POWERING PROGRESS *together*

MRA Scientific Retreat & Patient Forum

February 26-28, 2025

Melanoma Research Alliance

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Melanoma Research Alliance
730 15th Street, NW
Washington DC, 20005

Letter from MRA's Chief Science Officer

Every year the Melanoma Research Alliance (MRA) looks forward to bringing together the melanoma community at our annual Scientific Retreat. The 2025 Scientific Retreat was held February 26 – 28 in Washington, D.C. and brought together over 300 academic researchers and clinicians, pharmaceutical and biotech representatives, partners and donors, and patient advocates to engage in scientific discourse, collaboration, and learning.

At the Retreat, participants learned about the latest research breakthroughs in melanoma diagnosis, preventing disease progression, and advancing new treatments — with many updates stemming directly from the contributions of MRA-funded investigators. The event also highlighted perspectives from the patient advocate community, inspiring us all and emphasizing our shared mission to end suffering and death due to melanoma.

Topics presented at this year's Retreat crossed the melanoma continuum. From strategies to identify and overcome therapeutic resistance, to new technologies to identify diagnostic and prognostic biomarkers, and investigating the rare melanoma subtypes — a diverse overview of melanoma research was highlighted.

In addition to the formal sessions, the Retreat offered dedicated programming to support the next generation of scientific leaders. Highlights included a special breakfast for MRA Young Investigator Awardees focused on the theme, "The Use and Misuse of AI." An interactive Poster Session gave investigators the opportunity to present their research and engage in meaningful dialogue. The event also featured networking roundtables, encouraging focused discussions and interdisciplinary collaboration and idea sharing.

MRA remains deeply inspired by the connections made and insights gained at the Scientific Retreat — a gathering that continues to shape the future of melanoma research. The knowledge shared and collaborations sparked at this event are accelerating progress year after year.

While the advances discussed at the Retreat offer real hope, this is no time to pause. As national investments in cancer research are experiencing cuts, the need to support bold, lifesaving science is more critical than ever. MRA understands that funding is essential to ensure that research continues, discoveries are translated, and patients benefit from new breakthroughs.

We remain committed to **Powering Progress Together**, saving more lives, and changing the future for melanoma patients.

With gratitude,

Joan Levy

Joan Levy, PhD
Chief Science Officer

> "If I had gotten melanoma a decade earlier, I don't know that I'd be around to talk about it three and a half years later."
>
> **GEORGE MANNES**

POWERING PROGRESS TOGETHER:
MRA's 2025 Scientific Retreat

Each year, the Melanoma Research Alliance convenes leading voices from the global melanoma research community to share breakthroughs and challenges, spark new collaborations, and strengthen connections.

To officially kick off the 2025 Scientific Retreat, New York City-based writer and editor George Mannes shared his melanoma journey. An avid marathon and distance runner, George never imagined what he assumed was a blood blister on his big toe would end up changing his life. In 2021, he was diagnosed with acral melanoma — a rare and aggressive subtype that forms on the palms, soles, or under nails.

George's diagnosis came as a shock. Like many people, he associated skin cancer with sun exposure, never expecting the small lesion on his toe to be melanoma. Following surgery to amputate his toe, George began immunotherapy. Although the treatment initially helped, his cancer progressed.

Despite the setbacks, George and his care team pressed forward, trying multiple treatment combinations and enrolling in a clinical trial. Along the way, he has experienced both the physical toll of advanced melanoma and the emotional challenges of navigating a rare cancer. Through it all, George stays grounded in hope, fueled by the love of his family, his own determination to keep going, and the passion of his care team and researchers.

Today, George continues to navigate melanoma, grateful for his quality of life. "If I had gotten melanoma a decade earlier, I don't know that I'd be around to talk about it three and a half years later," he shared with the Retreat audience. "I'd like to thank each and every researcher for all the work they've done in the past, and all the work they are doing now. Work that is keeping me and other cancer patients alive and preserving our quality of life."

George's story is a powerful reminder of why continued research is so vital — giving patients more options, more time, and more hope for the future.

"One of the most valuable aspects facilitated during the MRA Scientific Retreat is the consequent spark of ideas and collaborations."

2025 SCIENTIFIC RETREAT PARTICIPANT

Peter Sorger, PhD - Harvard University

New developments may eventually expand access to expert-level detection while reducing unnecessary procedures.

The Changing Landscape of Melanoma Research:

From Biomarker Discovery to AI-Powered Early Detection

Recent developments in melanoma research are addressing persistent challenges in diagnosis, prediction, and prevention. Researchers are investigating novel biomarkers that could better stratify patients and predict treatment outcomes, particularly for immunotherapy. In addition, the application of artificial intelligence (AI) in dermatology has both promising opportunities and significant implementation hurdles that require careful navigation. These advances, alongside improvements in imaging technologies like 3D total body photography, may eventually expand access to expert-level detection capabilities while reducing unnecessary procedures.

Toward Better Prediction: Current Research in Melanoma Biomarkers

Dr. Peter Sorger from Harvard Medical School presented research on the development of prognostic biomarkers (which correlate with disease progression or recurrence) for early-stage melanoma and the potential of predictive biomarkers

(which predict therapeutic response) for treatment of Stage III melanoma with immunotherapies. He explained that accurately diagnosing early melanoma, determining stage, and developing a treatment strategy is dependent on the examination of tissue sections using criteria that are highly refined but involve methods developed 100-150 years ago. Dr. Sorger described how new multiplexed imaging methods that quantify many protein markers at the same time at a single-cell level in tissues promise to greatly improve classification and staging.

Dr. Sorger also presented the results of his team's advanced 3D imaging techniques, which can be used to visualize the orientation of tumor and immune cells and surrounding structures. Such methods make it possible to visualize cancers at an unprecedented level of detail and promise to reveal the fundamental biology of tumor initiation and spread. Such detailed understanding — performed primarily in a research setting — can reveal features such as the specific arrangements of immune cells killing a tumor cell, or the migration of rare tumor cells into surrounding tissue. Dr. Sorger suggested that, in the relatively near future, machine learning models trained on these detailed views of tumors cells, in combination with instruments and techniques designed for clinical translation, will enable a new generation of diagnostic methods able to realize the promise of individualized precision medicine.

Dr. Janis Taube, Johns Hopkins University, is working to bridge the gap between discovering biomarkers and getting approval for their clinical use from both local and federal regulatory groups. Her team developed an imaging test that analyzes several biomarkers within a melanoma simultaneously and is used to predict response to immunotherapy. They identified specific features that correlated with patient outcomes, allowing classification into poor, intermediate, or good prognosis groups.

In order for the test to be used on patient samples, the team performed a series of validation experiments. This includes determining the minimum sample requirements for reliable results, testing the performance on tumor samples that were collected from 5 to 20 years earlier, and comparing results obtained when the test was performed using different equipment and at different institutions. Dr. Taube and her team are now focused on additional validation experiments that are needed in order to receive the necessary approvals for the test to be used on samples from patients with melanoma.

Janis Taube, MD - Johns Hopkins University

The Role of AI in Research: Promises and Pitfalls

During the MRA Scientific Retreat, a panel discussion was held for young investigators to discuss the evolving role of AI in research, highlighting both the tremendous potential and significant challenges facing researchers and clinicians. The panel featured two dermatologists and a journal editor at the forefront of AI development and offered valuable insights into the current state and future applications of AI tools. Dr. Sara DiNapoli (MRA) moderated the panel, which included Dr. Alexia-Ileana Zaromytidou, *Nature Cancer*; Dr. Eugene Semenov, Massachusetts General Hospital, Harvard Medical School; and Dr. Veronica Rotemberg, Memorial Sloan Kettering Cancer Center.

The experts identified several key barriers to effective AI implementation in health care. Dr. Semenov highlighted

{L to R): Sara DiNapoli, PhD - Melanoma Research Alliance, Alexia-Ileana Zaromytidou, PhD - Nature Cancer, Eugene Semenov, MD, MA - Massachusetts General Hospital, and Veronica Rotemberg, MD, PhD - Memorial Sloan Kettering Cancer Center

infrastructure challenges: "It tends to require quite a bit of infrastructure in terms of storage, computing power, and someone to maintain all those systems, which is not cheap." Data sharing emerged as a significant research hurdle. Despite improvements in recent years, patient privacy concerns and institutional barriers often prevent the free exchange of information necessary to validate AI models across different settings. Dr. Rotemberg indicated "data sharing has improved, but especially for patient-related and genetic-related data, it's still very challenging to share across institutions and critical to proving that a model actually works."

The scientific community is progressing toward standardized assessments for AI models in publications to improve reproducibility. Dr. Zaromytidou emphasized the importance of transparency in published AI research. "For me, a red flag would be the absence of the code. You're essentially going by blind faith," she explained, adding that reputable journals now require authors to make their code available for peer review.

The panelists also addressed the appropriate use of AI tools in scientific writing. While AI can help with editing tasks, using it to generate original content raises serious ethical concerns. "AI cannot be an author. Authorship comes with responsibility, with accountability," Zaromytidou stated, explaining that *Nature Cancer* immediately rejects papers where generative AI tools were used to create text, in contrast to accepted uses such as AI-assisted copy editing of human-generated text.

The panel expressed optimism about AI's future in dermatology. Dr. Rotemberg highlighted diagnostics in melanoma from photos as an up-and-coming area: "There are not enough dermatologists. There are not enough experts, especially outside of large cities, and photos are such an accessible thing." However, the available AI tools are not yet able to work in the general population. Dr. Semenov added, "These models will continue to improve, and I think they will get to the point where they can be even more accurate than humans."

Prediction & Prevention: The Power of AI and the Power of the Eye

Dermatologist Allan Halpern of Memorial Sloan Kettering Cancer Center delivered a presentation on the future of melanoma prevention and early detection, focusing on how technology and AI are transforming the field.

> "The algorithms coming out of the challenges were better than dermatologists that we recruited from around the globe to compete against the algorithm."
>
> **DR. ALLAN HALPERN**

Allan Halpern, MD - Memorial Sloan Kettering Cancer Center

Dr. Halpern began by emphasizing that prevention is always preferable to treatment: "There is not a patient in this group...despite all the advances we're making in advanced disease, that wouldn't have preferred to never to have gotten their melanoma in the first place."

He explained that prevention falls into two categories:

- **Primary prevention:** Avoiding melanoma entirely, primarily through sun/UV protection.
- **Secondary prevention:** Early detection to catch melanoma when it can still be cured surgically with minimal intervention.

Detection becomes more challenging for high-risk individuals, particularly those with numerous or atypical moles. As one patient in the audience described, it's like "trying to find the changing tree in the forest." This is where advanced technologies become essential.

Two major technologies highlighted in Dr. Halpern's talk were total body photography and digital dermoscopy follow up. Total body photography was initially developed in the 1980s using regular photographs. This technique has since evolved into 3D imaging systems that can photograph a patient's entire body in milliseconds. Dr. Halpern explained, "Now every single spot has a unique coordinate" that allows for precise monitoring over time and may reduce the need for biopsy. Digital dermoscopy is a specialized imaging technique that allows dermatologists to see features invisible to the naked eye and track subtle changes in lesions.

Dr. Halpern also focused on the role of AI in melanoma detection. He co-founded the International Skin Imaging Collaboration in 2014, an archive of over 1.3 million skin images, including more than 10,000 melanoma images. This database has enabled the development of increasingly sophisticated AI algorithms. By 2018, these algorithms outperformed many dermatologists: "The algorithms coming out of the challenges were better than dermatologists that we recruited from around the globe to compete against the algorithm."

Despite this progress, Dr. Halpern acknowledged several challenges that remain:

- AI systems need data sets from multiple ancestral groups to be effective across all skin types and lesion varieties.
- Regulatory approval processes are complex for AI that continuously learns.
- There's a risk of overdiagnosis and unnecessary biopsies with increased detection.

Dr. Halpern emphasized the importance of balancing enhanced detection with patient-centered care. Screening too aggressively for melanoma can sometimes find harmless spots that are labeled as dangerous, leading to unnecessary stress and procedures for patients while making it harder for those who genuinely need care to get appointments with specialists.

The presentation concluded with a positive outlook on technology's potential to democratize access to expert-level melanoma detection while acknowledging the need for thoughtful implementation: "We need to give real thought to how this gets implemented in clinical practice."

Martin McMahon, PhD - University of Utah Huntsman Cancer Institute

Uncovering Hidden Pathways:
New Discoveries With Great Promise

Recent advances in melanoma research were presented at MRA's 2025 Annual Scientific Retreat. Scientists are making significant progress in four key areas: the evolution of targeted therapies, an unexpected connection between melanoma and Parkinson's disease, the unique challenges of mucosal melanoma, and how protein migration within cells might enable earlier detection and better treatments. These groundbreaking discoveries are transforming our understanding of melanoma and creating promising new pathways for patient care.

