



CURRENT AND EMERGING THERAPIES IN MPNS



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Philadelphia-negative myeloproliferative neoplasms (MPNs), including essential thrombocythemia (ET), polycythemia vera (PV), and primary myelofibrosis (PMF), are hematologic malignancies characterized by the abnormal proliferation of blood cells in the bone marrow. MPNs cause significant morbidity in the form of burdensome symptoms, potentially fatal cardiovascular complications, progression to more aggressive disease, and over time, can evolve to acute leukemia.

Although MPNs have diverse characteristics and outcomes, they are driven by shared mutations in *JAK2*, *CALR*, and *MPL* genes, mutations that originate in hematopoietic stem cells (HSC). This is important because HSCs have the ability to regenerate all of the different cell types, including those which are mutated. Developing curative therapies in MPNs would require drugs that target the MPN cell of origin, in this case the mutant HSCs.

Several drugs with various mechanisms have shown clinical efficacy (desired results) in the management of MPN symptoms, i.e., improving enlarged spleen (splenomegaly) and high red or white blood cell counts. Some have additionally shown deep molecular responses, which although controversial, may be considered surrogates for disease-modifying activity.

Currently, however, there is no definitive evidence that any one or combination of these clinical endpoints can reliably predict survival. While

these are all important benefits of treatment, we continue in pursuit of treatments capable of reducing disease complications, improving survival, and ideally curing MPNs. This synopsis provides an overview of the current and emerging MPN therapies and their clinical activity.

Conventional therapies such as aspirin, phlebotomy, cytoreductive agents, such as hydroxyurea, and interferon alpha (IFN) have been the mainstay of MPN treatment for decades. Significant advancements have been made in recent years with the development of novel targeted therapies and the recognition of IFN as an important disease-modifying agent.

Although IFN was used for decades in the treatment of PV, ET, and early primary and secondary myelofibrosis (MF), phase 3 randomized trials were only recently conducted. In PV, ropeginterferon alfa-2b treatment was associated with higher clinical, hematological, and molecular responses over hydroxyurea in “high-risk” patients and over phlebotomy alone in “low-risk” patients. The PROUD-PV study led to the first FDA approval of an IFN for PV in 2021. Although survival was not compared in these randomized studies, retrospective analyses of PROUD-PV and a large single center cohort identified event-free, myelofibrosis-free, and overall survival (OS) benefits with IFN.

The JAK inhibitors were the first targeted agents developed soon after the JAK2V617F driver mutation was identified (2005), now understood to be present in more than 60% of all MPNs. Ruxolitinib, an oral JAK1/JAK2 inhibitor, was the first approved in the U.S. for the treatment of intermediate- and high-risk MF after demonstrating significant improvement in symptom burden and spleen volume reduction (SVR), compared to placebo or best available therapy (BAT) in COMFORT-I and II trials, respectively. Analysis of long-term trial data identified an overall survival benefit. In PV, ruxolitinib is a second line treatment. A retrospective study identified its long-term

CONTINUED ON PAGE 3

MISSION

MPN Research Foundation stimulates original research in pursuit of new treatments — and eventually a cure — for myeloproliferative neoplasms (MPNs).

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The MPNRF Scientific Advisory Board (SAB) plays a key role in the organization's near-term and longer-term priorities and practices, ensuring that our global research strategy and annual research funding programs are well aligned with MPNRF's mission - to stimulate critical research and ultimately a cure for MPNs.

The SAB is a valued group of MPN thought leaders who volunteer their time and expertise to provide a broad range of medical, scientific, and strategic counsel to MPRNF staff and board of directors, with the ultimate goal of improving outcomes for patients.

Specific guidance is also offered on issues related to our role in the clinical trial process; such as engagement with regulatory agencies, the development of clinical trial endpoints, and the development of infrastructure to support ongoing research, i.e., optimizing patient registry for disease progression.

Individually and collectively, their valued knowledge and vision impacts MPNRF's strategic planning and execution of its objectives in multiple ways. For example, the 2022 Thrive Initiative, which funded an additional \$1.8 million through 11 MPN research projects, was born out of SAB discussions. Other ongoing contributions include the following:

- + Distinguishing areas of highest priority in current MPN clinical and basic research.
- + Recommending the most effective and highest impact mechanisms for research funding.
- + Identifying opportunities on the horizon to catalyze highly promising directions in MPN research.
- + Helping continuously improve MPNRF's peer review and funding selection process.
- + Monitoring and prioritizing current and future research and therapeutic developments to identify therapies being developed for other diseases, which may be relevant to the MPNs, i.e., immunotherapies and targeted therapies.
- + Identifying new research gaps and partnering opportunities to better address patient needs with regard to established and emerging MPN therapies.

