

Ruxolitinib treatment in patients with polycythemia vera reduces *JAK2* variant allele frequency and improves symptom burden and hematocrit control

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Abstract

Background: In RESPONSE/RESPONSE-2, ruxolitinib was superior to best available therapy (BAT) in patients with polycythemia vera (PV).

Objective: Report a post hoc pooled RESPONSE/RESPONSE-2 analysis.

Design: RESPONSE/RESPONSE-2 were randomized, open-label phase III trials. Adults with hydroxyurea-resistant/intolerant PV were randomized 1:1 to ruxolitinib or BAT; crossover to ruxolitinib was allowed after primary analysis.

Methods: *JAK2V617F* allele burden, hematocrit control, and Myeloproliferative Neoplasm Symptom Assessment Form total symptom score (MPN-SAF TSS) were assessed.

Results: Among 371 patients, mean *JAK2V617F* allele burden declined from baseline (ruxolitinib, 66.1%; crossover, 69.5%) through week 208 (41.4%; 37.1%). Significantly more ruxolitinib versus BAT patients achieved hematocrit control at week 28 (62.0% vs 18.2%; $p < 0.0001$) and $\geq 50\%$ reduction in MPN-SAF TSS at week 16 (48.7% vs 18.0%; $p < 0.0001$).

Conclusion: Patients receiving ruxolitinib experienced decreased *JAK2V617F* allele burden, durable hematocrit control, and better symptom improvements versus BAT, reinforcing ruxolitinib clinical benefit.

Trial registration: RESPONSE: <https://clinicaltrials.gov/study/NCT01243944>; RESPONSE-2: <https://clinicaltrials.gov/study/NCT02038036>.

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Plain language summary

Ruxolitinib reduces the number of cancer cells and improves symptoms in patients with polycythemia vera

Polycythemia vera (PV) is a rare type of cancer that causes patients to produce too many red blood cells. These patients often have difficult symptoms, such as fatigue and an inability to concentrate, and can develop an uncomfortably enlarged spleen. PV is caused by mutations in the Janus kinase 2 (*JAK2*) gene. Most people with PV have a specific change called *JAK2V617F*. The relative amount of *JAK2V617F* the body produces is known as the variant allele frequency (VAF). Patients with higher VAF have more severe symptoms and are more likely to have their PV transform into a more severe disease, such as myelofibrosis or leukemia. Studies have shown that reducing *JAK2V617F* VAF provides meaningful clinical benefit.

Ruxolitinib is an oral drug that treats PV by blocking the effects of mutations in the *JAK2* gene. Here we analyzed data from two clinical trials, RESPONSE and RESPONSE-2, to determine how effective ruxolitinib is as a treatment for PV. Our analysis showed that patients treated with ruxolitinib had improvements in *JAK2V617F* VAF. Ruxolitinib also improved symptoms and helped restore healthy red blood cell levels. These results provide further evidence that treatment with ruxolitinib provides clinical and quality-of-life benefits for patients with PV.

Keywords: allele burden, Janus kinase, MPN-SAF, myeloproliferative neoplasm, patient-reported outcome

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Introduction

Polycythemia vera (PV) is a chronic myeloproliferative neoplasm driven by clonal mutations in Janus kinase 2 (*JAK2*), with the vast majority of patients carrying the *JAK2V617F* allele.¹ The resulting overactivation of the JAK/STAT pathway causes the defining signs of PV: erythrocytosis and elevated hematocrit.¹ Patients with PV often experience burdensome symptoms, most commonly fatigue, concentration problems, itching, and night sweats, and often develop splenomegaly and related abdominal discomfort and early satiety.² PV also carries a risk of transformation into acute myeloid leukemia or myelofibrosis,³ increases the risk of thrombosis,⁴ and reduces overall survival.⁵ *JAK2V617F* variant allele frequency (VAF) correlates with the severity of PV signs and symptoms, with higher VAF associated with greater risk of leukemic and fibrotic transformation, thrombosis, elevated red and white blood cell counts, splenomegaly, and pruritus, although the VAF level corresponding to minimal residual disease remains unclear.⁶