The Trajectory of Pathway-Targeted Therapy in Melanoma

Dr. Martin McMahon, University of Utah Huntsman Cancer Institute, presented how melanoma treatment has transformed over the past 20 years. The discovery of *BRAF* gene mutations in melanoma was reported in 2002. Soon after, researchers developed drugs targeting the mutationally activated *BRAF* oncoprotein. These treatments showed impressive results in patients, but a significant challenge was quickly identified: drug resistance. Dr. McMahon explained that melanoma can develop "10 or 15 mechanisms of resistance" even within a single patient, making long-lasting responses challenging to achieve with single medications.

Even with these challenges, there have been remarkable success stories. Researchers developed combination approaches to improve responses, particularly by pairing *BRAF* and MEK inhibitors. This strategy has become "one of the standards of care for patients with *BRAF*-driven melanoma who are either ineligible for frontline immunotherapy or for whom immunotherapy fails." Researchers are also exploring combinations of targeted therapies with immunotherapy drugs, which have shown promising early results.

Despite advances in targeted therapy for melanoma, researchers still don't fully understand why some tumors

don't respond to treatment and why some return after initially responding to therapies, making it challenging to develop effective combination approaches that work for everyone. One of the most exciting approaches is targeting RAS proteins, which drive about 20% to 25% of all melanomas. RAS proteins act as cellular switches that, when mutated in cancers like melanoma, continuously trigger pathways that drive uncontrolled melanoma cell growth and tumor development.

For years, scientists considered RAS oncoproteins undruggable, but researchers at Revolution Medicines in California have now developed a novel medication named daraxonrasib that works as a "molecular glue" to block cancer-causing signaling within *NRAS*-mutated melanoma cells. Daraxonrasib elicited dramatic tumor responses in mouse models of *NRAS*-driven melanoma, and in a patient who had previously been treated with three rounds of immunotherapy.

Although drug resistance remains a concern, researchers are actively studying ways to overcome it, and the field has progressed tremendously from the "dark old days" when doctors had to use treatments that would not work, to today's era of increasingly personalized medicine. As Dr. McMahon noted, melanoma researchers are "a resilient bunch" who continue pushing forward despite challenges, and who are driven by the essential goal of helping patients with melanoma.

The Link Between Melanoma and Parkinson's Disease

Multiple studies have observed a link between melanoma and Parkinson's disease, explained Dr. Deanna Benson, Icahn School of Medicine at Mount Sinai, who is investigating this link. The research focuses on a protein called LRRK2, which may be a point of connection between the two seemingly unrelated conditions.

Increased LRRK2 activity appears to be a common driver in Parkinson's disease. Mutations that elevate LRRK2 activity increase Parkinson's disease risk by 25% to 80%, depending on an individual's genetic background. Many patients with Parkinson's disease that is idiopathic (i.e., with no known cause) also show increased LRRK2 activity.

Deanna Benson, PhD - Icahn School of Medicine at Mount Sinai

Researchers initially expected mice with increased LRRK2 activity to develop melanoma tumors faster than normal mice. "We thought that the gain-of-function kinase activity would make tumors grow faster," Dr. Benson explained, but they found no difference in tumor growth rates. However, tumor growth decreased in mice when LRRK2 activity was inhibited using a drug called MLi-2. "Much to our surprise... growth was almost completely obliterated [when we inhibited LRRK2]."

LRRK2 inhibition does not directly affect melanoma cell growth. Instead, it influences the tumor microenvironment through nerve innervation (how nerves grow into the tumor) and immune cell infiltration. The team used advanced imaging techniques to observe that sympathetic nerve cells innervate tumors. These nerve patterns are heterogeneous and appear to increase with LRRK2 mutation. Nerve axons and tumor cells interact with each other and the surrounding matrix. Sometimes, individual axons wind around tumor cells, while at other times, they form separate zones. Dr. Benson noted, "the axons are interacting with the tumor cells and talking to each other."

Genevieve Boland, MD, PhD - Massachusetts General Hospital

> "We welcome collaboration. We want to all work together to ensure we're moving the needle forward and providing better care for our patients."
>
> **DR. GENEVIEVE BOLAND**

Preliminary data suggest possible differences in immune cell recruitment to tumors. The team is now investigating how nervous system activity might influence melanoma development, because LRRK2 mutations affect neurotransmitter release. They are also examining how tumor characteristics change spatially in relation to nerve axons and LRRK2 activity. Although the research is still preliminary, it suggests that LRRK2 inhibition might offer a novel approach to treating melanoma through its effects on the tumor microenvironment.

Understanding Mucosal Melanoma: A Research Update

Mucosal melanoma is a rare melanoma subtype that originates from the moist surfaces (mucosa) inside the body, including respiratory passages, sinuses, mouth, throat, genitals, and urinary tract. Unlike cutaneous melanoma, this subtype presents unique challenges for treatment and research.

"It takes a village," noted Dr. Genevieve Boland, Massachusetts General Hospital, explaining that patients with mucosal melanoma are initially seen by many different specialists, such as oral surgeons, head and neck surgeons, gynecologists, or urologists, depending on where the cancer appears. This fragmentation makes studying—and treating—the disease more difficult. In addition, mucosal melanoma lacks its own specific staging system and dedicated treatment guidelines, and the systems used for cutaneous melanoma do not work well for mucosal cases, making it harder for doctors to make clinical decisions or predict outcomes. Dr. Boland advocated for standardized approaches to better understand and treat this melanoma subtype.

She shared findings from a study of about 70 patients. The research revealed that patients with mucosal melanoma who have cancer in their lymph nodes generally have worse outcomes, which is not the case for patients with cutaneous melanoma, further demonstrating the differences between melanoma subtypes.

Importantly, mucosal melanomas don't respond as well to immune checkpoint inhibitors, which have been successful against cutaneous melanoma. This led Dr. Boland and her team to study differences in immune cells between mucosal and cutaneous melanomas. Using advanced imaging techniques, they discovered that while many mucosal melanomas are "immunologically cold" (with few immune cells), some show immune cell presence. The team is studying these differences across various mucosal sites to better understand the disease.

"We welcome collaboration," Dr. Boland concluded, emphasizing the importance of teamwork across institutions. "We want to all work together to ensure we're moving the needle forward and providing better care for our patients."

"Patients with a high cytoplasmic-to-nuclear ratio of the protein have a higher probability of disease progression"

DR. FÁTIMA GEBAUER

Fàtima Gebauer, PhD - Centre for Genomic Regulation

Tracking Movements: How Protein Location Could Unlock New Melanoma Treatments

Dr. Fátima Gebauer, Centre for Genomic Regulation, highlighted important research on an RNA-binding protein called CSDE1 (also known as UNR) that could lead to new ways to detect and treat melanoma. RNA-binding proteins have been relatively overlooked in melanoma research, which has mostly focused on genetic mutations.

Dr. Gebauer's work focuses on how CSDE1 affects the way cells react to external cues, and her team has found that CSDE1 behaves differently in healthy cells versus melanoma cells. In moles (nevi), CSDE1 is mainly found in or near the cell nucleus. In melanoma cells, CSDE1 moves to the cell's outer region (cytoplasm) and attaches to a structure called the endoplasmic reticulum. This location change could serve as an early warning sign of melanoma.

"Patients with a high cytoplasmic-to-nuclear ratio of the protein have a higher probability of disease progression," noted Dr. Gebauer, suggesting this could help predict which cases might become more aggressive.

Interestingly, CSDE1 acts like a "Dr. Jekyll and Mr. Hyde" protein: It promotes cancer growth in melanoma but suppresses tumors in other cancers. Dr. Gebauer's research also revealed that CSDE1 undergoes a specific chemical modification in melanoma cells but not in healthy cells. This modification allows it to interact with new partners that may explain its cancer-promoting effects in melanoma.

This research could lead to two important developments. First, it may provide a new way to diagnose melanoma earlier and identify aggressive cases. Second, it could lead to potential treatments targeting its modification in melanoma cells.

The team is now working with collaborators to understand precisely how CSDE1 interacts with its new partners, which could "provide a specific therapeutic avenue for the treatment of late-stage disease."

Ana Anderson, PhD - Brigham and Women's Hospital

> "We uncovered a pathway that's generally operative within the central nervous system but has been co-opted to suppress T-cell responses in the context of cancer."
>
> **DR. ANA ANDERSON**

Outsmarting Melanoma:

Reinvigorating the Immune Response

New research is uncovering complex connections between immune function and cancer treatment, making progress towards future therapeutic approaches. Researchers have identified unexpected mechanisms affecting how the immune system interacts with cancer cells, including the surprising role of opioid peptides in immune suppression. Studies exploring Notch proteins, RNA splicing modifications, and genetic targets are also expanding our understanding of cancer biology.

Researchers Discover an Unexpected Role for Opioids in Cancer Immunity

Dr. Ana Anderson, Brigham and Women's Hospital, found that as tumors grow, some immune cells called CD8+ T cells begin producing their own opioid peptides, which surprisingly leads to their dysfunction.

"We uncovered a pathway that's generally operative within the central nervous system but has been co-opted to suppress T-cell responses in the context of cancer," explained Dr. Anderson. This finding emerged from dynamic analyses of melanoma tumor progression; using advanced single-cell

sequencing, the team tracked changes in immune cells over time as tumors progressed. They discovered that the T cells acquire the ability to produce natural opioid peptides as tumors grow. These same T cells also express receptors that can respond to these opioids, essentially creating a self-suppressing loop.

"In settings of chronic T-cell stimulation, T cells become endowed with the ability to dampen inflammatory pain through the production of opioid peptides," Dr. Anderson notes. "This mechanism, which is normally operative in the nervous system, has been co-opted in cancer and poses a barrier for anti-tumor immunity."

The team confirmed their findings through multiple experiments. When T cells were artificially programmed to increase opioid production, their function was decreased, and tumors grew faster. Conversely, when they removed the ability of T cells to produce these opioids, T cells had improved function and were better able to control tumor growth.

Most importantly, the research showed that blocking opioid signaling with medication enhanced the effectiveness of anti-PD-1 (programmed cell death protein 1) immunotherapy. A retrospective analysis of almost 300 patients with melanoma who were treated with anti-PD-1 revealed that those who received opioid medications during immunotherapy had worse outcomes than matched patients who didn't receive opioids.

These findings have important implications for cancer treatment, particularly regarding pain management approaches for patients receiving immunotherapy. The study suggests that commonly prescribed opioid medications might inadvertently hinder the body's anti-cancer immune response, potentially compromising treatment effectiveness.

Breaking New Ground in Notch Protein Research for Melanoma Treatment

Researchers are exploring promising new approaches to target Notch proteins in melanoma treatment, reported Dr. Barbara Bedogni, University of Miami. Notch proteins, particularly Notch1, play critical roles in cancer cell survival and growth, as well as resistance to immunotherapy.

"Notch is highly active in a very high percentage of melanomas, so we think it's a good target," explained Dr. Bedogni. Her team found that tumors with high Notch1 activity tend to have less inflammation and respond poorly to immunotherapy treatments.

Initial attempts to block all Notch proteins using gamma secretase inhibitors (GSI) proved problematic. While GSIs showed some tumor control, they caused concerning side effects, including weight loss and immune suppression. "GSIs are not a good approach to reducing Notch activity because they cause immunosuppression overall," Dr. Bedogni noted.

This led the team to develop a specialized antibody targeting only Notch1, preserving the beneficial functions of other Notch proteins. The antibody successfully reduced tumor growth and decreased immunosuppressive cells in the tumor environment in mouse models. It also enhanced the killing ability of cancer-fighting T cells and boosted the effectiveness of anti-PD-1 immunotherapy when combined. Unlike GSIs, the antibody didn't cause intestinal toxicity or weight loss in laboratory mice, suggesting it may be safer for patients.

Barbara Bedogni, PhD - University of Miami

Interestingly, the team discovered that the antibody works through an unexpected mechanism: It disrupts cancer cells' ability to produce histone proteins needed for DNA packaging during cell division. "This causes genomic instability, which may then promote an inflammatory type of tumor microenvironment," making cancers more responsive to immunotherapy. Future work will continue to expand upon these findings.

Precision Engineering: Making Immune Cells Smarter Cancer Fighters

Dr. Benjamin Izar, Columbia University, described the progress his lab has made in understanding how cancer cells resist immunotherapies and developing new approaches to overcome this resistance.

