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FOR A BETTER TODAY.
INVESTING IN RESEARCH
FOR A BETTER TOMORROW.

REPORT: 2018 ASH MEETING

John Crispino, PhD and Rick Winneker, PhD

Once again, the annual American Society of Hematology (ASH) meeting was full of interesting science and clinical updates. This year brought an emphasis on advanced clinical studies of interferon, combination studies of investigational agents with ruxolitinib, and new potential therapeutic targets for the MPNs.

INTERFERON

There were three notable presentations regarding interferon testing in the MPNs, including two on interferon alpha-2 and one on ropeginterferon alfa-2b. Dr. Mascarenhas presented the results of the MPN-RC Phase 3 randomized trial of pegylated interferon alfa 2a (PEG) versus hydroxyurea (HU) for the treatment of high-risk PV and ET. The study revealed that the complete remission rates were similar for PEG and HU at one and two years, but that PEG was associated with a higher rate of grade 3/4 toxicity. In patients with significant baseline MPN symptom burden, both HU and PEG therapy had a beneficial impact on the symptom burden with different patterns of efficacy and toxicity reported between HU and PEG.

Results of the three-year Daliah Phase 3 trial of PEG in PV were presented by Dr. Knudsen. The study showed that the clinicohematologic response rate was higher, although not significantly, with interferon compared to HU. Moreover, the median reduction in *JAK2V617F* allele burden was greater with interferon therapy.

Finally, Dr. Gisslinger presented the three-year clinical data from the PROUD and CONTI-PV studies of Ropgeinterferon, reporting on efficacy, safety and molecular response. This study revealed that Ropgeinterferon provided durable hematologic responses and symptom control, was well tolerated, and had a striking effect on the mutant allele burden, with 66% of patients achieving molecular remission for mutant *JAK2* as well as a reduction in other disease associated genes such as *TET2*.

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CARRYING ON THE LEGACY OF ROBERT ROSEN

Molly Rosen Guy



Robert Rosen and his family.

When my dad was diagnosed with blood cancer in 1997, he was an active, nine-to-five business guy. A father of three and third generation Chicagoan, Robert Rosen had an MBA, a pilot's license and a house on Lake Michigan. He had recently played basketball in the Senior Olympics, but he was constantly fatigued. His fingertips tingled often.

The summer after my sophomore year of college, we were walking downtown when he first told me: *Something is not right in my body*. A few months later, he gathered the family together to say he'd been diagnosed with polycythemia vera, a gene mutation that affected his red blood cell production. I remember seeing my mom's mascara smeared all over her pillowcase when I went into the bedroom the next morning. She'd been crying all night.

(CONTINUED ON PAGE 5)

MISSION

The primary mission of the MPN Research Foundation is to stimulate original research in pursuit of new treatments — and eventually a cure — for myeloproliferative neoplasms (MPNs). In addition, the MPN Research Foundation promotes collaboration in the scientific community to accelerate research and serves as a powerful patient advocacy group for patients and their families.

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MPN RESEARCH FOUNDATION UPDATE

A periodic newsletter published by the MPN Research Foundation to provide members of the MPN community with information on current research and the Foundation's activities.

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MPN COMMUNITY TO MEET WITH FDA ON PATIENT-LED DRUG DEVELOPMENT

Michelle Woehrle, Executive Director



Michelle Woehrle

This fall, the MPN community will have the chance to tell Food and Drug Administration (FDA) regulators, academic researchers, and industry players exactly which problems they want solved regarding their PV, ET and MF.

For over a year, the FDA has been conducting internal meetings on Patient Focused Drug Development (PFDD). In 2018, these meetings were opened to provide patient advocates an opportunity to identify and organize external patient-focused collaborations to generate public input on other disease areas, using the process established by the FDA-led PFDD meetings as a model. In collaboration with MPN Advocacy and Education International and researchers Robyn Scherber and Ruben Mesa, the MPN Research Foundation has been working with FDA staff

to develop an agenda that will create opportunities for patients to be heard.

To attend the meeting in person, please reach out to me at mwoehrle@mpnrf.org or 773-453-9917 to discuss. We hope to provide a webcast for those who cannot attend. As the event gets closer, we will share more information. After the meeting, we'll share findings with the MPN community as a whole – especially with patients, but also with the industry, academic researchers, and regulators who are also part of our world.

