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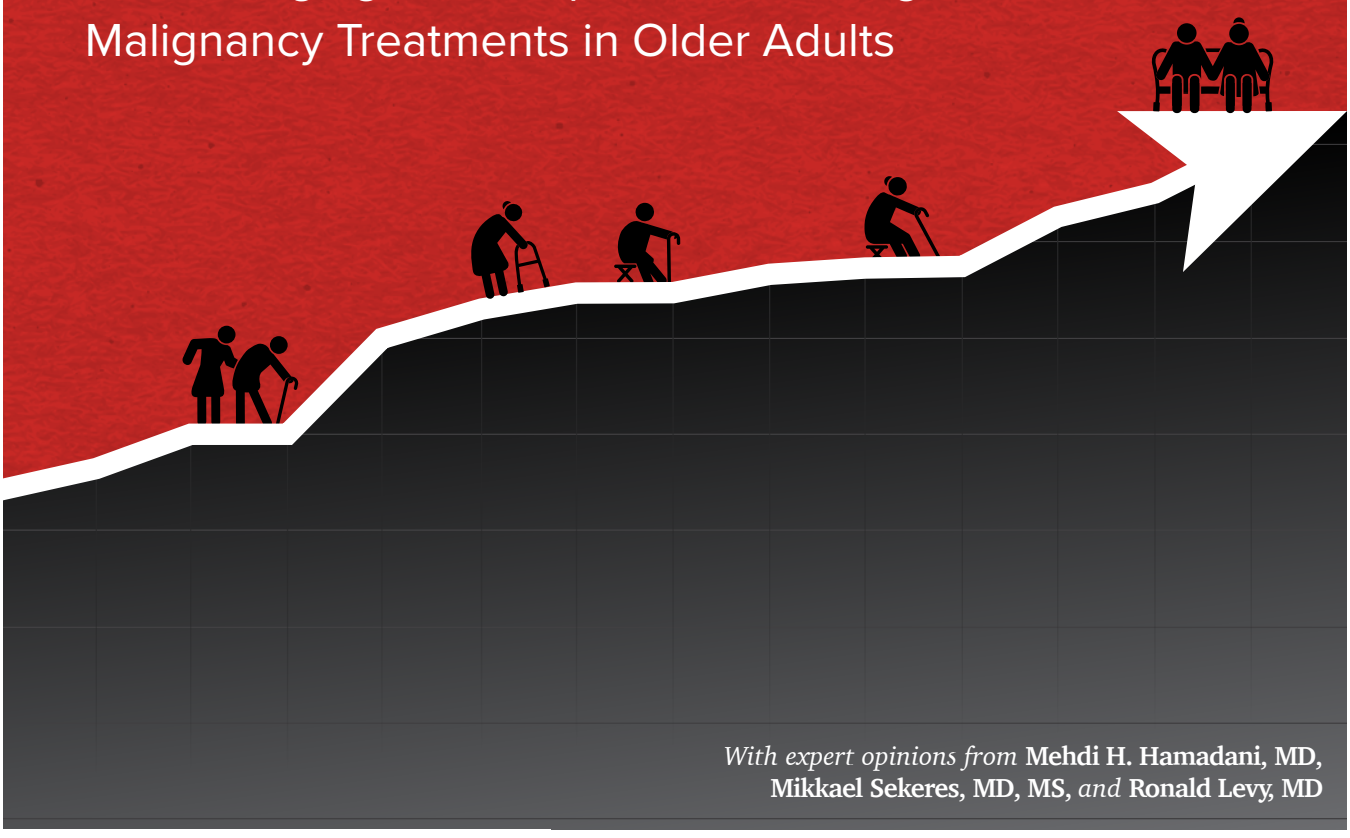

With expert opinions from **Mehdi H. Hamadani, MD, Mikkael Sekeres, MD, MS,** *and* **Ronald Levy, MD**

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Are We Treating Diseases—or Genotypes? The Evolving Logic of AML Therapy

PHYSICIAN'S WEEKLY

HOW AMBIENT AI HELPED SOME CLINICIANS RECONNECT WITH PATIENTS

Medicine was meant to be personal — so when did the screen become the center of care? As physician burnout rises and documentation demands pull clinicians further from patients, one health system's experience suggests technology may be helping medicine rediscover what it lost. Could AI be doing more than saving time? Explore how a new approach to documentation is reshaping the physician-patient connection and restoring meaning to care.

