Annals of Hematology (2026) 105:133
https://doi.org/10.1007/s00277-026-06873-w

RESEARCH

Overall survival with momelotinib vs. best available therapy in patients with ruxolitinib-experienced myelofibrosis: a matching-adjusted indirect comparison

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Received: 14 November 2025 / Accepted: 26 January 2026

Abstract

The Janus kinase (JAK) inhibitor ruxolitinib is a standard first-line therapy for patients with symptomatic and/or intermediate- to high-risk myelofibrosis (MF). However, the majority of patients discontinue ruxolitinib within 5 years of initiation, mainly due to lack or loss of response and/or therapy-related cytopenias. Additional treatments are needed to improve long-term outcomes, including overall survival (OS). Momelotinib, a JAK1/JAK2/activin A receptor type 1 inhibitor, has demonstrated benefits in reducing anemia and improving symptoms and spleen size in 3 phase 3 trials of patients with intermediate- to high-risk MF (SIMPLIFY-1, SIMPLIFY-2, and MOMENTUM). These studies also provide data on patients who received momelotinib after discontinuing ruxolitinib. In the absence of long-term head-to-head comparisons of momelotinib and other treatments after discontinuation of ruxolitinib, the present study compared OS in patients with ruxolitinib-experienced MF from the momelotinib phase 3 trials vs. those treated with best available therapy (BAT) after ruxolitinib from the RUX-MF retrospective real-world study. The comparison was performed using an unanchored matching-adjusted indirect comparison (MAIC). Additionally, an MAIC of OS was conducted in an anemic subgroup (hemoglobin < 10 g/dL). After adjustment for cross-trial differences, the MAIC results showed a favorable trend for momelotinib vs. BAT, both in the overall population and in the anemic subgroup, with hazard ratios < 1 across all analytical scenarios and all population-matching models with an effective sample size of ≥ 20. This study is a key addition to current evidence surrounding OS post ruxolitinib and highlights the benefit of momelotinib in this setting.

Keywords 2L · Anemia · Best available therapy (BAT) · Matching-adjusting indirect comparison (MAIC) · Momelotinib · Myelofibrosis

Introduction

Myelofibrosis (MF) is a chronic myeloproliferative neoplasm that originates in hematopoietic stem cells, the clonal proliferation of which leads to cytokine release, myeloid cell lineage hyperproliferation, and bone marrow fibrosis [1]. MF can present de novo as primary MF or can be secondary to other myeloproliferative neoplasms, namely essential thrombocythemia and polycythemia vera. The disease is driven by dysregulated Janus kinase (JAK)-signal transducer and activator of transcription signaling, which underlies the inflammation and fibrosis associated with MF [2]. The continued aberrant proliferation of abnormal cells and resultant scar tissue in the bone marrow lead to clinical manifestations such as anemia, hepatosplenomegaly, constitutional symptoms, and shortened survival [2–4]. Although allogeneic stem cell transplant is the only potentially curative option for patients with intermediate- or high-risk MF, it is suitable for few patients given its associated high risk of relapse, morbidity, and mortality [4].

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Published online: 21 February 2026

Treatment for MF includes JAK inhibitor therapies and is based on patients' symptom severity and disease risk category [5]. Ruxolitinib, a dual JAK1/JAK2 inhibitor, has been shown to effectively improve splenomegaly and MF-related symptoms and is one of the first-line therapies for symptomatic and/or intermediate- to high-risk MF [6]. While ruxolitinib improves symptoms and reduces splenomegaly in many patients, it can also cause or worsen anemia and thrombocytopenia [6–9]. The development or worsening of anemia can negatively impact overall survival (OS) [10]. As a result, ruxolitinib dose is reduced and eventually discontinued by most patients during the first 5 years of treatment, and additional treatments are required [11–13].

The management of patients with MF after ruxolitinib represents a substantial challenge in real-life clinical practice. In the RUX-MF retrospective real-world study, 40.8% of patients discontinued ruxolitinib after 3 years, and OS after ruxolitinib discontinuation was generally poor [13]. The median OS was approximately 3 years in patients who could receive treatment after ruxolitinib vs. < 4 months in patients who could not receive subsequent treatment [13, 14]. These outcomes underscore the need for more effective and safe treatments that can improve long-term outcomes in a post-ruxolitinib setting.

Momelotinib, a JAK1/JAK2/activin A receptor type 1 inhibitor, is approved in several regions for JAK inhibitor–naive and –experienced patients with intermediate- or high-risk MF with anemia [15–17]. Approval was granted based on anemia benefits and symptom and spleen responses demonstrated in 3 phase 3 trials (SIMPLIFY-1 [NCT01969838], SIMPLIFY-2 [NCT02101268], and MOMENTUM [NCT04173494]) [18–20]. In all 3 trials, patients who remained on study after week 24 received open-label momelotinib, including those who crossed over from the respective comparator arm.

SIMPLIFY-1 compared momelotinib with ruxolitinib in JAK inhibitor–naive patients with MF. Of the 216 patients who were randomized and received ruxolitinib, 197 (91.2%) crossed over to momelotinib in the open-label phase without ruxolitinib tapering or washout [18]. SIMPLIFY-2 examined the efficacy of momelotinib vs. best available therapy (BAT) in patients with MF who had suboptimal response or hematologic toxic effects with ruxolitinib. A total of 104 patients were previously treated with ruxolitinib and received momelotinib from baseline. Of 52 patients randomly assigned to BAT, 40 (76.9%) crossed over to momelotinib; for most of these patients (36/40), BAT was ruxolitinib [19]. MOMENTUM confirmed the anemia-related differentiated clinical benefits of momelotinib vs. danazol. The trial enrolled JAK inhibitor–experienced symptomatic (Total Symptom Score [TSS] ≥ 10) and anemic (hemoglobin [Hb] < 10 g/dL) adults, all of whom were previously treated with ruxolitinib, and 130 patients received momelotinib from baseline [20].

The crossover trial designs allow all 3 studies to provide information on patients receiving momelotinib after ruxolitinib discontinuation. In the absence of long-term evaluation of momelotinib and other treatments following ruxolitinib discontinuation, we conducted an indirect treatment comparison (ITC) across data sources to support treatment decision-making by patients and healthcare providers. The present study compared OS in patients with ruxolitinib-experienced MF from the momelotinib phase 3 trials vs. those treated with BAT after ruxolitinib, based on data from the RUX-MF retrospective real-world study.

