; Data Supplement, Table S5). No responses occurred in patients with negative or unknown CD70 (Data Supplement, Fig S3). Mean CAR T-cell expansion was greater with FC than FCA (Fig 2A, Data Supplement, Fig S5). Analysis of serum cytokines showed similar IL-15, higher IL-7, and lower IL-12/23 P40 levels with FCA versus FC (Data Supplement, Fig S6). FCA resulted in deeper endogenous T-cell depletion than FC, reducing target abundance and possibly contributing to the difference in CAR T-cell expansion (Data Supplement, Fig S7). Based on acceptable toxicity, encouraging ORR, and favorable CAR

TABLE 1. Baseline Characteristics in the Phase 1a, Phase 1b, and Total Populations of Patients Who Received ≥1 Dose of Lymphodepletion (safety analysis set)

Characteristic	Patients Enrolled in the Phase Ia Regimens (n = 34)[a]	Patients Enrolled in the Phase Ib Regimen (n = 22)[b]	Total (N = 50)[c]
Age, years, median (range)	62 (35-70)	56 (35-67)	60 (35-70)
≥65 years, No. (%)	9 (26.5)	3 (13.6)	12 (24.0)
Sex, male, No. (%)	30 (88.2)	20 (90.9)	44 (88.0)
ECOG performance status, No. (%)			
0	21 (61.8)	10 (45.5)	27 (54.0)
1	13 (38.2)	12 (54.5)	23 (46.0)
Race, No. (%)			
Asian	2 (5.9)	2 (9.1)	2 (4.0)
White	27 (79.4)	18 (81.8)	42 (84.0)
Other	1 (2.9)	2 (9.1)	2 (4.0)
Not reported	4 (11.8)	0	4 (8.0)
Stage IV disease, No. (%)	34 (100)	22 (100)	50 (100)
Baseline LDH > ULN, No. (%)	3 (8.8)	5 (22.7)	8 (16.0)
Prior nephrectomy, No. (%)	29 (85.3)	22 (100)	45 (90.0)
CD70 status, No. (%)			
Positive	26 (76.5)	22 (100)	42 (84.0)
TPS ≥50	20 (58.8)	17 (77.3)	33 (66.0)
Negative	5 (14.7)	0	5 (10.0)
Unknown	3 (8.8)	0	3 (6.0)
IMDC risk at screening, No. (%)			
Favorable	13 (38.2)	8 (36.4)	19 (38.0)
Intermediate	17 (50.0)	14 (63.6)	27 (54.0)
Poor	3 (8.8)	0	3 (6.0)
Not available	1 (2.9)	0	1 (2.0)
Time since original diagnosis, months, median (range)	42.1 (12.1-216.3)	81.9 (20.2-210.9)	64.9 (12.1-216.3)
Lines of prior therapy, median (range)	3 (1-8)	4 (1-11)	4 (1-11)
Prior therapies, No. (%)			
Anti–PD-(L)1 therapy	34 (100)	22 (100)	50 (100)
Anti–CTLA-4 therapy	24 (70.6)	12 (54.5)	31 (62.0)
Cabozantinib	26 (76.5)	18 (81.8)	39 (78.0)
≥1 TKI	34 (100)	22 (100)	50 (100)
≥2 TKIs	18 (52.9)	18 (81.8)	32 (64.0)
≥3 TKIs	7 (20.6)	13 (59.1)	17 (34.0)
mTOR inhibitor	9 (26.5)	15 (68.2)	20 (40.0)
Belzutifan	3 (8.8)	9 (40.9)	10 (20.0)
CTLA-4 inhibitor, PD-1 inhibitor, TKI, and belzutifan	3 (8.8)	7 (31.8)	8 (16.0)

Abbreviations: CTLA-4, cytotoxic T-lymphocyte antigen 4; ECOG, Eastern Cooperative Oncology Group; FC500, fludarabine 30 mg/m^2/d plus cyclophosphamide 500 mg/m^2/d once daily for 3 days; IMDC, International Metastatic Renal Cell Carcinoma Database Consortium; LDH, lactate dehydrogenase; mTOR, mammalian target of rapamycin; PD-1, programmed cell death protein 1; PD-(L)1, programmed cell death protein/ligand 1; TKI, tyrosine kinase inhibitor; TPS, tumor proportion score; ULN, upper limit of normal.