MPNRF is grateful for the commitment and contributions of each of the SAB members, listed in the column to the left. ■

potential for achieving deep, even complete molecular responses coupled with a myelofibrosis-free survival benefit, but further data on its long-term safety and OS benefit in PV are required.

More recently, JAK inhibitors fedratinib and pacritinib were approved for MF by the U.S. Food and Drug Administration (FDA), based on superior SVR to placebo and BAT in the JAKARTA and PERSIST-2 trials, respectively. Because pacritinib is a selective JAK2 and IRAK1 inhibitor with potentially lower likelihood for treatment-induced thrombocytopenia (when platelet counts are too low), the PERSIST-2 trial compared pacritinib to BAT (including ruxolitinib) in thrombocytopenic MF patients.

These positive results led to approval of pacritinib as a first-line treatment of thrombocytopenic MF patients. Retrospective analysis of PERSIST-2 data indicated additional benefit of pacritinib in treatment of anemia, with the hypothesized mechanism being inhibition of the ACVR1 pathway. Lastly, momelotinib is an emerging JAK and ACVR1 inhibitor with positive clinical response, including anemia benefit, shown in the SIMPLIFY-1 and MOMENTUM trials. It is currently in the FDA approval review process.

Emerging therapies in MPNs, primarily MF, now include bromodomain and extra-terminal (BET) inhibitors that target the epigenetic regulators of gene expression, thus changing the level of expression of those target genes. These emerging therapies include CPI-0610, ABBV-744, and INCB054329, in different stages of development for MF and ET. CPI-0610 (pelabresib) is currently in a phase 3 randomized, double-blind, placebo-controlled trial in combination with ruxolitinib as upfront treatment for MF (MANIFEST-2) (NLM, NCT04603495). The phase 2 results showed significant spleen volume reduction, symptom response, anemia benefit, and fibrosis reversion as monotherapy or in combination with ruxolitinib, in patients who either had never taken a JAK inhibitor, it was not effective, or it stopped working after a time (relapsed/refractory to ruxolitinib).

Other agents in phase 3 studies for myelofibrosis include navitoclax (TRANSFORM-2) (NLM, NCT04468984), imetelstat (IMpactMF) (NLM, NCT04576156), navtemadlin (BOREAS) (NLM, NCT03662126), and luspaterecept (INDEPENDENCE) (NLM, NCT04717414).

Navitoclax is a novel BCL-2 and BCL-X inhibitor that also showed significant clinical responses, molecular responses, and fibrosis reversion when added to ruxolitinib in the phase 2 study for patients with relapsed or refractory disease. In a post-hoc analysis, patients who achieved both molecular response and fibrosis reversion on this study had a higher overall survival.



Imetelstat inhibits telomerase, an enzyme crucial for maintaining telomere length in cancer cells. As cancerous cells divide, telomeres, a region of repetitive DNA at the ends of chromosomes, become shorter. Telomerase is increased to prevent this shortening and its associated cancer cell survival. The phase 2 study design was unique in randomizing patients to either of two dose levels of imetelstat and incorporating overall survival as a secondary endpoint. Given the encouraging survival results, the phase 3 was designed with overall survival being the primary endpoint.

Navtemadlin is an inhibitor of the MDM2, an oncoprotein (associated with the growth of cancer cells), and negative regulator of p53, a protein that acts as a tumor suppressor. The study was advanced to phase 3 after preliminary safety and efficacy was established in phase 1b/2 for treatment of p53 wild-type MF, relapsed or refractory to ruxolitinib.

Both imetelstat and navtemadlin were granted fast-track designation by the FDA. Finally, luspaterecept is a TGF superfamily ligand trap – which effectively reduces TGF signaling. It is already FDA approved for treatment of patients with transfusion dependent anemia from myelodysplastic syndromes (MDS) and beta thalassemia, an inherited blood disorder characterized by low or dysfunctional hemoglobin. The phase 3 randomized, placebo-controlled trial for MF, combined with a JAK inhibitor, is open to enrollment after results of phase 2 identified safety and notable improvements in anemia and transfusion burden.