Travis Bias, DO, MPH

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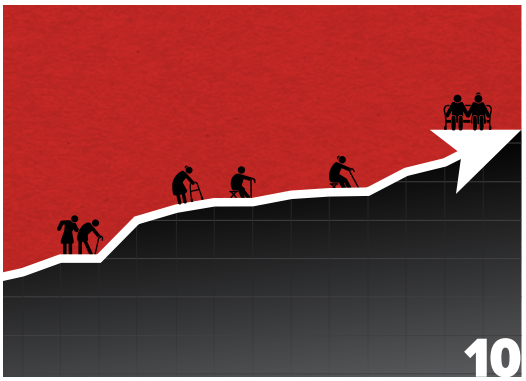

Redefining Age:

The Changing Landscape of Hematologic Malignancy Treatments in Older Adults

By 2030, rates of multiple myeloma are expected to jump 77% in adults aged 65 and older, compared with a 57% increase among younger patients. The aging United States population is presenting oncologists with a growing problem: how to treat this vulnerable population without compromising outcomes.

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Re-evaluating the Role of High-Dose Methotrexate for CNS Prophylaxis in High-Risk Large B-cell Lymphoma

*By **Charles Gaulin, MD***


Matthew R. Wilson

Secondary central nervous system (CNS) lymphoma relapse remains an unmet need associated with poor outcomes in patients with large B-cell lymphoma (LBCL).[1] Patients identified at the highest risk for CNS relapse—and who may therefore derive the greatest benefit from prophylaxis—are defined by a high CNS International Prognostic Index (CNS-IPI), high-risk anatomical involvement (eg, testis), and high-risk genomic subtypes.[2]

For decades, the standard approach to preventing CNS relapse relied on intrathecal chemotherapy, a practice that eventually fell out of favor due to lack of efficacy across several studies.[3] This failure, and the fact that the majority of secondary CNS relapses are parenchymal, prompted a widespread shift toward systemic intravenous high-dose methotrexate (HD-MTX). Despite its inclusion across several guidelines, HD-MTX prophylaxis was never validated by prospective, randomized controlled trials. The rationale was largely extrapolated from its proven efficacy in primary CNS lymphoma and supported by retrospective institutional series.[2]

As clinical practice evolved, large-scale real-world data called into question the benefit of HD-MTX as a prophylactic strategy.[4,5] Despite this, proponents of HD-MTX maintained that "ultra-high-risk" (UHR) patients, whose baseline CNS relapse risk reaches 32%,[6] may still derive benefit. At the European Hematology Association 2026 Congress, an international retrospective analysis introduced a major shift in clinical practice: omitting routine HD-MTX prophylaxis is a reasonable, evidence-based choice, even for the highest-risk patients.

The concurrently published study by Dr. Matthew R. Wilson and colleagues[7] represents the largest comparative dataset compiled to date, pooling data from 56 global sites to evaluate 1,923 patients meeting strict criteria for UHR. Patients were classified as UHR if they had at least one of the following features: a CNS-IPI score of 5-6, testicular, breast, or renal/adrenal involvement, or three or more extranodal sites.

Within this cohort, 872 patients received prophylactic HD-MTX (73% received a cumulative dose >6 g/m^2), while 1,051 patients received no HD-MTX prophylaxis. There was no statistically significant difference in the 3-year CNS relapse rate between the no-HD-MTX and HD-MTX groups (9.3% vs 8.1%; adjusted HR, 1.10; 95% CI 0.80-1.53). When isolated parenchymal or leptomeningeal events were evaluated, the rates remained statistically indistinguishable at 5.9% without HD-MTX versus 5.7%. A landmark analysis designed to eliminate immortal time bias confirmed these results, showing a 3-year CNS relapse rate of 6.7% versus 6.6%.

Not a single UHR subgroup demonstrated a statistically significant reduction in CNS events with HD-MTX. For patients with testicular involvement—historically considered an absolute indication for prophylaxis—there was a non-significant numerical trend toward reduction in overall CNS relapse (9.4% vs 6.8%), but this completely disappeared when isolated CNS relapse was evaluated (6.1% vs 6.3%).

To contextualize these findings within routine practice, I spoke with the study's lead author, Dr. Matthew R. Wilson, consultant hematologist at Beatson West of Scotland Cancer Centre in Glasgow, to discuss the practical implications of changing this longstanding treatment paradigm.