The loss of a protein called CD58 on cancer cells creates a "double whammy" effect that makes tumors highly resistant to immune checkpoint therapy. When cancer cells lose CD58, they gain more PD-L1 (programmed cell death ligand 1, a protein that helps cancer cells hide from the immune system). CD58 also helps immune cells find and stick to cancer cells to attack it, so when cancer cells lose this protein, your immune system has trouble locating and fighting the tumor. "We found that CD58 is central to antigen presentation and T-cell adhesion," explained Dr. Izar. "Loss of this protein significantly impairs T-cell infiltration into tumors."

Benjamin Izar, MD, PhD - Columbia University

Researchers in Dr. Izar's lab found that a protein called CMTM6 controls how CD58 and PD-L1 move around the cell. CMTM6 helps bring these proteins back to the cell surface after temporarily storing them inside the cell. When CD58 is missing, there is more CMTM6 available to transport PD-L1 to the surface, which makes it harder for immune cells to recognize and attack the cancer.

Building on these findings, the team developed a novel approach to improve cell therapy treatments. Rather than creating complex engineered T cells, they asked: "What is the absolute minimum change we could make to improve T-cell function?" Using precise gene editing, they introduced thousands of tiny mutations in genes important for T-cell function. The most promising results came from mutations affecting PI3Kδ, an enzyme central to T-cell activation.

"We found that a single amino acid change in this enzyme dramatically improved T-cell function," Dr. Izar said. "Most exciting was that these minimally engineered T cells could overcome resistance caused by loss of CD58 in cancer cells."

Another research group independently validated this finding and identified the same mutation as beneficial, suggesting this strategy has real promise for improving cancer immunotherapies. The findings point toward new therapeutic approaches that could help patients who don't respond to current immunotherapies.

Awakening the Immune Response: New Genetic Targets for Melanoma Treatment

Dr. Marcus Bosenberg, Yale University, said that researchers have discovered promising new approaches for melanoma treatment by studying how specific genes affect tumor growth and the immune response. Genetic screens were conducted to identify genes that, when removed, can cause melanoma tumors to be recognized and destroyed by the immune system.

One key discovery involves a gene called *SETDB1*, which modifies histones—the proteins that maintain DNA structure within cells. When researchers knocked out this gene in melanoma cells, the tumors completely regressed in mice. "The results were really quite dramatic," noted Dr. Bosenberg.

"When we had the knockouts, the mice survived, which means their tumors had fully regressed."

The team found that removing *SETDB1* causes melanoma cells to produce more type 1 interferon, a signaling molecule that helps immune function. This leads to increased expression of major histocompatibility complex (MHC) class I molecules on cancer cells, making them more visible to immune cells called CD8 T cells, which can then destroy the cancer. They also found that expression of endogenous retroviruses, which are ancient viral DNA fragments in our genome, was activated with *SETDB1* loss. This can trigger immune sensors and boost anti-cancer responses.

Dr. Bosenberg's lab is also studying another histone-modifying gene called *KDM5B*. When removed from cancer cells, tumors shrank and did not regrow. In a "convergence of similar data," the team found that *KDM5B* loss also resulted in activation of interferon type 1 signaling and activation of endogenous retroviruses. Additionally, they discovered that *KDM5B* and *SETDB1* can interact in melanoma cells and that *KDM5B* may help anchor *SETDB1* at certain regions in the genome.

These findings could lead to new therapeutic approaches for patients with melanoma. Future strategies might include developing drugs targeting *SETDB1* or *KDM5B* or creating vaccines targeting the endogenous retroviral proteins.

Unlocking the Power of RNA Splicing in Cancer Treatment

Dr. Rotem Karni, Hebrew University of Jerusalem, discussed exploring exciting new ways to fight cancer by targeting how our genes work. His team is studying RNA splicing, a crucial step in gene expression where parts of genetic sequences are removed or included to create different proteins from the same gene.

"RNA splicing can determine cell fate and plays a critical role in cancer," explained Dr. Karni. His team has discovered that certain splicing factors can act as oncogenes (cancer-promoting genes) in breast cancer, liver cancer, and other tumor types.

Their innovative approach focuses on enhancing immunotherapy, which has revolutionized cancer

Marcus Bosenberg, MD, PhD - Yale University

treatment but still has limitations. Many common cancers don't respond well to immunotherapy, and even in responsive cancers like melanoma, it only works for a fraction of patients.

The team has developed a method to force cancer cells to skip certain parts of genes (exons), creating frame shifts that generate "foreign" peptides. These new peptides can be recognized by the immune system, making the cancer cells more visible to immune cells.

"By manipulating the splicing process, we can generate strong neoantigens so immunotherapy will work more effectively," Dr. Karni said. In their experiments with mice, tumors with artificially induced exon skipping showed significantly more potent immune responses than control tumors.

The most promising results came when combining this approach with already established immunotherapy, such as checkpoint inhibitors. In mouse models of melanoma, the combination therapy showed dramatic tumor shrinkage, suggesting a powerful synergistic effect.

"The combination makes it even better. We think this is a strategy to enhance the activity of checkpoint inhibitors for rare melanomas like mucosal melanoma," shared Dr. Karni.

This innovative research opens new possibilities for improving cancer immunotherapy, especially for hard-to-treat cancers with limited treatment options.

Yana Najjar, MD - University of Pittsburgh

Advancing Priority Research:

Overcoming Immune Resistance and Metastatic Disease

Exciting new melanoma research aims to address important biological questions that might uncover new therapeutic approaches for melanoma patients who are not responding well to current treatments. This might aid in finding therapeutics to treat tumors that are resistant to immunotherapies as well in tumors that are progressing locally or to distant tissues in the body.

New Approach for Immune Resistant Melanoma

Dr. Yana Najjar, UPMC Hillman Cancer Center, explained that researchers are making important strides in understanding why some melanoma patients do not respond to immunotherapy and how to identify treatment for them. Dr. Najjar's team studies how cancer cells use oxygen and create hypoxic (low-oxygen) environments that help these cells resist immune treatments.

"We now know from the 10-year update that patients treated with dual checkpoint blockade have about a 50% chance of being alive 10 years out," notes Dr. Najjar. "However, more than half of patients either show primary or secondary resistance to immunotherapy."

In collaboration with Dr. Greg Delgoffe's lab, the team has discovered that melanoma tumors that have high oxidative metabolism, or use a lot of oxygen, create hypoxic conditions in the surrounding tissue. This oxygen-deprived environment weakens the immune cells that would normally fight cancer. Patients whose tumors work this way have poorer outcomes, with reduced progression-free and overall survival.

To address this challenge, Dr. Najjar developed a clinical trial using axitinib and anti-PD1 immunotherapy in patients with melanoma whose tumors had stopped responding to immunotherapy. The hypothesis was that axitinib would modulate hypoxia in the tumor environment, thus sensitizing melanoma tumors to anti-PD-1 inhibitors. The trial enrolled 31 patients, including half with acral or mucosal melanoma, two rare melanoma subtypes.

The results were promising, and patients who showed clinical benefit (partial response or stable disease) demonstrated either a reduction or stabilization of intra-tumoral hypoxia compared with their baseline measurements before treatment. "In patients with a confirmed partial response, there's a significant reduction

in intra-tumoral hypoxia at week 12 compared with baseline," explained Dr. Najjar.

Dr. Najjar's research also focuses on understanding oxidative metabolic effects in melanoma that is progressing from the primary tumor. Part of her studies look at "in-transit metastases," tumor nodules that develop between the primary tumor site and the nearest lymph node. These represent a unique opportunity to study how melanoma evolves as it spreads.

"We started to wonder whether we might be able to utilize in-transit melanoma metastases as a model to assess the impact of hypoxia on the propagation of melanoma metastases from one site to the next," Dr. Najjar shared.

Early findings suggest that as melanoma spreads further from the primary site, the tumor nodules become more hypoxic and immune cells become more dysfunctional. This ongoing research aims to better understand how melanoma evades the immune system as it progresses away from the primary site.

Repurposing Drugs for Leptomeningeal Disease: New Hope for Patients

Leptomeningeal disease occurs when cancer spreads to the membranes covering the brain and spinal cord, explained Dr. Inna Smalley, Moffitt Cancer Center. This condition typically leads to severe neurological symptoms and poor survival rates, with most treatments proving ineffective. The leptomeninges are the two innermost protective membranes that cover the brain and spinal cord, containing cerebrospinal fluid (CSF) that provides nutrients and removes waste products.

Dr. Smalley's team performed a comprehensive analysis of leptomeningeal disease across different cancer types, including melanoma, lymphoma, and breast cancer. The study revealed a dysfunctional immune environment in these patients. Most T cells (important immune cells) were exhausted, dying, or inactive. Patients who survived longer had more active, proliferating T cells, suggesting that immune function directly impacts survival.

In addition, unusually high concentrations of branched-chain keto acids were found in the spinal fluid of people with leptomeningeal disease. Typically associated with rare metabolic disorders and not cancer, these

Inna Smalley, PhD - Moffitt Cancer Center

compounds are highly neurotoxic and may contribute to the severe neurological symptoms experienced by patients with leptomeningeal melanoma. They also can be very immunosuppressive correlating with the presence of inactive or exhaustive T cells in the CSF of leptomeningeal patients.

When the researchers exposed nerve cells to the keto acids in laboratory experiments, they observed decreased cell viability. In animal models of leptomeningeal disease, they found that higher levels of the keto acids were coupled with worse neurological function.

The team explored whether an existing treatment for metabolic disorders, sodium phenylbutyrate, might help patients with leptomeningeal disease. Using animal models, they tested this treatment, which lowers keto acid levels in the CNS. Sodium phenylbutyrate reduced keto acid levels almost to normal and some animals even had complete resolution of central nervous system symptoms. Furthermore, there were significant improvements in survival.

"We're hoping this data puts forward a potentially actionable therapeutic opportunity through drug repurposing," said Dr. Smalley, suggesting that existing medications might be adapted to help patients with leptomeningeal melanoma. This approach offers a promising new therapeutic direction for treating this aggressive cancer complication potentially improving patient survival and quality of life.

"We would not be able to make these discoveries without the invaluable contribution of patients and their families," Dr. Smalley emphasized, highlighting the collaborative nature of this work.

Growing knowledge of the unique genomic profiles and biological behaviors of rare melanomas is paving the way for more effective targeted treatments.

Boris Bastian, MD, PhD - University of California, San Francisco

Rare Melanomas in Focus:

Breakthroughs Powered by Collaboration

Recent research into rare melanoma subtypes has yielded significant breakthroughs in understanding these challenging cancers. Scientists are uncovering distinct genomic evolution patterns in acral melanoma, promising therapeutic advancements for uveal melanoma, and innovative approaches to better understand drivers and resistance mechanisms of the different rare melanoma subtypes through collaborative research efforts. This growing knowledge of the unique genomic profiles and biological behaviors of rare melanomas is paving the way for more effective targeted treatments.

Genomic Storms: New Insights into Acral Melanoma Evolution

Acral melanomas arise on the non-hair-bearing skin on the soles of the feet, palms of the hands and nailbeds and are genomically distinct from cutaneous melanoma. Recent research on acral melanoma has revealed significant insights into its genomic evolution patterns, explained Dr. Boris Bastian, University of California, San Francisco. He described a study focused on characteristic genomic alterations called “hailstorms,” which are complex changes in regions of chromosomes that contain specific genes existing in multiple copies (commonly referred to as being amplified). Many of these gene amplifications involve oncogenes or mutated genes that can potentially cause cancer. Through analysis of tumor samples representing different progression stages from 37 acral melanoma patients, researchers discovered that hailstorm patterns occur before acral melanoma becomes apparent and they stay the same as this subtype of melanoma grows and spreads throughout the body.

Dr. Bastian proposed several possible mechanisms for hailstorm formation. Acral melanomas typically develop in areas of the body subject to physical force, which might cause changes in the genetic material. Furthermore, and in early acral melanoma development, cells can double their entire genetic material, meaning that they have four sets of chromosomes instead of the normal two.

This makes the cells prone to dividing errors, leading to abnormal DNA structures that contribute to melanoma development.

Unlike cutaneous melanoma, telomerase (TERT) alterations appear to be the earliest oncogenic events in acral melanoma, typically through creating extra copies of this gene. Telomerase adds protective caps to the ends of chromosomes preventing them from shortening and causing cell death. Due to the TERT alterations observed, acral melanomas have longer protective DNA caps on the ends of their chromosomes than regular cutaneous melanomas. This might help stabilize damaged genomic material in acral melanomas by adding new protective caps to broken DNA ends, allowing cells to survive, rapidly divide, and eventually become cancerous.

The study also revealed significant heterogeneity within primary tumors. Changes (mutations) in a specific cellular pathway (called MAP kinase) occur after acral melanoma has already started to form, unlike the early-arising MAP kinase pathway mutations found in cutaneous melanoma. Dr. Bastian showed data that MAP kinase mutations may be present in certain areas of the tumor and not in others. Therefore, multiple cancer cell populations within the same tumor may be driven by different genomic changes. This genomic heterogeneity within the same tumor may explain why targeted treatments specific for MAP kinase alterations often don't work well in acral melanoma.

