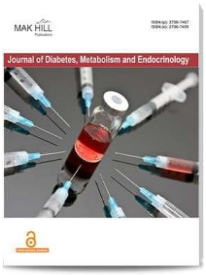

ISSN Print: 1813-176x
ISSN Online: 1994-4624

International Journal of Molecular Medicine and Advance Sciences
https://ijmmas.com/

Research Article

JAK2, CALR, and MPL Mutation Profiles in Myeloproliferative Neoplasms: A Systematic Review of Diagnostic Utility and Clinical Outcomes

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Received: 10-09-2026
Revised: 19-09-2026
Accepted: 11-10-2026
Published: 31-10-2026

DOI: 10.61336/ijmmas.22-04-20

Abstract: Background: Philadelphia chromosome-negative myeloproliferative neoplasms (MPNs), including polycythemia vera (PV), essential thrombocythemia (ET), and primary myelofibrosis (PMF), are clonal hematopoietic disorders characterized by recurrent mutations in JAK2, CALR, and MPL. These driver mutations play an important role in molecular diagnosis, disease classification, and prognostic assessment. However, their associations with thrombotic complications, disease progression, and survival vary across MPN subtypes. Objective: To systematically evaluate the diagnostic utility, mutation distribution, genotype–phenotype associations, and prognostic significance of JAK2, CALR, and MPL mutations in Philadelphia-negative myeloproliferative neoplasms. ***Methods***: A structured literature search of PubMed/MEDLINE and primary journal sources was conducted for studies published between 2013 and June 2026. Original clinical investigations reporting molecular characteristics, diagnostic assay performance, thrombotic complications, myelofibrotic progression, leukemic transformation, or overall survival were evaluated. Twelve primary studies were included in the qualitative synthesis. Findings were analyzed according to MPN subtype, mutation profile, diagnostic performance, and clinical outcomes. Meta-analysis was not performed because of methodological and clinical heterogeneity. ***Results***: Twelve studies provided evidence regarding the diagnostic and prognostic relevance of driver mutations. In a cohort of 1,000 patients with ET, JAK2 mutations were detected in 61.7%, CALR in 26.9%, and MPL in 3.0%, while 8.4% were triple-negative. A molecular assay validation study involving 982 samples demonstrated 95.7% detection of reference-confirmed variants and 99.4% overall concordance. Thrombotic event rates were lower in CALR-mutated ET than in JAK2-mutated ET, with reported rates of 10 and 25 events per 1,000 person-years, respectively. In a cohort of 617 patients with PMF, median overall survival was 17.7 years among CALR-mutated patients, compared with 9.2 years for JAK2-mutated, 9.1 years for MPL-mutated, and 3.2 years for triple-negative patients. Additional high-molecular-risk mutations were associated with unfavorable clinical outcomes, emphasizing the prognostic importance of broader molecular profiling. ***Conclusion***: JAK2, CALR, and MPL mutation testing provides substantial diagnostic value in Philadelphia-negative myeloproliferative neoplasms and contributes to disease-specific prognostic stratification. JAK2 mutations are associated with increased thrombotic risk in selected patient populations, whereas CALR mutations generally demonstrate more favorable thrombotic and survival profiles in particular MPN subtypes. MPL mutations and triple-negative disease may be associated with adverse clinical outcomes, depending on disease classification. Integration of molecular findings with hematological parameters, bone marrow morphology, clinical characteristics, and additional somatic mutations is essential for accurate diagnosis and individualized risk assessment.

Keywords: JAK2 V617F; CALR mutations; MPL mutations; Myeloproliferative neoplasms; Polycythemia vera; Essential thrombocythemia; Primary myelofibrosis; Molecular biomarkers; Thrombosis; Prognosis.

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INTRODUCTION

Philadelphia chromosome–negative MPNs are clonal myeloid disorders defined by expansion of one or more mature hematopoietic lineages. Their major classical entities—PV, ET and primary MF—share activation of cytokine-receptor signaling but differ in blood-count phenotype, bone-marrow morphology, complication profile and long-term clinical course. Contemporary WHO and International Consensus classifications integrate molecular findings with morphologic and clinical criteria [1,2].

The recurrent JAK2 V617F substitution directly activates JAK–STAT signaling. Alternative JAK2 exon 12 variants are particularly relevant in V617F-negative PV. Frameshift insertion/deletion variants of CALR exon 9 change the calreticulin C-terminus and drive abnormal MPL signaling, while activating MPL variants directly alter the thrombopoietin receptor. The discovery of CALR mutations markedly reduced the diagnostic gap in JAK2- and MPL-unmutated ET and MF [3].