"HD-MTX may lack efficacy, despite its widespread adoption in high-risk patients over the last 20 years … However, given the dismal outcomes in patients suffering CNS relapse, there has been understandable reluctance to completely stop its use."

—Matthew R. Wilson, MBChB, Beatson West of Scotland Cancer Centre

Dr. Gaulin: In our clinics, testicular involvement has long been viewed as an absolute mandate for CNS prophylaxis. Given the historical data, particularly the IELSG30 trial,[8] how should the hematology community interpret your findings in this specific subgroup?

Dr. Wilson: This remains a challenging question. As you mentioned, this is the only DLBCL [diffuse large B-cell lymphoma] subgroup for which prospective trial evidence suggests a potential benefit of CNS prophylaxis. The IELSG30 trial was a single-arm study recruiting patients with low-risk/early-stage primary testicular lymphoma, with the treatment incorporating intrathecal prophylaxis, contralateral testicular radiotherapy, and IV methotrexate given at a dose of 1.5g/m^2

at the end of R-CHOP treatment. Although the absence of any reported CNS relapses in the trial is very encouraging, it is difficult to know which aspect of the treatment protocol is actually mitigating this risk. In our study, we found no statistically significant reduction in patients with testicular involvement who received HD-MTX versus those who did not, including in propensity-matched analyses. Overall, our findings strongly suggest that HD-MTX does not reduce CNS relapse in this patient group. I understand that some clinicians may feel more comfortable following the IELSG30 trial protocol, but I believe that in patients adequately screened to exclude CNS involvement at baseline, there is now evidence to support the omission of HD-MTX in this group.

Dr. Gaulin: The median cumulative dose of HD-MTX delivered was 6 g/m^2. Do you think the cumulative dose of HD-MTX could have impacted the CNS relapse rate in your cohort?

Dr. Wilson: This is an important question and one that we address in the manuscript. We performed analyses of total cumulative HD-MTX dose received and the number of doses received of at least 3 g/m^2 (widely considered the most effective dose in CNS lymphoma). We found no reduction in CNS relapse in patients receiving a total cumulative dose of 6 g/m^2 or more versus less than 6 g/m^2 and no signal towards benefit with increasing number of doses at 3 g/m^2.

Dr. Gaulin: Given your data, how do you see emerging diagnostic and therapeutic platforms fit into the future of CNS prophylaxis?

Dr. Wilson: We have been struggling with the question of CNS prophylaxis in DLBCL for many years now. Increasingly, we have become aware that HD-MTX may lack efficacy, despite its widespread adoption in high-risk patients over the last 20 years. However, given the dismal outcomes in patients suffering CNS relapse, there has been understandable reluctance to completely stop its use, especially given the lack of viable alternatives. It is essentially impossible to conduct an adequately powered prospective trial to answer this question, so we believe this is likely to be the best-quality evidence we will ever have showing no efficacy. Given the significant toxicity involved, this study should essentially halt HD-MTX use in this indication for the vast majority of patients.

Moving forward, we should focus more heavily on baseline CNS screening in the highest risk patients. It is likely we have been under-recognizing baseline occult CNS disease, and such patients may benefit from dose-intensified, CNS-penetrating, first-line chemotherapy regimens. Novel technologies, including CSF ctDNA analysis, may improve our screening sensitivity going forward and more accurately define molecular subgroups at the highest risk of CNS disease. There are multiple trials in first-line DLBCL incorporating novel agents capable of crossing the blood-brain barrier, which are likely to change the treatment landscape in the next few years,[9] and, I suspect, will also reduce rates of CNS relapse. Importantly, as a lymphoma community, we should continue to address this specific question in prospective clinical trials, if possible, but our data can also be used to justify a trial design that does not incorporate HD-MTX.

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Are We Treating Diseases—or Genotypes? The Evolving Logic of AML Therapy

*By **Amer M. Zeidan, MBBS, MHS***

Amer M. Zeidan
MBBS, MHS

Long gone are the days in which all fit patients with acute myeloid leukemia (AML) automatically get intensive chemotherapy (IC) with the 7+3 regimen. What we are seeing now is the accelerating shift toward individualizing the choice of AML therapy, guided by the genetic profile of the leukemia cells to modify the IC backbone, increasing reliance on "biologic" fitness and not merely physical fitness in assessing risk-benefit ratio of using IC, and the increasing use of measurable residual disease (MRD) measurements to guide dynamic risk stratification and subsequent therapy recommendations, including proceeding to allogeneic hematopoietic cell transplantation (allo-HSCT) when possible.