Methods

Study population

Individual patient data for those who received momelotinib after discontinuation of ruxolitinib are available from SIMPLIFY-1 [18] (conducted between 2013 and 2016), SIMPLIFY-2 [19] (conducted between 2014 and 2016), and MOMENTUM [20] (conducted between 2020 and 2021) (Supplementary Table 1). The comparator RUX-MF study (NCT06516406) [13] includes data from participants treated according to standard clinical practice at various European hematology centers, as previously described [21]. Of the original population of 524 patients who initiated ruxolitinib in Palandri et al. (2020) [13], OS was evaluated in 267 patients who discontinued ruxolitinib in the chronic disease phase and were subsequently treated with either conventional or novel agents (excluding momelotinib as listed in Supplementary Table 2), with data available for up to 105 months [14]. Additionally, OS data were provided for a subgroup of 174 anemic patients (those with Hb < 10 g/dL at baseline) for subpopulation analyses. Summary aggregate baseline characteristics were also made available for both the overall and anemic populations. While the present analysis is based on 267 patients from the RUX-MF study [14], that study overall included 1055 patients treated with ruxolitinib across 26 European hematology centers from 2013 until death or February 2, 2024 (data cutoff) [22].

Outcomes

The RUX-MF retrospective real-world study reported OS starting from discontinuation of ruxolitinib, whereas the momelotinib trials reported it from the start of administration of the investigational drug. OS was therefore redefined starting from ruxolitinib discontinuation (or with momelotinib start date for the few patients—13 in SIMPLIFY-2 and

3 in MOMENTUM—for whom ruxolitinib discontinuation data was missing). OS was compared both in the overall population and the anemic subpopulation (Hb < 10 g/dL) of momelotinib patients who discontinued ruxolitinib in SIMPLIFY-1 [18], SIMPLIFY-2 [19], and MOMENTUM [20] and BAT patients in Palandri et al. (2025) [14].

Statistical analysis

An unanchored matching-adjusted indirect comparison (MAIC) analysis was used, leveraging the individual patient data available from the momelotinib trials and aggregate data available from Palandri et al. (2025) [14] to obtain estimates of comparative effectiveness. To account for baseline differences between trial populations, individual momelotinib trial patients were reweighted so that weighted means/medians and/or standard deviations for continuous variables and proportions for binary/categorical variables of key baseline characteristics in the SIMPLIFY-1, SIMPLIFY-2, and MOMENTUM trials match those reported in Palandri et al. (2025) [14]. The weights obtained were also used to calculate the effective sample size (ESS) achieved. Weight distribution plots are shown in Supplementary Figs. 1–4.

Selection of patient characteristics included as matching factors in the MAIC analysis was based on key prognostic factors and potential effect modifiers in MF identified based on clinical judgement and biological plausibility. Key prognostic factors for OS in MF, such as age, sex, Dynamic International Prognostic Scoring System (DIPSS) risk category, Hb level, platelet count, circulating blast count, and JAK2 status were considered in the design and analysis of the ITC. While certain variables were unavailable or inconsistently reported across data sources, a broader set of covariates beyond those strictly prognostic for OS were incorporated to make the matched comparison more robust and mitigate potential bias. Covariates that were available and defined comparably across the momelotinib and BAT data sources included the following: age, sex, TSS, Hb level, platelet count, palpable spleen length, white blood cell (WBC) count, body mass index (BMI), MF subtype, and DIPSS score.

Several population-matching models considering different combinations of baseline characteristics were evaluated (Supplementary Table 3). The analyses considered population matching on as many prognostic factors relevant to OS as possible, aiming to both maintain a balanced population between those receiving momelotinib and those receiving BAT and maintain the ESS as high as possible. Based on the ESS calculations, model C (hereafter referred to as model 1) and model E (hereafter referred to as model 2) were selected for further analysis. Model 1 matched on more characteristics (age, sex, TSS, Hb level < 10 g/dL, platelet count < 100×10^9/L, palpable spleen length ≥ 10 cm, WBC count > 25×10^9/L, BMI > 25 kg/m^2, MF subtype, and DIPSS score), while model 2 matched only for age, sex, BMI > 25 kg/m^2, MF subtype, and DIPSS but had a higher ESS. Standardized mean differences were calculated as previously described [23].

The base case analytical scenario that was considered in the ITC examined patients who received momelotinib within 1 month since ruxolitinib discontinuation from SIMPLIFY-1, SIMPLIFY-2, and MOMENTUM. This corresponds to the median time from ruxolitinib discontinuation to the start of a salvage therapy (1 month [range, 0–45 months]) in the RUX-MF cohort. Scenario analyses to assess the impact of excluding ruxolitinib-randomized patients in SIMPLIFY-1 (to remove potential survival effects of patients who were JAK inhibitor naive or those who were treated with ruxolitinib for only 24 weeks prior to study) and momelotinib-randomized patients in MOMENTUM (to remove potential effects of COVID-19) were also evaluated as well as a supplementary analysis of patients who received momelotinib regardless of when they discontinued ruxolitinib (Fig. 1 and Supplementary Table 4).

After baseline characteristics were balanced across the momelotinib arms of SIMPLIFY-1, SIMPLIFY-2, and MOMENTUM and the BAT arm of the RUX-MF study, comparisons of outcomes were conducted using the weights obtained in the matching process for the momelotinib arms and the aggregate results reported for BAT. Standard errors, 95% confidence intervals (CIs), and *P* values for these indirect comparisons were based on a robust sandwich estimator. Differences in OS between momelotinib and BAT were analyzed using weighted Cox proportional hazards models to estimate hazard ratios (HRs) and 95% CIs. Statistical significance was based on a 2-sided *P* value <0.05. Parallel naive comparisons were also done in the trial populations before matching (using an unweighted version of the momelotinib data and the aggregate results reported for BAT) and reviewed alongside the matched results.

Ethical statement

This study complied with all applicable laws regarding patient privacy. No direct subject contact or primary collection of individual human subject data occurred. IRB approvals for the SIMPLIFY-1 [18], SIMPLIFY-2 [19], and MOMENTUM [20] clinical trials and the RUX-MF study were obtained by individual study sites [24]. All study results are presented as aggregate analyses that omit patient identification; therefore, informed consent and ethics committee or IRB approval were not required.