[a]Two patients included in the phase 1a safety analysis set received lymphodepletion but did not receive ALLO-316: one withdrew consent after they were found to be positive for COVID-19 disease, and one did not meet eligibility for ALLO-316 administration because of elevated transaminases.

[b]Two patients included in the phase 1b safety analysis set received lymphodepletion but did not receive ALLO-316: one because of progression of disease (altered mental status during lymphodepletion prompted discovery of brain metastases on imaging) and the other because of liver and kidney failure during lymphodepletion.

[c]Six patients treated with FC500 once daily for 3 days and 80 × 10^6 CAR T cells in a single dose in phase 1a were analyzed as part of both phase Ia and phase Ib; data for these patients are included in both the phase Ia and phase Ib columns.

TABLE 2. TEAEs With Incidence ≥20% in the Total Population and Any AESIs in Patients Who Received ≥1 Dose of Lymphodepletion (safety analysis set)

	Patients Enrolled in the Phase Ia Regimens (n = 34)[a]		Patients Enrolled in the Phase Ib Regimen (n = 22)[b]		Total (N = 50)[c]	
AE	Any Grade	Grade 3 to 5	Any Grade	Grade 3 to 5	Any Grade	Grade 3 to 5
TEAEs, No. (%)	34 (100)	30 (88.2)	22 (100)	21 (95.5)	50 (100)	46 (92.0)
Neutropenia	20 (58.8)	18 (52.9)	18 (81.8)	18 (81.8)	33 (66.0)	31 (62.0)
WBC count decreased	17 (50.0)	15 (44.1)	16 (72.7)	16 (72.7)	29 (58.0)	27 (54.0)
Anemia	18 (52.9)	12 (35.3)	13 (59.1)	9 (40.9)	27 (54.0)	18 (36.0)
Fatigue	23 (67.6)	1 (2.9)	5 (22.7)	0	26 (52.0)	1 (2.0)
Nausea	20 (58.8)	0	8 (36.4)	0	25 (50.0)	0
Thrombocytopenia	14 (41.2)	9 (26.5)	13 (59.1)	7 (31.8)	24 (48.0)	14 (28.0)
Pyrexia	14 (41.2)	1 (2.9)	8 (36.4)	0	20 (40.0)	1 (2.0)
Peripheral edema	14 (41.2)	0	8 (36.4)	0	18 (36.0)	0
ALT increased	11 (32.4)	5 (14.7)	7 (31.8)	2 (9.1)	16 (32.0)	7 (14.0)
Headache	13 (38.2)	0	5 (22.7)	0	16 (32.0)	0
Arthralgia	10 (29.4)	0	8 (36.4)	0	15 (30.0)	0
AST increased	10 (29.4)	5 (14.7)	6 (27.3)	2 (9.1)	15 (30.0)	7 (14.0)
Constipation	12 (35.3)	0	4 (18.2)	0	15 (30.0)	0
Cough	11 (32.4)	0	3 (13.6)	0	13 (26.0)	0
Insomnia	10 (29.4)	0	3 (13.6)	0	12 (24.0)	0
Diarrhea	8 (23.5)	0	4 (18.2)	0	11 (22.0)	0
Dyspnea	9 (26.5)	1 (2.9)	3 (13.6)	0	11 (22.0)	1 (2.0)
Lymphopenia	8 (23.5)	8 (23.5)	5 (22.7)	5 (22.7)	11 (22.0)	11 (22.0)
Hyponatremia	7 (20.6)	1 (2.9)	4 (18.2)	0	10 (20.0)	1 (2.0)
Hypotension	8 (23.5)	2 (5.9)	3 (13.6)	0	10 (20.0)	2 (4.0)
Myalgia	10 (29.4)	0	4 (18.2)	0	10 (20.0)	0
Vomiting	6 (17.6)	0	4 (18.2)	0	10 (20.0)	0
AESIs, No. (%)						
CRS	21 (61.8)	1 (2.9)	15 (68.2)	0	31 (62.0)	1 (2.0)
Infection	23 (67.6)	12 (35.3)	11 (50.0)	9 (40.9)	30 (60.0)	19 (38.0)
IEC-HS	6 (17.6)	1 (2.9)	8 (36.4)	2 (9.1)	12 (24.0)	3 (6.0)
ICANS	1 (2.9)	0	4 (18.2)	0	4 (8.0)	0
GVHD	0	0	0	0	0	0