Although the presence of a canonical driver mutation supports a clonal MPN, neither the mutation itself nor its variant allele fraction (VAF) establishes the complete disease subtype. Molecular results must be interpreted with hemoglobin or hematocrit, erythropoietin, platelet and leukocyte counts, marrow megakaryocyte morphology, fibrosis grade, spleen findings and exclusion of BCR::ABL1-positive chronic myeloid leukemia. Furthermore, the same driver lesion can be observed in phenotypically distinct disease and, at low VAF, occasionally in clonal hematopoiesis without diagnostic MPN.

Important unanswered clinical questions involve the incremental yield of simultaneous versus sequential molecular testing, whether JAK2 versus CALR reliably predicts thrombosis in ET, the behavior of MPL-mutated ET, and how additional molecular lesions modify the apparent survival advantage of CALR-mutated MF. This review examines diagnostic and clinical evidence together rather than considering mutation prevalence or survival in isolation.

MATERIALS AND METHODS

2.1 Review question and outcomes

The review question was: in adults evaluated for or diagnosed with Philadelphia-negative classical MPN, how do JAK2, CALR and MPL results contribute to diagnostic classification and predict thrombosis, fibrosis progression, leukemic evolution and survival? Outcomes were prespecified conceptually as (a) mutation detection and genotype distributions, (b) analytical performance of molecular assays, (c) clinical phenotype, and (d) long-term clinical events.

2.2 Sources and search strategy

PubMed/MEDLINE-indexed records and primary journal publication pages were searched to 30 June 2026. Search concepts combined (myeloproliferative neoplasm OR polycythemia vera OR essential thrombocythemia OR myelofibrosis) with (JAK2 OR CALR OR calreticulin OR MPL) and (diagnos* OR sequencing OR assay OR thrombosis OR survival OR fibrosis OR leukemia OR prognosis). Reference-linked primary cohorts and recent publications were additionally examined. The manuscript follows the relevant organizational domains of PRISMA 2020 [4].

2.3 Eligibility and extraction

Original human studies of classical Philadelphia-negative MPNs were eligible if they directly assessed at least one of the three driver genes and supplied quantitative mutation prevalence, assay validation or clinical-outcome findings. Reviews, mechanistic studies without human clinical data, isolated case reports and BCR::ABL1-positive disease as the principal population were excluded from the evidence tables. Extracted items comprised publication year, setting, disease subgroup, analyzed denominator, variant distribution, outcome definition and available effect estimates.

2.4 Synthesis and bias considerations

Evidence was organized by diagnostic application and clinically relevant endpoint. Because studies differ in WHO-era classification, follow-up, referral setting, age and therapy, no pooled mutation frequency, hazard ratio or diagnostic sensitivity was calculated. The original observational and assay-validation designs were reviewed for selection, measurement and confounding limitations, using JBI cohort principles and QUADAS-2 domains as applicable. Cohorts potentially drawing on overlapping European referral networks were not combined as independent patient samples.