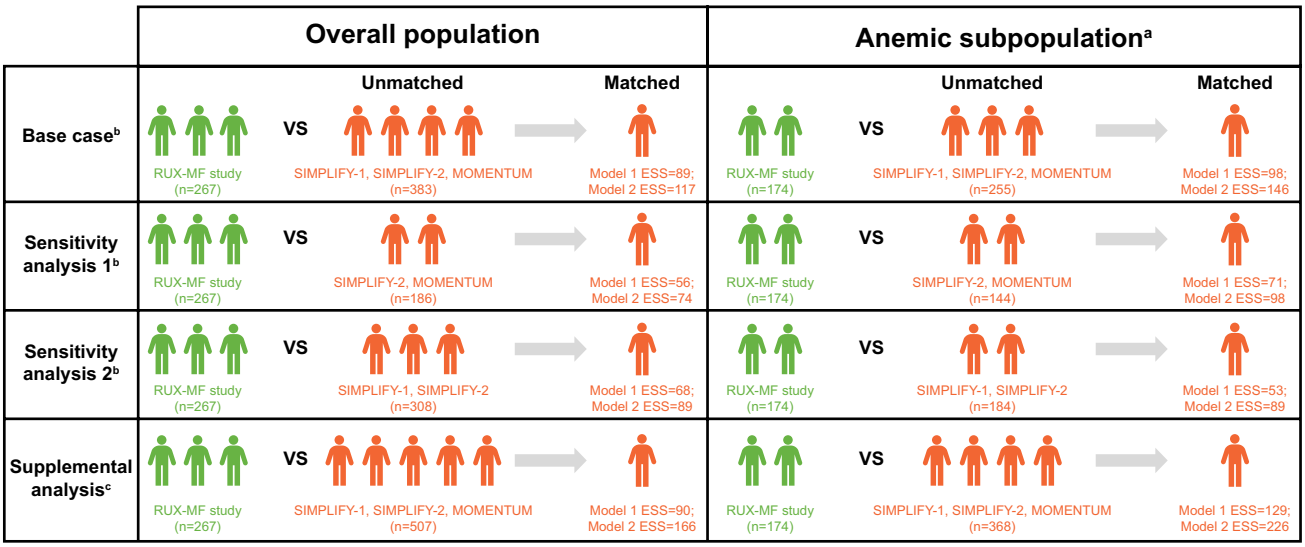


Fig. 1 Study Design. ESS, effective sample size; Hb, hemoglobin; N, number; RUX, ruxolitinib. [a] Patients in the anemic subpopulation had Hb < 10 g/dL. [b] Includes patients who received momelotinib within 1 month after RUX discontinuation. [c] Includes all patients who received momelotinib regardless of when they discontinued RUX

Results

Overall population

A comparison of baseline characteristics in the BAT and momelotinib overall populations showed that the characteristics were consistent after matching using both models 1 and 2 (Table 1). Across all models and scenarios explored, survival improvement favored momelotinib in the MAIC. In the unmatched analysis, OS favored momelotinib (n=383) vs. BAT (n=267), with an HR (95% CI) of 0.388 (0.311–0.483) (Fig. 2 and Fig. 3). In model 1, the momelotinib ESS was 89 after matching and showed a statistically significant improvement in OS with momelotinib vs. BAT, with an HR (95% CI) of 0.525 (0.375–0.737; P<.001). In model 2 (ESS = 117), the HR (95% CI) in favor of momelotinib was 0.486 (0.353–0.668; P<.001).

Consistent with the primary analysis, in sensitivity analysis 1, which excluded patients randomized to ruxolitinib in the SIMPLIFY-1 trial, the HR (95% CI) was 0.474 (0.298–0.755; P=.002) for model 1 (ESS = 56) and 0.517 (0.345–0.775; P=.001) for model 2 (ESS = 74) (Fig. 3). In sensitivity analysis 2, which excluded patients randomized to momelotinib in the MOMENTUM trial, the HR (95% CI) was 0.561 (0.39–0.808; P=.002) for model 1 (ESS = 68) and 0.529 (0.375–0.747; P<.001) for model 2 (ESS = 89) (Fig. 3). Additionally, in a supplemental analysis including all patients who received momelotinib regardless of when they discontinued ruxolitinib, the HR (95% CI) was 0.503 (0.356–0.711; P<.001) for model 1 (ESS = 90) and 0.467 (0.352–0.618); P<.001) for model 2 (ESS = 166) (Fig. 3).

Anemic subpopulation

In the anemic subpopulation, some key baseline characteristics were more closely aligned prior to matching, resulting in a larger matched population despite a smaller starting population. Similar to the overall population, survival improvement was observed in the anemic subgroup treated with momelotinib. In the unmatched analysis, OS favored momelotinib (n = 255) vs. BAT (n = 174), with an HR (95% CI) of 0.395 (0.305–0.513; P<.001). The ESS was 98 after matching to model 1 and showed a statistically significant improvement in OS with momelotinib vs. BAT, with an HR (95% CI) of 0.565 (0.413–0.772; P<.001). In model 2 (ESS = 146), the HR (95% CI) in favor of momelotinib was 0.503 (0.379–0.668; P<.001) (Fig. 4 and Fig. 5).

Sensitivity analysis 1 in the anemic subpopulation was consistent with the primary analysis, with an HR (95% CI) of 0.522 (0.362–0.752; P<.001) for model 1 (ESS = 71) and 0.519 (0.374–0.719; P<.001) for model 2 (ESS = 98) (Fig. 5). In sensitivity analysis 2, there was loss of significance for model 1, where the HR (95% CI) was 0.698 (0.476–1.024; P=.066) for model 1 (ESS = 53) and 0.563 (0.411–0.773; P<.001) for model 2 (ESS = 89) (Fig. 5). In a supplemental analysis of all patients in the anemia subgroup who received momelotinib regardless of when they discontinued ruxolitinib, the HR (95% CI) was 0.447 (0.327–0.611; P<.001) for model 1 (ESS = 129) and 0.399 (0.304–0.523; P<.001) for model 2 (ESS = 226) (Fig. 5).

