Real-world effectiveness and safety of belantamab mafodotin monotherapy in patients with relapsed/refractory multiple myeloma treated in Europe

Belantamab mafodotin is an antibody-drug conjugate targeting B-cell maturation antigen. It consists of a humanized, afucosylated, IgG antibody conjugated to the microtubule inhibitor monomethyl auristatin F and has multiple mechanisms of action.[1] From August 2020 to February 2023 in the United States (US) and August 2020 to February 2024 in Europe, belantamab mafodotin was available as monotherapy for patients with relapsed/refractory multiple myeloma (RRMM) who had received at least four prior lines of therapy including an immunomodulatory agent, a proteasome inhibitor, and an anti-CD38 monoclonal antibody.[2,3] Conditional approval was based on the results of the phase II DREAMM-2 trial.[4] In the phase III DREAMM-3 trial, belantamab mafodotin did not meet the primary endpoint of progression-free survival resulting in its withdrawal from European and US markets.[2,3,5]

Belantamab mafodotin demonstrated robust clinical efficacy in combination regimens for second-line or later RRMM in the DREAMM-7 and DREAMM-8 phase III trials, and the combinations have been approved in multiple countries for patients with RRMM, including Europe.[6]

To mitigate and manage ocular adverse events (AE), which have been reported with antibody-drug conjugates containing a monomethyl auristatin F payload, including belantamab mafodotin,[7] the European label recommended ophthalmic examinations before the first four belantamab mafodotin doses and as clinically indicated thereafter.[8]

Real-world data on belantamab mafodotin monotherapy provide valuable insights to inform clinical decisions, particularly management of ocular AE, with recently approved combination regimens. Study objectives were to describe the real-world use, safety, and effectiveness of belantamab mafodotin monotherapy in patients treated across multiple European countries.

This was a multinational, multicenter, non-interventional, prospective study of adults with RRMM who received belantamab mafodotin in routine clinical care in Europe. *Online Supplementary Figure S1A* depicts the study design, eligibility criteria, and endpoints. All sites obtained Independent Ethics Committee approval. The study duration was planned to be a maximum of 2 years and 3 months per site; however, the study was closed early based on the European Medicines Agency's decision not to renew the license for belantamab mafodotin monotherapy.[2,5]

At data cutoff (June 7, 2024), 84 patients had been enrolled across seven countries and all had received one or more dose of belantamab mafodotin (*Online Supplementary Figure S1B*). The median follow-up (impacted by the early termination of the study) was 7.8 months (interquartile range [IQR], 4.6-13.1).

The median age at diagnosis was 63.5 years (IQR, 58.0-71.5). At initiation of belantamab mafodotin treatment, the patients' median age was 72.0 years (IQR, 64.5-78.0), 46 (54.8%) patients were female, and most patients had an Eastern Cooperative Oncology Group performance status of 0 (N=24 [28.6%]) or 1 (N=20 [23.8%]) (data missing for 25 [29.8%]) (Table 1).

A history of eye disease (including dry eye/eye injuries affecting best-corrected visual acuity [BCVA]) was present in 27 (32.1%) patients at baseline (*Online Supplementary Table S1*), with the eye diseases known for 26 (31.0%) patients.

The median number of prior lines of therapy was four (range, 2-11). All patients had received a prior immunomodulatory agent (60.7% were refractory to lenalidomide; 69.0% to pomalidomide) and a prior proteasome inhibitor (42.9% were refractory to bortezomib; 52.4% to carfilzomib), and 82 (97.6%) had already been treated with anti-CD38 (78.6% were refractory to daratumumab) (Table 1). Concomitant treatments for multiple myeloma were received by 32 (38.1%) patients and concomitant eye medications were received by 69 (82.1%) patients.

Most patients received an initial belantamab mafodotin dose of 2.5 mg/kg (77 [91.7%]) and most were planned to be treated once every 3 weeks (Q3W schedule) (73 [88.1%]).

The median duration of treatment (time-to-event analysis) and median duration of active exposure (period that belantamab mafodotin was considered to have a treatment effect) were both 4.1 months (IQR, 2.3-7.6).

