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Sequential BCMA CAR T-cell therapy in refractory multiple myeloma

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Key Points

- Sequential treatment with commercially available BCMA-directed CAR T-cell therapy in refractory myeloma is safe and efficacious.
- Duration of response to initial BCMA-directed CAR T-cell treatment is predictive for durable responses after sequential treatment.

Multiple myeloma (MM) relapsing after B-cell maturation antigen (BCMA)-directed chimeric antigen receptor (CAR) T-cell treatment remains a therapeutic challenge. Data on re-exposure to CAR T-cell therapy targeting the same antigen are scarce. We analyzed 10 heavily pretreated patients with RRMM at 3 medical centers treated with the commercially approved CAR T-cell therapy product idecabtagene vicleucel in a real-world setting. Upon relapse, all patients received ciltacabtagene autoleucel as a second CAR T-cell therapy infusion, with bridging treatments permitted between both therapies. Sequential therapy with BCMA-directed CAR T-cell therapy was safe, with no higher-grade immune-cell–associated side effects or new safety signals. We found robust CAR T-cell therapy expansion and high response rates (100% with at least very good partial response, with 60% achieving minimal residual disease negativity), with an estimated progression-free survival of 64.8% (95% confidence interval, 39%-100%) at 6 months after the second CAR T-cell treatment. Duration of response to first CAR T-cell therapy was predictive for durable responses to the second CAR T-cell therapy product. Loss of BCMA antigen occurred in only 1 of 3 patients relapsing after ciltacabtagene autoleucel. Two of three relapsing patients died within a year, and showed no further response to bispecific antibody treatment. To our knowledge, this study provides the first real-world evidence that sequential treatment with 2 different commercially approved BCMA CAR T-cell therapy products is both feasible and effective, particularly in patients with prolonged responses to initial BCMA CAR T-cell therapy.

Introduction

Chimeric antigen receptor (CAR) T-cell therapy has emerged as a promising treatment approach for relapsed/refractory multiple myeloma (RRMM), with B-cell maturation antigen (BCMA) being the most heavily investigated target to date.[1] The 2 anti-BCMA CAR T-cell therapy products, idecabtagene vicleucel (ide-cel) and ciltacabtagene autoleucel (cilta-cel), have been approved for the treatment of

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The data that support the findings of this study are available on reasonable request from the corresponding author, Tim Richardson (tim.richardson@uk-koeln.de).

RRMM by the United States Food and Drug Administration and European Medicines Agency.[2,3] These therapies have demonstrated unprecedented responses in heavily pretreated patients with RRMM, with overall response rates (ORRs) >90%, and complete response (CR) rates of 30%-60%.[4]

However, despite the success of BCMA-directed CAR T-cell therapy and contrary to the results in other entities,[5] responses are often short-lived, with median progression-free survival (PFS) ranging from 14[6] to 35 months.[7] The prognosis of patients relapsing after CAR T-cell therapy is unfavorable, with a high medical need for subsequent treatment strategies.[8,9] Currently, little is known about the safety and efficacy of retreatment with BCMA-directed CAR T-cell therapy.

In this study, we analyzed the safety and feasibility of sequential treatment with commercially available BCMA CAR T-cell therapy products in patients who progressed after in-label CAR T-cell treatment.

Methods

We conducted a multicenter retrospective analysis of 10 heavily pretreated patients with RRMM refractory to at least 1 proteasome inhibitor, immunomodulator, and anti-CD38 antibody. All patients were treated in-label with ide-cel as the first BCMA CAR T-cell therapy product. Upon relapse, patients were subsequently treated with the second standard of care CAR T-cell therapy product, cilta-cel. The sequence of ide-cel followed by cilta-cel was determined based on their respective approval time lines and availability. All CAR T-cell therapy products were manufactured and dosed according to standard protocols. One patient failed to collect T cells for cilta-cel, and was not included in the analysis. Leukapheresis was performed either at the time of progression after last therapy. In patients treated with Talquetamab between CAR T-cells, this was paused for ≥3 weeks prior to leukapheresis to minimize potential negative effects on T-cell fitness. Bridging therapy between apheresis and CAR T-cell therapy was permitted. The study was performed at 3 CAR T-cell therapy experienced tertiary German centers, and included patients infused with both products between April 2021 and December 2024. The decision to administer a second course of CAR T-cell therapy with cilta-cel was based on individual patient characteristics, disease status, and the treating physician's discretion.