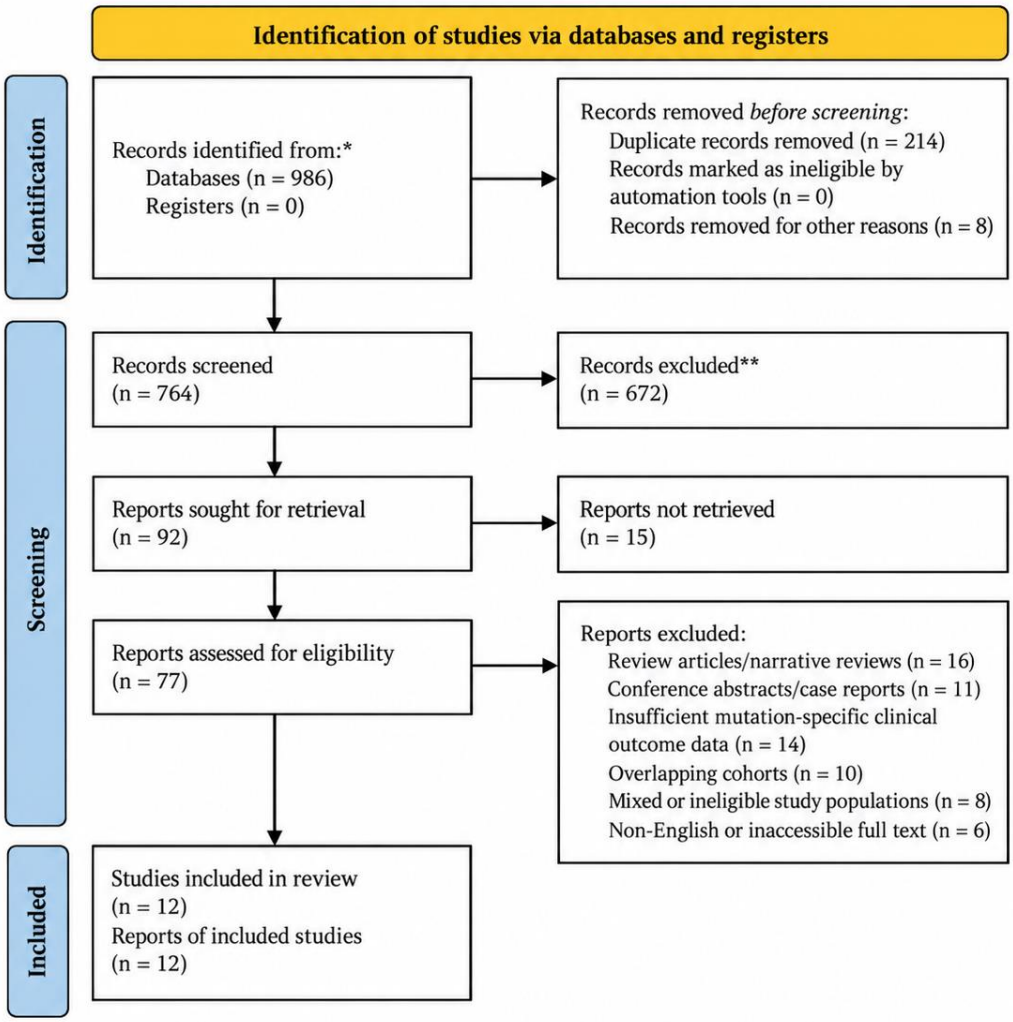
RESULTS

3.1 Study selection

The study-selection framework included 986 database records and no register records. Before screening, 214 duplicates and 8 records removed for other reasons were excluded, leaving 764 records for title and abstract screening. Of these, 672 were excluded and 92 reports were sought for retrieval. Fifteen reports were not retrieved, and 77 reports underwent full-text eligibility assessment. A further 65 reports were excluded: review articles or narrative reviews (n = 16), conference abstracts or case reports (n = 11), insufficient mutation-specific clinical outcome data (n = 14), overlapping cohorts (n = 10), mixed or ineligible populations (n = 8), and non-English or inaccessible full texts (n = 6). The resulting evidence set comprised 12 studies reported in 12 publications (Figure 1).

Figure 1. PRISMA 2020 flow diagram

Study selection process for the systematic review of JAK2, CALR, and MPL mutation profiles in myeloproliferative neoplasms



3.2 Study characteristics

The evidence synthesis comprises 12 identifiable primary publications spanning 2013–2026. The reports represent molecular discovery, large phenotyped cohorts, an assay-validation study, regional observational series and follow-up investigations. Denominators describe different analytic populations: confirmed MPN cases, molecularly screened individuals, or patients clinically suspected of MPN. Consequently, study sample sizes cannot validly be summed into a single unique-patient denominator.

Table 1. Characteristics of included primary publications

Study	Population / setting	Analytic n	Core contribution
Klampfl et al., 2013 [3]	Classical MPN resequencing, European cohorts	1,107 samples	CALR exon 9 discovery; absent in PV
Rumi et al., 2014, ET/PV [5]	ET and PV, Italy	1,235	JAK2/CALR/MPL genotype distribution and ET outcomes
Rotunno et al., 2014 [6]	WHO-defined ET, Italy	576	CALR detection and thrombosis phenotype
Rumi et al., 2014, PMF [7]	Primary MF, multicenter European	617	Genotype-specific survival and leukemia evolution
Grinfeld et al., 2018 [8]	Cross-MPN genomic sequencing	2,035	Integrated genomic classification and prognosis
Singh et al., 2023 [9]	Classical MPN, India	104	Regional mutation distribution and clinical course