Treatment regimens for PV aim to reduce hematocrit and normalize blood counts to minimize the risk of thrombotic events and to resolve disease-related symptoms.^{7,8} First-line treatment options for patients with PV are aspirin and phlebotomy,⁹ with hydroxyurea and interferons recommended for those with high-risk status (history of thrombosis or age ≥ 60 years).⁹ However, approximately 25% of patients become resistant to or intolerant of hydroxyurea,¹⁰ and interferon treatment, including ropeginterferon, is associated with side effects that cause 20%–30% of patients to discontinue treatment.^{11,12}

Ruxolitinib is a potent and selective JAK1/JAK2 inhibitor approved for treatment of PV in adults who have had an inadequate response to or are intolerant of hydroxyurea.¹³ In the RESPONSE and RESPONSE-2 clinical trials, ruxolitinib was superior to best available therapy (BAT) across the primary and secondary endpoints in both trials, including providing hematocrit control and complete hematologic response, improving PV-related symptoms, and reducing splenomegaly.^{14,15} The phase II MAJIC-PV study showed that ruxolitinib treatment was superior to BAT in achieving *JAK2V617F* VAF partial molecular response (PMR), a threshold correlated with improvement in survival outcomes.¹⁶ The same study also corroborated the key RESPONSE findings, reporting hematologic control with significant and durable symptom reductions in patients treated with ruxolitinib. Here we report the efficacy of ruxolitinib at reducing *JAK2V617F* VAF, controlling hematocrit, and improving symptoms in adults with PV who were resistant to or intolerant of hydroxyurea in a pooled analysis of data from RESPONSE and RESPONSE-2, making this the largest analysis of ruxolitinib in clinical trial patients with PV.

Methods

Study designs

Full trial designs have been reported previously.^{14,15} Briefly, both RESPONSE and RESPONSE-2 were randomized, controlled, open-label, multicenter phase III trials. The trials had similar designs but differed in that RESPONSE patients were required to have

splenomegaly to be enrolled, whereas patients enrolled in RESPONSE-2 did not have palpable splenomegaly. In both trials, patients were randomized 1:1 to receive ruxolitinib 10mg twice daily or BAT until the primary endpoint was recorded (week 32 in RESPONSE; week 28 in RESPONSE-2), after which patients in the BAT group could cross over to receive ruxolitinib if they did not achieve the primary endpoint, if BAT was shown ineffective, or for safety-related reasons. The studies continued through an extended-treatment phase until their completion at week 256 in RESPONSE or week 260 in RESPONSE-2.

This analysis was based on data from previously published phase III trials (RESPONSE and RESPONSE-2).^{14,15} As previously described, the study protocols were approved by an institutional review board or independent ethics committee for all study sites before patient enrollment. This study was conducted in accordance with the Declaration of Helsinki. Patients provided written informed consent for inclusion in RESPONSE and RESPONSE-2; informed consent for this analysis was not required given the use of anonymized data from the previously published trials.

Endpoints and assessments

In this pooled analysis, *JAK2V617F* VAF was analyzed in patients receiving ruxolitinib (at baseline through up to 4 years of follow-up) or BAT (at baseline through up to 80 weeks of follow-up). PMR was defined as $\geq 50\%$ reduction from baseline in *JAK2V617F* VAF; patients achieving an undetectable *JAK2V617F* VAF level were considered to have a complete molecular response. The percentage of patients achieving hematocrit control was assessed at weeks 28 and 80, defined as hematocrit $< 45\%$ that was maintained since week 16 with ≤ 1 phlebotomy occurring post-randomization and before week 4. Patient-reported symptom severity was analyzed at baseline and weeks 16 and 30 (common time points between RESPONSE and RESPONSE-2) with the Myeloproliferative Neoplasm Symptom Assessment Form total symptom score (MPN-SAF TSS).¹⁷

Statistical analyses

Hematocrit control rate and symptom response rate 95% CIs were calculated using the Clopper–Pearson exact method. Odds ratio and 95% CI

for comparisons of the percentage of patients achieving $\geq 50\%$ reduction from baseline in MPN-SAF TSS were calculated by the Mantel–Haenszel method. The full analysis set was used to determine the hematocrit control rate. For molecular response, patients with baseline data and ≥ 1 post-baseline measurement were evaluated. To calculate the symptom response rate, patients with data at baseline and available data at weeks 16 or 30 were included.