Abbreviations: AESI, adverse event of special interest; CRS, cytokine release syndrome; FC500, fludarabine 30 mg/m^2/d plus cyclophosphamide 500 mg/m^2/d once daily for 3 days; GVHD, graft-versus-host disease; ICANS, immune effector cell–associated neurotoxicity syndrome; IEC-HS, immune effector cell–associated hemophagocytic lymphohistiocytosis-like syndrome; TEAE, treatment-emergent adverse event.

[a]Two patients included in the phase Ia safety analysis set received lymphodepletion but did not receive ALLO-316: one withdrew consent after they were found to be positive for COVID-19 disease, and one did not meet eligibility for ALLO-316 administration because of elevated transaminases.

[b]Two patients included in the phase Ib safety analysis set received lymphodepletion but did not receive ALLO-316: one because of progression of disease (altered mental status during lymphodepletion found to be brain metastases on imaging), and one because of liver and kidney failure during lymphodepletion.

[c]Six patients treated with FC500 once daily for 3 days and 80 × 10^6 CAR T cells in a single dose in phase Ia were analyzed as part of both phase Ia and phase Ib; data for these patients are included in both the phase Ia and phase Ib columns.

T-cell kinetics, the FC500 80 × 10^6 CAR T-cell regimen was selected for expansion in phase Ib.

Phase Ib Expansion Phase

Phase Ib included 23 patients: Six from phase Ia who received FC500 80 × 10^6 CAR T cells and 17 additional patients. Twenty-two patients received lymphodepletion and were evaluable for safety; 20 received ALLO-316 (Fig 1). Patients in Ib tended to be younger, had higher ECOG performance status and baseline lactate dehydrogenase levels, longer time since diagnosis, and more prior lines of therapy; other baseline characteristics were similar between phases Ib and Ia (Table 1). All patients evaluable for safety had CD70-positive tumors; 17 (77.3%) were CD70-high.

TABLE 3. Confirmed Best Overall Response per RECIST v1.1 in Phase Ia, Phase Ib, and the Total Population of all Patients and Patients With a CD70 TPS of ≥50% Who Received ALLO-316 (mITT; efficacy analysis set)

Any CD70 TPS	Patients Treated With the Phase Ia Regimens (n = 32)	Patients Treated With the Phase Ib Regimen (n = 20)	Total (n = 46)[a]
Objective response rate, No. (%)	5 (15.6)[b]	5 (25.0)	8 (17.4)
95% CI	5.3 to 32.8	8.7 to 49.1	7.8 to 31.4
Disease control rate, No. (%)[c]	19 (59.4)	10 (50.0)	27 (58.7)
CR	0	0	0
PR	5 (15.6)	5 (25.0)	8 (17.4)
SD	14 (43.8)	5 (25.0)	19 (41.3)
PD	11 (34.4)	9 (45.0)	16 (34.8)
Not evaluable	2 (6.3)	1 (5.0)	3 (6.5)

CD70 TPS ≥50 Population	Patients Treated With the Phase Ia Regimens (n = 19)	Patients Treated With the Phase Ib Regimen (n = 16)	Total (n = 31)
Objective response rate, No. (%)	5 (26.3)[b]	5 (31.3)	8 (25.8)
95% CI	9.1 to 51.2	11.0 to 58.7	11.9 to 44.6
Disease control rate, No. (%)[c]	13 (68.4)	8 (50.0)	19 (61.3)
CR	0	0	0
PR	5 (26.3)	5 (31.3)	8 (25.8)
SD	8 (42.1)	3 (18.8)	11 (35.5)
PD	4 (21.1)	7 (43.8)	9 (29.0)
Not evaluable	2 (10.5)	1 (6.3)	3 (9.7)