These genomic findings have important implications for diagnostic testing and identifying the appropriate targeted therapies for acral melanoma. The genomic evolution pattern in acral melanoma differs significantly from that of the more common cutaneous melanoma, potentially requiring different therapeutic approaches.

Better Understanding of Uveal Melanoma

Dr. Andrew E. Aplin, Thomas Jefferson University, focused on promising advancements in uveal melanoma research. Uveal melanoma is the most common eye tumor in adults and makes up around 4% of melanoma diagnoses. Metastasis to the liver is also quite common with uveal melanoma. Tebentafusp (KIMMTRAK), a type of immune therapy used to treat advanced uveal melanoma and Hepzato, a liver directed therapy for unresectable liver metastatic tumors, have recently been approved by the US Food and Drug Administration.

Thomas Jefferson University has a strong collaborative approach to uveal melanoma, which combines clinical expertise with laboratory research. Research teams are addressing questions across the spectrum of uveal melanoma, from genomic pathways to understanding tumor dormancy. This coordinated effort ensures that scientific discoveries move efficiently from the laboratory to clinical applications that can benefit patients.

Research has made great strides in understanding the genomic basis of uveal melanoma. Approximately 90% of uveal melanomas have mutations in the *GNAQ* and *GNA11* genes, which trigger complex cellular changes that contribute to cancer progression. In addition, alterations in chromosome 3 and damage to a gene called *BAP1* can make uveal melanoma more likely to spread and harder to treat. These genomic insights are helping researchers develop more targeted treatment approaches that address the specific biological drivers of uveal melanoma.

An interesting aspect of uveal melanoma is how cancer cells can spread early but remain dormant for years or decades before growing, particularly in the liver. To

Andrew Aplin, PhD - Thomas Jefferson University

study this challenging phenomenon, the Aplin lab has collaborated with the Aguirre-Ghiso group at Albert Einstein College of Medicine. This research team has developed unique "reporter models" using fluorescent markers to track dormant versus active cancer cells. After implanting the specially labeled uveal melanoma cells in mice, most remained dormant for up to three months. By four months, the majority were growing and increasing in number.

This research revealed important differences between dormant and active cancer cells. Dormant cells show downregulation of cell cycle genes, meaning the genes that control cell division are less active. Upregulation of the transcription factor called NR2F1, which regulates the expression of different genes, appears crucial for maintaining dormancy. When researchers reduced levels of NR2F1, cancer cells grew more effectively in the liver, forming larger tumors.

The YAP1/TAZ/TEAD signaling pathway is activated in proliferating cells and conversely this pathway was downregulated in dormant cells. Inhibiting this pathway with drugs called TEAD inhibitors was expected to slow the growth of proliferating uveal cells. TEAD inhibitors didn't work well by themselves against slowing the growth of uveal melanoma cells, but combining them with another type of drug (MEK inhibitors) slowed cancer growth. This research suggests that targeting multiple pathways simultaneously may be more effective than single-drug approaches, opening new possibilities for treatment of uveal melanoma reawakening in the liver.

Understanding Cancer Cell Diversity and Treatment Resistance

Dr. Junyue Cao, Rockefeller University, focused on developing tools to study drug resistance in cancer cells. Significant progress has been made in understanding why cancer cells behave differently within the same tumor and why some cells resist treatment while others do not. This diversity among cancer cells, called heterogeneity, is a major focus of current research and could lead to better treatments.

"This different cellular state will result in different cancer cell behaviors, from proliferation to metastatic capacity, and also results in different response rates to treatment," explained Dr. Cao. "We are interested in characterizing this heterogeneity and understanding how this cellular heterogeneity contributes to different responses to treatment."

New technologies have been developed to analyze millions of individual cancer cells within a tumor simultaneously. These advanced methods allow researchers to see differences between cells that weren't visible before. One exciting development is single-cell functional genomics, which helps identify why some cancer cells resist drug treatment and continue to grow. Using CRISPR technology,

Junyue Cao, PhD - Rockefeller University

The Cao lab has developed an affordable, efficient technique called PerturbSci, which can examine hundreds of thousands of cells from a tumor in a single experiment.

(L to R): Vito Rebecca, PhD - Johns Hopkins University School of Medicine, Kasey Couts, PhD - University of Colorado, Keiran Smalley, PhD - Moffitt Cancer Center, and Joan Levy, PhD - Melanoma Research Alliance

researchers have demonstrated how some cancer cells escape treatment with drugs targeting the *BRAF* mutation. Although these drugs inhibit many cancer cells within a tumor, certain cells develop resistance, which can cause the cancer to return.

One exciting development is single-cell functional genomics, which helps identify why some cancer cells resist drug treatment and continue to grow. However, current single-cell functional genomics technology is quite expensive and time consuming, limiting its usefulness in research. To address this limitation, the Cao lab has developed an affordable, efficient technique called PerturbSci, which can examine hundreds of thousands of cells from a tumor in a single experiment. This method reveals which genes are active in resistant cells and how these cells change over time when exposed to treatment. Many different genomic changes can cause drug resistance, but they often activate a smaller set of shared regulators. Targeting these shared regulators could overcome resistance caused by various genomic mutations.

Recent experiments in the Cao lab showed that combining existing *BRAF*-targeted drugs with treatments aimed at these shared regulators can significantly reduce drug resistance. When several regulators were targeted simultaneously, the effect was even more potent. "This confirms their potential function in driving drug resistance in melanoma cells," said Dr. Cao. This research represents an essential step toward developing more effective cancer treatments that can overcome drug resistance, potentially leading to better outcomes for patients with melanoma.

Rare Melanomas: Cracking the Code Through Collaboration

A panel discussion at the Melanoma Research Alliance's 2025 Patient Forum focused on the three rare melanoma sub-types, acral, mucosal, and uveal melanomas. Dr. Joan Levy, MRA, moderated the panel, which featured Dr. Vito Rebecca, Johns Hopkins University; Dr. Kasey Couts, University of Colorado; and Dr. Keiran Smalley, Moffitt Cancer Center.

In work that MRA has supported, Dr. Levy explained that research identified distinct genomic profiles in different melanoma subtypes, helping identify specific targets for drug development. To advance specific treatments for different melanoma subtypes to the clinic, MRA also created preclinical model catalogs on its website that can help researchers access models for rare melanomas in order to test specific drugs. Furthermore in 2022, MRA became the sponsor of the "RARE Registry," a direct-to-patient platform where people with acral, cutaneous, and mucosal melanoma can share their experiences and disease journeys by answering questions in specific surveys. In return, participants of RARE have access to aggregated, de-identified participant data about their condition, receive clinical trial alerts, and contribute to data from which researchers and clinicians can learn more about these melanoma subtypes, including the patient perspective. The registry was inspired by patient advocates and later joined by medical and scientific advisors to ensure that RARE remains true to its mission. New features of RARE will soon expand to include

Vito Rebecca, PhD - Johns Hopkins School of Medicine

capturing more clinical information through electronic medical record integration and the ability for patients to have their tumor and normal tissues sent to a centralized biorepository. The collected samples will be shared with researchers worldwide and lead to a better understanding of these rare and often more difficult-to-treat melanoma subtypes.

Dr. Rebecca said that at Johns Hopkins, his laboratory is investigating what causes acral melanoma, how it metastasizes, and why existing therapies for cutaneous melanoma do not work as effectively for patients with acral melanoma. He is testing different drugs and combinations of therapies in specialized mouse models that enables acral melanoma to grow on the foot pads of mice, recapitulating the anatomical location that it is found in human disease.

Dr. Couts shared that at Colorado University, researchers have built a skin cancer biorepository of tumor samples collected from patients with cutaneous melanoma as well as all rare melanoma subtypes. Using this tissue they have created 175 melanoma patient-derived xenograft (PDX) mouse models. This includes about 30 PDX models for mucosal melanoma that were derived from mucosal tumor tissue isolated from different anatomical sites. PDX models are laboratory mice with transplanted human cancer tissue that maintain the original tumor's genetic and biological characteristics. This allows researchers to study cancer biology and test potential treatments in living systems that closely mimic human disease. Mucosal melanoma, a rare cancer that develops in mucous membranes throughout the body, occurs in different anatomical locations, including the mouth, nasal cavity, ear canal, genital tract, and anorectal region, each of which has a unique microenvironment that influences tumor behavior and treatment response. The researchers are studying how these anatomically diverse mucosal melanomas differ genomically and may require different types of treatment. Dr. Couts emphasized the importance of collaboration and sharing of samples to advance research on these rare melanoma subtypes.

Dr. Levy noted, "We're going to have to study these rare subtypes collaboratively because one institution will not have enough samples to study a given rare melanoma — it will require samples from multiple institutions that will have to be shared by researchers."

Dr. Smalley focuses his research on uveal melanoma, with additional interest in acral melanoma. "We were struck by the enormous progress that we made in treating patients who had cutaneous melanomas," he explained, noting how bleak the outlook had once been. Progress has been slower for uveal melanoma, with two

Kasey Couts, PhD - University of Colorado

Keiran Smalley, PhD - Moffitt Cancer Center

> "We were struck by the enormous progress that we made in treating patients who had cutaneous melanomas."
>
> **DR. KEIRAN SMALLEY**

FDA approvals only in the last 3 years. His lab's research focuses on understanding why high-risk uveal melanomas metastasize, particularly to the liver, and investigating the immune environment of uveal melanoma. "It's not as immunogenic as cutaneous melanoma," he noted, possibly due to lower tumor mutational burden and the fact that "the eye is an immune-privileged site, and so is the liver." For acral melanoma, they're applying similar techniques to understand why patients "are not responding as effectively to the newly approved immunotherapies."

For acral melanoma, there has been "something of a renaissance" in the last 5 years after being understudied since its discovery. Recent research has revealed that acral melanoma has a different mutational landscape from cutaneous melanoma, with varying driver mutations at a variety of frequencies. "The sequence of mutational events is strikingly different between cutaneous and acral melanoma, essentially showing that it's a different disease altogether," said Dr. Rebecca. Melanocytes in the foot (where acral melanomas originate) have "completely different" properties than melanocytes in sun-exposed sites.

Mucosal melanoma research, which is described as "the rarest of the rare melanomas," has lagged behind other types of melanomas. Over the past 5 years, through global collaboration and sharing of samples, researchers have gathered enough mucosal melanoma samples to conduct significant genomic studies that distinguish mucosal from other melanoma subtypes. Clinical trials specifically for patients with mucosal melanoma, who were previously often excluded from research, have now been established.

Dr. Smalley noted that industry has been increasingly interested in uveal melanoma. Several clinical trials for uveal melanoma are ongoing at Moffitt Cancer Center, with diverse targets. The key to research progress in rare melanomas is collaboration and sharing resources and models. "We can learn so much by analyzing specimens," noted Dr. Smalley. Using advanced technology to study immune environments and genomic changes, researchers can share emerging data with each other to collectively identify new treatment targets from limited specimens.

Sapna Patel, MD - The University of Colorado

Industry Roundtable:

Harnessing Collective Power for Rare Melanomas

Despite significant advances in melanoma treatment over the past 15 years, patients with rare melanoma subtypes like acral, mucosal, and uveal melanoma often face barriers to accessing approved therapies and being included in clinical trials. Furthermore, researchers need the correct resources and tools to identify new and potentially distinct drug treatments more appropriate for these patients. To address these issues, during the 2025 Scientific Retreat, the Melanoma Research Alliance convened a roundtable discussion of approximately 35 representatives from industry, academia, and patient advocacy, which was co-chaired by Dr. Sapna Patel, University of Colorado and Dr. Joan Levy, MRA.

Drug Access for Rare Melanoma Subtypes

Dr. Patel kicked the roundtable off by asking "Once drugs are approved, how easy is it for us to utilize them in the rare melanoma populations?" This sparked discussion about whether drugs approved for "metastatic melanoma" — like the recently approved cellular based tumor infiltrating lymphocyte (TIL) therapy, Amtagvi, or the dual checkpoint inhibitor drug, Opdualag — should automatically be available for all melanoma subtypes. Several participants expressed hesitation about using treatments such as cellular therapies in rare subtypes like uveal melanoma without either sufficient data or robust efficacy data, even in a small number of patients. While combination immune checkpoint therapy has some precedent, more invasive treatments such as TIL therapy may require stronger evidence.