Read more about the FDA's Patient Focused Drug Development meetings online at:

<https://www.fda.gov/ForIndustry/UserFees/PrescriptionDrugUserFee/ucm453856.htm>. ■

A LOOK AT OUR 2019 PROJECT PRIORITIES

Continuing our tradition of issuing a request for proposals for the most exciting MPN science, the MPN Research Foundation is looking to fund **approximately 5 new projects for \$1 million over 2 years**. The focus areas we are soliciting ideas for are those which we believe to have the most potential impact for people with MPN, and which are not already being explored. In 2019 this includes:

- Immunotherapeutic approaches for MPNs
- Targeting the driver mutations associated with MPN (primarily JAK2, CALR, and MPL)
- New pathways or targets other than mentioned above that impact the disease
- Repurposing existing FDA-approved drugs for use in MPN
- Development of biomarkers of disease progression
- Stem cell transplantation
- Quality of life research

As in years past, we anticipate receiving more proposals than we are able to fund. We work with a panel of MPN scientists who score the proposals, and the final funding decision is made by our board of directors (comprised of patients and their families). This patient-researcher team is capable and motivated to select projects that will lead the way in improving the lives of people with MPN. We are proud that the Leukemia & Lymphoma Society has remained a committed partner for this program.

Expect an announcement about who will be funded in July 2019. You can review the complete list of research MPNRF has funded on our website:

<http://www.mpnresearchfoundation.org/Research-Funded>. ■

NOVEL SINGLE AGENT CLINICAL STUDIES

PRM-151 is a natural human protein that has been shown to reverse fibrosis in pre-clinical studies. Dr. Verstovsek provided an update on the status of 18 patients who had been treated in a Phase 1/2 study for up to 140 weeks. Overall, PRM-151 was well tolerated and the treatment was associated with a reduction in spleen volume and symptom burden in a subset of patients, but most notably a reduction in the fibrosis grade in the majority of patients on study.

The results of the Phase 1 study of the aurora kinase A inhibitor alisertib, presented by Dr. Gangat, were similar in that alisertib was well tolerated, resulted in a reduction in spleen size and symptom burden in a subset of patients, and notably reduced the degree of fibrosis in 5 of 7 patients for whom sequential marrows were available. These two agents thus represent potential anti-fibrotic agents.

Another novel agent reported was LCL161, which is an antagonist of inhibitor of apoptosis proteins that results in increased cell death. Results from a Phase 2 study, presented by Dr. Pemmaraju, demonstrated an objective response rate of 30% which included a number of anemia and symptom responses.

Finally, Dr. Pemmaraju also presented a poster featuring results from an ongoing Phase 1/2 clinical trial of Tagraxofusp (SL-401) in patients with intermediate or high risk relapsed/refractory MF. Tagraxofusp is a novel targeted therapy directed to the interleukin-3 receptor- α (CD123), a target expressed in some myeloid malignancies including MF. While this study is still enrolling patients, Tagraxofusp monotherapy has demonstrated improvements in splenomegaly, with a manageable safety profile in this patient population.

COMBINATION THERAPIES

Searching for a drug that will increase the effectiveness of JAK inhibition and potentially cure patients continues to be a major effort. Notable combinations included thalidomide, a PI3K pathway inhibitor, and 5-azacytidine. There was also a report of the combination of ruxolitinib with pegylated interferon alpha 2a, referred to as the Ruxopeg study. In each case, there appeared to be promising preliminary efficacy signals, although further study will be required to determine just how much each of the new therapies enhances JAK inhibition.

NOVEL PRE-CLINICAL TARGETS AND NEW BASIC BIOLOGY INSIGHTS

One of the most exciting presentations came from the work of Dr. Constantinescu and colleagues, who wondered whether mutant CALR protein was secreted from cells, and if so, whether it had any tumor inducing properties. This study revealed that indeed mutant CALR protein is released from cells, where it is then free to induce JAK/STAT signaling in nearby cells. The cells that already harbor a CALR mutation were more sensitive to the secreted CALR than normal cells. This study shows that mutant CALR can act as a "rogue cytokine" to potentiate the growth of the tumor.