Progress made in MF treatment provides an optimistic outlook for ET and PV patients. Beyond conventional cytoreductive treatment for ET, emerging agents in clinical trial include ropeginterferon alfa-2b, pelabresib, and bomedemstat (a lysine-specific demethylase-1 [LSD1] inhibitor). In PV, there is accumulating interest in treatment targeting iron metabolism to normalize erythropoiesis (production of red blood cells) and correct iron deficiency.

Also noteworthy are the profound hematocrit responses, phlebotomy independence rates, and iron normalization in patients treated with rusfertide, which mimics hepcidin, an iron-regulating hormone. Currently in phase 2 clinical trial, the ongoing phase 3 (VERIFY) (NLM, NCT05210790) will determine safety and efficacy of this agent as monotherapy, to replace the need for phlebotomy, or as an add-on to cytoreductive therapy in refractory patients.

While several drugs are in the clinical trial pipeline, there is also a multitude of promising preclinical research examining novel MPN treatments. Immunotherapy approaches, including vaccines and monoclonal antibodies, are high on this list. For CALR mutant MPNs, a clinical trial is highly anticipated for the recently discovered anti-CALR antibody that may selectively target mutant cells including HSCs.

In summary, the treatment landscape for MPNs is rapidly evolving, with a shift towards targeted drugs and combinations that provide improved efficacy and tolerability to current and conventional therapy, as well as disease-modifying activity to improve the overall survival of MPN patients. With several candidate drugs in the investigational pipeline, the outlook for MPN patients is becoming increasingly optimistic, with the potential for improved outcomes and enhanced quality of life.

This article appears with a full list of references on the MPNRF website. ■

2021-2023 MPN CHALLENGE AWARDS PROJECT UPDATES

MPN Challenge is our open request for research applications that allows us to fund the most pressing MPN science. Focus areas change each round as we learn more about unmet needs in MPN research. Previously these have included CRISPR, selective JAK2 inhibition, inflammation, interferon, and more. Researchers are awarded two-year grants and are required to make regular progress reports. Below are one-year summaries of their 2021-2023 progress.



Josef Prchal, MD | Professor, Division of Hematology and Hematologic Malignancies | University of Utah & Huntsman Cancer Center, Salt Lake City, UT | **Role of Iron Deficiency in Thromboses of Polycythemia Vera**

Blood clots (thrombosis) in arteries and veins are major causes of morbidity and mortality

in polycythemia vera (PV). However, the molecular mechanism of thrombosis in PV is unknown. The process of phlebotomy, which removes excess red blood cells from the blood, is generally performed to maintain hematocrit at levels below 45%, with the goal of preventing thrombosis.

In Chuvash erythrocytosis, a rare congenital polycythemia, the incidence of thrombosis is higher than in the more common PV, with increased hypoxia (lack of oxygen) inducible transcription factors HIF-1 and HIF-2. And phlebotomy did not prevent thrombosis but instead facilitated it. Repeated phlebotomies induced iron deficiency and further increased the level of HIF-1 and HIF-2. It is hypothesized that increased signaling in granulocytes (a type of white blood cell) and platelets, perhaps with an additional contribution of inflammation, plays a central role in the development of thrombosis in PV.

If this hypothesis is proven, it would radically change how we treat PV and would preclude common use of phlebotomies, except in unusual situations. This group is investigating how increase of hypoxia inducible factors, or HIFs, due to phlebotomy-induced iron deficiency, increases HIF-regulated expression of prothrombotic genes, leading to increased risk of thrombosis. Dr. Prchal reports that so far, the generated data supports this hypothesis.



Anandi Krishnan, PhD | Instructor, Department of Pathology | Stanford University, Stanford, CA | **Platelet, Blood and Plasma Signatures of MPN Subtype-Specific Risk**

This project sets out to distinguish associations between MPN molecular signature and disease phenotype (characteristics) with implications toward early detection, novel therapies, and improved clinical interpretation of disease natural history, while serving as a proxy for disease severity.

The objective is to translate and validate a clinically and biologically relevant set of RNA-sequencing derived molecular markers in platelets from banked MPN whole blood and matched plasma samples.

Personalized medicine in MPNs requires further investigation of genetic heterogeneity (diversity) among patients. While current clinical DNA sequence and targeted panel data successfully identify novel genetic variants, there is an unmet need for clinically relevant signatures that bridge the knowledge gap between the patient mutational data and their evolving clinical traits.