Results

Patient demographics and baseline characteristics

Overall, 371 patients were included in the pooled analysis (ruxolitinib, $n=184$; BAT, $n=187$). Patient demographics and key characteristics at baseline were generally well balanced between the ruxolitinib and BAT cohorts (Table 1). Among all patients, the mean *JAK2V617F* VAF was 67.9%, and the mean hematocrit was 43.3 g/L.

JAK2V617F VAF

The mean *JAK2V617F* VAF decreased from 66.1% at baseline to 41.4% at 208 weeks (4 years) in patients randomized to ruxolitinib, from 69.5% to 37.1% at 208 weeks among patients in the ruxolitinib crossover group, and from 69.9% at baseline to 55.9% at week 80 during treatment with BAT (Figure 1(a)). Overall, 38.9% of patients randomized to ruxolitinib achieved PMR as best response, and 57.1% achieved a $\geq 25\%$ decrease in *JAK2V617F* VAF. Among patients in the ruxolitinib crossover group, best response was PMR in 35.3% and a $\geq 25\%$ *JAK2V617F* VAF decrease in 48.7% of patients. Analysis of the BAT group was limited by small patient numbers, as most patients crossed over to ruxolitinib,^{14,18} with best response of PMR in 4 patients (2.6%) and a $\geq 25\%$ *JAK2V617F* VAF decrease in 11 (7.2%). Median (range) time to first PMR was 112 (28–261) weeks among the 68 evaluable patients in the ruxolitinib-randomized group and 130 (24–234) weeks among the 53 patients in the crossover group. A best *JAK2V617F* VAF level $< 10\%$ was achieved by 38 patients (20.7%) in the ruxolitinib-randomized group, 14 (9.0%) in the crossover group, and 7 (3.7%) in the BAT group, whereas 4 (2.2%), 0, and 0, respectively, achieved a best *JAK2V617F* VAF level $< 1\%$. At last measurement, mean *JAK2V617F* VAF reduction from baseline was -28.8% among

Table 1. Patient demographics and baseline characteristics.

Variable	Ruxolitinib (n = 184)	Best available therapy (n = 187)	Total (N = 371)
Age, median (range), years	62.0 [26–90]	62.0 [33–87]	62.0 [26–90]
<60y, n (%)	76 (41.3)	69 (36.9)	145 (39.1)
≥60y, n (%)	108 (58.7)	118 (63.1)	226 (60.9)
Male, n (%)	105 (57.1)	127 (67.9)	232 (62.5)
Race, n (%)			
White	165 (89.7)	162 (86.6)	327 (88.1)
Black	1 (0.5)	0	1 (0.3)
Asian	15 (8.2)	21 (11.2)	36 (9.7)
Other	3 (1.6)	4 (2.1)	7 (1.9)
JAK2V617F VAF, mean (SD), % ^a	66.1 (25.8)	69.9 (25.4)	67.9 (25.7)
Hct, mean (SD), g/L ^b	43.3 (2.0)	43.4 (2.0)	43.3 (2.0)
MPN-SAF TSS, mean (SD)	10.3 (15.1)	8.7 (12.7)	–
^a Ruxolitinib, n = 181; best available therapy, n = 180; total, n = 361. ^b Ruxolitinib, n = 183; best available therapy, n = 186; total, n = 369. Hct, hematocrit; MPN-SAF TSS, Myeloproliferative Neoplasm Symptom Assessment Form total symptom score; VAF, variant allele frequency; Y, years.			

patients randomized to ruxolitinib and –24.5% in the crossover group (Figure 1(b)). The median (range) daily ruxolitinib dose at the time of best VAF response was 20 (5–50) mg for both the ruxolitinib-randomized and crossover populations.

Hematocrit control

At week 28, a significantly greater percentage of patients achieved hematocrit control with ruxolitinib treatment (62.0%; 95% CI: 54.5%, 69.0%) than with BAT (18.2%; 95% CI: 12.9%, 24.5%; $p < 0.0001$; Figure 2). Durable hematocrit control was maintained to week 80 by 50.5% (95% CI: 43.1%, 58.0%) and to week 208 by 33.7% (95% CI: 26.9%, 41.0%) of patients randomized to receive ruxolitinib, whereas nearly all patients in the BAT arm crossed over to ruxolitinib before week 80. For the ruxolitinib-randomized group, 62.3% (38/61) of evaluable patients who achieved PMR also achieved hematocrit control, versus 61.8% (76/123) of those without molecular response; the corresponding percentages for the BAT group were 75.0% (3/4) and 16.9% (31/183), respectively.