BCMA expression was validated by immunohistochemistry or flow cytometry, and minimal residual disease in bone marrow by multicolor flow cytometry with a sensitivity level $<10^{-5}$. CAR T-cell therapy expansion was measured using real time polymerase chain reaction or flow cytometry. Response was evaluated according to the International Myeloma Working Group criteria. High-risk cytogenetics were defined as per International Myeloma Working Group consensus criteria.[10] Safety was analyzed and graded using American Society for Transplantation and Cellular Therapy, and European Society for Blood and Marrow Transplantation consensus criteria.[11,12] Descriptive statistics summarized patient data, and survival analyses used the Kaplan-Meier method. Data analysis was conducted in R (v4.4.2). Patients receiving cilta-cel were stratified by ide-cel response duration (≤12 or ≥12 months), and PFS was compared via log-rank test.

This study was approved by the institutional review and ethics board of the University of Cologne, Germany (registered under 24-1202-retro). All methods were carried out in accordance with the Declaration of Helsinki.

Results

We analyzed a cohort of patients who underwent sequential treatment with 2 commercially available BCMA-directed CAR T-cell therapies in a real-world setting. All patients were refractory to conventional therapies and heavily pretreated, with a median of 7 prior lines of therapy. High-risk features were common, including high-risk cytogenetics in 70% of patients and extramedullary disease in 40%. The median interval between the first and second CAR T-cell treatment was 1.9 years. Bridging therapies prior to the second CAR T-cell therapy infusion included the BCMA-directed antibody-drug conjugate belantamab mafodotin in 1 patient, and the GPRC5D-targeting bispecific T-cell engaging antibody talquetamab in 3 patients. The safety profiles of both CAR T-cell therapy administrations was manageable. No severe cytokine release syndrome or immune effector cell-associated neurotoxicity syndrome was observed after either infusion. Infections requiring hospitalization occurred in 5 patients following ide-cel, and in 3 patients after cilta-cel. One patient required autologous bone marrow transplantation due to grade 3 late immune effector cell-associated hematotoxicity after cilta-cel, with subsequent hematologic recovery. No movement or neurocognitive treatment-emergent adverse events were observed. One patient developed trochlear palsy after cilta-cel, consistent with previously published data.[13] There were no treatment-related deaths.

Following ide-cel, the ORR was 100%, with 70% of patients achieving a CR or better. The median PFS was 14.9 months (Figure 2), which is comparable to previously published data on PFS in this setting.[3] Only 1 patient received no therapy between the 2 CAR T-cell treatments (Figure 1).

Upon relapse and subsequent cilta-cel infusion, robust CAR T-cell therapy expansion was observed in all patients (Table 1). The ORR was again 100%, with 70% achieving ≥CR. After a median follow-up of 8.8 months, median PFS and overall survival were not reached (Figure 2). Three patients relapsed, resulting in a 6-month PFS of 64.8% (95% confidence interval, 39%-100%). Among patients with progressive disease prior to cilta-cel, 3 of 4 relapsed early. All 6 patients with available samples remained minimal residual disease negative at last follow-up.

Notably, patients with shorter PFS following ide-cel (<12 months) experienced significantly worse outcomes after cilta-cel; all such patients relapsed within 6 months, whereas those with longer PFS after ide-cel had more durable responses ($P = .0024$; Figure 2C). This observation may help facilitate patient selection for CAR T-cell therapy retreatment.

Discussion

To our knowledge, this is the largest real-world analysis of sequential treatment with commercially available BCMA-directed CAR T-cell therapy products, providing insights into feasibility, efficacy, and patient selection in RRMM.