Abbreviations: CR, complete response; FC500, fludarabine 30 mg/m^2/d plus cyclophosphamide 500 mg/m^2/d once daily for 3 days; mITT, modified intention-to-treat; PD, progressive disease; PR, partial response; SD, stable disease; TPS, tumor proportion score.
[a]Six patients treated with FC500 and 80 × 10^6 CAR T cells in a single dose in phase Ia were analyzed as part of both phase Ia and phase Ib; data for these patients are included in both the phase Ia and phase Ib columns.
[b]Confirmed PRs observed in four patients who received FC (1 in FC300 + 80 × 10^6 CAR T cells, two in FC500 + 80 × 10^6 CAR T cells, and one in FC500 + 120 × 10^6 CAR T cells) and in one patient who received FCA (40 × 10^6 CAR T cells). All CAR T cells regimens were administered in a single dose.
[c]Disease control rate defined as a confirmed CR, PR, or SD for at least 42 days.

Safety

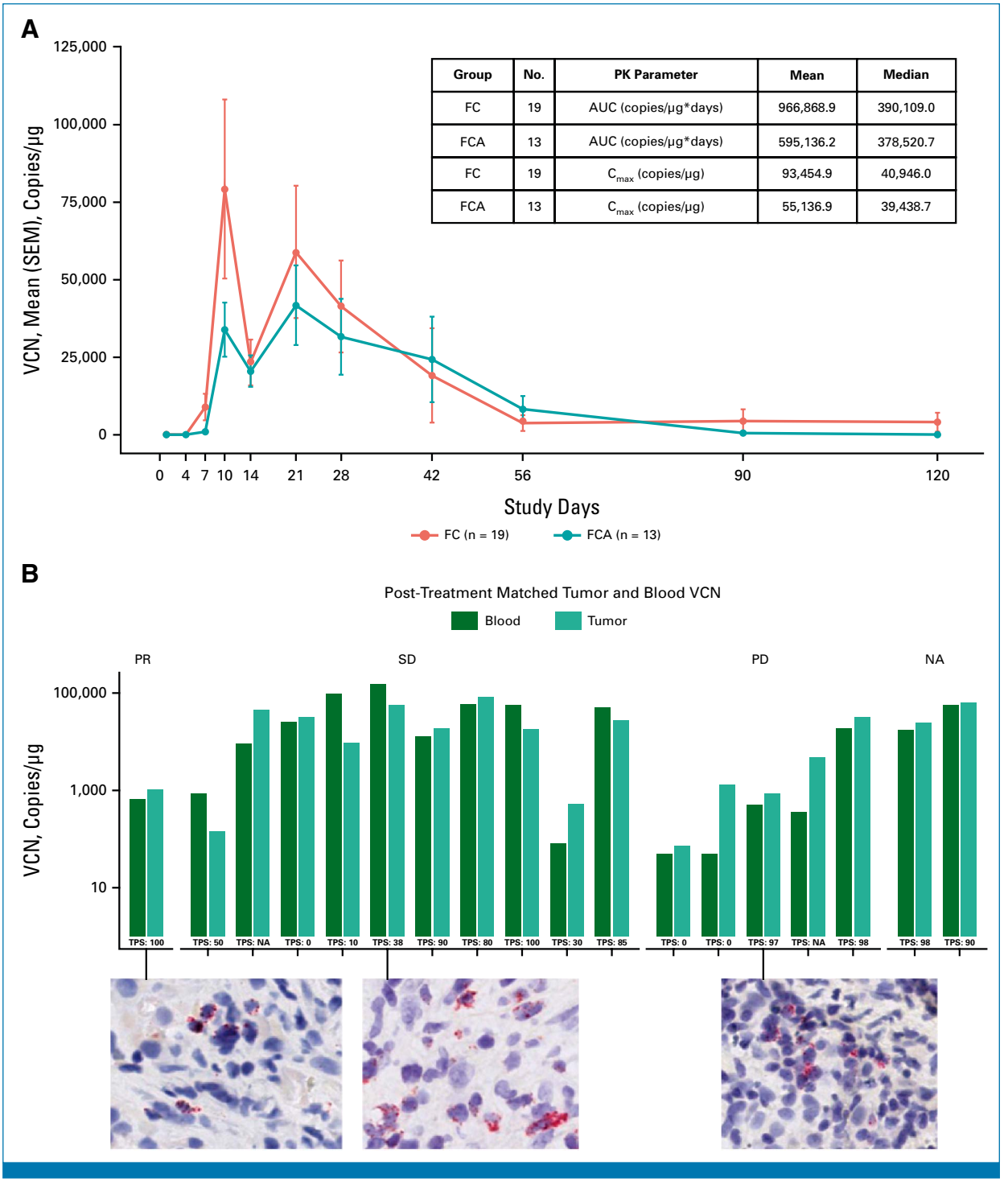
No unexpected safety findings were observed (Table 2). Most grade ≥3 TEAEs were hematologic. Grade 1 or 2 CRS was the most common AESI (15 patients [68.2%]; no grade ≥3). Other AESIs included infection in 11 patients (50.0%; grade ≥3, nine patients [40.9%]), IEC-HS in eight (36.4%; grade ≥3, two patients [9.1%]), and ICANS in four (18.2%; no grade ≥3). No GVHD occurred. Median times to onset of CRS, ICANS, and IEC-HS were 6, 10, and 18 days, respectively. No grade 5 treatment-related AEs occurred.