The story of *FLT3*-mutated AML is a telling one, illustrating the highly dynamic and transformative changes in AML management and outcomes. The C10603/RATIFY trial, originally published in 2017, was the first randomized phase 3 trial to demonstrate that adding an FLT3 inhibitor to standard chemotherapy could change outcomes in *FLT3*-mutant AML.

In the trial, 717 patients aged 18 to 59 years were randomly assigned to receive standard 7+3 induction and high-dose cytarabine consolidation with either midostaurin or placebo. Midostaurin reduced the risk of death by 22%, with a 4-year overall survival (OS) rate of 51.4% versus 44.3% for placebo. Recently presented 10-year follow-up data offer some of the longest mature outcome data available for any targeted agent in this disease, with a 10-year OS estimate for midostaurin-treated patients of 43.7%, compared with 38.6% for patients treated with placebo.

Since the original approval of midostaurin for *FLT3*-mutant AML in 2017, the field has moved fast. The QUANTUM-First trial established quizartinib as another option of an FLT3 inhibitor in the frontline setting, and gilteritinib as a monotherapy demonstrated OS benefit compared with salvage chemotherapy in relapsed or refractory *FLT3*-mutant AML through the ADMIRAL trial. For patients with *IDH1* or *IDH2* mutations, phase 1/2 studies led to regulatory approvals of ivosidenib and enasidenib, targeted agents that would have been inconceivable to develop without the molecular diagnostic infrastructure to identify the relevant patient population in the first place. For patients with core binding factor AML, the ALFA-0701 trial supported the addition of gemtuzumab ozogamicin to chemotherapy in the first-line setting. Each of these trials represents a case in which a specific genetic finding changed not only prognosis but also clinical action.

None of this works without fast, reliable genetic testing, and that remains one of the more persistent practical problems in AML care. The disease progresses quickly. Patients are often ill at presentation, decisions often need to be made within days, and the information needed to make those decisions correctly may not yet be available. Some institutions run next-generation sequencing in-house and can return results within a week or two, but others rely on external laboratories and may wait longer.

Testing results also need to be communicated clearly. A report that buries a clinically actionable *FLT3* or *IDH* mutation in technical language or fails to flag a *TP53* alteration in a way that prompts reconsideration of intensive therapy can do real harm. Beyond interpretation, there are access issues, particularly in the United States, where patients' insurance coverage, hospital formulary restrictions, and inpatient-versus-outpatient drug availability can all determine whether a patient actually receives the targeted therapy their genotype indicates they should have.

Genetic testing at diagnosis does more than identify targets for add-on inhibitors. Perhaps the clearest example involves *TP53*-mutated AML. These patients have a poor prognosis with standard IC, and the question of whether IC does more harm than good in this population has driven real practice change. The PARADIGM study assessed whether biologic fitness (accounting for genomic risk alongside physical performance status) better predicts benefit from intensive induction than conventional assessments alone, with the implication that some patients, especially those with adverse risk disease, who appear physically fit enough for IC should not receive it based on their mutational profile. That reframing, from "can this patient tolerate intensive therapy" to "is intensive therapy likely to help this patient," is an important conceptual shift in the field.

Genetic testing also shapes decisions that come after remission. In *FLT3-ITD*–mutated AML, the question of whether to recommend allo-HSCT during a first complete remission (CR1) has become more nuanced with the availability of MRD testing and FLT3 inhibitors. The UK NCRI AML17 and AML19 trials showed that patients with co-occurring *NPM1* mutations who achieved MRD negativity in peripheral blood after two induction cycles had 3-year OS rates above 80% without transplant, comparable to those who did proceed to allo-HSCT, sparing them the long-term morbidity that transplantation carries.

On the other hand, QUANTUM-First trial data suggest that quizartinib and allo-HSCT during CR1 each independently reduce the risk of death for patients with *FLT3-ITD* AML and that the combination produces the greatest benefit. These data collectively suggest that genetic testing alone cannot yet answer who needs a transplant, but it is an important part of the conversation. Furthermore, the randomized MORPHO trial suggested that for patients with *FLT3*-mutated AML undergoing transplant, detectable *FLT3-MRD* by a sensitive next-generation sequencing polymerase chain reaction test in the peritransplant setting could be a biomarker of benefit from the FLT3 inhibitor gilteritinib during posttransplant maintenance.