Table 1 Patient characteristics

OVERALL POPULATION						
	BAT	**Momelotinib (unmatched)**	**SMD**	**Momelotinib (matched)**		**SMD[a]**
	RUX-MF *(n = 267)*	**S1, S2, MOMENTUM *(n = 383)***	**BAT vs. Momelotinib (unmatched)**	**Model 1 (ESS = 89)**	**Model 2 (ESS = 117)**	**BAT vs. Momelotinib (matching model 2)**
Age, mean, years	71.5	66.5	0.540	**71.5**	**71.5**	**0.000**
Male, %	58.4	55.6	0.057	**58.4**	**58.4**	**0.000**
Hb < 10 g/dL, %	65.2	66.8	−0.034	**65.2**	67.3	−0.044
WBC > 25×10^9/L, %	20.6	11.4	0.253	**20.6**	10.7	0.275
PLT < 100×10^9/L, %	46.8	31.1	0.326	**46.8**	33.8	0.267
Spleen length ≥ 10 cm, %	47.2	43.4	0.076	**47.2**	46.1	0.022
TSS ≥ 20, %	37.5	31.9	0.118	**37.5**	26.6	0.235
BMI > 25 kg/m^2, %	37.1	60.9	−0.490	**37.1**	**37.1**	**0.000**
Primary MF, %	54.7	56.7	−0.040	**54.7**	**54.7**	**0.000**
Intermediate-2 risk, %	51.7	53.8	−0.042	**51.7**	**51.7**	**0.000**
High risk, %	15.4	28.5	−0.321	**15.4**	**15.4**	**0.000**
ANEMIC SUBPOPULATION						
	BAT	**Momelotinib (unmatched)**	**SMD**	**Momelotinib (matched)**		**SMD[a]**
	RUX-MF *(n = 174)*	**S1, S2, MOMENTUM *(n = 255)***	**BAT vs. Momelotinib (unmatched)**	**Model 1 (ESS = 98)**	**Model 2 (ESS = 146)**	**BAT vs. Momelotinib (matching model 2)**
Age, mean, years	71.6	68.1	0.414	**71.6**	**71.6**	**0.000**
Male, %	56.9	58.0	−0.022	**56.9**	**56.9**	**0.000**
Hb < 10 g/dL, %	100	100	0.000	**100**	100	0.000
WBC > 25×10^9/L, %	23.0	10.1	0.352	**23.0**	10.5	0.340
PLT < 100×10^9/L, %	51.1	31.7	0.402	**51.1**	32.0	0.395
Spleen length ≥ 10 cm, %	51.7	46.2	0.110	**51.7**	43.8	0.159
TSS ≥ 20, %	45.4	36.5	0.182	**45.4**	37.9	0.153
BMI > 25 kg/m^2, %	36.8	56.6	−0.405	**36.8**	**36.8**	**0.000**
Primary MF, %	56.9	59.6	−0.055	**56.9**	**56.9**	**0.000**
Intermediate-2 risk, %	71.3	53.7	0.370	**71.3**	**71.3**	**0.000**
High risk, %	23.0	37.6	−0.322	**23.0**	**23.0**	**0.000**

Bolding indicates characteristics matched in each model

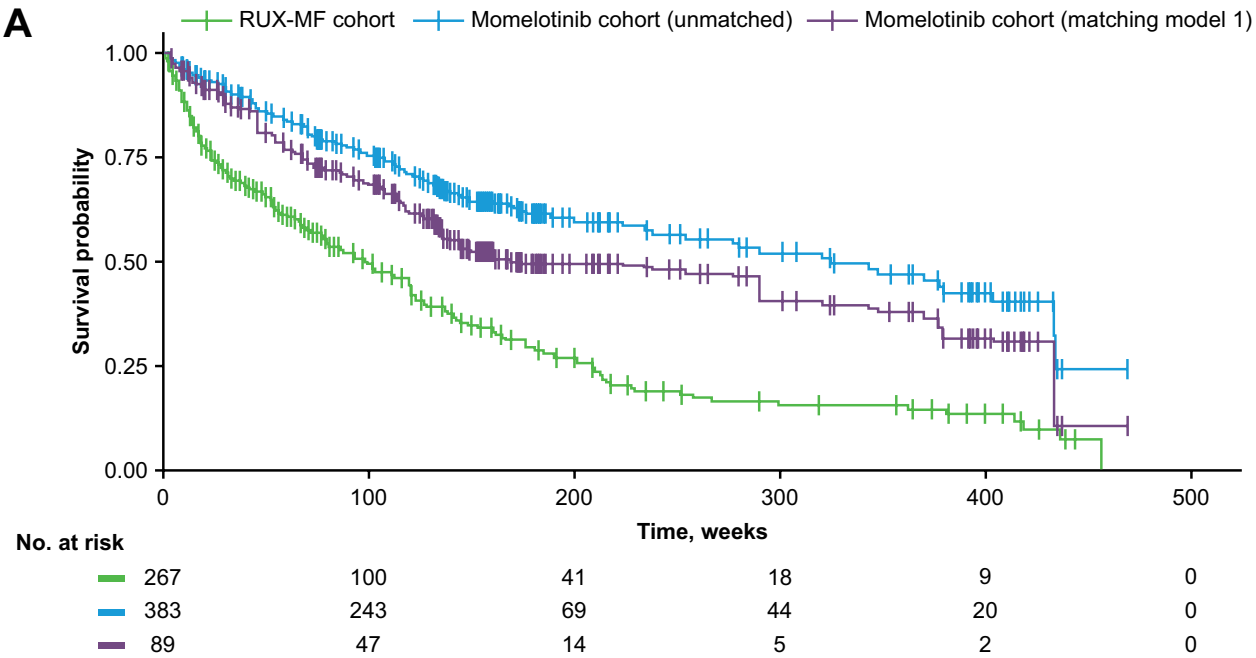
BAT, best available therapy; BMI, body mass index; ESS, effective sample size; Hb, hemoglobin; MF, myelofibrosis; PLT, platelet; S1, SIMPLIFY-1; S2, SIMPLIFY-2; SMD, standardized mean difference; TSS, Total Symptom Score; WBC, white blood cell

[a] SMDs are 0 in Model 1 because the population means are matched and therefore the differences in means are zero (not shown). The same is true for Model 2 for the variables that have been matched (bold cells)