At baseline, prior to initiating belantamab mafodotin therapy, 65 (77.4%) patients had an ophthalmic examination. Of patients who received a second, third, and fourth dose of therapy, 57/76 (75.0%), 41/59 (69.5%), and 31/40 (77.5%) had an ophthalmic examination between administration of the prior dose and administration of the second, third, or fourth dose, respectively. Ophthalmic examination rates remained high for patients with ongoing eye disease at belantamab mafodotin initiation and in patients who had an ocular AE at any time during the follow-up period (Figure 1A).

Ocular AE occurred in 58 (69.0%) patients (all treatment-related), 19 (22.6%) of which were grade ≥3 (Table 2); ocular AE generally occurred after the first or second dose. The most common ocular AE was keratopathy (42 [50.0%]), which was mostly mild or moderate at maximum severity as per the National Cancer Institute Common Terminology

Criteria for Adverse Events (NCI-CTCAE) scale (Table 2). Rates of keratopathy on treatment were relatively higher among patients with an ongoing ophthalmic disease at initiation (14/23 [60.9%]) (*Online Supplementary Table S2*) than among patients without a history of ophthalmic disease (26/57 [45.6%]). Overall, the first ocular AE resolved or was resolving by the last known visit in 29/58 (50.0%) patients. Considering keratopathy alone, the first incidence of keratopathy resolved in 22/42 (52.4%) patients.

Impacts of ocular AE on daily living were followed until resolution of the event or until last study visit. Among 42 patients with keratopathy, the most common impact on daily living was eye irritation/pain, which occurred in 13 (31.0%) patients; ten (23.8%) patients reported reading

Table 1. Demographic and disease characteristics.

Characteristic	Overall study population N=84
Age at belantamab mafodotin initiation, years, median (IQR)	72.0 (64.5-78.0)
Age at belantamab mafodotin initiation, N (%)	
18 to <65 years	21 (25.0)
65 to <75 years	30 (35.7)
75 to <80 years	20 (23.8)
≥80 years	13 (15.5)
Age at initial MM diagnosis, years, median (IQR)	63.5 (58.0-71.5)
Female, N (%)	46 (54.8)
ECOG performance status at belantamab mafodotin initiation, N (%)	
0	24 (28.6)
1	20 (23.8)
2	14 (16.7)
3	1 (1.2)
4	0
Missing	25 (29.8)
Time since initial MM diagnosis, months, median (IQR)	79.0 (53.2-119.3)
Extramedullary disease[a] between initial MM diagnosis and belantamab mafodotin initiation, N (%)	
Yes	14 (16.7)
No	66 (78.6)
Unknown	4 (4.8)
ISS stage at initial MM diagnosis, N (%)	
I	21 (25.0)
II	14 (16.7)
III	24 (28.6)
Missing	25 (29.8)
MM subtype at initial MM diagnosis, N (%)	
IgA	13 (15.5)
IgD	1 (1.2)
IgG	43 (51.2)
IgM	0
Biclonal (IgG, IgA)	0
Light chain	18 (21.4)
Other[b]	9 (10.7)
Cytogenetic risk between initial MM diagnosis and belantamab mafodotin initiation, N (%)[c]	
High cytogenetic risk[d]	23 (27.4)
High risk per IMWG criteria[e]	17 (20.2)
Standard risk	61 (72.6)

Characteristic	Overall study population N=84
≥1 prior treatment, N (%)	
Immunomodulator	84 (100.0)
Refractory to pomalidomide	58 (69.0)
Refractory to lenalidomide	51 (60.7)
Anti-CD38 exposure	82 (97.6)
Refractory to daratumumab	66 (78.6)
Proteasome inhibitor	84 (100.0)
Refractory to bortezomib	36 (42.9)
Refractory to carfilzomib	44 (52.4)
Chemotherapy	66 (78.6)
Stem cell transplant	41 (48.8)
Bispecific antibody[f]	1 (1.2)
CAR T-cell therapy[g]	2 (2.4)
Histone deacetylase treatment	1 (1.2)
N of prior lines of therapy, N (%)	
2[h]	7 (8.3)
3[h]	8 (9.5)
4	36 (42.9)
5	15 (17.9)
6	7 (8.3)
>6	11 (13.1)
Refractory status, N (%)[i]	N=78
Triple refractory or greater	78 (100.0)
Triple refractory	29 (37.2)
Quadruple refractory	28 (35.9)
Penta refractory	21 (26.9)