Symptom response

A significantly greater percentage of patients randomized to ruxolitinib achieved a ≥50% reduction from baseline in MPN-SAF TSS at weeks 16 and 30 ($p < 0.0001$; Figure 3(a)). Consistent with this finding, patients randomized to ruxolitinib experienced a greater mean improvement from baseline in MPN-SAF TSS compared with BAT at weeks 16 and 30 ($p < 0.0001$; Figure 3(b)). For the ruxolitinib-randomized group, 49.2% (30/61) of evaluable patients who achieved PMR also achieved symptom control, versus 34.1% (42/123) of those without molecular response; the corresponding percentages for the BAT group were 0 and 11.5% (21/183), respectively.

Discussion

In this pooled analysis of data from the RESPONSE and RESPONSE-2 clinical trials—the largest clinical trial population of ruxolitinib in patients with PV to date—JAK2V617F VAF improved over time with up to 4 years of treatment with ruxolitinib. Furthermore, long-term results showed continuous improvement in

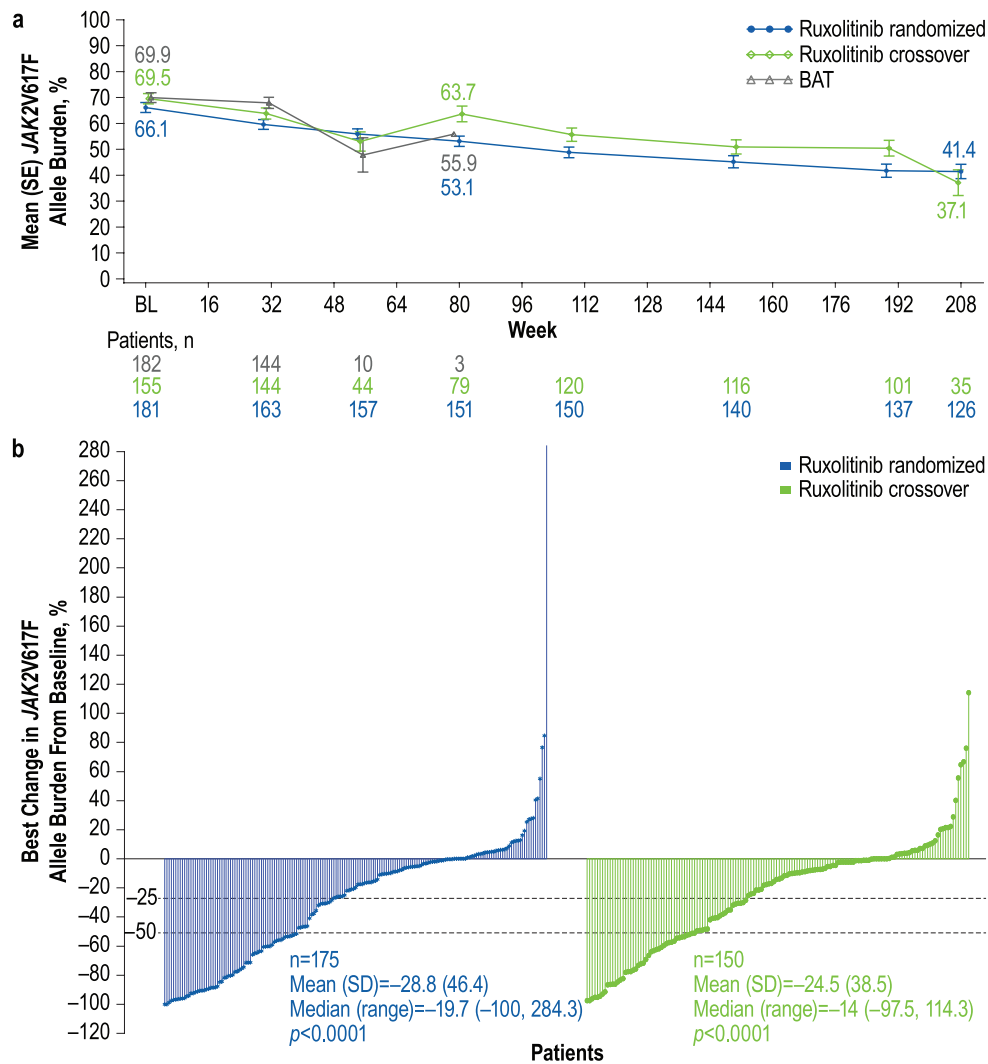


Figure 1. JAK2V617F VAF. (a) Absolute JAK2V617F VAF level. (b) Best change in JAK2V617F VAF from baseline. The BAT group includes all patients randomized to BAT with censoring at crossover to ruxolitinib; the ruxolitinib crossover group includes all patients randomized to BAT who eventually crossed over to ruxolitinib, including time on BAT. BAT, best available therapy; BL, baseline; VAF, variant allele frequency.

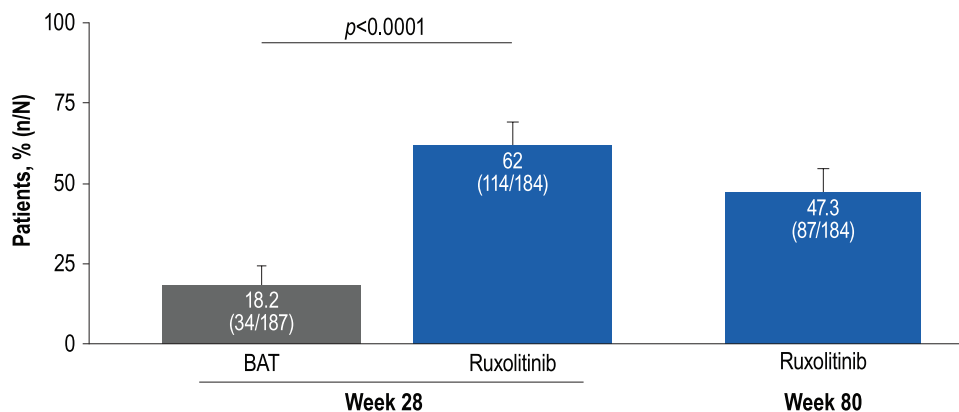


Figure 2. Hematocrit control. Error bars are 95% CI. BAT, best available therapy.

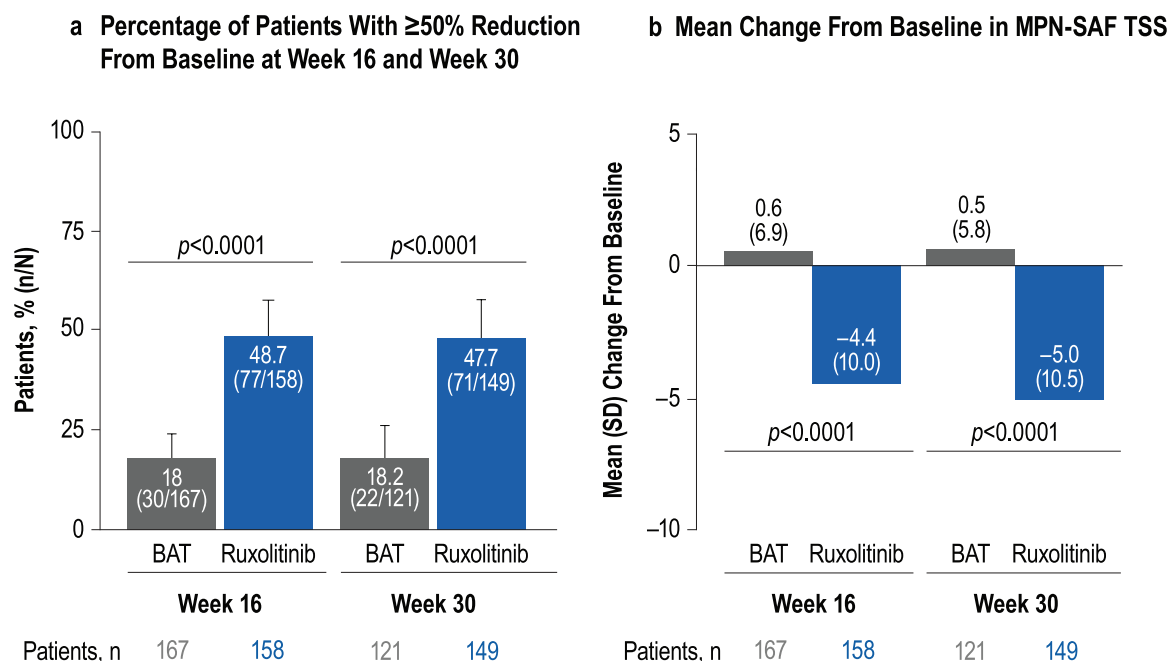


Figure 3. Symptom response. (a) Percentage of patients with $\geq 50\%$ reduction from baseline at weeks 16 and 30. (b) Mean change from baseline in MPN-SAF TSS. Panel a: Error bars are 95% CI. Panel b: week 30 includes week 32 of RESPONSE and week 28 of RESPONSE-2.