A central issue discussed was insurance denials for USA Food and Drug Administration (FDA)-approved melanoma drugs when used in rare subtypes. Dr. Patel explained that even when a drug is approved for metastatic melanoma, patients with rare subtypes often face insurance denials because these individuals weren't included in the original clinical trials that led to FDA approval. "We're talking about drugs that are approved for melanoma, but still, there seems to be a barrier getting it for the rare melanoma

Mark Luna-Vargas, PhD - SkylineDx and Graham Ferrier, PhD - Pfizer

population, for no reason other than insurance has said no." She emphasized that nothing about the FDA approval process or industry intentions aims to prevent use in rare melanoma subtypes. Layered on top of this are the appeals that clinicians go through with insurance companies to try to have insurance decisions reversed and be granted approval to treat patients with a rare melanoma with a particular approved drug.

Dr. Levy asked if foundations could play a role in assisting clinicians and their staff with these appeals. Dr. Patel suggested that foundations could have some template language and documents, such as a letter of medical necessity, available for clinicians to use for a particular drug appeal. In addition, foundations can secure industry assistance to get the medication for a patient. Dr. Levy noted that MRA has been asked to help in certain cases with some success in having a drug ultimately approved for a patient.

Dr. Graham Ferrier, Pfizer, offered a practical solution: "We've had a lot of success in running small investigator-sponsored trials, or partnering with academia, to produce data, and in some instances that data can be used to get recommendations within guidelines and compendia allowing some patients with rare cancer subtypes to get access to the treatment."

Dr. Jaymin Patel, Immunocore, noted two main challenges for treating rare melanoma: 1) access to novel therapies and 2) sufficient clinical data to assess activity relative to other therapies. Running clinical trials for rare diseases with small patient populations is challenging from a pharmaceutical industry perspective. One solution could be creating an intermediary system where doctors could access drugs from different companies (with perhaps a limit of 30 patients per drug), collect structured data on outcomes, and use that evidence to support broader access and potentially change treatment guidelines. There were suggestions that data collection and storage should not reside with companies or even government; it was more favorable to store the data with a neutral third party like foundations.

Industry representatives acknowledged that even small studies could generate enough data to secure National Comprehensive Cancer Network (NCCN) guideline inclusion, which insurance companies typically recognize. This approach was identified as more practical than attempting to include rare subtypes in large Phase 3 trials. Dr. Alexander Shoushtari, Memorial Sloan Kettering Cancer Center said, "You don't need that many patients to broaden, just thirty or so. Then you have the data and the access (to the drug). Patients are happy, doctors are happy."

Clinical Trial Design and Inclusion

Dr. Sapna Patel noted that while the melanoma field has made tremendous progress, there has been a tendency

Jaymin Patel, MA - Immunocore

to test investigational drugs in cutaneous melanoma first and wait until later to attempt to use them in rare subtypes. Dr. Shoushtari challenged pharmaceutical companies to be more inclusive in their approach to rare melanomas. He emphasized that patients with rare melanomas currently receive inadequate standard treatments, and if new drugs are effective, these patients deserve the opportunity to benefit from them, too.

The group discussed the challenges of including patients with rare melanoma subtypes in clinical trials. Many industry representatives cited concerns about these individuals potentially having worse outcomes that could negatively impact overall trial results.

Dr. Shoushtari challenged this perspective: "There is no scientific reason to exclude rare subtypes for certain therapies, particularly those with clear shared molecular targets with the more common cutaneous melanoma." He noted examples where patients with rare melanomas were arbitrarily excluded from trials despite sharing the same molecular targets.

Carving out a certain number of spots for patients with rare melanoma — especially where the drug target is justified in an early melanoma all-comers trial — makes sense to see if there is at least a signal in a rare subtype. Dr. Marlana Orloff, Thomas Jefferson University, explained, "That could give us the confidence to move forward with a dedicated trial in the subtype. Because the truth is that the uveal immunotherapies that have gone the distance started because there was a signal in a melanoma all-comers trial. That's the Tebe story [tebentafusp, specifically the branded name KIMMTRAK]. That's now the RP2 story. You know, that could be the Immatic story."

Dr. Patel described a clinical trial called Dual Anti-CTLA4 and Anti-PD-1 Blockade for Rare Tumors (DART). This approach, called a "basket trial", tests a given drug or drug combination in multiple cancers. In the DART basket trial, several rare cancers showed encouraging results with the treatment combination. If there is a rationale for certain drugs to be tested across different melanoma subtypes, a basket trial could be used. This type of trial has the potential to be run through government-funded initiatives and only relies on industry supplying the study drug.

The topic of including rare subtypes in Phase 3 studies, even in separate cohorts, was addressed. "We really want to design simple trials that answer the question for patients with rare melanoma subtypes, and for me, that's not tagging on a rare cohort to a Phase 3 clinical trial", said Dr. Rohit Lal from Merck. "There are just so many complexities when it comes to Phase 3 clinical trials, never mind what the FDA will look at. When you've got a Phase 3 clinical trial, then authorities from around the world

Alexander Shoushtari, MD - Memorial Sloan Kettering Cancer Center

are looking at that data. We really want simple clinical trials for these rare subtypes, and that for us is through independent, sponsored research (ISR)." Unfortunately, some companies only release the drug to study in an ISR after the drug is approved in a particular indication.

Dr. Orloff noted: "I think the other thing is that we've all seen in uveal patients enrolled on a random Phase 1 trial where there's been a signal in a few uveal patients that were treated, or even a uveal trial and there was a signal, and then the drug is abandoned. I have been keeping a running list and there are about twelve drugs that we were very excited about once upon a time for uveal melanoma, and for whatever reason, things did not move forward. I think there are drugs out there that have shown good signals that we need some sort of strategy for how to move forward and try to resurrect these in some way."

Laboratory Science, Model Systems, and Collaboration

The discussion shifted to preclinical research needs. Dr. Silvio Gutkind, University of California, San Diego, highlighted the need for better development and sharing of model systems: "We have cell lines. It would be ideal to collaborate with our clinicians who see patients in developing organoids, new cell lines, and PDX [patient-derived xenografts] mouse models and most importantly, sharing the models in the research community. We are lacking a lot of that collaboration."

Keiran Smalley, PhD - Moffitt Cancer Center

He emphasized that drug combinations are likely necessary to effectively treat rare melanomas, which requires greater collaboration between pharmaceutical companies: "It's doubtful any drug will work on its own. It would likely be a drug combination because we need to target the primary target and certainly compensatory mechanisms leading to resistance, as they are activated right away."

Dr. Keiran Smalley, H. Lee Moffitt Cancer Center and Research Institute, echoed these points: "For those interested in some of the rarest subtypes, we have human uveal or acral/mucosal cell lines. The thing we lack is some kind of syngeneic mouse model." A syngeneic mouse model maintains a fully functioning immune system, better representing what happens in human patients and offers the ability to test combination approaches with immune-targeting drugs.

Dr. Smalley also called for more formal research collaboration: "This research community is relatively small. We all know who we are, but I'm unsure if we've ever collaborated formally." He suggested dedicated working meetings at international conferences to facilitate interactions among researchers and also standardization of cell lines to facilitate comparison of results across research groups.

Since mucosal melanoma can arise in the mucosal linings of different tissues, Dr. Ravi Amaravadi, University of Pennsylvania, highlighted the heterogeneity challenge even within a specific rare subtype. There is substantial heterogeneity in the mucosal population, particularly regarding the origin of the melanoma. This complexity makes it challenging to ensure preclinical research findings generated in specific mucosal models are relevant to the broader patient population with mucosal melanoma.

Dr. William Harbour, University of Texas Southwestern Medical Center, highlighted another challenge: "We have a list of high-value targets that we've described and identified…but it doesn't necessarily translate to industry. We must fit what industry is doing into the uveal context, rather than saying 'here are the high-value targets."

Richard Carvajal, MD - Northwell Health

He noted that some of the most promising therapeutic targets identified in academic labs don't align with industry priorities.

He also emphasized the importance of correlative biomarkers: "Even with the best organoids and the best mouse models, I suspect that no more than 10% to 20% of preclinical compounds tested with encouraging data will show efficacy in patients." He noted that while industry often supports clinical trials, they rarely fund the biomarker studies needed to understand trial results, especially when response rates are low.

> "For those of us who treat rare subtypes of certain oncologic diseases, it doesn't feel rare to us. This is our everyday, and it feels really good to have so many stakeholders at the table."
>
> **DR. SAPNA PATEL**

Dr. Levy shared several MRA initiatives to address these gaps:

- A virtual catalog on MRA's website listing available cell lines and PDX models to share for acral and mucosal melanoma, with plans to expand to uveal melanoma.
- An upcoming Request for Applications to support the development and characterization of rare melanoma preclinical models, with a requirement that the models be shared with the research community.
- A direct-to-patient registry for patients with acral, mucosal, and cutaneous melanomas (with a separate registry for uveal melanoma available through A Cure In Sight).

Dr. Sapna Patel concluded: "For those of us who treat rare subtypes of certain oncologic diseases, it doesn't feel rare to us. This is our everyday, and it feels really good to have so many stakeholders at the table."

She emphasized that organizations like MRA are well-positioned to help link institutions together, noting that the sites that can enroll patients with rare melanomas may not always be the same ones that can perform the necessary scientific analyses. The meeting highlighted the significant challenges faced by rare melanoma subtypes and concrete steps that could improve access, research, and ultimately better outcomes for these patient populations.

Lynn Schuchter, MD - University of Pennsylvania

Science Fuels the Future:

Transforming Melanoma Treatment and Survival

Melanoma treatment is undergoing a paradigm shift from traditional postsurgical (adjuvant) approaches to therapy given before a surgical procedure (neoadjuvant). Advances in neoadjuvant therapy are associated with the ability to assess the effectiveness of a treatment in a short period of time and then tailor subsequent treatments to the individual patient. Melanoma patients today have more options than ever before: with approved immunotherapies including different types of immune checkpoint inhibitors, oncolytic viral therapy, and the first tumor-infiltrating lymphocyte (TIL) cellular therapy. Promising new therapeutic approaches are being tested in the clinic including personalized cancer vaccines, different immunotherapy combinations, T-cell receptor (TCR)-based approaches, and improved oncolytic viruses. Researchers continue to develop better ways of selecting the most appropriate treatments for individual patients through use of biomarkers, emphasizing the importance of understanding the biological mechanisms behind treatment responses and resistance.

Treatment Revolution: How Neoadjuvant Approaches Are Reshaping Melanoma Management

Dr. Lynn Schuchter, Director of the Tara Miller Melanoma Center at the University of Pennsylvania and former president of the American Society of Clinical Oncology, provided a keynote lecture at the Retreat focused on the evolving landscape of melanoma treatment. She illustrated the growing shift from traditional adjuvant to neoadjuvant therapy. Adjuvant therapy typically involves surgery followed by one year of treatment with either single-agent immunotherapy or targeted therapy for *BRAF*-mutated melanoma. These approaches have demonstrated a 50% to 60% reduction in recurrence risk and prolongation of relapse free survival at 5 years of follow-up although without proven overall survival benefit.

Besides shrinking tumors before surgery and facilitating treatment personalization, the emerging neoadjuvant approach provides valuable research specimens for

translational studies, and it may be more effective due to the presence of more tumor antigens for the neoadjuvant treatment to react to than giving treatment in the adjuvant setting once the tumor is removed. Research on neoadjuvant therapy is ongoing and evidence from clinical trials to support its use is still growing. One such example is the randomized Phase 3 NADINA trial, in which optimized ipilimumab/nivolumab administered before surgery was compared with standard adjuvant nivolumab after surgery. In the NADINA study, patients receiving two cycles of neoadjuvant treatment with major pathological responses (no evidence of tumor in the surgical specimen or less than 10% tumor cells remaining) received no additional adjuvant therapy after surgery, which was clinically innovative and could improve the patient's experience. Results from the trial showed promising event-free survival in Stage III melanoma patients receiving neoadjuvant therapy.

Pathologic response assessment in melanoma has become critical, with responses categorized based on the percentage of viable tumor cells that remain after a patient has been treated, and it strongly correlates with better outcomes. This paradigm shift has significant implications for various specialists. Pathologists must now follow protocols collecting very specific criteria for specimens from patients who receive neoadjuvant therapy. Surgeons are encouraged to initially perform biopsies rather than immediately remove the whole tumor. This allows for neoadjuvant treatment to be considered, and for some patients, it may mean less extensive surgery after all. Medical oncologists face decisions about treatment duration, either to deescalate therapy for patients who show good pathological response or even escalate or switch therapies for patients who do not. Safety considerations also remain essential, as results from the Nadina trial with neoadjuvant combination immunotherapy (ipilimumab/nivolumab) showed higher toxicity rates compared to the single-agent nivolumab adjuvant approach. The field is rapidly transitioning toward considering neoadjuvant approaches for appropriate patients. Success with this new approach requires close collaboration between medical oncologists and surgeons, careful patient selection, and ongoing clinical trials to further refine how neoadjuvant therapy can be tailored to patients.