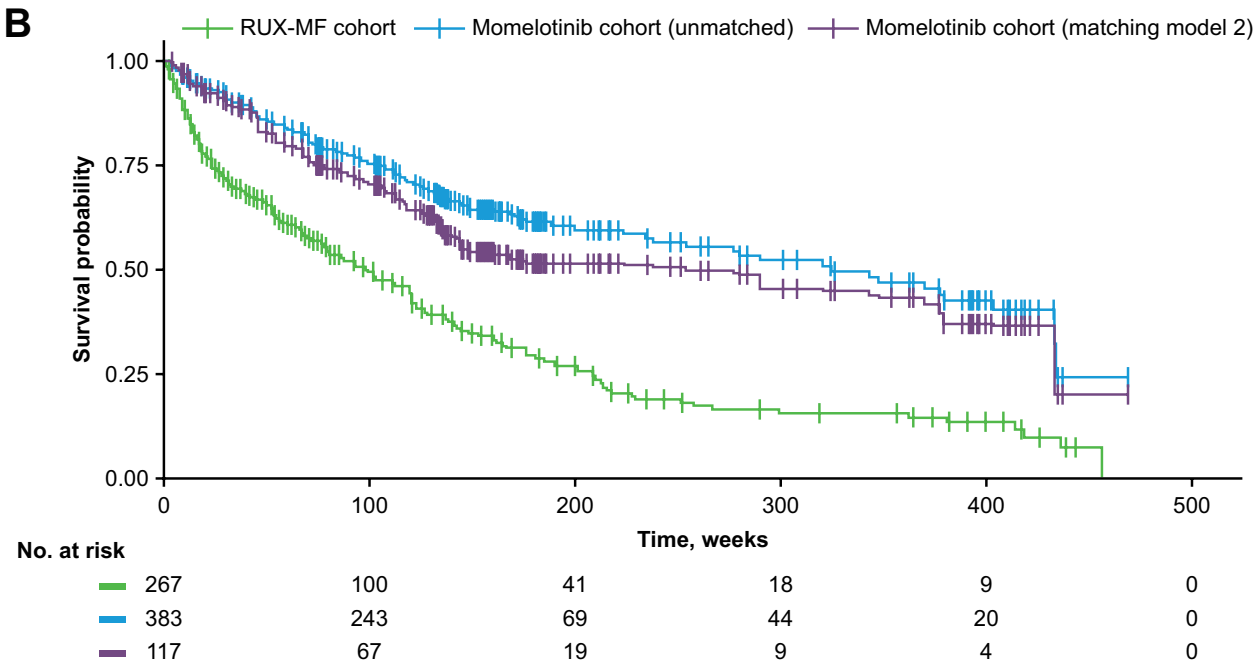
Discussion

The MAICs from this study provided favorable results for survival expectation with momelotinib vs. BAT across all analytical scenarios (base case and all sensitivity/supplemental analyses), with HRs < 1. For both matching models with an ESS of ≥ 20 (models 1 and 2), *P* values were statistically significant (< 0.05). Results favoring momelotinib were also observed in the anemic subgroup, which is particularly relevant because momelotinib is specifically approved in this patient population [25].

This study is a valuable addition to existing evidence that demonstrates the negative impacts of anemia and ruxolitinib discontinuation on OS in patients with MF [13]. Anemia in patients treated with ruxolitinib has consistently been correlated with poor OS and worse outcome in patients with MF [24, 26–28]. A multivariate model developed by Maffioli et al. (2022) recognized lower ruxolitinib dosage, palpable spleen length reduction ≤ 30%, and transfusion requirement as risk factors that negatively impact OS following ruxolitinib discontinuation in a mixed cohort of patients with MF [11]. Findings suggested that most patients with anemia receiving ruxolitinib treatment should transition to a different therapy, which can improve anemia and maintain or improve spleen and symptom responses [11, 29]. A revised model developed to evaluate patients with



Unmatched HR (95% CI)	Unmatched *P* value	Matched HR (95% CI)	MAIC *P* value	n	ESS
0.388 (0.311-0.483)	<.001	0.525 (0.375-0.737)	<.001	383	89.406



Unmatched HR (95% CI)	Unmatched *P* value	Matched HR (95% CI)	MAIC *P* value	n	ESS
0.388 (0.311-0.483)	<.001	0.486 (0.353-0.668)	<.001	383	116.922

Fig. 2 OS from ruxolitinib discontinuation (base case analytical scenario) in the overall population using (**a**) matching model 1 (matched on age, sex, TSS, Hb level, platelet count, palpable spleen length, WBC count, BMI, MF subtype, and DIPSS score) and (**b**) matching model 2 (matched on age, sex, BMI, MF subtype, and DIPSS score). Data cutoff: April 4, 2024. BMI, body mass index; CI, confidence interval; DIPSS, Dynamic International Prognostic Scoring System; ESS, effective sample size; Hb, hemoglobin; HR, hazard ratio; MAIC, matching-adjusted indirect comparison; MF, myelofibrosis; N, number; OS, overall survival; TSS, Total Symptom Score; WBC, white blood cell

intermediate-1–risk MF confirmed these findings, highlighting a clinical need for effective therapies following ruxolitinib discontinuation [30]. Our study addresses this gap, demonstrating that momelotinib may offer superior survival compared to other therapies and could represent a preferred option in this difficult to treat population. Transition to momelotinib therapy from prior ruxolitinib treatment was explored in SIMPLIFY-1. This transition was done without washout or tapering and did not result in symptoms associated with ruxolitinib withdrawal, and control of spleen volume was maintained [29]. Most patients tolerated momelotinib at a full dose, including those previously on low-dose ruxolitinib, and transition to momelotinib was associated with improvement in anemia and a shift toward transfusion independence [29].

The sensitivity and supplemental analyses chosen for this study reinforced the favorable OS results observed for momelotinib vs. BAT in a number of scenarios that may be encountered in clinical practice. In SIMPLIFY-1, patients were JAK inhibitor naive and may have been earlier in their disease course and/or still responding to ruxolitinib; therefore, a subsequent treatment was not yet needed. Additionally, ruxolitinib was only administered for 24 weeks. Excluding this cohort of patients for sensitivity analysis 1 confirmed that this shorter exposure did not drive the OS benefit. Notably, although switching to momelotinib after week 24 was not mandated, nearly all patients transitioned to momelotinib, reinforcing the need for more effective options. Similarly, sensitivity analysis 2 confirmed that OS was not impacted by outside factors, as it excluded patients in MOMENTUM who were enrolled during the COVID-19 pandemic. For the anemic subpopulation, the loss of statistical significance in model 1 during sensitivity analysis 2 may be explained, in part, by the fact that MOMENTUM