BAT, best available therapy; MPN-SAF TSS, Myeloproliferative Neoplasm Symptom Assessment Form total symptom score.

$JAK2V617F$ VAF over time without plateau. Ruxolitinib was also associated with significant improvements in hematocrit control and symptom burden compared with BAT, consistent with findings from the RESPONSE and RESPONSE-2 primary analyses.^{14,15}

The $JAK2V617F$ VAF reduction findings reported here align with those from MAJIC-PV, an open-label, randomized, controlled trial of ruxolitinib versus BAT in the treatment of PV.¹⁶ Importantly, crossover was not permitted in MAJIC-PV, allowing for clearer interpretation of long-term follow-up data than with RESPONSE or RESPONSE-2, which permitted crossover from BAT to ruxolitinib. In the MAJIC-PV trial, up to 5 years of treatment with ruxolitinib was associated with more frequent and greater reductions in $JAK2V617F$ VAF compared with BAT (Table 2).¹⁶ Specifically, 56% of patients (median follow-up time, 48 months) who received ruxolitinib achieved a $>50\%$ reduction in $JAK2V617F$ VAF compared with 25% of those who received BAT (median follow-up time, 36 months; $p < 0.001$).¹⁶ MAJIC-PV also demonstrated that a $>50\%$ reduction in $JAK2V617F$ VAF is an important threshold that translates into meaningful clinical benefit. Patients treated with ruxolitinib who achieved this

threshold experienced significantly improved progression-free survival ($p = 0.001$), event-free survival (a composite of major thrombosis, major hemorrhage, transformation, or death; $p = 0.006$), overall survival ($p = 0.04$), complete response at 1 year ($p = 0.04$), and fewer hemorrhagic events ($p = 0.04$) compared with those with $<50\%$ reduction in $JAK2V617F$ VAF.¹⁶ In the full study population, thromboembolic event-free survival and overall event-free survival were significantly longer with ruxolitinib treatment ($p = 0.05$ and $p = 0.03$, respectively) compared with BAT, whereas progression-free survival and overall survival did not significantly separate between groups during the study time frame.¹⁶ These results were also corroborated by findings from a recent meta-analysis, which reported that lower $JAK2V617F$ VAF is a predictor for improved clinical status, associated with lower white blood cell and hematocrit levels, as well as lower risk of leukemic and fibrotic transformation, thrombosis, splenomegaly, and pruritus.⁶ An earlier analysis of the RESPONSE study also suggested a connection between allele burden reduction and spleen size, such that 80% of patients treated with ruxolitinib who achieved a $\geq 20\%$ allele burden reduction also had a $\geq 35\%$ reduction in spleen volume.¹⁹

Table 2. Common outcomes between the pooled RESPONSE/RESPONSE-2 analysis and the MAJIC-PV trial.

Variable	Pooled RESPONSE/RESPONSE-2: ruxolitinib (n = 184)	MAJIC-PV: ruxolitinib (n = 93)
JAK2V617F VAF	39% achieved ≥50% VAF reduction as best improvement within 4 years	56% achieved >50% VAF reduction at median follow-up of 4 years
Hct control	62% achieved Hct control at week 28 ^a	Mean Hct remained <40% throughout study
Patients achieving MPN-SAF TSS of ≥50%, %	48% at week 30	61% at ≥1 time point
^a Hct control was defined as Hct <45% maintained since week 16 with ≤1 phlebotomy occurring post-randomization and before week 4. Hct, hematocrit; MPN-SAF TSS, Myeloproliferative Neoplasm Symptom Assessment Form total symptom score; VAF, variant allele frequency.		