Melanoma Research Today: Progress, Challenges, and the Road Ahead

Dr. Allison Betof Warner, Stanford University, detailed the remarkable progress in melanoma treatment while acknowledging the challenges ahead. She drew a stark contrast between past and present: "If you came to one of our clinics anywhere in the 1990s, the early 2000s… the standard of care was to get chemotherapy, and the average survival that we would quote to a patient with advanced melanoma was about 6 months."

The introduction of immune checkpoint inhibitors (ICIs) transformed treatment outcomes dramatically. Combination therapies extended median overall survival from 6 to 72 months, a transformation Dr. Betof Warner called "monumental in our field." Yet she emphasized, "we still have a long way to go."

Dr. Betof Warner shared decade-long melanoma-specific survival data for patients treated with immune checkpoint inhibitors. The CheckMate 067 study revealed that 43% of patients receiving ipilimumab plus nivolumab combination therapy were alive at the 10-year mark, including 52% with melanoma-specific survival at 10 years. "It is a huge milestone, but not nearly what we want it to be as we want it to be 100% of patients," said Dr. Betof Warner. She noted that patients who reached the 3-year mark without disease progression had excellent longer-term prospects of being disease free at 10 years.

Melanoma has one of the highest risks for spreading to the central nervous system — such as the brain — among common solid cancers. Dr. Betof Warner presented encouraging updated data very recently published from the ABC trial testing ipilimumab plus nivolumab combination therapy versus nivolumab alone in melanoma patients with asymptomatic brain metastases. The results showed a median overall survival (OS) of 64 months—more than 5 years—in patients with brain metastases. This represented what Dr. Betof Warner called a huge step forward from the previous 4-month median OS.

The U.S. Food and Drug Administration (FDA) approval of tumor-infiltrating lymphocyte (TIL) therapy (lifileucel) in 2024 marked another milestone as the first cell therapy

Allison Betof Warner, MD, PhD - Stanford University

> Combination therapies **extended median overall survival from 6 to 72 months**, a transformation Dr. Betof Warner called "monumental in our field."

for solid tumors. This one-time treatment showed promising results in patients who had progressed on standard therapies, with a 78% disease control rate (measuring tumors shrinking and tumors that are not growing but stable).

Dr. Betof Warner described an innovative small pilot study of modified TIL therapy for melanoma brain metastases, which showed encouraging safety and initial efficacy despite the challenging nature of treating these patients. Looking toward the future, she highlighted ongoing work in rare melanoma subtypes, novel therapeutics including oncolytic viruses and cancer vaccines, advanced cell therapies, and improved approaches to managing treatment toxicity.

Concluding with a patient-centered vision emphasizing early detection, she said, "Please put me out of work. I can happily find something else to do if we can detect melanoma early, and we don't need as many advanced treatments."

Beyond Today: Emerging Melanoma Treatments

Dr. Omid Hamid, Cedars-Sinai the Angeles Clinic and Research Institute, acknowledged treatment advances and highlighted innovative treatment approaches using different types of immunotherapies. With the success of new treatments, patients with advanced or metastatic melanoma are now surviving for many years. He also pointed out that what we have discovered in melanoma has become standard of care in many other solid cancers.

Dr. Hamid discussed using circulating tumor DNA and gene expression profiling to identify high-risk patients earlier. This research aims to determine which patients benefit from treatment after surgery to lower the chance of their melanoma progressing. "We have now begun identifying patients who need treatment earlier, who don't respond to anti-PD-1 (programmed cell death protein 1) and are the primary resistant population and may need other types of therapies," he said.

Several clinical trials of therapeutic vaccines that contain neoantigens—proteins specific to a patient's particular tumor are ongoing. These vaccines are individualized for each patient, and they can be less toxic than other forms of treatment for melanoma. In the Phase 2b KEYNOTE-942 trial, a vaccine that used technology similar to what was used to produce mRNA vaccines against COVID-19 was found to significantly reduce risk of melanoma recurrence when combined with pembrolizumab. It is now being studied in a large Phase 3 trial that has already enrolled all patients.

New combinations of immunotherapy drugs also show promise. A triple combination with LAG3-PD1-CTLA4 inhibitors (three different immune checkpoint inhibitors) had a 4-year survival rate 20 points greater than the standard two-drug combination of anti-CTLA4 and anti-PD-1. However, many patients still do not do well on immunotherapy and predictive biomarkers are in development to identify them earlier. Said Dr. Hamid, "We now understand that those patients need to be studied in a different fashion and that they need other options or other combinations to ignite their immune system."

> "Spending a few days walking among some of the smartest people in melanoma, but also walking with melanoma patients and their advocates, just reminds us of what really matters."
>
> **DR. HUSSEIN TAWBI**

Omid Hamid, MD - Cedars-Sinai the Angeles Clinic and Research Institute

He also described various approaches to enhancing T cell activity, including T cell receptor (TCR) based therapy targeting a tumor-specific protein called PRAME, (preferentially expressed antigen in melanoma). Early studies in solid cancers demonstrated responses in cutaneous melanoma as well as uveal melanoma. Other types of TCR approaches act as "matchmakers," bringing tumors expressing a specific protein and T cells together causing T-cell mediated tumor death. The PRISM-MEL-301 trial is testing whether combining IMC-F106C (a novel T cell redirecting therapy targeting PRAME) with nivolumab improves outcomes compared with standard treatment in patients with previously untreated metastatic melanoma.

New and improved oncolytic viruses — viruses injected into tumors causing shrinkage — are being tested in the clinic. One virus, RP1 in combination with the anti-PD1 drug, Nivolumab, in patients who failed prior anti-PD1 therapy, showed shrinkage of not only injected tumors but tumors not injected that have spread to distant organs like the liver. These results are now being confirmed in a larger Phase 3 trial. Furthermore, a next generation RP1, RP2, with features to enhance the immune system locally, is being tested in a Phase 2 trial in metastatic uveal melanoma in combination with Nivolumab.

Science Fuels the Future

Dr. Hussein Tawbi from MD Anderson moderated a panel discussion at MRA's Scientific Retreat by underscoring that "spending a few days walking among some of the smartest people in melanoma, but also walking with melanoma patients and their advocates, just reminds us of what really matters." Science is fueled by the desire and passion among the broad team of melanoma clinicians and researchers to bring additional treatments and ensure better outcomes for melanoma patients—the theme of this panel discussion.

Progress has been made in the last year to get the cellular tumor infiltrating lymphocyte (TIL) therapy to melanoma patients with the approval of lifileucel. Dr. Betof Warner enthusiastically shared that the infrastructure has been established at many leading academic centers and requires an experienced clinical team to be able to treat refractory melanoma patients with standard of care TIL. She went on to say clinicians are learning a lot as they treat patients, such as: Who are the right patients? What is the right timing of this treatment? The field needs to continually collect this critical information.

Panelist Dr. Jason Luke, University of Pittsburgh, pointed out that the melanoma field is one of the only ones that does not use biomarkers to stratify patients to receive immunotherapy such as immune checkpoint inhibitors. He emphasized the need for better patient selection through biomarkers, stating that "Who is going to be a great responder to anti-PD-1, or who's going to progress quickly are important questions to answer," because patients who won't respond well need to transition to different treatment options quickly.

(L to R): Taha Merghoub, PhD - Weill Cornell Medicine, Patrick Ott, MD, PhD - Dana-Farber Cancer Institute, Jason Luke, MD - University of Pittsburgh, Allison Betof Warner, MD, PhD - Stanford University, and Hussein Tawbi, MD, PhD - MD Anderson Cancer Center

Dr. Luke also mentioned the importance of HLA profiling. HLA is the genetic matching system that determines which patients can receive specific T cell receptor (TCR)-T cell therapies, because these treatments only work with certain HLA types. He argued that all patients with melanoma ought to be getting HLA profiling because "there's a whole wave of novel therapeutics that are coming forward that are going to be dependent upon this additional information needed to qualify for these treatments." TCR-T cells unlike TILs involve genetically modifying a patient's T cells so they can be more specific to recognize the tumor. Dr. Luke commented that one of the TCR-T cell therapies being tested works in more than half of the patients treated who have stopped responding to other treatments.

The potential of developing other immunotherapies such as cancer vaccines for melanoma that can generate a novel immune response that is potentially long lasting and diverse was discussed by Dr. Patrick Ott, Dana-Farber Cancer Institute. Dr. Ott said the key to improving cancer vaccines is finding tumor-specific antigens that can trigger immune responses. The field can sequence tumors and find mutations but faces uncertainty about which mutations to target for a vaccine approach and dealing with hundreds to thousands of potential mutations in a given melanoma tumor, with current selection algorithms being far from ideal.

Dr. Ott described advances in reducing vaccine manufacturing timelines to just a few days after tumor samples are received, which have made personalized approaches viable even in neoadjuvant settings. Tumor heterogeneity and clonal evolution make targeting multiple mutations necessary, giving personalized vaccines a significant advantage over approaches that target a single mutation (like *BRAF*). This personalized vaccine approach allows treatment for each patient's unique tumor profile rather than limiting it to specific mutations and is being tested in the clinic with encouraging results emerging from early clinical trials.

"Discovery medicine and science to improve melanoma treatments is critically needed," said Dr. Taha Merghoub, Weill Cornell Medicine. Dr. Merghoub discussed the importance of discovery medicine and science in improving melanoma treatments. He emphasized that to advance therapies (like cellular therapy and vaccines), researchers need to understand the biological mechanisms behind treatment responses and resistance. He argued that many clinical trials fail because they only measure clinical outcomes without investigating the underlying biological mechanisms. Despite the costs, conducting biological analyses alongside clinical trials is worthwhile because it can reveal biological activity even when clinical responses aren't observed, providing opportunities to enhance treatments.

The panelists agreed that it is also vital to study trials that have negative results, and in particular, try to determine why the treatment did not work. As Dr. Betof Warner commented, "Did the drug not hit the target, or did hitting the target not work?" The answer to that question will help researchers design better treatments and trials in the future. It takes a team of researchers and clinicians working together with patient advocates and foundations to ensure that necessary resources can be secured to address all critical questions that will fuel new therapeutics to patients.

The **MRA Melanoma > Exchange** Patient and Advocate Forum

MRA's **Melanoma > Exchange Patient and Advocate Forum**, held in-person in Washington DC and virtually on February 26th, 2025, brought together hundreds of melanoma patients, survivors, advocates, and their loved ones to provide lay-friendly, state-of-the-science education, promote collaboration, and provide networking opportunities across the melanoma community.

The Forum brought advocates together for both the in-person and simulcast program, with virtual attendees spanning the United States and 15 countries. Participants left with practical tips and strategies to get the most out of their care while navigating the challenges of melanoma diagnosis, treatment, and beyond.

Videos from the **2025 Melanoma > Exchange Patient and Advocate Forum** are available at **CureMelanoma.org/25ForumRecordings**

Alexandra-Chloé Villani, PhD - Massachusetts General Hospital

Balancing Benefit and Risk:

Managing Immunotherapy Side Effects in Melanoma

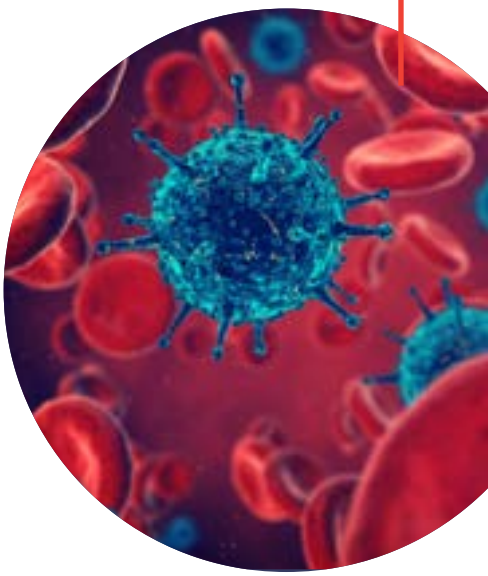
In recent years, immunotherapy has revolutionized melanoma treatment, offering new hope to patients with advanced disease. However, these powerful treatments can sometimes trigger immune-related adverse events (irAEs) that affect various organs. Understanding, predicting, and managing these side effects has become crucial in melanoma care.

> Understanding, predicting, and managing side effects of immunotherapy has become crucial in melanoma care.

The Immunotherapy Revolution

During her presentation at MRA's 2025 **Melanoma > Exchange Patient Forum**, Dr. Alexandra-Chloé Villani, a scientific researcher at Massachusetts General Hospital (MGH), shared valuable insights about the delicate balance between effective treatment and managing potential complications.