An abstract presented by Dr. Maslah described the rationale for and preliminary results of a pre-clinical study of the combination of interferon

alpha and arsenic. Although interferon has significant anti-tumor activity and the ability to reduce the JAK2V617F mutant allele burden, the addition of arsenic significantly enhanced this effect and also led to a profound loss of tumor initiating cells. The results suggest that combining these approved agents is worthy of further investigation.

Other pre-clinical abstracts identified new potential therapeutic modalities including inhibitors of PIM kinase and a protein named PRMT5, which modifies other proteins in cells to regulate their activity. Inhibition of these two proteins effectively reduced JAK2V617F MPN in animal models and support further investigation of these pathways in MPN patients. Additional preclinical studies presented support the potential clinical utility of inhibitors of Axl kinase (with BGB324), SHP2 (with SHP-099) and the NFkB pathway (with pevonedistat) in combination with JAK inhibition.

Lastly, Dr. Kralovics presented his work on an MPN tumor cell neoantigen discovery platform as a means to ultimately develop personalized vaccines and/or adoptive cell-based therapies. He was able to identify a rich source of neoantigens in an estimated 60% of patients tested, in particular those with mutations in *CALR*, *MPL* and *SF3B1*.

DISEASE PROGNOSIS AND PROGRESSION

There was no shortage of presentations addressing MPN disease prognosis and the factors that contribute to disease progression. It was great to see so many major academic centers, both US and abroad, contributing to this area of research. Prognostic scoring systems for the MPNs continue to evolve with the introduction of additional patient, hematologic, karyotypic and gene mutational analyses to further enhance predictions of disease progression and overall survival.

One example to highlight is the mutation enhanced international prognostic scoring system for ET and PV developed by Dr. Tefferi (Mayo Clinic) and Dr. Vannucchi (University of Florence). By looking at the factors that led to fibrotic and leukemic progression and overall survival in a large cohort of patients, a simplified system of four risk factors has been created to predict low, intermediate and high-risk groups of patients. These studies and others have suggested that spliceosome mutations enhance survival prediction in ET and PV and identify those at risk for fibrotic progression, and that TP53 mutations may predict leukemic transformation in ET. Mutations in *AXL1*, *EZH2*, *IDH1*, *IDH2*, *RAS* pathway gene and elevated IL-8 serum levels have been associated with disease progression and adverse outcomes in patients with MPNs.

The majority of studies in this area of research are retrospective, so additional large patient prospective studies are still needed to truly understand the specific causative role of these mutations in MPN progression. The time may soon be approaching when genomic-based classification systems predicting differences in disease outcomes may transcend the original MPN diagnosis. This information will allow for better patient selection for more aggressive therapies, those more likely to respond to traditional therapies, e.g. JAK inhibitors and interferon, and potentially open the pathway for early disease treatment and prevention strategies. ■

MPN CHALLENGE GRANTS: CELEBRATING OUR FIRST YEAR OF PROGRESS

Rick Winneker, PhD

In the fall of 2017, the MPN Research Foundation, with support from The Leukemia & Lymphoma Society, announced five new MPN Challenge Grant awardees. These are two-year awards of \$100,000 per year. Highlights for the first year of research progress are below.



INFLAMMATION AS A DRIVER OF CLONAL EXPANSION IN MYELOPROLIFERATIVE NEOPLASM

Angela G. Fleischman, MD, PhD, University of California, Irvine

The goal is to define how chronic inflammation drives the emergence of hematopoietic stem cell (HSC) clones containing disease driver mutations (i.e. JAK2V617F) while exhausting the wild type stem cells. Inflammatory stimuli do not induce replicative stress in JAK2V617F HSCs as it does in wild type HSCs. This may protect JAK2V617F HSCs from exhaustion and provide a selective advantage over wild type HSCs. The anti-oxidant N-acetylcysteine can protect wild type HSCs from inflammation and extend the lifespan of JAK2V617F mutant mice. The investigators have opened a nutrition interventional trial to study how a low inflammatory diet may impact inflammation, symptoms, and blood counts in MPN patients.