A higher incidence of IEC-HS was reported in phase Ib versus Ia, likely because of improved diagnosis and coding of IEC-HS and its inclusion in MedDRA. Most IEC-HS events were mild to moderate. Two high-grade IEC-HS events occurred: grade 4 IEC-HS based on gastrointestinal bleeding with subsequent improvement and grade 3 IEC-HS based on hypotension, which improved without vasopressors.

Preliminary Efficacy

A confirmed PR was reported in five of 20 patients in phase Ib (ORR, 25.0% [95% CI, 8.7 to 49.1]; five of 16 [31.3%; 95% CI, 11.0 to 58.7] in the CD70-high group; Table 3). Median DOR was not estimable (NE; 95% CI, 6.9 to NE; Data Supplement, Fig S8). No progression events occurred in responders after a minimum follow-up of 8 months (Fig 3). No responses occurred in patients with CD70 TPS <50% (n = 4). Seven of 20 evaluable patients (35.0%) had a >30% reduction in target lesion size from baseline (Fig 3). Responses were observed regardless of prior therapy and typically occurred within 28 days of ALLO-316 infusion (Fig 3; Data Supplement, Fig S9). Patients with a best overall response of stable disease experienced disease control for a median of 3.75 months (range, 3.19-4.01). The median OS was 15.2 months (95% CI, 4.6 to NE) in the overall phase Ib population and NE (95% CI, 3.2 to NE) in the CD70-high group (Data Supplement, Fig S10).

Paired tumor/blood samples demonstrated infiltration of ALLO-316 cells into tumor tissue in responders and nonresponders (Fig 2B, Data Supplement, Fig S11). RNAscope multiplex analysis showed that replicating endogenous T cells and CAR T cells were present (Data Supplement, Fig S11). Responders had a higher maximum concentration (C_{max}) and AUC over 28 days (AUC_{28}) compared with nonresponders, and CAR T-cell frequency fell to near



Group	No.	PK Parameter	Mean	Median
FC	19	AUC (copies/µg*days)	966,868.9	390,109.0
FCA	13	AUC (copies/µg*days)	595,136.2	378,520.7
FC	19	C_{max} (copies/µg)	93,454.9	40,946.0
FCA	13	C_{max} (copies/µg)	55,136.9	39,438.7