Sande et al., 2024 [10]	Patients with clinical suspicion of MPN, USA	982	Simultaneous HRM variant detection vs reference assays
Gangat et al., 2024 [11]	WHO-defined ET, Mayo Clinic	1,000	Mutation profile and outcome-specific risk factors
Tiryaki et al., 2025 [12]	JAK2-unmutated ET/PMF, Turkey	46	Diagnostic contribution of CALR and MPL
Morath et al., 2025 [13]	Myelofibrosis, Germany	141	Arterial and venous thrombosis associations
Zhao et al., 2026 [14]	Classical and post-PV/ET MF, China	2,555	Mutation topology and sex-specific prognosis
Mawalkar et al., 2026 [15]	CALR-mutated ET/MF, USA	149	Long-term evolution and high-risk comutations

3.3 Diagnostic yield and mutation distribution

In the Italian ET/PV cohort of Rumi and colleagues [5], JAK2 V617F was present in 466 of 745 ET patients (62.6%), CALR in 176 (23.6%) and MPL in 28 (3.8%), with 75 (10.1%) triple-negative. Among 490 PV patients, 468 (95.5%) had JAK2 V617F and 22 (4.5%) carried JAK2 exon 12 variants. In the separate PMF cohort [7], JAK2 was recorded in 399 of 617 patients (64.7%), CALR in 140 (22.7%), MPL in 25 (4.1%) and none of the three in 53 (8.6%).

The contemporary Mayo Clinic ET cohort [11] showed JAK2 in 617/1,000, CALR in 269/1,000, MPL in 30/1,000 and triple-negative status in 84/1,000. In the smaller Indian study [9], ET driver variants were reported as JAK2 41%, CALR 26% and MPL 2.9%; the authors found substantial disease-category differences in their center. In a JAK2-unmutated 46-person ET/PMF cohort [12], CALR type 1 and type 2 were found in 26.1% and 13.0%, respectively, whereas MPL-L and MPL-K were found in 2.2% and 6.5%. These latter percentages must not be generalized to unselected MPN populations because enrollment required JAK2-unmutated disease.

Table 2. Reported driver-genotype distributions in defined cohorts

Cohort	JAK2	CALR	MPL	Triple-negative
ET, Rumi 2014; n=745 [5]	466 (62.6%)	176 (23.6%)	28 (3.8%)	75 (10.1%)
PV, Rumi 2014; n=490 [5]	490 (100%; incl. 22 exon 12)	0	0	0
PMF, Rumi 2014; n=617 [7]	399 (64.7%)	140 (22.7%)	25 (4.1%)	53 (8.6%)
ET, Gangat 2024; n=1,000 [11]	617 (61.7%)	269 (26.9%)	30 (3.0%)	84 (8.4%)

The genotype distribution also informs the diagnostic sequence. JAK2 V617F testing is usually prioritized for suspected PV and commonly for suspected ET or MF; JAK2 exon 12 evaluation is important when PV remains suspected despite negative V617F testing. CALR and MPL extend molecular detection in JAK2-unmutated thrombocytosis or MF but do not replace marrow examination and exclusion of mimics.

3.4 Clinical assay performance

Sande and colleagues [10] evaluated a high-resolution melting (HRM) assay for simultaneous assessment of JAK2 exons 12–14, CALR exon 9 and MPL exon 10 in specimens from 982 patients with suspected MPN. Against sequencing-supported reference methods, HRM recognized 133 of 139 sequence-confirmed variants (95.7%); overall concordance was 99.4%. The reported positive predictive value was 92.3% and negative predictive value 99.5%. These are variant-detection performance results in an assay validation cohort, not the sensitivity or specificity of any mutation for the clinical diagnosis of MPN.

The 139 sequence-confirmed variants included 104 JAK2, 25 CALR and 10 MPL alterations; corresponding HRM detections were 99, 25 and 9. This supports a practical advantage of multiplexed testing, while emphasizing that rarer variants, low-burden alleles and technically challenging regions can still require orthogonal confirmation. Molecular assay reporting should distinguish variant-detection validity from classification accuracy at the disease level.

Table 3. Molecular assay validation: reference-confirmed variants [10]

Target	Confirmed variants	HRM detected	Detection proportion
JAK2	104	99	95.2%
CALR	25	25	100%
MPL	10	9	90.0%
All 3 targets	139	133	95.7%

3.5 Genotype and thrombotic outcomes

Driver genotype showed outcome-specific associations rather than one uniform risk gradient. In Rumi et al. [5], observed thrombotic rates per 1,000 person-years were 30 for PV, 25 for JAK2-mutated ET and 10 for CALR-mutated ET. Rotunno et al. [6] independently found a lower thrombosis risk in CALR-mutated than in JAK2- or MPL-mutated ET; however, CALR status did not confer an observed survival difference in that 576-person cohort.