	RUX-MF (N=267)							
Analytical scenario	**Unmatched, HR (95% CI)**	**Unmatched *P* value**	**N**	**Matching model**	**Forest plots (matched)**	**Matched, HR (95% CI)**	**MAIC *P* value**	**ESS**
Base case[a]	0.388 (0.311-0.483)	<.001	383	1		0.525 (0.375-0.737)	<.001	89
				2		0.486 (0.353-0.668)	<.001	117
Sensitivity analysis 1[b]	0.518 (0.396-0.676)	<.001	186	1		0.474 (0.298-0.755)	.002	56
				2		0.517 (0.345-0.775)	.001	74
Sensitivity analysis 2[c]	0.373 (0.295-0.47)	<.001	308	1		0.561 (0.39-0.808)	.002	68
				2		0.529 (0.375-0.747)	<.001	89
Supplemental analysis[d]	0.376 (0.307-0.462)	<.001	507	1		0.503 (0.356-0.711)	<.001	90
				2		0.467 (0.352-0.618)	<.001	166

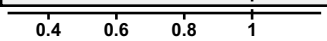
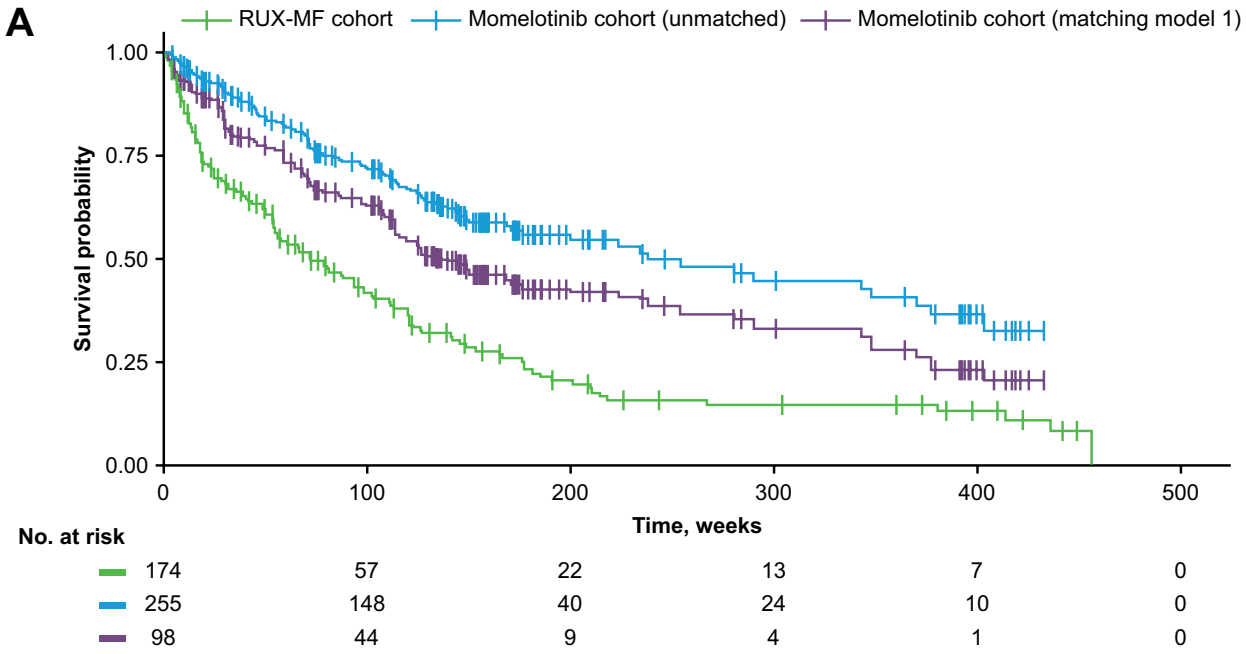
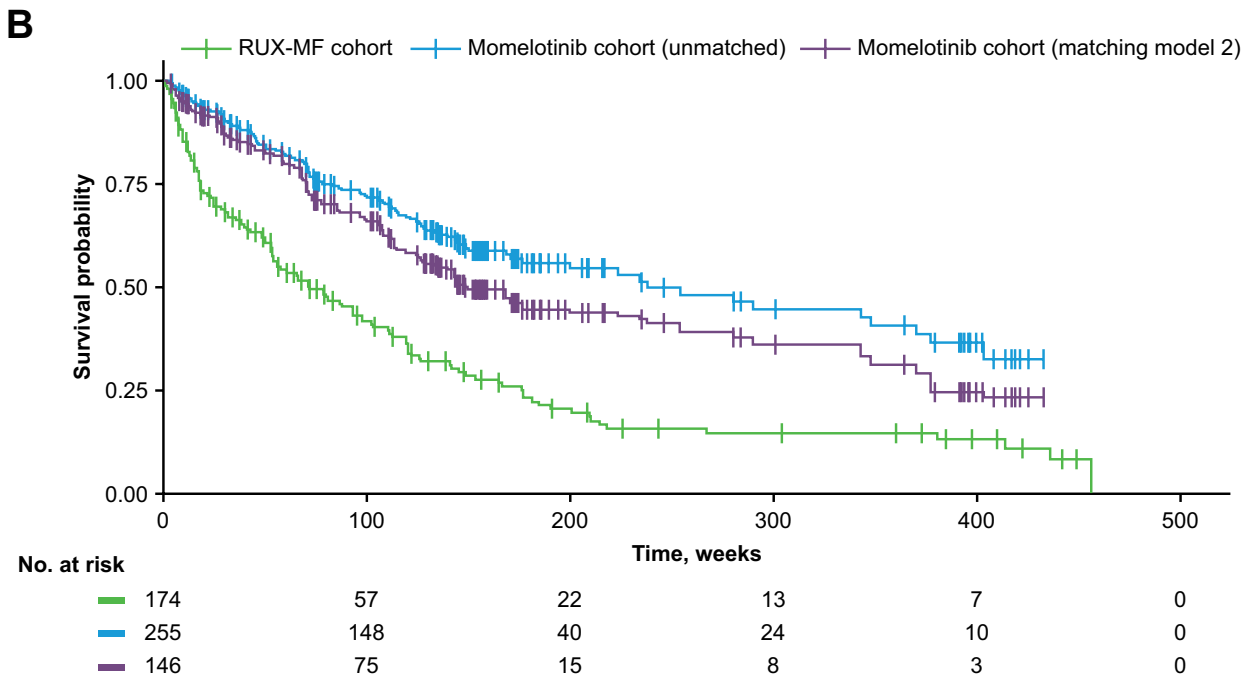