[a]Extramedullary disease was not further classified as soft tissue masses not contiguous with the bone or extraskeletal disease. [b]Other was reported as IgGκ (N=2) and one each of IgGλ, Bence-Jones, plasma cell leukemia (micromolecular), micromolecular κ, plasmocytoma type κ, since 2015, multiple light chain myeloma κ, non-secretory no phenotype, and type λ. [c]A patient could be included in both high cytogenetic risk and high risk per IMWG categories. The manner of cytogenetic risk determination was not collected. [d]High-risk cytogenetics: t(4;14), t(14;16), del17p, or 1q+. [e]High-risk cytogenetics per IMWG: t(4;14), t(14;16), or del17p. [f]Not targeting B-cell maturation antigen. [g]Idecabtagene vicleucel. [h]Twelve patients who received fewer than four prior lines of therapy were included despite being major protocol violations; three patients in Spain who received fewer than four prior lines of therapy were included but not considered protocol violations as per belantamab mafodotin labeling in Spain, in which belantamab mafodotin was indicated for patients with four or more prior therapies and therefore could include patients who received four or more individual agents regardless of therapy line.[18] [i]Refractory status was missing for six patients; percents are calculated based on patients for whom refractory status are available. Triple, quadruple, and penta refractory refer to patients who were refractory to three, four or five classes of therapy, respectively. IQR: interquartile range; MM: multiple myeloma; ECOG: Eastern Cooperative Oncology Group; ISS: International Staging System; IMWG: International Myeloma Working Group; CAR: chimeric antigen receptor.

impairment, two (4.8%) reported driving impairment, one (2.4%) reported a need for caregiver support, eight (19.0%) reported other impacts, and 15 (35.7%) patients reported no significant impact (Figure 1B).

Ocular AE led to treatment delay in 37 (44.0%) patients, dose reduction in 13 (15.5%), and treatment discontinuation in seven (8.3%) (Table 2). Among patients with keratopathy who had a dose delay (N=29), the median duration of delay was 22.3 days (IQR, 17.5-35.0).

Among 62 patients evaluable for response, the overall response rate was 38.7% (N=24; 1 patient [1.6%] had a complete response, 10 [16.1%] had a very good partial response, 13 [21.0%] had a partial response). The median duration of response was 10.7 months (95% CI: 3.94-not reached). The median real-world progression-free survival was 4.5 months (95% CI: 3.5–5.2) in the overall study population and the median overall survival was not estimable (95% CI: 11.0 months-not estimable). These findings have important implications for the integration of belantamab mafodotin into combination regimens, especially in the light of the robust efficacy demonstrated in recent phase III trials.[9-11]

Patients in this study were slightly older than patients in the DREAMM-2 and DREAMM-3 trials of belantamab mafodotin 2.5 mg/kg Q3W monotherapy and in the real-world study of belantamab mafodotin use in the US,[4,5,12] but similar to the median age reported in real-world studies of patients with multiple myeloma in Europe.[13]

All patients for whom information on refractory status was available were at least triple-class refractory. All patients had received an immunomodulator and a proteasome in-