Certain types of Immunotherapies work by removing checkpoints, or "brakes," from the immune system, allowing them to recognize and attack cancer cells. Dr. Villani emphasized that this immunotherapy approach fundamentally differs from other therapies because it does not target anything specific to the tumor. Instead, it unleashes the body's own immune response. "This is a good news story," Dr. Villani reassured the audience.



"Since immune checkpoint inhibitors were approved for melanoma in 2011, this class of drugs has been approved for 109 indications across different cancer types."

Dr. Villani's presentation focused on how immune checkpoint inhibitors work and why they sometimes cause side effects. She explained that these immune checkpoint molecules typically serve essential protective functions in the body by:

- Slowing down immune responses to prevent damage to healthy tissues.
- Helping the immune system distinguish between the body's own healthy cells and foreign cells so it doesn't attack the healthy cells.
- Preventing excessive immune responses once infections or allergic reactions are resolved.

Melanoma cells can exploit these protective mechanisms by expressing molecules that tell T cells not to attack them. Checkpoint inhibitor drugs block this deception, allowing T cells to recognize and destroy cancer cells. However, by interfering with these protective mechanisms without a tool for modulation, these treatments can sometimes lead to side effects. Immunotherapy does not work for everyone, and different checkpoint inhibitors are often combined to improve their effectiveness.

Understanding Immune-Related Adverse Events

Depending on the dose and combination of immunotherapy drugs, up to 90% of patients on these therapies may develop some form of side effect. These can range from mild skin rashes to potentially life-threatening inflammation of organs such as the heart, lungs, or nervous system. Dr. Villani noted that side effects are becoming increasingly relevant as immunotherapy, such as immune checkpoint inhibitors, expands beyond treatment of metastatic disease to adjuvant and neoadjuvant settings. "The consideration of risk-to-benefit ratio is a whole different story if you are in a curative or preventive setting," she explained.

Dr. Villani shared the story of David, a 64-year-old businessman whose melanoma journey inspired her work and the work of her colleagues at MGH in this field. David initially responded well to immunotherapy for metastatic melanoma but later had severe complications. First, he developed pneumonitis (lung inflammation), then colitis (colon inflammation), and finally inflammation of his nerves. Despite aggressive treatment with steroids and other immunosuppressive medications, David's condition worsened, and he ultimately passed away. His autopsy revealed no remaining cancer cells, as the treatment had effectively eliminated his melanoma, but his organs showed extensive inflammation from an overactivated immune system.

This case highlighted several important aspects of managing immunotherapy side effects:

- The need for multidisciplinary care teams with expertise across different organ systems.
- The importance of developing better treatment solutions for side effects beyond broad immunosuppression.
- The necessity for more research to understand why some patients develop multiple different toxicities while others have none.

Timing and Types of Side Effects

Most checkpoint inhibitor immunotherapy-related side effects occur within the first six months of treatment, although some can develop later or be chronic. Dr. Villani presented data showing that different side effects tend to emerge at different time points:

- Skin reactions often occur first.
- Gastrointestinal and liver issues typically follow.
- Endocrine problems such as thyroid dysfunction may develop later.

Some side effects resolve completely with proper management, while others, particularly those that affect endocrine organs such as the thyroid or pancreas, may cause permanent damage requiring lifelong hormone replacement therapy. This underscores the importance of early detection of side effects and intervention.

Chronic side effects from immunotherapy, which can affect up to 43% of patients, were previously underreported for several reasons: They can occur months after treatment ends, early clinical trials focused on patients with limited survival time, side effects were

"...The future of improving efficacy is by mitigating these side effects, so that we can keep patients on these drugs safely for a longer time."

DR. ALEXANDRA-CHLOÉ VILLANI

often confused with other health problems, and until October 2024, there wasn't even an official medical billing code to track these problems in health care systems.

Building Solutions: The Severe Immunotherapy Complications Service

In 2017, Dr. Villani in parternship with Dr. Kerry Reynolds and colleagues established the MGH Severe Immunotherapy Complications (SIC) Service to address these challenges. This multidisciplinary team includes oncologists, specialists from various departments (gastroenterology, pulmonary, cardiology, etc.), nurses, and researchers collaborating to provide comprehensive care for patients who experience immunotherapy complications. The team also conducts research to better understand the root causes of these toxicities, with the goal of developing improved treatments to better prevent and manage them.

The service operates in both inpatient and outpatient settings with established protocols for patient care, research sample collection, and data analysis. Dr. Villani described how this integrated approach has improved patient outcomes and accelerated research efforts.

"It takes a village to make this happen," she explained, describing a case where over 14 specialists and researchers worked together to rapidly diagnose and treat a patient with muscle and joint inflammation. The team was able to not only provide excellent clinical care but also collect tissue samples, which led to important scientific insights.

Advancing Science Through Research

To improve the treatment of immunotherapy side effects, doctors need several key advances: tools to quickly distinguish side effects from opportunistic infection or melanoma progression, methods to predict which patients will develop specific side effects, and more targeted treatments that go beyond steroids (which can possibly reduce the melanoma-fighting response). Because these side effects do not appear in laboratory mice, researchers need partnerships with patients and families to collect samples and understand the root causes and ultimately develop better treatments that don't require stopping the life-saving melanoma immunotherapy.

Dr. Villani's work focuses on understanding the biological mechanisms behind different types of immune-related side effects. Her lab uses cutting-edge single-cell genomics technology to analyze affected tissues at high levels of detail.

She compared this approach to analyzing a fruit salad, where each piece of fruit represents an individual cell. "Traditional methods would be like blending the entire fruit salad into a smoothie and analyzing the average content." Using this new single-cell genomics technology is like emptying the bowl onto a table, organizing and regrouping each piece of fruit based on similarity, and examining its quality (healthy versus rotten).

This detailed analysis allows researchers to:

- Identify the specific types of immune cells that drive inflammation in each organ.

> "With this big data approach, we can define targets that could be followed up on in clinical trials."
>
> **DR. ALEXANDRA-CHLOÉ VILLANI**

- Determine whether similar mechanisms are at work across different types of toxicities.
- Discover potential biomarkers that might predict which patients are at risk to develop toxicities.
- Find targeted treatment approaches to address side effects without diminishing the anti-cancer effects.

Dr. Villani shared that her team has already made significant discoveries about the mechanisms driving heart inflammation (myocarditis) and colon inflammation (colitis) in patients receiving immunotherapy. They found that the biology driving anti-tumor responses differs from that causing heart inflammation, suggesting that it may be possible to separate the beneficial effects of treatment from the harmful ones.

"This work is really important in terms of setting the next phase," she noted. "With this big data approach, we can define targets that could be followed up on in clinical trials."

Looking Ahead: Challenges and Opportunities

Despite the progress made, significant challenges remain. Dr. Villani highlighted several areas where further work is needed:

- Establishing larger patient cohorts to validate findings and discover predictive biomarkers for patients at risk of developing side effects.
- Developing better classification systems and terminology to distinguish these side effects from anti-tumor immune response (i.e. treatment efficacy).
- Securing funding for research on toxicity management, which often receives less attention than developing new cancer treatments.
- Building partnerships between patients, clinicians, and researchers to advance understanding.

"I personally believe that the future of improving efficacy is by mitigating these side effects, so that we can keep patients on these drugs safely for a longer time," Dr. Villani stated.

She emphasized that patients could play a crucial role by reporting symptoms promptly to their health care providers. Early intervention is key, because side effects are generally easier to manage when caught early.

The Road Forward

Dr. Villani concluded by reminding the audience that despite the challenges, immunotherapy — in particular immune checkpoint inhibitors — represents a tremendous advancement in cancer treatment. The goal is to make these life-saving treatments safer and more effective for more patients.

The field is making significant progress toward understanding and managing immune-related side effects through integrated clinical care, innovative research approaches, and collaboration between patients and medical professionals. This work promises to further transform the treatment landscape for melanoma and many other cancers, allowing more patients to benefit from these powerful therapies with fewer complications.

By raising awareness about immunotherapy side effects and supporting research efforts to address them, the melanoma community can help ensure that it reaches its full potential as a cornerstone of cancer treatment.

(L to R): Lynn Schuchter, MD - University of Pennsylvania, Toni English, Robin Zimmerman, Brittanny Groover - Patient Advocates, and Stephanie Kauffman - MRA

Healing Connections: Navigating Melanoma as a Team

When facing a melanoma diagnosis, the strength of one's relationship with their medical team is crucial. At the Melanoma Research Alliance's 2025 **Melanoma > Exchange Patient Forum**, a panel session moderated by MRA's President and Chief Operating Officer Stephanie Kauffman explored the critical bonds between patients with melanoma, their caregivers, and the medical professionals they interact with throughout their journey. Each panelist shared unique perspectives on building effective partnerships to ensure optimal patient outcomes.

The panel discussed melanoma care and examined relationships between patients, caregivers, and medical providers. The participants shared experiences about diagnosis, treatment, and communication challenges. Their insights emphasized the importance of healthcare teams working with patients to improve outcomes.

Personal Pathways in the Melanoma Experience

Brittanny Groover, melanoma survivor and patient advocate, shared her experience being diagnosed with stage III disease in 2021 after missing a skin check during the pandemic. Her journey was "rocky" with her initial medical team, mainly when dealing with side effects from immunotherapy treatment. This experience underscored the importance of advocating for oneself and finding the right medical team.

Robin Zimmerman, survivor and sun safety advocate, brought dual perspectives as both a caregiver and patient. He first supported his wife Bonnie, who was diagnosed with melanoma in 2004 and eventually passed away in 2008 after the cancer metastasized to her liver. Later, Zimmerman became a patient himself when a spot on his back was found to be an early-stage melanoma. His experience highlighted the stark contrast between late- and early-stage diagnoses. He has become a dedicated advocate for sun safety and early detection, continuing his melanoma education for over 17 years.

Toni English, mucosal melanoma survivor and rare melanoma advocate, shared her journey with the rare subtype, diagnosed in 2015 with an initial prognosis of just six months to live. After surgery and radiation, she was declared cancer-free, only to discover a year later that the cancer had returned with metastases in her brain, lungs, and kidney. After unsuccessful immunotherapy treatments, she joined a clinical trial for tumor-infiltrating lymphocyte (TIL) therapy in 2018. Six months later, all her tumors had disappeared. English has now been

Lynn Schuchter, MD - University of Pennsylvania

> "It's really important that we're sitting, looking at our patients, talking, asking questions and giving them time to answer."
>
> **DR. LYNN SCHUCHTER**

cancer-free for over six years and dedicates her time to advocating for and supporting those newly diagnosed with mucosal melanoma and sharing her expertise on MRA's RARE Melanoma Registry as a patient advisor.

Dr. Lynn Schuchter, medical oncologist and Director of the Tara Miller Melanoma Center at the University of Pennsylvania, emphasized the importance of teamwork in treating this complex disease. She noted that treating melanoma requires a team to take care of the patient and that collaboration among medical professionals is crucial for achieving the best outcomes.

Complexity of Diagnosis and Treatment

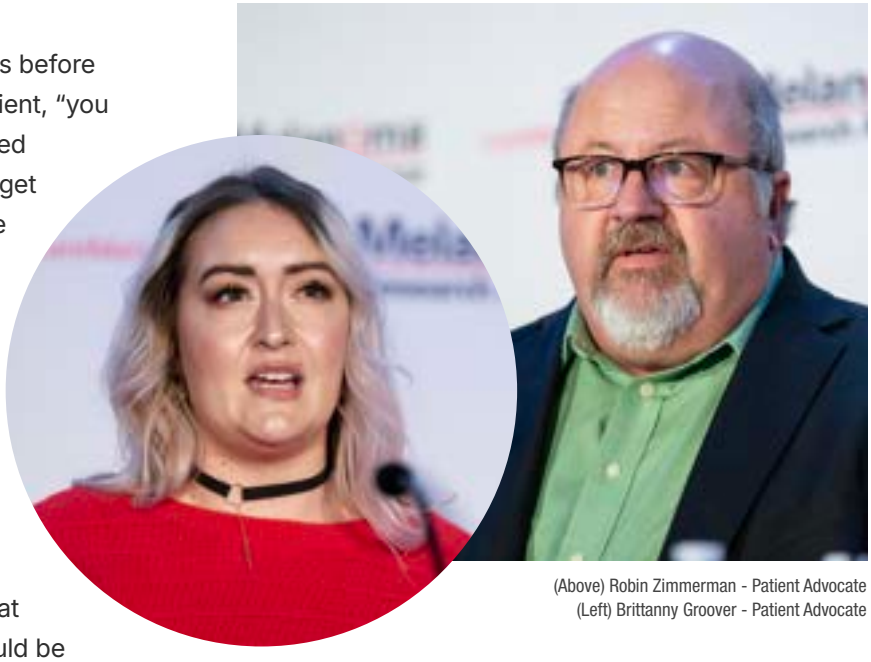
A significant portion of the discussion addressed the common misconception that melanoma is "just skin cancer." Zimmerman admitted that when his wife was first diagnosed 20 years ago, they thought the same, due to a lack of information and education at the time. Groover shared how she now uses her visible scar to start conversations about melanoma's seriousness. English highlighted the challenges associated with rare subtypes of the disease that many doctors have never heard of, which makes education and patient advocacy even more critical.