Fig. 3 Summary of MAICs of OS With Momelotinib vs. BAT in the Overall Population. 2L, second line; BAT, best available therapy; CI, confidence interval; ESS, effective sample size; HR, hazard ratio; JAK, Janus kinase; MAIC, matching-adjusted indirect comparison; N, number; OS, overall survival. [a] The base case analysis compared patients who received momelotinib within 1 month since ruxolitinib discontinuation from SIMPLIFY-1, SIMPLIFY-2, and MOMENTUM vs. 267 patients who received 2L BAT from the RUX-MF study. [b] Sensitivity analysis 1 compared patients who received momelotinib within 1 month since ruxolitinib discontinuation from SIMPLIFY-2 and MOMENTUM vs. 267 patients who received 2L BAT from the RUX-MF study (excluding SIMPLIFY-1 removes potential survival effects of patients who were JAK inhibitor naive or those who were treated with ruxolitinib for only 24 weeks prior to study). [c] Sensitivity analysis 2 compared patients who received momelotinib within 1 month since ruxolitinib discontinuation from SIMPLIFY-1 and SIMPLIFY-2 vs. 267 patients who received 2L BAT from the RUX-MF study (excluding MOMENTUM removes potential effects of COVID-19). [d] The supplemental analysis compared all patients who received momelotinib from SIMPLIFY-1, SIMPLIFY-2, and MOMENTUM regardless of time since ruxolitinib discontinuation vs. 267 patients who received 2L BAT from the RUX-MF study (allowed assessment of importance of time since ruxolitinib discontinuation to start of momelotinib or BAT)



Unmatched HR (95% CI)	Unmatched *P* value	Matched HR (95% CI)	MAIC *P* value	n	ESS
0.395 (0.305, 0.513)	<.001	0.565 (0.413, 0.772)	<.001	255	98.476



Unmatched HR (95% CI)	Unmatched *P* value	Matched HR (95% CI)	MAIC *P* value	n	ESS
0.395 (0.305, 0.513)	<.001	0.503 (0.379, 0.668)	<.001	255	146.241

Fig. 4 OS from ruxolitinib discontinuation (base case analytical scenario) in the anemic subpopulation using (**a**) matching model 1 (matched on age, sex, TSS, Hb level, platelet count, palpable spleen length, WBC count, BMI, MF subtype, and DIPSS score) and (**b**) matching model 2 (matched on age, sex, BMI, MF subtype, and DIPSS score). Data cutoff: April 4, 2024. BMI, body mass index; CI, confidence interval; DIPSS, Dynamic International Prognostic Scoring System; ESS, effective sample size; Hb, hemoglobin; HR, hazard ratio; MAIC, matching-adjusted indirect comparison; MF, myelofibrosis; N, number; OS, overall survival; TSS, Total Symptom Score; WBC, white blood cell

specifically enrolled patients with Hb < 10 g/dL, while SIMPLIFY-1 and SIMPLIFY-2 were not restricted to this population. As a result, the momelotinib ESS was the lowest across the scenarios examined, likely contributing to loss of significance.

Finally, differences in washout/transition periods between the different trials (e.g., patients in SIMPLIFY-1 and SIMPLIFY-2 had no washout of prior ruxolitinib, but patients in MOMENTUM did) did not lead to bias in the MAIC, which was confirmed by the supplemental analysis. Sensitivity analysis 1 and the supplemental analysis also allowed consideration of whether prior ruxolitinib treatment and time since ruxolitinib discontinuation to start of momelotinib or BAT might be important factors, and also assessed the potential impact of the crossover design of the momelotinib clinical trials. Results in both instances were consistent with the base case scenario and favored momelotinib. Overall, the findings highlight the benefit of momelotinib in a post-ruxolitinib setting and demonstrate its potential to improve long-term OS vs. BAT, regardless of when it is offered.

This study is subject to potential limitations inherent to MAIC analyses, including unmeasured confounders and biases due to cross-trial differences. The momelotinib trials were phase 3 randomized clinical trials, whereas the RUX-MF study was an observational, real-world study, with all treatment decisions based on the clinical judgment of the hematologist. Due to variability in clinical practice that is both informative and expected, it was assumed that characteristics of the patients from the momelotinib trials were comparable to the overall population of patients reported in the RUX-MF study. Specific matching factors in the ITC were selected a priori and based on clinical plausibility of relevant prognostic factors or potential effect modifiers in

	RUX-MF (N=174)							
Analytical scenario	**Unmatched, HR (95% CI)**	**Unmatched *P* value**	**N**	**Matching model**	**Forest plots (matched)**	**Matched, HR (95% CI)**	**MAIC *P* value**	**ESS**
Base case[a]	0.395 (0.305-0.513)	<.001	255	1		0.565 (0.413-0.772)	<.001	98
				2		0.503 (0.379-0.668)	<.001	146
Sensitivity analysis 1[b]	0.511 (0.377-0.693)	<.001	144	1		0.522 (0.362-0.752)	<.001	71
				2		0.519 (0.374-0.719)	<.001	98
Sensitivity analysis 2[c]	0.389 (0.293-0.516)	<.001	184	1		0.698 (0.476-1.024)	.066	53
				2		0.563 (0.411-0.773)	<.001	89
Supplemental analysis[d]	0.35 (0.275-0.445)	<.001	368	1		0.447 (0.327-0.611)	<.001	129
				2		0.399 (0.304-0.523)	<.001	226

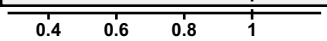


Fig. 5 Summary of MAICs of OS With Momelotinib vs. BAT in the Anemic Subpopulation. 2L, second line; BAT, best available therapy; CI, confidence interval; ESS, effective sample size; HR, hazard ratio; JAK, Janus kinase; MAIC, matching-adjusted indirect comparison; N, number; OS, overall survival. [a] The base case analysis compared patients who received momelotinib within 1 month since ruxolitinib discontinuation from SIMPLIFY-1, SIMPLIFY-2, and MOMENTUM vs. 174 patients who received 2L BAT from the RUX-MF study. [b] Sensitivity analysis 1 compared patients who received momelotinib within 1 month since ruxolitinib discontinuation from SIMPLIFY-2 and MOMENTUM vs. 174 patients who received 2L BAT from the RUX-MF study (excluding SIMPLIFY-1 removes potential survival effects of patients who were JAK inhibitor naive or those who were treated with ruxolitinib for only 24 weeks prior to study). [c] Sensitivity analysis 2 compared patients who received momelotinib within 1 month since ruxolitinib discontinuation from SIMPLIFY-1 and SIMPLIFY-2 vs. 174 patients who received 2L BAT from the RUX-MF study (excluding MOMENTUM removes potential effects of COVID-19). [d] The supplemental analysis compared all patients who received momelotinib from SIMPLIFY-1, SIMPLIFY-2, and MOMENTUM regardless of time since ruxolitinib discontinuation vs. 174 patients who received 2L BAT from the RUX-MF study (allowed assessment of importance of time since ruxolitinib discontinuation to start of momelotinib or BAT)

MF among those available in the trials being compared. Clinical input was prioritized in this selection of factors because limited evidence was identified in the literature on prognostic factors and effect modifiers in MF to guide the choice of matching factors. While most factors in DIPSS/DIPSS-plus risk stratification were accounted for, lack of adjustment for key prognostic variables, including mutational risk, cytogenetic abnormalities, comorbidities, transfusion dependence, and depth of prior ruxolitinib response, limit comparability between the populations and may bias the observed treatment effect.