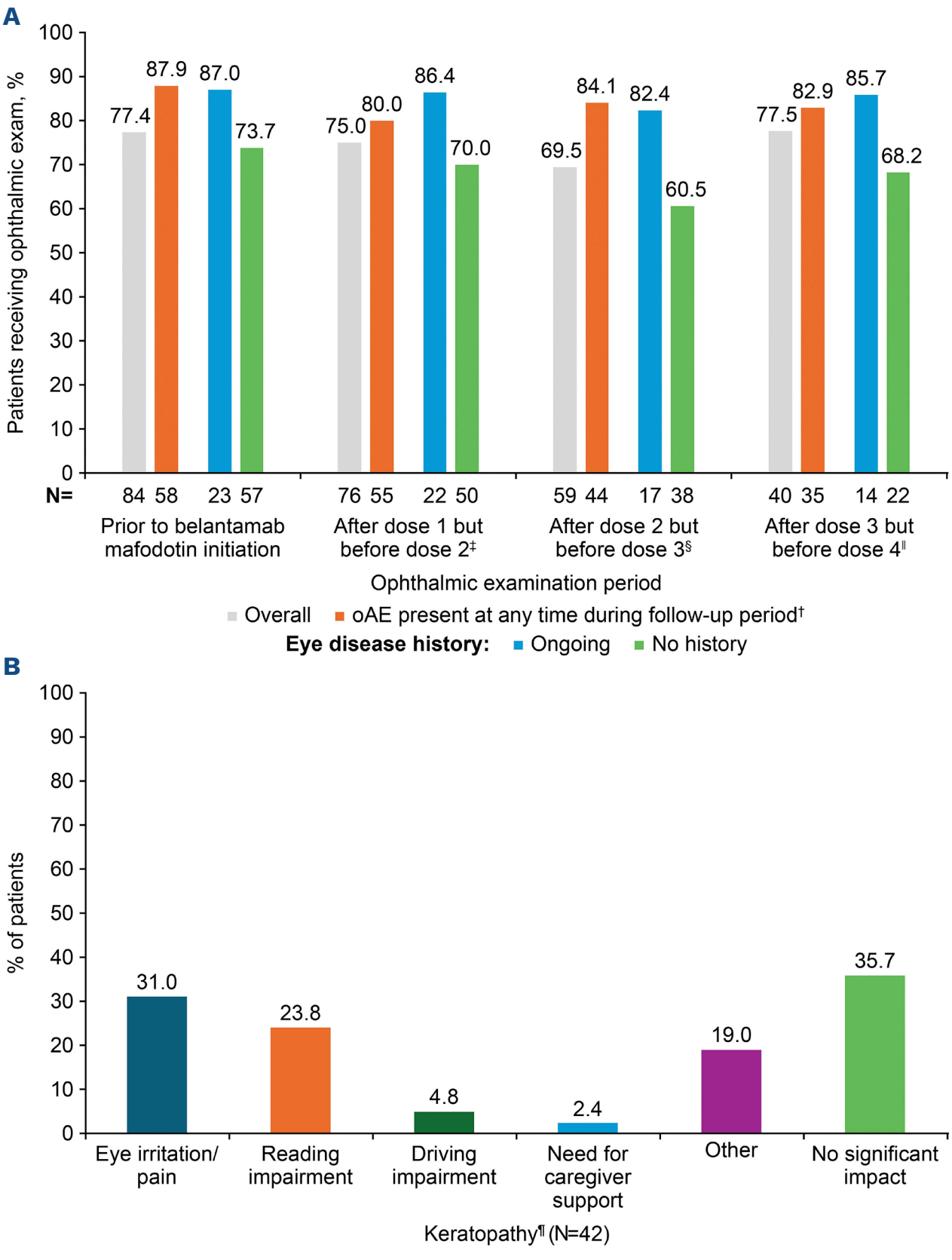

Figure 1. Real-world ophthalmic examinations in patients with relapsed/refractory multiple myeloma receiving belantamab mafodotin in Europe. (A) The proportion of patients receiving ophthalmic examinations overall, based on ocular adverse events (oAE) and on eye disease history. (B) The proportion of patients reporting oAE with an impact on daily living. A limitation of these analyses is the small number of patients analyzed. †Patients who experienced an oAE at any time during the period of belantamab mafodotin administration; the analysis did not link the timing of the oAE to the ocular examination. ‡Percentages are calculated based on the number of patients who received a second dose. §Percentages are calculated based on the number of patients who received a third dose. ‖Percentages are calculated based on the number of patients who received a fourth dose. ¶The impact of daily living was missing for nine patients with keratopathy. oAE: ocular adverse event.

hibitor in a prior line of therapy, and nearly all had had prior anti-CD38 exposure. Although patients refractory to these agents can achieve responses with later lines of therapy or retreatment, the responses are typically shorter than those achieved with the initial treatment.[14,15] Belantamab mafodotin has been evaluated in the second-line or later setting as part of combinations in the phase III DREAMM-7 and DREAMM-8 studies, and demonstrated significant survival benefits.[9-11]