FIG 2. Tumor infiltration, expansion, and persistence of ALLO-316 CAR T cells. (A) Mean (SEM) VCN over time by lymphodepletion regimen in patients from phase Ia who received ALLO-316 (n = 32); AUC is area under the expansion curve through 28 days postinfusion. (B) VCN measurements in matched tumor and blood samples post-treatment, along with representative chromogenic in situ hybridization images of CAR T-cell RNA in tissue sections. CAR, chimeric antigen receptor; FC, fludarabine 30 mg/m^2 and cyclophosphamide 300 or 500 mg/m^2 once daily for 3 days; FCA, fludarabine 30 mg/m^2, cyclophosphamide 300 or 500 mg/m^2, and ALLO-647 30 mg once daily for 3 days; NA, not applicable; PD, progressive disease; PR, partial response; SD, stable disease; TPS, tumor proportion score; VCN vector copy number.

the lower limit of detection by day 240 (Data Supplement, Fig S12). There was a trend toward greater CAR T-cell expansion in CD70-high tumors versus CD70-low tumors (median C_{max}: CD70 ≥50, 53,034 copies/µg [n = 31]; CD70 < 50, 32,865 copies/µg [n = 12]; Data Supplement, Fig S12).

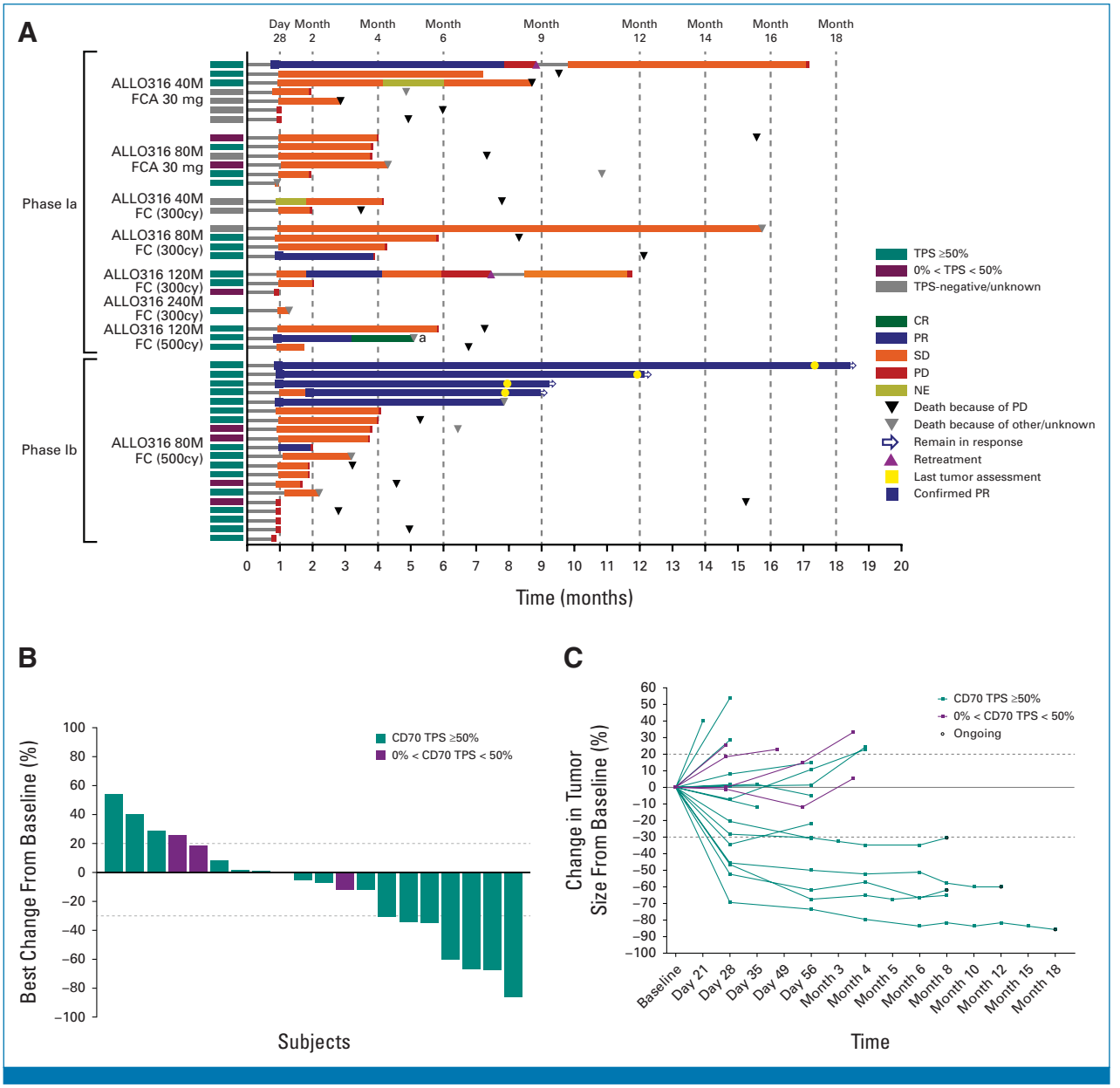


FIG 3. Efficacy of ALLO-316 CAR T cells. (A) Time to response in patients who received ALLO-316 (mITT; efficacy analysis set; n = 46). (B) Best percentage change from baseline in tumor size for patients in the phase Ib population who received ALLO-316 (n = 20). (C) Change from baseline in tumor size over time for each patient in the phase Ib population who received ALLO-316 (n = 20). [a]The patient had a response of CR, but died on day 154 of septic shock in the context of ongoing COVID-19 disease before a confirmatory scan could be performed. The fatal event of septic shock was considered unrelated to ALLO-316 by the investigator and sponsor. CAR, chimeric antigen receptor; CR, complete response; FC, fludarabine 30 mg/m^2 and cyclophosphamide 300 or 500 mg/m^2 once daily for 3 days; FCA, fludarabine 30 mg/m^2, cyclophosphamide 300 or 500 mg/m^2, and ALLO-647 30 mg once daily for 3 days; mITT, modified intention-to-treat; NE, not evaluable; PD, progressive disease; PR, partial response; SD, stable disease; TPS, tumor proportion score.