Furthermore, the process of matching, while essential for comparing across treatment populations, can sometimes result in a small ESS when there are substantial differences between populations. Our analyses considered population matching on as many prognostic factors relevant to OS as possible, aiming to both maintain a balanced population between momelotinib and BAT and retain as large an ESS as possible. While a relatively small ESS may influence the results to some extent, this limitation is inherent to ITCs and was part of the broader considerations when interpreting findings. In addition, although the RUX-MF dataset was based on patients treated prior to the approval of JAK inhibitors other than ruxolitinib, many patients were treated with investigational JAK2 inhibitors or ruxolitinib rechallenge (Supplementary Table 2). Future studies may be warranted to expand on the relative benefits of other JAK inhibitors post ruxolitinib now that they are approved (i.e., fedratinib, momelotinib, pacritinib). In the RUX-MF study, ruxolitinib rechallenge was performed in a small proportion of patients, which creates a dataset that is not strictly "post ruxolitinib," potentially introducing variability due to the patients who were re-exposed to ruxolitinib and may not behave like the patients who definitively discontinued it. Furthermore, the MOMENTUM study follow-up was substantially shorter than in all other studies (Supplementary Table 1). Based on the feasibility assessment performed prior to conducting this analysis, no conclusive evidence was identified that the RUX-MF data would not be comparable with the momelotinib data.

While the trials used in this analysis did not provide long-term comparative outcomes, this MAIC suggests that momelotinib may offer a greater OS benefit than BAT in patients with MF previously treated with ruxolitinib, both in the overall cohort and the anemic population. Future analyses may include evaluating subgroups based on transfusion status and real-world studies to validate the OS results. Together with the results of SIMPLIFY-2 and MOMENTUM, these data further support the use of momelotinib as effective treatment in patients with MF and anemia after ruxolitinib failure, with the potential to positively impact survival expectation.

Supplementary Information The online version contains supplementary material available at https://doi.org/10.1007/s00277-026-06873-w.

Acknowledgements Medical writing support was provided by Prasanthi Mandalay, PhD, of Nucleus Global, an Inizio company, and funded by GSK.

Author contributions All authors were involved in the design of the work, interpretation of data, and manuscript development.

Funding These analyses were supported by GSK.

Data availability The data that support the findings of this analysis are available upon reasonable request. Information on GSK's data sharing commitments and requesting access to anonymized individual participant data and associated study documents can be found at [https://www.gsk-studyregister.com/en/](https:/www.gsk-studyregister.com/en).

Declarations

Ethics approval This study complied with all applicable laws regarding patient privacy. No direct subject contact or primary collection of individual human subject data occurred. All study results are presented as aggregate analyses that omit patient identification; therefore, informed consent and ethics committee or IRB approval were not required.

Financial interests Venediktos Kapetanakis and Balázs Dobi report employment with Evidera, a business unit of Thermo Fisher Scientific, which was contracted to conduct this study by GSK. Massimo Breccia reports honoraria from Novartis, Incyte, GSK, AOP Orphan, AbbVie, Bristol Myers Squibb, Pfizer, Servier, and Otsuka. Catherine Ellis, Nicholas Ballew, Tom Liu, Kelesitse Phiri, Fulya Sen Nikitas, Shiyuan Zhang, and Dwaipayan Patnaik report employment with and stock/stock options in GSK. Giuseppe A. Palumbo reports honoraria for advisory boards and speakers bureau from AbbVie, AOP Orphan, AstraZeneca, BeiGene, Bristol Myers Squibb, GSK, Incyte, MorphoSys, and Novartis and financial support for travel from AbbVie, AOP Orphan, BeiGene, Bristol Myers Squibb, Johnson & Johnson, Novartis, and Stemline Menarini. Elisabetta Abruzzese reports consultancies and advisory boards from Ascentage, Bristol Myers Squibb, GSK, Incyte, Novartis, and Pfizer. Massimiliano Bonifacio reports honoraria from Amgen, Ascentage, Blueprint Medicines, Bristol Myers Squibb, GSK, Incyte, Novartis, and Pfizer. All authors acknowledge medical writing support for this manuscript, which was funded by GSK.

Competing interests Venediktos Kapetanakis and Balázs Dobi report employment with Evidera, a business unit of Thermo Fisher Scientific, which was contracted to conduct this study by GSK. Massimo Breccia reports honoraria from Novartis, Incyte, GSK, AOP Orphan, AbbVie, Bristol Myers Squibb, Pfizer, Servier, and Otsuka. Catherine Ellis, Nicholas Ballew, Tom Liu, Kelesitse Phiri, Fulya Sen Nikitas, Shiyuan Zhang, and Dwaipayan Patnaik report employment with and stock/stock options in GSK. Giuseppe A. Palumbo reports honoraria for advisory boards and speakers bureau from AbbVie, AOP Orphan, AstraZeneca, BeiGene, Bristol Myers Squibb, GSK, Incyte, MorphoSys, and Novartis and financial support for travel from AbbVie, AOP Orphan, BeiGene, Bristol Myers Squibb, Johnson & Johnson, Novartis, and Stemline Menarini. Elisabetta Abruzzese reports consultancies and advisory boards from Ascentage, Bristol Myers Squibb, GSK, Incyte, Novartis, and Pfizer. Massimiliano Bonifacio reports honoraria from Amgen, Ascentage, Blueprint Medicines, Bristol Myers Squibb, GSK, Incyte, Novartis, and Pfizer. All authors acknowledge medical writing support for this manuscript, which was funded by GSK.

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