At odds with the European label which recommended ophthalmic monitoring before the first four doses and as clinically indicated to manage ocular AE,[8] the US label required ophthalmic monitoring before each dose, with a Risk Evaluation and Mitigation Strategy program in place to ensure that the examinations were conducted.[16] Despite these differences, patients in Europe still had a high rate of ophthalmic monitoring prior to each of the first four belantamab mafodotin doses (≥69.5%), especially among patients with ongoing ophthalmic disease at treatment initiation or an ocular AE at any time during belantamab mafodotin treatment (≥80.0%). Patients with ongoing ophthalmic disease at belantamab mafodotin initiation had higher rates of keratopathy than patients without a history of ophthalmic disease, supporting the need for close and active ophthalmic monitoring in patients with ophthalmic disease at treatment initiation. While ocular AE are an important consideration when treating patients with antibody-drug conjugates, including belantamab mafodotin, these events can be adequately managed with proper monitoring and dose modification, and the less stringent monitoring recommendations in Europe than in the US does not seem to have affected tolerability overall or increased ocular risk.[12] Additionally, the rate of ocular AE (69.0%) was similar to that in the DREAMM-2 (74.0%) and DREAMM-3 (66.0%) monotherapy studies despite the use of various concomitant multiple myeloma therapies in the current study.[4,5]

Reading and driving impairment are particularly important symptoms of ocular AE when considering impacts on daily living. Although the evaluation was limited by small numbers of patients and missing data, patients with keratopathy tended to have moderate rates of reading impairment and low rates of driving impairment. A study of patient-reported experiences on belantamab mafodotin indicated that these symptoms resolve over time.[17]

The real-world nature of this study introduces several limitations. As assessment and monitoring criteria are not as stringent as required in clinical trials, bias in reporting, delays in monitoring, or under-identification of ocular AE and disease progression may have occurred. The use of electronic health records for collection of retrospective data also has inherent limitations, including the potential for missing or misclassified information. Despite the possibility that assessments of effectiveness were limited by missing data, the real-world progression-free survival in this study (median 4.5 months) was consistent with that reported in other real-world studies of belantamab mafodotin monotherapy in heavily pretreated patients.[12] In addition, non-ocular AE were not assessed, and the NCI-CTCAE grading criteria for AE were designed for use in clinical trials and not real-world studies. Lastly, the early closure of the study limited the number of patients enrolled and follow-up time, restricting the robustness of efficacy outcomes. A strength of the study was the use of prospective data; specific guidance for use of belantamab mafodotin beyond that included in the label was also not

Table 2. Ocular adverse events.

Ocular adverse events	Overall study population N=84
Patients with any ocular AE, N (%)	58 (69.0)
Treatment-related	58 (69.0)
NCI-CTCAE grade ≥3	19 (22.6)
KVA grade ≥3	19 (22.6)
Leading to dose reduction	13 (15.5)
Leading to treatment interruption/delay	37 (44.0)
Leading to treatment discontinuation	7 (8.3)
Leading to study withdrawal	0
Leading to death	0
Keratopathy, N (%)	42 (50.0)
Mild	11 (13.1)
Moderate	22 (26.2)
Severe	9 (10.7)
Other, N (%)	16 (19.0)
Mild	8 (9.5)
Moderate	6 (7.1)
Severe	1 (1.2)
Missing	1 (1.2)
Corneal erosions or defects, N (%)	7 (8.3)
Mild	2 (2.4)
Moderate	4 (4.8)
Severe	1 (1.2)
Blurred vision events, N (%)	6 (7.1)
Mild	3 (3.6)
Moderate	1 (1.2)
Severe	1 (1.2)
Missing	1 (1.2)
Change in BCVA, N (%)	4 (4.8)
Mild	0
Moderate	2 (2.4)
Severe	2 (2.4)
Dry eye events, N (%)	1 (1.2)
Mild	0
Moderate	1 (1.2)
Severe	0
Photophobia, N (%)	1 (1.2)
Mild	1 (1.2)
Moderate	0
Severe	0

All percentages calculated using N=84 as the denominator. AE: adverse event; NCI-CTCAE: National Cancer Institute Common Terminology Criteria for Adverse Events; KVA: keratopathy and visual acuity; BCVA: best-corrected visual acuity.

provided to participating sites, which allowed the study to assess real-world treatment decisions.