INHIBITION OF DEUBIQUITINATING ENZYMES (DUBS) AS A NOVEL TARGETED THERAPY FOR JAK2-DEPENDENT MYELOID MALIGNANCIES

James D. Griffin, MD, Martin Sattler, PhD, Sara J. Buhrlage, PhD & Ellen L. Weisberg, PhD, Dana-Farber Cancer Institute

The goal is to cause degradation of the mutant JAK2 protein by identifying drugs that inhibit the DUB that normally stabilizes it. Two independent chemical series have been identified that degrade mutant JAK2 and cause increased killing of mutant JAK2 expressing cells compared to wild-type JAK2. The current focus is to identify the exact DUB involved and chemically optimize the inhibitors for the candidate DUBs.



FUNCTIONAL AND MOLECULAR DISSECTION OF THE FIBROTIC TRANSFORMATION AND CLONAL SELECTION IN MYELOPROLIFERATIVE NEOPLASMS

Rebekka K. Schneider, MD & Rafael Kramann, MD, Erasmus University Medical Center, Rotterdam, The Netherlands and RWTH Aachen University, Aachen, Germany

This project is focused on how an altered bone marrow (BM) microenvironment and inflammation play a key role in fibrotic progression. Initial results show that the transcription factor Gli1 marks the critical stromal effector cells in BM fibrosis and that CXCL4 expression pattern in the BM may allow for the discrimination of different stages of fibrosis. CXCL4 knockout in hematopoietic cells also reduces fibrosis severity in an animal model of disease.



CHARACTERIZING MYELOPROLIFERATIVE NEOPLASM NEOANTIGENS AND T CELL RESPONSES FOR THERAPEUTIC APPLICATION

Vivian G. Oehler, MD & Marie Bleakley, MD, PhD, Fred Hutchinson Cancer Research Center

The goal is to identify neoantigens derived from mutations that are shared among MPN patients as targets for T cell immunotherapy using a novel antigen discovery platform. HLA-typed hematopoietic antigen-presenting cells transduced to express segments of 17 recurrently mutated genes in MPNs are used to stimulate T cell responses against MPN neoantigen epitopes. This platform should lead to the discovery of both neoantigens and T cells specific for those neoantigens. So far, one novel epitope from a recurrent U2AF1 spliceosome mutation, along with T cells specific for mutant U2AF1, has been discovered.



LEVERAGING NFKB PATHWAY DYSREGULATION FOR THERAPEUTIC BENEFIT IN MYELOPROLIFERATIVE NEOPLASMS

Stephen Oh, MD, PhD, Washington University in St. Louis

The goal is to target the NFkB pathway and its impact in MPN patients. Studies have confirmed that the drug pevonedistat inhibits the NFkB pathway and restricts growth of an MPN cell line and patient cells. It also inhibits production of multiple inflammatory cytokines more potently than ruxolitinib. Studies in animal models with pevonedistat alone and in combination with ruxolitinib are ongoing and planned. A Phase 1 study of pevonedistat in myelofibrosis patients has been opened. ■

CLINICAL TRIAL HIGHLIGHTS



Research on Myeloproliferative Neoplasms has picked up heavily over the last few years. There are several MPN Clinical Trials underway. A complete list can be found on our website: www.mpnresearchfoundation.org/Clinical-Trials.

The trials listed below may be of interest as they are currently enrolling patients:

KRT-232 vs Ruxolitinib

Sponsor: Kartos Therapeutics, Inc.
Contact: John Mei
650-542-0136 | jmei@kartosthera.com
Diagnosis Type: Polycythemia Vera

KRT-234

Sponsor: Kartos Therapeutics, Inc.
Contact: John Mei
650-542-0136 | jmei@kartosthera.com
Diagnosis Type: Primary Myelofibrosis, Post-Polycythemia Vera MF, Essential Thrombocythemia MF

CALR Exon 9 Mutant Peptide Vaccine

Sponsor: Inge Marie Svane
Contact: Jacob H. Grauslund, MD
452-010-4504 | jgra0033@regionh.dk
Contact: Hans Hasselbalch, MD
454-732-4800 | hkhl@regionsjaelland.dk
Diagnosis Type: Essential Thrombocythemia, Myelofibrosis