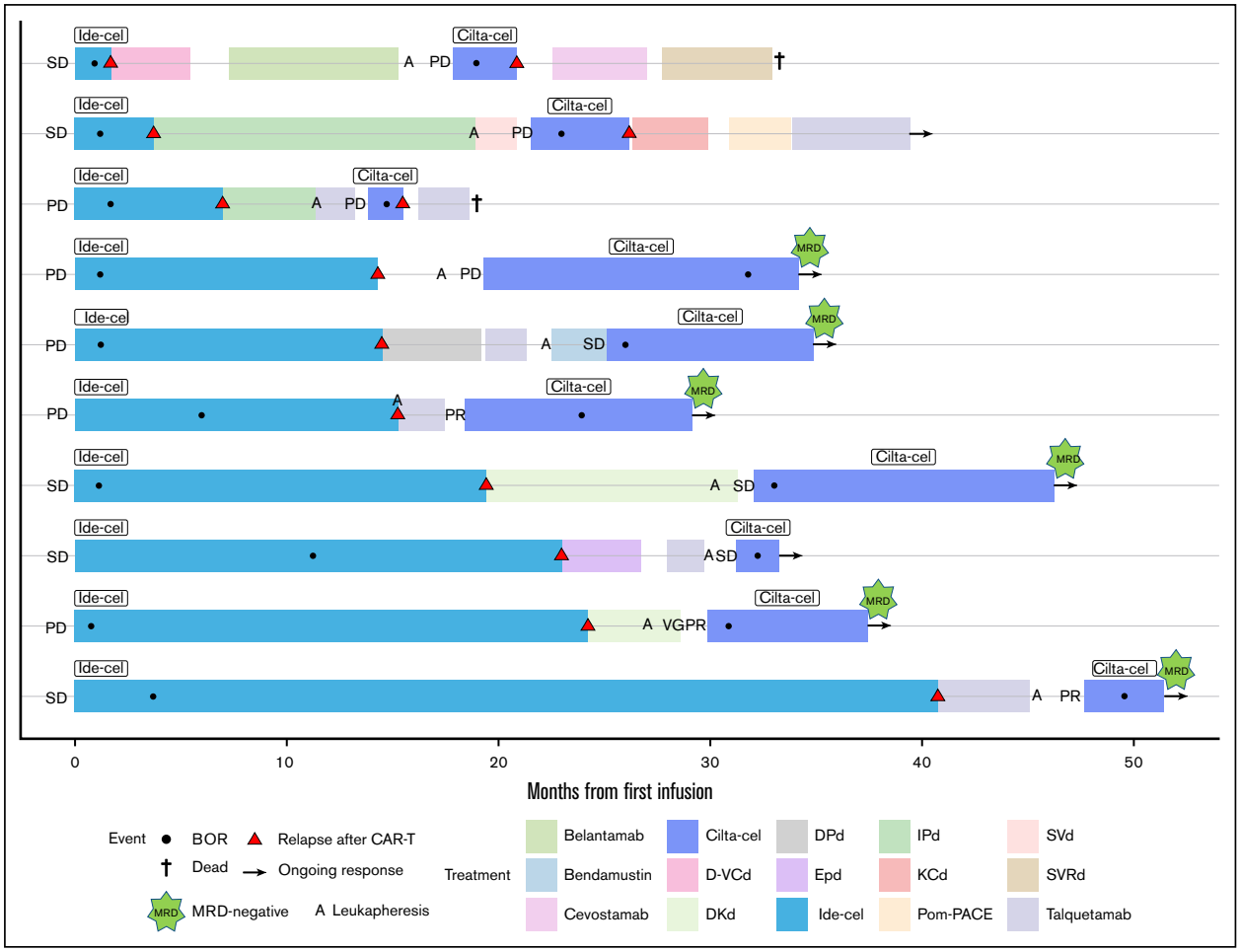

Figure 1. Swimmer plot starting at first CAR T-cell therapy infusion. Remission status prior to CAR T-cell therapy infusions, subsequent lines of therapy, depth, timing, and duration of response to CAR T-cell therapy, MRD assessment, and death are depicted. A, leukapheresis; BOR, best overall response; C, cyclophosphamide; d, dexamethasone; D, daratumumab; E, elotuzumab; I, isatuximab; K, carfilzomib; MRD, minimal residual disease; P, pomalidomide; PD, progressive disease; pom-PACE, pomalidomide with cisplatin, doxorubicin, cyclophosphamide, and etoposide; PR, partial response; R, lenalidomide; S, selinexor; SD, stable disease; V, bortezomib; VGPR, very good partial response.