Dr. Schuchter addressed the challenges providers face when explaining complex diagnoses and treatment options while trying to build patient trust. She emphasized the importance of taking time to talk with, educate, and listen to patients. "It takes time to fully understand the diagnosis," she noted, stressing that electronic medical records can sometimes hinder genuine communication. "It's really important that we're sitting, looking at our patients, talking, asking questions and giving them time to answer." She also advised that creating the right treatment plan takes several visits.

The panelists shared advice based on their own treatment journeys. Groover discussed her experience with side effects. "I developed colitis, which is a potential side effect of immunotherapy, and it ended up going untreated for six months," she explained. Her oncologist at the time dismissed her concerns, but Groover wished she was "brave enough to stand up for myself." Groover stressed the importance of advocating for oneself and not being reluctant to seek a second opinion or switch doctors. "You can find a new doctor if things are not working out," she emphasized.

Toni English - Patient Advocate

English urged patients to write down questions before an appointment and emphasized that as a patient, "you are part of the care team." She also encouraged patients not to be afraid to ask questions and get a second opinion, particularly if they have rare cancers. Dr. Schuchter reminded attendees to take advantage of the whole care team, noting that nurses, nurse practitioners, pharmacists, and social workers are all valuable resources for information and support.



(Above) Robin Zimmerman - Patient Advocate
(Left) Brittanny Groover - Patient Advocate

The panelists also discussed the importance of involving the right medical professionals in melanoma care. Dr. Schuchter emphasized that all healthcare providers treating a patient should be informed about their cancer diagnosis, because it could impact treatment decisions. Zimmerman added that even a patient's dentists should be informed about their cancer diagnosis, because they need to know what medications they are taking, and may not have access to electronic health records to see their treatment plan. English highlighted the value of tumor boards, where different specialists collaborate to determine the best treatment approach.

Crucial Role of Caregivers

The discussion also covered the crucial role of caregivers. Zimmerman shared how he quietly took over household tasks while his wife was ill to prevent her from feeling like a burden. English emphasized how the mucosal melanoma community "puts caregivers on a pedestal" — recognizing the emotional weight they carry while trying to remain strong for their loved ones. "Having a good caregiver allows you to be the patient," English noted, adding that it's equally important for caregivers to have opportunities to decompress and receive support themselves. Dr. Schuchter acknowledged the importance of caregivers and agreed that they also need attention and support because they manage complex situations and can become emotionally and physically exhausted in the process. Identifying who makes up a support team, be that a spouse, friend, or child, and understanding what both the patient and the caregivers need is essential for effective care.

How to Handle Digital Demands

The panel also addressed the impact of digital healthcare tools such as patient portals and telemedicine on patient quality of life. Dr. Schuchter acknowledged both the benefits and challenges, noting that while immediate access to test results is beneficial, it can also be stressful when patients see results before providers have a chance to review them. An additional layer of complexity is that sometimes machine learning does the first review of test results, and this needs to be disclosed to patients.

Groover strongly advocated for digital tools, stating that the availability of telehealth and electronic records is a requirement for her when choosing doctors: "I need all of my medical information right here 24/7/365." English appreciated having access to test results before appointments to research unfamiliar terms and prepare questions. Zimmerman also said that telehealth appointments are surprisingly comfortable, noting that conversing through a screen made it easier to discuss health concerns and take notes during the conversation. Access to test results and other data is a valuable tool to most patients, but clinical interpretation, and in-person consultation are equally important.

In closing remarks, panelists agreed that every patient journey is different and emphasized the importance of finding what works best for the individual through open and honest communication with their care team.

POWERING PROGRESS *together*

MRA Scientific Retreat

February 26-28, 2025

AGENDA

Wednesday, February 26

4:00-8:00pm	Retreat Registration opens
5:30-6:00pm	Sponsor Toast/Reception
6:00-7:30pm	Opening Reception

Thursday, February 27

6:30am-6:00pm	Registration
7:00-8:45am	General Breakfast
7:00-8:45am	Young Investigators Breakfast (by invitation)
9:00-9:15am	**OPENING REMARKS DAY 1** **Marc Hurlbert**, MRA CEO **George Mannes**, Patient Advocate **Joan Levy**, MRA CSO
9:15-9:45am	**KEYNOTE LECTURE 1 :** **Lynn Schuchter**, University of Pennsylvania *Shaping the future of melanoma care: neoadjuvant and adjuvant therapy*
9:45-11:50am	**Scientific Session 1: *Strategies to Identify and Overcome Therapeutic Resistance*** Chair: **John Kirkwood**, University of Pittsburgh
9:45-10:10	**Ana C. Anderson**, Brigham and Women's Hospital *Dynamic immune landscapes during melanoma progression reveal a role for endogenous opioids in promoting CD8+ T cell dysfunction*
10:10-10:30	**Benjamin Izar**, Columbia University *Dissecting and overcoming cancer immune evasion due to defects in the CD58-CD2 axis*
10:30-10:50	**Barbara Bedogni,** University of Miami *Overcoming immunotherapy resistance by selective inhibition of Notch1*
10:50-11:05	Break
11:05-11:30	**Marcus Bosenberg**, Yale University *Targeting epigenetics to enhance anti-melanoma immunity*
11:30- 11:50	**Yana Najjar**, University of Pittsburgh *The role of hypoxia in the evolution of melanoma metastases*
11:50-12:00	**Amie Bunker**, CDMRP Melanoma Research Program *2025 Funding Opportunities from the CDRMP Melanoma Research Program*

12:00-1:20pm **Networking Lunch and General Roundtables**

- RARE Melanoma Patient Registry
- Genomics — Role of genetics, genomics & epigenetics; single cell technologies
- Biomarkers — 'liquid biopsy' ctDNA, and tumor biomarkers
- irAE — understanding immune-related adverse events
- Brain metastasis, leptomeningeal disease, in-transit disease, and tumor dormancy
- Neoadjuvant and adjuvant therapy
- Cell based therapy (CAR-T, NK cells, TILs)
- Targets & drug discovery for new treatments
- Clinical trials — patient recruitment and decentralized trials
- Tumor microenvironment and microbiome
- Diversity — Women & Underrepresented groups in melanoma research & care
- Uveal melanoma (Rayburn Room)
- Early Detection & Diagnosis (AI, imaging, machine learning)
- Vaccines and intralesional therapies

1:30-2:45pm **Scientific Session 2: *Rare Melanomas***
Chair: **Keiran Smalley**, Moffitt Cancer Center

1:30-1:55 **Boris C. Bastian**, University of California, San Francisco
Mutational mechanisms in acral melanoma

1:55-2:20 **Genevieve Boland**, Massachusetts General Hospital
Understanding the biology of mucosal melanoma

2:20-2:45 **Andrew E. Aplin**, Thomas Jefferson University
Mechanisms of dormancy and growth in uveal melanoma

2:45-3:00 Break

3:00-4:00pm **Scientific Session 3: *New Technologies to Identify Diagnostic and Prognostic Biomarkers***
Chair: **Marc Hurlbert**, MRA Chief Executive Officer

3:00-3:20 **Peter K. Sorger**, Harvard University
Investigating mechanisms of melanoma initiation and progression to improve diagnosis and risk assessment

3:20-3:40 **Janis M. Taube**, Johns Hopkins University
Analytic and clinical validation of a 6-plex mIF assay for predicting response to anti-PD-1-based therapy

3:40-4:00 **Panel Discussion**

4:00-5:00pm **Scientific Session 4: *Highlighting Early-Stage Investigators***
Chair: **Ryan Sullivan**, Massachusetts General Hospital

4:00-4:15 **Debattama Sen**, Massachusetts General Hospital
Reviving tumor immunity: epigenomic approaches to reprogram T cell exhaustion

4:15-4:30 **Junyue Cao**, Rockefeller University
Functional characterization of genetic regulators driving melanoma drug resistance via single-cell high-content CRISPR screening

4:30-4:45	**Pietro Berico**, New York University *A targetable developmental program co-regulates angiogenesis and immune evasion*
4:45-5:00	**Inna Smalley**, Moffitt Cancer Center *The unique microenvironment of CNS melanomas promotes tumor growth, drug resistance, and immune suppression*
5:00-5:15pm	**Concluding Remarks**
5:15-6:15pm	**Poster Session and Reception**

Friday, February 28

6:30-10:00am	Registration
7:00-8:50am	General Breakfast
7:00-8:50am	Industry Roundtable Breakfast (by invitation only)
9:00-9:05am	**OPENING REMARKS DAY 2** **Sara DiNapoli**, MRA Director, Scientific Programs
9:05-9:35am	**KEYNOTE LECTURE 2 :** **Martin McMahon**, University of Utah *The past, present, & future of pathway-targeted therapeutics in melanoma — a personal perspective*
9:35-11:20am	**Scientific Session 5: *Novel Targets in Melanoma*** Chair: Rhoda Alani, Boston University
9:35-10:00	**Fátima Gebauer**, Centre for Genomic Regulation *CSDE1 as a potential biomarker and therapeutic target in melanoma*
10:00-10:20	**Vivek Unni**, Oregon Health & Science University *Linking melanoma and Parkinson's disease: The role of alpha-synuclein in DNA double-strand break repair*
10:20-10:35am	Break
10:35-10:55	**Deanna L. Benson**, Icahn School of Medicine at Mount Sinai *A strategy to identify shared risk for melanoma and Parkinson's*
10:55-11:20	**Rotem Karni**, Hebrew University of Jerusalem *Targeting RNA processing to enhance mucosal melanoma immunotherapy*
11:20am-12:30pm	**CLOSING PANEL** **Science Fuels the Future: Sustaining the Virtuous Cycle in Drug Development** *Chair:* **Hussein Tawbi**, MD Anderson Cancer Center *Panelists:* **Allison Betof Warner**, Stanford University School of Medicine **Jason Luke**, University of Pittsburgh **Patrick Ott**, Dana-Farber Cancer Institute **Taha Merghoub**, Weill Cornell Medicine
12:30-12:35pm	**Concluding Remarks** **Stephanie Kauffman,** MRA President & COO
12:35-1:35pm	Lunch and departures

Melanoma Exchange
Patient & Advocate Forum

February 26, 2025

AGENDA

11:30-11:45am	**Registration & Check In**
11:45-1:00pm	**Networking Lunch**
1:00-1:10pm	**OPENING REMARKS** *Stephanie Kauffman*, MRA President & Chief Operating Officer *Dana Deighton*, MRA Director, Communications & Engagement
1:10-2:00pm	**Melanoma Research Today: Progress, Challenges, & the Road Ahead** *Allison Betof Warner, MD*, Stanford Medicine
2:00-2:35pm	**A Delicate Balance: Tackling Treatment Resistance & Adverse Effects** *Alexandra Chloe Villani, PhD, Massachusetts General Hospital*
2:35-3:20pm	**Prediction & Prevention: The Power of AI & the Power of the Eye** *Allan Halpern, MD*, Memorial Sloan Kettering Cancer Center
3:20-4:05pm	**Beyond Today: Emerging Melanoma Treatments** *Omid Hamid, MD,* The Angeles Clinic and Research Institute, Cedars-Sinai
4:05-4:50pm	**Rare Melanomas: Cracking the Code through Collaboration** Moderator: *Joan Levy, PhD*, MRA Chief Science Officer Panelists: *Kasey Couts, PhD*, University of Colorado, *Vito Rebecca, PhD,* Johns Hopkins University *Keiran Smalley, PhD*, Moffitt Cancer Center
4:50-5:50pm	**Partners in Care: Navigating the Patient, Caregiver & Medical Team Relationship** *Moderator: Stephanie Kauffman,* MRA President & COO Panelists: *Lynn Schuchter, MD,* Penn Medicine *Toni English, Brittanny Groover, & Robin Zimmerman*, Patient Advocates
5:50-6:00pm	**Closing Remarks** *Renee Orcione,* MRA Associate Director, Communications & Engagement
6:00-7:30pm	**Patient, Advocate, & Researcher Reception** *Penn Avenue Terrace — Lobby Level*

POWERING PROGRESS *together*

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