Ruxolitinib + Pevonedistat

Sponsor: Washington University School of Medicine
Contact: Stephen Oh, MD, PhD
314-747-7960 | stoh@wustl.edu
Diagnosis Type: Myelofibrosis

Ruxolitinib + Chemotherapy

Sponsor: City of Hope Medical Center
Contact: Haris Ali | 626-256-4673
Diagnosis Type: Myelofibrosis

Rigosertib

Sponsor: MD Anderson
Contact: Jorge Cortes | 713-794-5783
Diagnosis Type: Myelofibrosis ■

CARRYING ON THE LEGACY OF ROBERT ROSEN

(FROM PAGE 1)

My dad's diagnosis coincided with a life transition. He had recently sold his real estate business and was searching for renewed purpose in life.

"I'd always wanted to do something for the greater good," he said years later. "And now there was a chance to do that, and to help myself and other patients in the process."

And so the MPN Research Foundation was born. Dad had no background in science, medical research or grant programs. But he was hell-bent on learning. He built a team focused on connecting patients with researchers, with a goal of ensuring that no patient would feel as hopeless as he did upon diagnoses. Nearly twenty years, \$13 million, and 60 funded research projects later, he has done just that.

Here's the thing. Much of MPN happens beneath the surface. Organs enlarge, joints swell, red blood cells thicken inside the bone marrow. Most people didn't even know Dad was sick. He didn't look sick. He didn't act sick. But he was sick. And in the summer of 2017, twenty years after his original diagnosis, his polycythemia vera converted to myelofibrosis, and then to leukemia. He moved to New York to prepare for a stem cell transplant, and four months later he died.

This progression – which is something patients fear most, and researchers don't understand – happened right before my eyes. It was terrifying. It was the worst thing that's ever happened to me. And to my family.

That's why I'm writing to you today – to do what Dad cannot. To ask for your help. My dad had access to the best doctors in the world, and still he died before his time. Each year, hundreds of thousands of people await a similar fate. **Our goal is to raise \$500,000 for the Robert Rosen Memorial Fund so that his legacy will not be in vain.**

My dad's story, and the stories of thousands of other patients, show us the road ahead. We need to make stem cell transplant, the only cure for MPNs, safer and more successful. We need to better understand disease progression and stop it in the first place. And we need to continue to put pressure on the medical community to better address patient needs, and to improve quality of life for people living with these chronic cancers. Medical research is expensive, and no one person can find a cure on their own. My dad knew this, and built the MPN Research Foundation based on this fact.

I used to tell Dad: *Make the mission of the foundation personal. Make it about you. Make yourself the face of this disease.* But being front and center was not his style. He was too humble. Or maybe he was scared. Or maybe, in his case, it's the same thing.

Your gift to the Robert Rosen Memorial will keep the best minds in scientific and translational research fixed on the issues of MPNs. We still have a lot of work to do before a cure is found, but every dollar raised for the MPN Research Foundation gets us a little closer.

Let's do this together. For everyone who is still suffering from blood cancer.

It's what my dad would want. ■

MPN RESEARCH FOUNDATION

CONNECT WITH US ONLINE



TO LEARN MORE ABOUT THE ROBERT ROSEN MEMORIAL FUND
OR TO MAKE A DONATION, VISIT

WWW.MPNRF.ORG/ROBERT-ROSEN-MEMORIAL-FUND

PUTTING YOUR DATA TO WORK FOR YOU: HOW TO DOWNLOAD YOUR *myMPN* INFO

Lindsey J. Whyte, Longitudinal Research Project Manager

Many thanks to all who continue to visit myMPN regularly and update your profiles. Have you visited the registry lately to fill out a *How Do You Feel, Today?* survey or record a recent event such as a blood draw or medicine change? Using myMPN as a repository for your health data can make preparing for your next doctor visit so much less stressful! Did you know that you can download a report of all your survey data directly from your myMPN dashboard at any time? Here's how.

WHERE TO FIND YOUR DATA



When you log into your profile, you will see your myMPN dashboard. At the upper left corner, there is a button called "My Data."