DISCUSSION

The results of TRAVERSE showed that ALLO-316 had encouraging antitumor activity and toxicity that was manageable with tailored monitoring and treatment in patients with heavily pretreated CD70-high ccRCC. The optimal regimen was identified as lymphodepletion with fludarabine 30 mg/m^2/d plus cyclophosphamide 500 mg/m^2/d once daily for 3 days plus ALLO-316 at a single dose of 80 × 10^6 CAR T cells.

CRS and neurologic toxicity, including ICANS, are common with autologous CAR T-cell products. The incidence of CRS with axicabtagene ciloleucel in non-Hodgkin lymphoma is 90% (grade ≥3, 9%), and the incidence of neurologic toxicity is 78% (grade ≥3, 25%).[24] In TRAVERSE, high-grade CRS and ICANS occurred in 2% and 0% of patients, respectively. High-grade IEC-HS was observed in 6%, but outcomes improved after implementation of diagnostic criteria and a tailored treatment algorithm. In phase Ib, IEC-HS was

diagnosed early, often while asymptomatic, and treated with therapies including the JAK inhibitor, ruxolitinib. With these interventions, IEC-HS was largely controlled in the final 20 patients enrolled, with some receiving IEC-HS care in the ambulatory setting. No grade 5 IEC-HS events occurred. Similar to approved CAR T-cell products,[24-27] infections occurred in 60% of patients (grade ≥3, 38%), indicating the need for prophylaxis, close monitoring, and rapid treatment.

Preliminary efficacy in phase Ib was encouraging. An ORR of 31.3% was reported in patients with CD70 TPS ≥50%, with no responses in patients with CD70 TPS <50%. Responses also appeared durable. These findings were particularly encouraging given the heavily pretreated phase Ib population and exceed the response rates observed with VEGFR TKIs and HIF-2α inhibitors in earlier lines.[5,6] ALLO-316 is also a single, one-time infusion, potentially resulting in long treatment-free intervals. A best overall response of progressive disease was observed for 34.8% of patients, suggesting that some ccRCC may be primarily refractory to CAR T-cell therapy; this could be due to intratumor variability of CD70 expression and/or an inability to overcome the local immunosuppressive environment.