Recent work from this group shows that profiling the MPN platelet transcriptome (RNA sequencing information) in a large cohort of essential thrombocythemia (ET), polycythemia vera (PV), and myelofibrosis (MF) patients – contrasted with healthy donors – identified a robust core set of highly sensitive molecular signatures that were both shared and progressive across the MPN spectrum, and more importantly, associated with disease clinical severity.

The primary impact of this project is the discovery of enkurin (ENKUR) – a calcium signaling gene – as a potential new peripheral biomarker, therapeutic strategy, and a possible clue to the development of disease (pathogenesis) in MPNs. The team has developed ex vivo and in vitro systems to extend their prior data from MPN patient platelet RNA sequencing, discovering select proteostasis-associated markers at RNA and/or protein levels in platelets, parent megakaryocytes, and whole blood specimens. They plan to continue to build their efforts toward a better understanding of enkurin and associated molecular pathways and aim to develop assays (a type of measurement procedure) for clinical laboratory evaluation of MPN patient disease progression.



Sara Buhrlage, PhD | Assistant Professor, Biological Chemistry and Molecular Pharmacology | Dana-Farber Cancer Institute, Boston, MA | **JOSD1 as a Novel Targeted Therapy for JAK2V617F Dependent MPNs**

The goal of this project is to develop JOSD1 inhibitors, characterized by improved potency and selectivity toward JOSD1 as a therapeutic target for treatment of MPNs harboring mutated JAK2 genes. These studies provide a novel strategy to obtain a real therapeutic window in targeting mutated JAK2 in MPN, potentially filling a critically unmet clinical need.

Small molecule inhibitors of the activity of mutated JAK2 provide clinical benefit. However, because they also impair the function of the non-mutated form of JAK2, which normal cells require, clinical usefulness is limited. This project proposes a strategy to target mutated JAK2 for degradation, using the cell's intracellular machinery to do so, while sparing non-mutated JAK2.

The work of this multidisciplinary team of chemists, biologists, and clinicians shows that inhibitors of the deubiquitinase (DUB) JOSD1 selectively inhibit the growth of mutated JAK2-driven malignant cells without impacting the growth of cells expressing non-mutated JAK2.

To date, the work is focused on optimization of lead inhibitors. This is to enable translational studies, which apply these basic science discoveries to human health and clinical practice. The team discovered that JOSD1 inhibitor exhibited additional inhibition of several DUBs, not detected using a previous method for selectivity profiling. They identified chemical modification that achieves derivatives with excellent selectivity for JOSD1. And they identified a second chemically-distinct inhibitor series for JOSD1.



Stephen Oh, MD, PhD | Associate Professor, Division of Hematology | Washington University School of Medicine, St. Louis, MO | **Functional Interrogation of an Aberrant DUSP6-RSK1 Signaling Axis Driving MPN Pathogenesis**

This project expects to address the role of DUSP6 in MPN disease development and progression, and to identify vulnerabilities that can potentially be used for therapeutic benefit. The group has identified abnormally increased expression of DUSP6 – a phosphatase enzyme that regulates the ERK signaling pathway – in CD34+ cells (blood forming stem cells) of MPN patients. They identified increased DUSP6 expression in JAK2 inhibitor-persistent cells. And in preliminary studies, they found that genetic and pharmacologic inhibition of DUSP6 inhibits MPN cell proliferation in conjunction with suppression of multiple downstream signaling effectors (i.e., pERK, pRSK1, pS6). Effectors are proteins that evoke cellular responses to signaling molecules.

In preliminary in vivo studies, inhibition of DUSP6 improved MPN disease features, reduced disease burden, and extended survival. Based on these findings, the researchers hypothesize that: (1) DUSP6 drives MPN disease progression via aberrant (abnormal) dysregulation of an ERK-RSK1-S6

signaling axis; (2) targeted inhibition of DUSP6 may represent a viable therapeutic strategy in MPNs.

The team is now addressing these hypotheses by employing MPN patient samples and animal models in conjunction with genetic and pharmacological manipulation.



Bridget Marcellino, MD, PhD | Assistant Professor, Division of Hematology & Oncology | Icahn School of Medicine at Mount Sinai, New York, NY | **Characterization of Immune Landscape and Determinants of Immune Dysregulation in MPNs**

This group aims to explore the immune landscape of MPNs and develop strategies to harness the immune system to treat MPN patients. The outcome of this project will provide a multipronged approach to both understand the immune dysfunction in MPNs and develop novel immunotherapeutic approaches.