Similar to findings in this pooled RESPONSE analysis, MAJIC-PV showed statistically significant improvements in fatigue, itching, bone pain, early satiety, night sweats, and weight loss with ruxolitinib compared with BAT.¹⁶ MAJIC-PV also reported that the improvements in MPN-SAF TSS were durable with ruxolitinib treatment, with scores significantly improved compared with BAT after 2 months of treatment and lasting a mean of 52 months.¹⁶

A limitation of this study is the difference in study design between RESPONSE and RESPONSE-2, which may have led to slightly different ruxolitinib exposure durations (8 vs 7 months, respectively). Patients in RESPONSE-2 had 1 month less ruxolitinib exposure at the time of primary endpoint evaluation. The crossover designs of RESPONSE and RESPONSE-2 also limited the interpretability of the data from the BAT cohort after the primary endpoint was collected. Finally, selection bias may have affected the results, as is possible with longitudinal analyses.

Conclusion

In this pooled analysis, reductions in JAK2V617F VAF were consistently observed through week 208 in patients treated with ruxolitinib, including those who crossed over from BAT, comparable with results from MAJIC-PV. More than a third of patients in the ruxolitinib-randomized group achieved PMR, which MAJIC-PV demonstrated is associated with better long-term outcomes, including improved survival, hemorrhage, and disease progression outcomes, as well as increased likelihood of achieving hematologic

response. Patients with PV treated with ruxolitinib achieved durable hematocrit control through week 80 and had better symptom control through week 30 compared with BAT. These results provide further evidence of the clinical and quality-of-life benefits of ruxolitinib after failure of initial cytoreductive treatment in patients with PV.

Declarations

Ethics approval and consent to participate

This analysis was based on data from previously published phase III trials (RESPONSE and RESPONSE-2), which were conducted in accordance with the Declaration of Helsinki. Study protocols were approved by an institutional review board (IRB) or independent ethics committee for all study sites before patient enrollment. This analysis was based on data from previously published phase III trials (RESPONSE and RESPONSE-2); patients provided written informed consent for inclusion into these studies. Informed consent and further IRB/ethics approval for this analysis were not required, given the use of anonymized data from the previously published RESPONSE and RESPONSE-2 trials.

Consent for publication

Not applicable.

Author contributions

Claire N. Harrison: Data curation; Formal analysis; Methodology; Visualization; Writing – review & editing.

Jean-Jacques Kiladjian: Conceptualization; Investigation; Methodology; Supervision; Writing – review & editing.

J. E. Hamer-Maansson: Conceptualization; Investigation; Methodology; Supervision; Writing – review & editing.

Ahmad Naim: Conceptualization; Investigation; Methodology; Supervision; Writing – review & editing.

Francesca Palandri: Conceptualization; Investigation; Methodology; Supervision; Writing – review & editing.

Francesco Passamonti: Conceptualization; Investigation; Methodology; Supervision; Writing – review & editing.

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Competing interests

CNH received honoraria from BMS, Novartis, CTI, GSK, AOP, Galecto, AbbVie, and MorphoSys; served on the speakers bureau for BMS, Novartis, CTI, GSK, AOP, Galecto, AbbVie, and MorphoSys; and received research funding from Novartis. J-JK acted as a consultant and received honoraria from Novartis, BMS, GSK, AOP Health, AbbVie, Incyte, and PharmaEssentia. JEH-M and AN are employees and shareholders of Incyte. FPal acted as a consultant and received honoraria from AbbVie, Amgen, AOP, BMS Celgene, CTI, GSK, Karyopharm, and Novartis. FPas has received honoraria from AbbVie and Janssen.

Availability of data and materials

Incyte Corporation (Wilmington, DE, USA) is committed to data sharing that advances science

and medicine while protecting patient privacy. Qualified external scientific researchers may request anonymized datasets owned by Incyte for the purpose of conducting legitimate scientific research. Researchers may request anonymized datasets from any interventional study (except phase I studies) for which the product and indication have been approved on or after January 1, 2020, in at least one major market (e.g., US, EU, and JPN). Data will be available for request after the primary publication or 2 years after the study has ended. Information on Incyte's clinical trial data sharing policy and instructions for submitting clinical trial data requests are available at: <https://www.incyte.com/Portals/0/Assets/Compliance%20and%20Transparency/clinical-trial-data-sharing.pdf?ver=2020-05-21-132838-960>

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