When you click that button, you will have the option to select any survey responses you want to view. When "Show Responses" is selected, you can see the data that you have entered. To show your doctor, scroll to the bottom of the page and click "Print to PDF" or "Save to Excel." You'll then see a dialog box (you may need to scroll back to the top of the screen) that asks to confirm whether you want to export only the selected data or all data. Make your selection and look for the prompt to see the file in the chosen program.

ONCE I HAVE THE DOWNLOAD, WHAT DO I DO WITH IT?

At this point, if you've exported to Excel, you will have the opportunity to make a chart or prepare the data in any number of different ways. The more surveys you complete, the more options you will have to organize survey responses and present them to your caregivers.

For example, if you've filled out twenty *How Do you Feel, Today?* surveys over ten months, you can create a line chart showing your level of fatigue over time. If, during that time, particular events such as a medicine change, transfusion or phlebotomy occur, you can note those events on the chart and determine how those treatments have affected your fatigue level. Seeing your data in this way helps you to better understand and appreciate the importance of being proactive about how you feel, communicating that to your doctor and ensuring that the treatments have the desired effects. If you see that their recommendations aren't working, you can show that to the doctor and work with them to find better options that do work.

If you chose PDF, you can either download the file and store for your records, upload to a secure file site for your doctor to see, or print a copy to bring to your appointment. PDFs display only the questions and your responses and may be lengthy to read. Nevertheless, a doctor or other caregiver may find it useful in assessing your condition during the time since your last appointment.

We've heard from myMPN participants that they find a lot of value in having their data available to them this way. We'd love to hear if you've found methods for recording and referring back to your data later. If you would like to share, please email us at myMPN@mpnrf.org.

And don't forget to log in to myMPN and update your profile with the latest on how you are feeling and any recent health events. If you haven't yet started with myMPN, there's no time like the present! Visit the registry at www.myMPN.org or www.mpnrf.org/mympn-register. ■

PROVIDING EDUCATION AND RESOURCES FOR A BETTER TODAY. INVESTING IN RESEARCH FOR A BETTER TOMORROW.

Partner. Advocate. Friend. At the MPN Research Foundation, we're committed to standing with you in the fight against polycythemia vera (PV), essential thrombocythemia (ET) and myelofibrosis (MF) – the group of blood cancers collectively known as myeloproliferative neoplasms.

WWW.MPNRESEARCHFOUNDATION.ORG



CHANGE YOUR PROGNOSIS

MPN RESEARCH CONSORTIUM REBOOTS, FOCUSING ON MYELOFIBROSIS



Rick Winneker, PhD

I recently had the privilege to represent the MPN Research Foundation at an External Advisory Board meeting with the MPN Research Consortium (MPN-RC) team. MPN-RC is the only independent, academic, multi-centered, externally funded collaboration that is focused exclusively on the study of MPNs. It has received continuous National Cancer Institute funding since 2006, with the current grant renewal awarded in March 2018 for five additional years and ~\$19 million of support.

While MPN-RC has contributed significantly to furthering our understanding of the MPNs since its inception, it has refocused its efforts in this latest version to improve the survival of patients with myelofibrosis (MF). They have assembled an outstanding group of laboratory and clinical scientists from eleven institutions in North America who are working collaboratively to develop and evaluate therapeutic strategies for patients with MF. They have chosen to focus on MF since it is the MPN with the shortest survival, and currently allogeneic hematopoietic stem cell transplantation is the only approach which substantially alters its natural history.

Four major project areas and a series of strong core facilities will collectively contribute to developing the strategies and clinically testing the drugs, which will act by directly targeting malignant hematopoietic stem cells and/or by correcting their tumor promoting micro-environments to ultimately deplete the numbers of cancer stem cells. This is the path needed to get to long-term remissions and possible cures.

Just as important, the lessons learned from these studies will be useful to scientists and clinicians studying a large number of other cancers. We have longstanding and close ties to many of the clinicians and scientists that are part of this highly interactive team, such as Ron Hoffman, MD, Ross Levine, MD, John Crispino, PhD, Anna Rita Migliaccio, PhD, Ruben Mesa, MD, John Mascarenhus, MD and Raajit Rampal, MD – just to name a few. I look forward to future meetings and continued progress for the MPN Research Consortium. ■

NO ONE SHOULD FIGHT CANCER ALONE

Lexi Moore,
Community Engagement & Outreach Coordinator

The MPN Research Foundation is excited to announce a new partnership with Imerman Angels (IA), a one-on-one support service aimed at connecting "Mentor Angels" with cancer fighters, survivors and caregivers. Through our partnership, these services are available to all MPN patients and caregivers, regardless of what stage you're at in your cancer journey.