Regulation of disease-specific immune networks in MPNs and how they might drive MPN development (pathogenesis) and progression is currently unknown. This research team will first study the immune landscape of the MPNs by immunophenotyping and transcriptionally profiling the immune cells of MPN patients. Additionally, they will determine the potential of checkpoint receptor inhibitor therapy to activate T and “natural killer” cell responses against MPN cells. Natural killer cells are white blood cells associated with enzymes that can kill tumor cells. Finally, they will test the efficacy of donor NK cells against MPN cells using in vitro and xenograft models.

CITE-sequencing (a unique, more efficient method of cellular indexing) is being used on a newly acquired cohort of 14 MPN patients to get more information from a single cell. This allows for maximizing the data acquired from limited available samples. ■



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ET PATIENT EMBRACES HEALTH AND FITNESS



"When they diagnosed me with ET, I couldn't even say what I had: essential thrombocythemia. I had no idea what an MPN was at that time. I was just in shock," says Alex, a 51-year-old Australian fitness and now nutrition buff who has had ET since 2017. "It was a bit overwhelming."

Alex recently told MPNRF that he clearly remembers his hematologist handing him a booklet when he first spoke to him of his diagnosis, which

came shortly after routine bloodwork led to a red flag for high platelets. He immediately focused on a leukemia organization logo at the top.

"I thought, wow! And I just looked at him... is this cancer? And his words: yeah, it is, but it's the best kind of cancer." Alex thought about if there is such a thing as a 'good' cancer. Then he asked if there was anything he could do about it. He wasn't prescribed any medications, and today is still only on aspirin for anticoagulation (blood thinners). He was simply told to "try and be as healthy as you can."

He has taken that advice to heart every day since.

Alex describes himself as "not really impacted by this disease." As a soccer (football) player, he had noticed his shins were always bruised, and that they didn't heal as well as his teammates. "My fingers and toes would have that purple discoloration," and he had minor episodes of gout. Today, he also suffers from brain fog, tiredness, and affected eyesight.

Coupling the research he did about ET with two intense bouts of gout, he decided to actively adjust his lifestyle. "I really started to work on the health and fitness thing because I could," he says, "which in the long run, it's helped my health."

Alex reports that his platelet counts have been at a reduced level for a few years now. "I got involved in this routine for the two or three months that we were locked down during the pandemic. And at the back end of that, just as Covid finished, I went to do my blood tests..."

"What's happened?" That was his doctor's opening comment when he called with the lab results. "And I'm thinking the worst. He said, 'your platelet levels have dropped, like 150,000 in three months.' The only thing I could think of was I was less stressed... And I was substituting

some of the stuff I was eating with some of the stuff my partner was eating, primarily around vegetables, raw vegetables."

Alex is confident that changing his lifestyle, including daily workouts and eating differently, more in line with the Mediterranean diet, has kept him healthy and able to enjoy living an active life. His doctor has embraced Alex's progress. His previous three-month interval medical visits are for now just twice a year.

With a large following as a middle-aged exercise and healthy eating role model, Alex shares his tips with others who are newly diagnosed or in early treatment for ET, or any other MPN.

- #1** Make a follow-up appointment soon after your initial diagnosis to throw your 1,000 questions at your physician – all of the questions you thought of after you went home and researched on the internet.
- #2** Explore the idea that clinical trials could be your best option for new drug therapies. Though Alex was never told about clinical trials, he knows about them now and recognizes that a diverse pool of participants is needed to lead to better treatments. He is currently looking at any appropriate studies he might participate in.
- #3** There is always going to be uncertainty in life, and you can't control that.

"I'm not really worried about the uncertainty anymore," says Alex. "I'll deal with it when it happens. So I can only do the best, do what's working for me now and get on with life. That's my view."

For more information about nutrition and MPNs, specifically the Mediterranean diet, you can go to www.youtube.com and search "Angela Fleischman: Nutrition and MPNs." The related nutritional research was funded by MPNRF. Results of her Nutrient Trial will be published soon. ■



CLINICAL TRIALS UPDATE

CLINICALTRIALS.GOV GETS USER FRIENDLY MAKEOVER

Have you visited clinicaltrials.gov lately? This valuable global resource maintained by the National Institutes of Health (NIH) recently received an update. Now more than ever it is a great place for MPN patients, caregivers, and clinicians to regularly visit for new and updated clinical trial information.