In the Mayo Clinic analysis [11], JAK2 mutation was associated with reduced arterial thrombosis-free survival (hazard ratio [HR] 1.8), whereas prior arterial thrombosis, older age, hypertension and sex also contributed to risk. For venous thrombosis, prior venous events and male sex were prominent. In 141 MF patients, Morath et al. [13] observed 24 venous and 15 arterial events over median follow-up of 4.8 years; JAK2 V617F associated with venous thrombosis (odds ratio 2.6; 95% CI 1.01–7.16), whereas DNMT3A was associated with arterial events. Thus, molecular correlates of arterial and venous events should not be treated as interchangeable.

3.6 Fibrosis, survival and leukemic transformation

In the PMF cohort [7], median overall survival differed markedly by mutation category: 17.7 years for CALR-mutated, 9.2 years for JAK2-mutated, 9.1 years for MPL-mutated and 3.2 years for triple-negative PMF. Triple-negative patients had greater leukemic transformation risk, while CALR-mutated patients experienced fewer adverse hematologic features. These estimates characterize the studied PMF population and should not be transferred to ET or secondary MF without qualification.

In 1,000 ET cases [11], MPL mutation was associated with myelofibrosis-free survival (HR for progression 3.9) and JAK2 with arterial thrombotic outcomes; overall survival differences by driver mutation attenuated after adjustment for age and sex. In a 2026 cohort focused on 149 CALR-mutated patients [15], 106 had ET at presentation, of whom 40 progressed to MF; reported median time to MF was 20.4 years and 20-year overall survival was 81%. High-molecular-risk comutations were associated with inferior leukemia-free and overall survival (HR 3.82; 95% CI 1.30–11.18). Such referral-center results reflect long follow-up and selection and should not be interpreted as an unselected population transformation rate.

Grinfeld et al. [8] analyzed 2,035 patients and found driver mutations in JAK2, CALR or MPL as the sole molecular abnormality in 45%. Additional driver events, genomic subgroups and clinical variables strengthened individualized prognostic modeling. Zhao et al. [14] studied 2,555 patients and demonstrated sex-related differences in molecular features and prognosis, further supporting adjustment for patient-level factors alongside driver status.

Table 4. Quantitative clinical outcome associations reported by primary studies

Study / disease	Endpoint	Published estimate	Interpretation
Rumi 2014; ET/PV [5]	Thrombosis / 1,000 person-years	PV 30; JAK2-ET 25; CALR-ET 10	CALR-ET lower observed rate
Rumi 2014; PMF [7]	Median OS by driver	CALR 17.7 y; JAK2 9.2 y; MPL 9.1 y; TN 3.2 y	Strong disease-specific gradient
Gangat 2024; ET [11]	Arterial thrombosis	JAK2 HR 1.8	Predictor among clinical variables
Gangat 2024; ET [11]	Fibrotic progression	MPL HR 3.9	Subtype-associated progression
Morath 2025; MF [13]	Venous thrombosis	JAK2 OR 2.6 (95% CI 1.01–7.16)	Association in 141-patient cohort
Mawalkar 2026; CALR-MPN [15]	Adverse HMR comutations	HR 3.82 (95% CI 1.30–11.18)	Comutations refine driver-only risk

3.7 Study-level appraisal and evidence limitations

Table 5 reports the principal methodological considerations relevant to interpretation. The table is descriptive rather than a validated final overall risk-of-bias score because a complete independent domain-by-domain assessment would require the complete protocol, screening decisions and source material for every included report.

Table 5. Study-level risk-of-bias and applicability considerations

Study	Assessment framework	Principal concern
Klampfl 2013 [3]	JBI observational / discovery	Mixed sampled disease subtypes; discovery enrichment
Rumi ET/PV 2014 [5]	JBI cohort	Historical diagnostic criteria; overlapping prior research population
Rotunno 2014 [6]	JBI cohort	Retrospective outcome comparison; treatment confounding
Rumi PMF 2014 [7]	JBI cohort	Era-specific classification and supportive therapy