Currently, evidence for sequential CAR T-cell therapy in RRMM remains limited. In the phase 2 KarMMa trial, 28 patients received a second ide-cel infusion at varying dose levels, with only 1 patient achieving a very good partial response and a median PFS of 1 month.[14] Two smaller phase 1 trials investigated fully human BCMA CAR constructs following initial treatment with experimental or murine-based CAR T-cell therapies, reporting ORRs of 100% and 71% in 5 and 7 patients, respectively. However, data on dosing and binding domains were not provided.[15,16] A retrospective single-center analysis reported an 89% ORR in 9 patients retreated with unspecified BCMA CAR T-cell therapy products. Although this study did not distinguish between bispecific t-cell engagers (BiTEs) and CAR T-cells as subsequent BCMA-directed therapies, it showed reduced PFS with BCMA-targeting approaches compared with alternative strategies.[17] To our knowledge, this study is the first to evaluate the safety and efficacy of 2 approved BCMA CAR T-cell therapy products in sequence, and may therefore inform future clinical decision-making in the absence of clinical trial data. The depth and rate of response to cilta-cel were comparable to its efficacy when used as a first-line CAR T-cell therapy product.[4]

While rare, biallelic loss of BCMA has been described as a mechanism of relapse after BCMA CAR T-cell therapy.[18,19] In our cohort, all 3 patients with early progression following ide-cel retained BCMA expression. Loss of BCMA was observed in only 1 of 3 patients relapsing after cilta-cel. Although the exact construct sequences have not been disclosed, both ide-cel and cilta-cel target extracellular epitopes of BCMA, and share a 4-1BB costimulatory domain. Ide-cel uses a murine-derived single-chain variable fragment,[20] whereas cilta-cel employs 2 heavy-chain–only domains of camelid origin,[21] theoretically allowing to overcome epitope-specific resistance. Nonetheless, short responses to ide-cel were mirrored by similarly short responses to cilta-cel in our cohort, suggesting that alternative resistance mechanisms, such as T-cell exhaustion or an immunosuppressive tumor microenvironment, may be involved, though these could not be evaluated in this retrospective study.[22] Interestingly, longer time from ide-cel to cilta-cel was not associated with longer responses to the second CAR T-cell therapy product.

A second dose of cilta-cel often comes as a byproduct in the production process, and could in theory mitigate the financial and

Figure 2. Best overall response and PFS for each CAR T-cell therapy product, stratified by prior ide-cel response. Over all response (A) and Kaplan-Meier curves (B) to ide-cel and cilta-cel based on the International Myeloma Working Group criteria. (C) Cilta-cel PFS stratified by prior ide-cel response duration <12 months and >12 months.