A major impediment with CAR T-cell therapies is allorejection of donor-derived products, analogous to host-versus-graft reactions. Strategies to overcome this include high-dose cytotoxic chemotherapy-based lymphodepletion,[28] deletion of HLAs,[29,30] and incorporation of anti-allorejection molecules.[31,32] ALLO-316 was designed with rejection avoidance properties intrinsic to the CD70 CAR itself.[20] As endogenous T cells detect non-self HLA molecules on CAR T cells, they upregulate CD70, both marking them for elimination by ALLO-316 and providing a growth stimulus to the CAR T cells. Evidence from TRAVERSE supporting this novel mechanism includes elevated CAR T-cell peak expansion and AUC_{28}, which notably exceed those observed in trials of autologous CAR T cells for hematologic malignancies,[33-36] durable CAR T-cell persistence, and the paradoxically superior expansion seen with the less-intense lymphodepletion regimen (FC). This unique attribute of ALLO-316 likely explains the robust CAR T-cell expansion not observed with other CD70 CAR T cells.[16]

While CAR T-cell products targeting CD19 and BCMA have transformed treatment for lymphoma and myeloma, there are no approved CAR T-cell products for solid tumors in the US and Europe.[37] Satricabtagene autoleucel, an autologous product against claudin-18.2, produced an ORR of 22% in gastric and gastroesophageal junction adenocarcinoma versus 4% in the active control arm, but had limited durability (median DOR, 5.52 months).[38] Safety was acceptable, but CRS occurred in 95% of patients (grade ≥3, 5%).[38] Satricabtagene autoleucel was recently approved in China for patients with HER2-negative advanced gastric or gastroesophageal junction adenocarcinoma who have failed at least two prior lines of systemic therapy and whose tumors express claudin-18.2. Locally administered CAR T cells targeting EGFRvIII and wild-type EGFR demonstrated activity in glioblastoma multiforme, but durability was limited by rapid antigen loss and adaptive resistance; no grade ≥3 AEs or DLTs occurred.[39] In the COBALT-RCC study, allogeneic CD70-directed CTX130 induced one durable CR in 16 patients with ccRCC (ORR, 6.3%).[16] Half of patients had CRS (all grade 1 or 2), and 19% had serious infections; no ICANS or GVHD occurred.[16]

Within this landscape, our experience with ALLO-316 supports continued evaluation of allogeneic anti-CD70 CAR T cells. The results offer preliminary evidence that allogeneic CAR T cells can overcome longstanding barriers to efficacy in solid tumors when paired with elements that support CAR T-cell expansion and persistence. The success of CAR T-cell therapy also depends on optimization of safety. IEC-HS is a potentially fatal syndrome, but was successfully managed in TRAVERSE using a tailored diagnostic and treatment algorithm implemented mid-trial.

While the results of TRAVERSE require validation in larger RCC studies, ALLO-316 could also be explored in less heavily pretreated patients and in other CD70-positive tumors. Primary limitations include the small population, relatively short follow-up, lack of a control arm and rigorously defined CD70 cutoff, and investigator response assessment. The protocol underwent several early amendments; however, all patients in phase Ib were enrolled and treated consistently. Ninety percent of patients were male, and the population was predominantly White, which limits the generalizability of these findings. Although 38.0% of trial participants had an IMDC risk score of favorable, which may not reflect real-world patients with heavily pretreated, metastatic ccRCC, four of five responders (80.0%) in phase Ib had an intermediate risk score. With real-world use, the need for CD70 IHC testing could delay urgent access for some high-risk patients. However, when validated, CD70-targeted positron emission tomography imaging could potentially allow for noninvasive assessment to determine patient eligibility.

TRAVERSE provides proof-of-concept clinical and translational evidence for the role of allogeneic CAR T-cell therapy in solid tumors. As a one-time, off-the-shelf treatment that rapidly induces durable responses in multidrug-resistant metastatic ccRCC, ALLO-316 represents a novel and promising treatment option for patients with CD70-expressing cancers that warrants further clinical development.

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AUTHORS' DISCLOSURES OF POTENTIAL CONFLICTS OF INTEREST

Allogeneic CD70-Targeted CAR T-Cell Therapy for Advanced Renal Cell Carcinoma: Results From the Phase I TRAVERSE Trial

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