Grinfeld 2018 [8]	JBI prognostic cohort	Referral setting; prognostic model transportability
Singh 2023 [9]	JBI cohort	Small disease subgroups and regional referral selection
Sande 2024 [10]	QUADAS-2 diagnostic domains	Variant-testing endpoint rather than clinical MPN diagnosis
Gangat 2024 [11]	JBI cohort	Long accrual era; temporal treatment and ascertainment differences
Tiryaki 2025 [12]	JBI cross-sectional	Small JAK2-negative selected sample
Morath 2025 [13]	JBI cohort	Limited thrombotic event numbers; observational confounding
Zhao 2026 [14]	JBI cohort	Single-center study; complex covariate and sex effects
Mawalkar 2026 [15]	JBI cohort	Tertiary-referral enrichment; long follow-up selection

DISCUSSION

4.1 Diagnostic value differs from prognostic value

The central clinical implication is the need to separate a mutation's capacity to establish clonality from its capacity to predict a future clinical event. JAK2, CALR and MPL mutations are highly informative molecular criteria, yet the presence of a mutation cannot by itself distinguish prefibrotic MF from ET, identify PV without appropriate erythrocytosis assessment, or establish a complete prognostic category. The WHO and ICC frameworks therefore remain deliberately multiparametric [1,2].

A laboratory-centered approach also distinguishes high analytical concordance from clinical sensitivity. The 982-specimen HRM validation [10] demonstrates that a combined assay can reliably detect canonical variants relative to molecular comparators. It does not establish that a negative molecular panel excludes all MPNs, particularly when the phenotype strongly suggests clonal disease or when noncanonical variants are present. Negative molecular results therefore trigger renewed clinical and morphologic assessment rather than automatic exclusion.

4.2 Driver-specific prognosis is modified by disease context

JAK2-mutated ET has repeatedly been associated with a more erythrocytic and leukocytotic phenotype and greater thrombotic risk than CALR-mutated ET [5,6,11]. Yet the degree of risk is shaped by age, previous thrombosis, hypertension and treatment. Conversely, the CALR survival advantage seen in primary MF [7] may be weakened when additional lesions accumulate. Findings from the 2026 CALR-specific series [15] emphasize that a "favorable" driver genotype is not necessarily a favorable complete genomic profile.

MPL-mutated disease deserves separate interpretation. Its small prevalence in ET and PMF leads to modest subgroup numbers, but the Mayo study observed a marked association with myelofibrotic progression [11]. This should encourage careful baseline histologic phenotyping rather than extrapolation from the much larger JAK2 subgroup. A small JAK2-unmutated pilot study [12] further illustrates how selection strategy can alter the apparent frequency of MPL and CALR variants.

4.3 Implications for pathology and molecular hematology practice

A defensible workflow starts with complete blood count, peripheral smear, clinical context and exclusion of reactive causes. JAK2 V617F analysis is an efficient initial test in suspected classical MPN. When clinically indicated, JAK2 exon 12, CALR exon 9 and MPL exon 10 testing extend sensitivity, while marrow morphology resolves many borderline ET/prefibrotic-MF classifications. Assay reports should specify gene, transcript, variant nomenclature, analytical detection threshold, VAF when validated, and any limitations relevant to low-frequency variants.

For prognosis, mutation testing should be integrated with disease-specific clinical risk models and, particularly in MF or evolving MPN, extended myeloid sequencing when it is likely to alter management. Repeated molecular profiling may inform clinical evolution, but decisions about transplantation or cytoreductive treatment must not be inferred from a single isolated genotype statistic.

4.4 Heterogeneity and generalizability

Studies varied in disease definitions, baseline selection, molecular techniques, length of follow-up, treatment, and use of alternative end points. The presence of overlapping Italian and multi-institutional cohorts makes simple addition of sample sizes misleading. Several relative effects lack uniform adjustment for competing risks and treatment, and some studies evaluated molecular assays whereas others enrolled confirmed diagnoses. These differences preclude a meaningful pooled clinical sensitivity or prognostic HR from the available aggregate information.

CONCLUSION

JAK2, CALR and MPL testing is fundamental to molecular characterization of classical Philadelphia-negative myeloproliferative neoplasms. Evidence from large observational cohorts demonstrates substantial heterogeneity in mutation prevalence across

PV, ET and myelofibrosis, while laboratory-validation studies demonstrate high variant-detection performance for appropriately designed multiplex assays. JAK2 is particularly associated with thrombotic phenotypes in ET and myelofibrosis; MPL can identify an ET subgroup at risk of fibrotic progression; and CALR-associated survival patterns depend on disease phase and accompanying molecular abnormalities. Combining driver genotype with marrow pathology, hematologic parameters, clinical risk factors and additional somatic mutations provides a more clinically meaningful framework than driver status alone.

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