Mentor Angel connections are made all across the U.S., as well as internationally, and are based on match criteria including gender, lifestyle, age, and cancer experience. Through the world's largest peer-to-peer database, Imerman Angels is able to help cancer fighters and caregivers create their own systems of mutual support.

"We could not be more excited about this partnership with MPNRF," says Jackie Herigodt, Director of Programs and Outreach for Imerman Angels. "The Foundation works tirelessly to ensure that those who are impacted by MPNs have a full breadth of support, which makes this collaboration so vital to us. Together, we can reach many more people."

Both the MPN Research Foundation and Imerman Angels share the belief that no one should face cancer alone. IA's free services help anyone touched by any type of cancer, anywhere in the world, and at any stage. Whether you were diagnosed 30 days ago or 30 years ago, Imerman Angels can help connect you to our growing MPN community. ■

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THE FUTURE IS BRIGHT: TURNING YOUR DIAGNOSIS INTO INSPIRATION

Jennifer Acker, Content Writer for MPN Research Foundation



Simran Bhardwaj

Like many teenagers, Simran Bhardwaj loves going to the movies as much as she loves adventures, hiking, running cross country, and playing both soccer and the violin. But at just sixteen, she is razor-focused on her future career.

Though she was diagnosed with Essential Thrombocythemia (ET) at fourteen, Simran isn't using her diagnosis as an excuse. Just the opposite – she hopes to someday help children like herself by becoming a doctor.

"My MPN has helped me with something – deciding my future," she says. "My first hematologist, Dr. Stuart Winter, was one of the best doctors I have ever met. Dr. Winter inspired me to want to become a pediatric hematologist or oncologist."

Before her diagnosis, Simran was nauseous and dealt with migraines that lasted up to two weeks. As a soccer player and runner, she was conditioned to running long distances, and it seemed strange that she was getting nauseous after practices. Initially Simran's doctor didn't find any cause for concern, but her mother insisted on a blood test.

That's when Simran discovered that she had a high platelet count.

Simran's father Manoj says there aren't many teenagers with ET, so the data isn't extensive and trying to determine the best care and treatment options can be difficult. The Bhardwajs are grateful for the treatment Simran has received at the University of New Mexico, especially the care of Dr. Stuart Winter, Simran's first pediatric hematologist. After receiving treatment, Simran now experiences fewer symptoms. "I'm able to live a fairly normal life, which is wonderful," she says.

Having a child with an MPN is rare, but attending a recent pediatric

conference related to MPNs gave Manoj the opportunity to connect with others. He suggests that parents keep meticulous medical records of their child and stay on top of everything, even if it means being a pain to the doctor. And his hope for the future is that the genetic mutation which causes ET will be identified.

"The MPN Research Foundation has helped bring folks together to create a community for support," he says. "We have started to get to know this community, and after having attended this conference, we are so appreciative that this was possible."



(L-R) Simran with her family: Brother Thaaaj, father Manoj, mother Urvashi, and Simran.

"MY MPN HAS HELPED ME WITH SOMETHING – DECIDING MY FUTURE. MY FIRST HEMATOLOGIST INSPIRED ME TO WANT TO BECOME A PEDIATRIC HEMATOLOGIST OR ONCOLOGIST."

Simran Bhardwaj, 16 years old, ET Patient

While it would be perfectly understandable for Simran to take it easy, that's not her style. Simran now hopes that the MPN community can one day help her achieve her career goal through practice methods such as shadowing and other clinical experiences.

"I'm exposing myself to medical opportunities, such as the medical club at school and the Youth Empowerment Program at the hospital I get treatment at," she adds.

A driven, spirited, and intelligent young woman, Simran has already learned one of life's toughest lessons – one that takes some people a lifetime to figure out. When you can identify the good in the tragic and recognize that challenges are opportunities for self-discovery and growth, you've got a special gift. ■