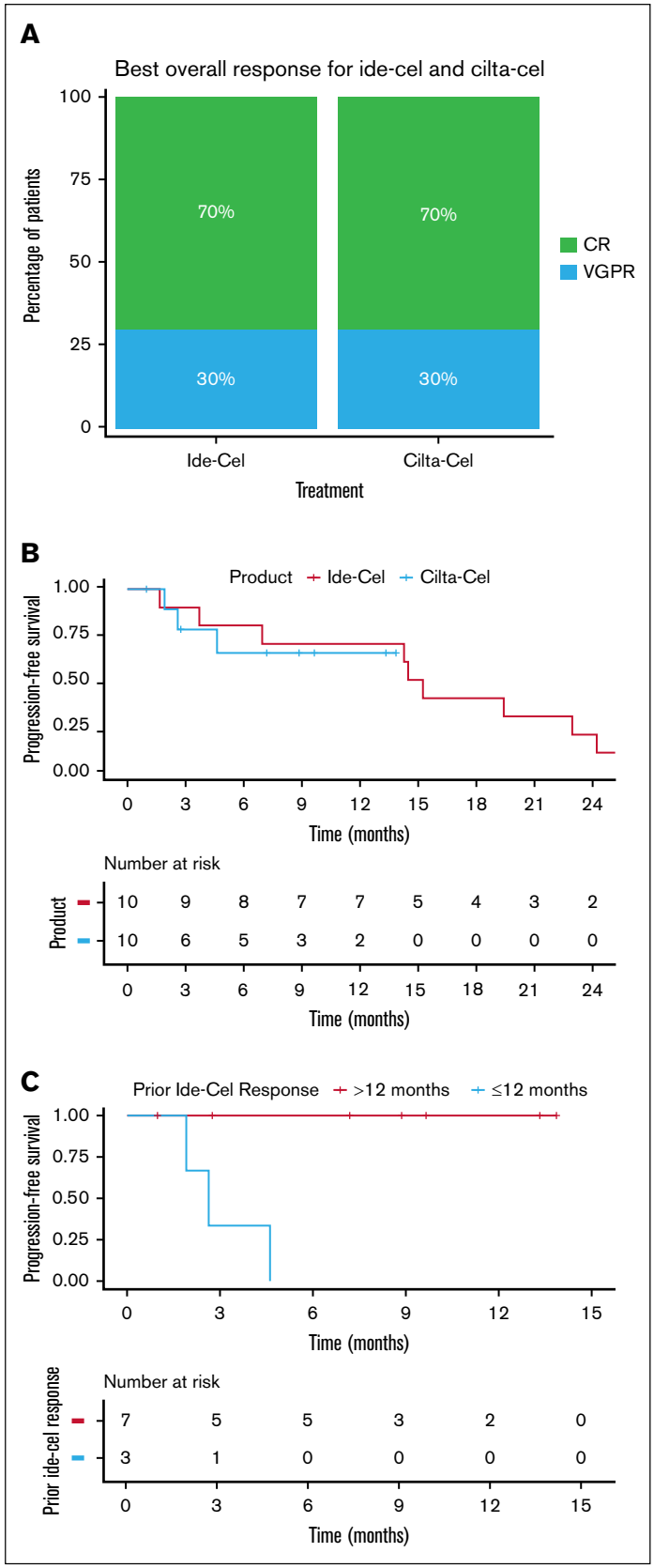

Table 1. Baseline characteristics of treated patients

Baseline characteristics at cilta-cel infusion	N = 10
Sex	4 females, 6 males
Age, median (range), y	63 (49-75)
Penta-refractory	7
High-risk cytogenetics	7
Double-hit cytogenetics	5
Extramedullary disease	4
Prior lines, median (range)	7 (5-12)
BCMA expression	8/8 (2 missing)
Treatment-related, median (range)	
Time between diagnosis and ide-cel infusion, y	5.8 (2.5-10.0)
PFS after ide-cel, mo	14.9 (1.7-40.4)
Time between ide-cel and cilta-cel infusion, y	1.9 (1.2-4.0)
Number of therapies between CAR T-cell products	1 (0-3)
CAR T-cell therapy expansion, C_{max} (PCR) cp/10E6 WBC	248 000 (27 500-1 410 000)
Disease status at cilta-cel infusion	4 PD, 3 SD, 2 PR, 1 VGPR
Feasibility & toxicity	
Vein-to-vein ide-cel, median (range), d	53.5 (45-88)
Vein-to-vein cilta-cel, median (range), d	61 (46-110)
CRS ide-cel	I° (n = 5), II° (n = 5)
ICANS ide-cel	0
CRS cilta-cel	I° (n = 3), II° (n = 3)
ICANS cilta-cel	0
Other neurotoxicity	1 trochlear palsy
In-hospital ide-cel, median (range), d	17 (10-23)
In-hospital cilta-cel, median (range), d	11 (0-16)

Data points are related to cilta-cel if not mentioned otherwise.
C_{max}, maximum concentration in copies per white blood cells (WBC); CRS, cytokine release syndrome; ICANS, immune effector cell–associated neurotoxicity syndrome; PCR, polymerase chain reaction; PD, progressive disease; PR, partial remission; SD, stable disease; VGPR, very good partial response.

logistical expenditure, even though recommended shelf life and availability are limited by the manufacturers. However, cilta-cel retreatment has been reported for 3 patients who had responded for ≥6 months to the first infusion in the CARTITUDE-1 trial. No patient responded, and all lacked CAR T-cell therapy expansion, with evidence of anti-CAR antibodies in only 1 patient.[23] These findings support a broader trend observed across CAR T-cell therapies: while retreatment with the same CAR construct appears to be largely ineffective, subsequent treatment with a different CAR may remain efficacious, at least in terms of ORRs, possibly due to immune responses developing against specific constructs. This pattern, also seen with CD19-directed CAR T-cells, is relevant for future clinical sequencing strategies, and the development of novel CAR T-cell therapy products. Data on re-exposure to cilta-cel are clearly needed. In a study investigating patients with chronic lymphocytic leukemia, B-cell non-Hodgkin lymphoma, and B-cell acute lymphoblastic leukemia, retreatment with CD19 CAR T-cells led to responses in 39%, with 20% achieving a CR. Factors associated with durable responses were increased CAR T-cell therapy doses, and a lymphodepletion with cyclophosphamide-fludarabine.[24]