The results of this study were generally consistent with those observed in belantamab mafodotin monotherapy clinical trials for RRMM,[4,5] and support the use of label-recommended monitoring strategies as a way for appropriate management and resolution of ocular AE with belantamab mafodotin in clinical practice. This experience may guide optimization of monitoring and safety with combination regimens.[8-11]

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Disclosures

MC has served as a consultant and/or on advisory committees for Janssen, Celgene, BMS, Sanofi, Amgen, Pfizer and Menarini Stemline and has received honoraria from Janssen, Celgene, BMS, Sanofi, Amgen, Pfizer and Menarini Stemline. FS has consulted for AbbVie, Celgene, GSK, Janssen and Oncopeptides, with ownership interests in Celgene, GSK, Janssen, Oncopeptides, Targovax and Sanofi; additionally, he receives institutional research funding from AbbVie, Amgen, BMS, Daiichi Sankyo, Europe GmbH, GSK, Janssen, Novartis, Oncopeptides, Pfizer, SkylineDx and Takeda. MDi has served as a consultant, received honoraria from, and was involved in speaker's bureaus or advisory committees for Amgen, Sanofi, Regeneron, Menarini, Takeda Pharmaceuticals, GSK, BMS, Janssen, Beigene, Swixx Biopharma and AstraZeneca and has had travel expenses covered by Amgen, BMS, Janssen and Takeda. MDe has been involved in speaker's bureaus or advisory committees for Amgen, BeiGene, BMS, Janssen and Takeda and has received honoraria from Amgen, BMS, Janssen, GSK, Sanofi and Stemline. FE reports involvement in advisory boards for Amgen, BMS, GSK, Janssen, Sanofi and Takeda. DK consults for GSK, iuvo Clinical (where he serves on a clinical advisory board) and Avenzo Therapeutics, Inc. and holds ownership interests in Calm Waters Therapeutics LLC, where he has also served on advisory committees. HCL reports consulting for BMS, Pfizer, Janssen, Regeneron, GSK, Sanofi, AbbVie, Takeda, Allogene Therapeutics, Menarini and Alexion Pharmaceuticals, with research funding from Amgen, BMS, Janssen, Regeneron and Takeda. RV has received grant support from BMS, Sanofi and Takeda, with honoraria from Adaptive, BeiGene, BMS, GSK, Janssen, Oncopeptides, Sanofi and Takeda. RG has received institutional research funding from Novo Nordisk, Lilly, Celgene, Merck, Takeda, AstraZeneca, Novartis, Amgen, BMS, MSD, Sandoz, AbbVie, Gilead Sciences, Roche and Daiichi Sankyo Europe GmbH; he has also received honoraria directly from Celgene, Roche, Merck, Takeda, AstraZeneca, Novartis, Amgen, BMS, MSD, Sandoz, AbbVie, Gilead Sciences, Daiichi Sankyo and Sanofi, with travel accommodation or expenses covered by Roche, Amgen, Janssen-Cilag, AstraZeneca, Novartis, MSD, Celgene, Gilead Sciences, BMS, AbbVie and Daiichi Sankyo Europe GmbH. TM reports receiving honoraria directly from GSK. EA has served on advisory boards for Amgen, Janssen, Pfizer, Sanofi and Takeda. AL has no conflicts of interest to disclose. LC-O'B, JBi, TdE, MF, JBy, SS, VSB, and JM are employees of and hold financial equities in GSK. JBy additionally reports ownership interest in Adaptimmune and Novartis. CYV reports employment at Syneos Health. MH reports research funding from AbbVie, Beigene, BMS, Cosette Pharmaceuticals, Daiichi Sankyo, GSK, Johnson & Johnson, SpringWorks Therapeutics and The Binding Site and has received honoraria for consultancy/participated in advisory boards for Curio Science LLC, Intellisphere LLC, BMS, Johnson & Johnson and GSK.

Contributions

MC, MDe, MDi, FE, DK, HCL, RV, LC-O'B, JBi, TdE, MF, JBy, CYV, JM and MH were involved in the study concept or design, data acquisition, data analysis, and data interpretation. RG, TM, EA, FS and AL were involved in data acquisition, analysis and interpretation.

SS and VSB were involved in data interpretation. All authors reviewed and revised the manuscript, approved the final version and agreed to submit the manuscript for publication.

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Data-sharing statement

Information about GSK's data-sharing commitments and access requests to anonymized individual participant data and associated documents can be requested for further research from *https://www.gsk-studyregister.com/en/*

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