With the number of clinical trials increasing ET, PV, and MF patients, it's the best resource about individual trials, including:

- Inclusion criteria (who qualifies for the trial).
- Exclusion criteria (events, treatments, or other factors that may exclude patient participation).
- Endpoints (what outcomes are being measured in the trial).
- Trial sites (what hospitals and cancer centers are participating).

Visit the new clinicaltrials.gov today to see for yourself, and visit MPNRF's clinical trial finder by Leal Health to enter your information and receive a list of trials that may be right for you!

TRIAL HIGHLIGHTS

With the help of patients who choose to participate, clinical trials determine whether a therapy is safe and effective in treating a particular disease. Trials are designed to test a medication or other medical treatment against a placebo (inactive look-a-like), other medication, or the standard medical treatment for a patient's condition. Data collected in clinical trials are necessary for regulatory agencies to review prior to approving any new therapy or a new use of a therapy previously approved to treat a different disease. The decision to participate in a clinical trial is one to consider carefully and discuss with your physician.

Abemaciclib plus Ruxolitinib (Phase 1)

Sponsor: Memorial Sloan Kettering Cancer Center Investigator
Initiated trial

Diagnosis: Myelofibrosis

Notes: For intermediate- to high-risk MF patients on a stable dose of ruxolitinib that is not producing an adequate response. Abemaciclib (previously approved for breast cancer treatment) is given in combination with

ruxolitinib. Both block specific proteins that play a role in cancer growth.

More Info: <https://clinicaltrials.gov/ct2/show/NCT05714072>

NS-018 Ilginatinib (Phase 2)

Sponsor: NS Pharma

Diagnosis: Myelofibrosis

Notes: For MF patients with severe thrombocytopenia (platelet count <50,000/ μ L) ilginatinib (NS-018) is a highly selective and potent inhibitor of Jak2.

More Info: <https://clinicaltrials.gov/study/NCT04854096>

VAC85135 (Phase 1)

Sponsor: Janssen

Diagnosis: Essential Thrombocythemia, Polycythemia Vera, Myelofibrosis

Notes: Novel vaccine regimen directed against MPN tumor cells administered with ipilimumab which helps activate the immune system to kill cancer cells. Ipilimumab is an approved therapy used to treat many different types of cancer.

More Info: <https://classic.clinicaltrials.gov/ct2/show/NCT05444530>

Ruxolitinib + CK0804 (Phase 1b)

Sponsor: Cellenkos, Inc.

Diagnosis: Myelofibrosis

Notes: Study of adding T-Regulatory cells (CK0804) to ruxolitinib for patients who did not have optimal response to ruxolitinib alone. Being derived from umbilical cord blood, T-Regulatory cells are suppressor cells that resolve inflammation through multiple direct and indirect interactions.

More Info: <https://clinicaltrials.gov/study/NCT05423691>

EXCEED ET (Phase 2b)

Sponsor: PharmaEssentia

Diagnosis: Essential Thrombocythemia

Notes: Study to determine the effectiveness and safety of ropeginterferon alfa-2b-njft in treating patients who have not received treatment for ET or who discontinued treatment when it stopped working or due to unbearable side effects. Note that this interferon formulation was approved by the FDA for use in PV in November 2021.

More Info: <https://www.exceedet.com/>



PERSONALIZED CLINICAL TRIAL FINDER

Launched in 2022, MPN Research Foundation clinical trial finder – powered by Leal Health – easily and quickly matches an MPN patient to trials they may qualify for based on the diagnosis and other information that they enter. After filling out a short profile, the patient may receive links to trial sites and a list that they can share with a doctor who manages their MPN. Leal Health provides live support in multiple languages. Get started today at www.mpnresearchfoundation.org/mpn-clinical-trials.



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SEPTEMBER IS BLOOD CANCER AWARENESS MONTH

Watch your email and MPNRF social media for our Blood Cancer Awareness Month campaign highlighting personal impacts and stories from those living with ET, PV, and MF and their care partners.

We will be sharing MPN facts, the latest thoughts on research and clinical advancements, and more. There will also be an opportunity to support MPNRF's investments in MPN research with a gift that is matched by some generous industry sponsors.