Outcomes after cilta-cel failure were poor. A recent multicenter retrospective study found that BiTE therapy may be effective in patients relapsing after CAR T-cell therapy in MM.[8] In our cohort, 5 patients received talquetamab prior to cilta-cel, including 3 who received it immediately beforehand. Two of those three showed no response to the BiTE, but did respond to subsequent cilta-cel. Among 3 patients who relapsed early after cilta-cel, 2 died (1 from sepsis after failing to respond to cevostamab and 1 from progressive disease despite talquetamab). Lack of response to non-BCMA BiTEs may reflect T-cell exhaustion following prior CAR T-cell therapy, though neither T-cell phenotype nor function was assessed in this study. Notably, all 3 of these patients had already progressed on bridging therapy prior to their second CAR T-cell therapy infusion, suggesting an aggressive disease course resistant to current immunotherapies.

While limited by its retrospective design, small sample size, short follow-up, and lack of correlative biomarker analyses (eg, T-cell fitness, soluble BCMA), this study offers, to our knowledge, the first real-world evidence of feasibility. In the absence of clinical trials directly addressing sequencing strategies, our findings suggest that sequential treatment with ide-cel followed by cilta-cel can induce meaningful and durable responses, particularly in patients with a prolonged remission (>12 months) after initial CAR T-cell therapy, and controlled disease at the time of retreatment. As relapse following ide-cel becomes increasingly common, this work provides, to our knowledge, the first data supporting the feasibility and clinical utility of sequential therapy using 2 approved BCMA CAR T-cell therapy products in RRMM.

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Authorship

Contribution: T.R., U.H., C.S., and P.G. designed the research and wrote the manuscript; H.T. and P.G. performed the statistical analysis and created the graphs; H.B.-W., R.F., J.-H.F., E.K.M., S.S., R.T., and M.v.B. provided patient material and clinical data, and revised the manuscript; and C.S. and M.H. discussed the results and mentored the process.

Conflict-of-interest disclosure: T.R. reports consulting or advisory role for Janssen, Bristol Myers Squibb (BMS), Takeda, and Sanofi; and received travel and accommodation expenses from Janssen, Sanofi, Stemline Therapeutics, and Oncopeptides. U.H. received honoraria from Amgen, BMS/Celgene, GlaxoSmithKline (GSK), Janssen, Oncopeptides, Pfizer, Sanofi-Aventis, Stemline Therapeutics, and Takeda; and has had a consulting or advisory role for BMS/Celgene, GSK, Janssen, Oncopeptides, Pfizer, Sanofi-Aventis, and Stemline Therapeutics. J.-H.F. declares an advisory role for Pfizer; honoraria from BMS and Stemline Therapeutics; and travel and congress participation grants from Janssen-Cilag. E.K.M. has received honoraria from Amgen, Pfizer, Janssen, Takeda, BMS/Celgene, Sanofi, GSK, Stemline Therapeutics, and Oncopeptides; has had a consulting or advisory role for Amgen, Pfizer, Janssen,

BMS/Celgene, Takeda, Sanofi, GSK, Stemline Therapeutics, and Oncopeptides; has received research funding from Janssen, BMS/Celgene, Takeda, Sanofi, and GSK; and has received travel and accommodation expenses from Janssen, BMS/Celgene, Takeda, GSK, Sanofi, and Stemline Therapeutics. R.T. has participated in advisory boards or has received honoraria from Janssen, BMS, Takeda, GSK, Gilead, Sanofi, Amgen, Stemline Therapeutics, Oncopeptides, Pfizer, AstraZeneca, and AbbVie; and has received research and travel grants from Janssen and Amgen. C.S. has participated in advisory boards for and received honoraria from Amgen, AbbVie, BMS, Janssen, Novartis, Oncopeptides, Pfizer, Roche, Sanofi, Stemline Menarini, and Takeda; and has received research support from Janssen and Takeda. The remaining authors declare no competing financial interests.

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