Finally, it would be incomplete to discuss molecular classification in AML without acknowledging that the current landscape is also, frankly, somewhat confusing. The simultaneous publication in 2022 of two classification systems, one from the WHO and one from the International Consensus Classification, created uncertainty about diagnostic criteria. The two systems agree on a great deal but differ in some ways: which mutations qualify for a diagnosis at lower blast percentages, what variant allele frequency thresholds matter, and how to handle subtypes such as *TP53*-mutated disease or the myelodysplastic syndrome (MDS)–AML overlap category. For a clinician trying to determine whether a patient has AML or MDS, those differences are not trivial. They affect clinical trial eligibility, drug reimbursement, and the conversation a physician has with a patient about what disease they actually have.

There is reason for optimism, though. A classification harmonization meeting took place in March 2026, bringing together representatives from both classification efforts to work toward a single, unified schema. The goal is not uniformity for its own sake; it's ensuring that a patient diagnosed at one clinic is understood to have the same disease when referred to another and that clinical trial eligibility is applied consistently across sites.

Looking ahead, AML treatment will continue moving toward oral targeted agents, reducing the intensity of therapy for selected patients, and combinations of agents chosen on the basis of a patient's specific mutational profile. We are not there yet, and many patients still need and benefit from standard intensive induction therapy such as 7+3, but the proportion managed primarily by genotype-guided strategies is growing. The challenge for the field is making sure the infrastructure to support that approach—the testing, the reporting, the access to drugs, and the classification systems that determine who gets what—keeps pace with the science.

"What we are seeing now is the accelerating shift toward individualizing the choice of AML therapy, guided by the genetic profile of the leukemia cells to modify the IC [intensive chemotherapy] backbone..."

Get to Know

Learn more about the leaders, innovators, and educators in hematologic oncology

Joshua Brody, MD

Joshua Brody, MD, director the Lymphoma Immunotherapy Program at The Tisch Cancer Institute at Mount Sinai, shares his work on cancer immunotherapy in lymphoma and the importance of collaborative science at the Brody Lab.

*By **Melissa Badamo***

When interviewed by his local Long Island newspaper for winning a Father's Day essay contest at 8 years old, **Joshua Brody, MD**, knew exactly what he wanted to do when he grew up: "I want to cure cancers like my dad."

"When we could not find a babysitter, my dad would take me into his lab," Dr. Brody recalled. "I would look into the microscope, and he would show me little bits of cancer."

Following in the footsteps of his dermatologist father, Dr. Brody pursued cancer immunotherapy during his fellowship at Stanford University School of Medicine and was drawn to hematology oncology due to the hands-on nature of the field. Now, he directs the Lymphoma Immunotherapy Program at The Tisch Cancer Institute at Mount Sinai in New York City, a position he was recruited to in 2011 by his mentor, **Miriam Merad, MD, PhD,** chair of the Department of Immunology and Immunotherapy at Icahn School of Medicine.

"You can cure cancer in many ways, but hematology oncology is the most hands-on way. It's the people that spend the most time with cancer patients."

"You can cure cancer in many ways, but hematology oncology is the most hands-on way. It's the people that spend the most time with cancer patients," Dr. Brody said fondly. "When you're coming up with solutions, it is helpful to get a real exposure to the problem. Cancer is a bad thing, but there's a lot more nuance to what specifically the problem is. The problem is that these medicines are good, but they're not safe enough or they fail because of a certain mechanism. Seeing patients is how you really see the problem. I have scientists that work around me that are trying to cure cancer, but they don't hear about the actual day-to-day limitations of where we are so far with cancer therapy."

The Next Wave of Cancer Immunotherapy

At the Brody Lab, Dr. Brody and his research team study and develop *in situ* vaccines for patients with lymphoma. A novel immunotherapy approach, these vaccines are developed with FLT3 ligand, the primary growth and differentiation factor of dendritic cells, and work by targeting the tumor with the patient's own immune system. In early clinical trials, *in situ* vaccines showed success in patients with advanced-stage lymphomas.

"We can bring the immune system to the tumor, where there is a source of tumor-specific antigens. The *in situ* approach is not making the vaccine in the lab, but making it in the site of the tumor," Dr. Brody explained. "We could mobilize a patient's dendritic cells, hopefully bring many of them to the tumor, load those dendritic cells with tumor antigens at the tumor site, and then activate those dendritic cells."

The Brody Lab is also working to overcome patient relapse due to CD20 loss in lymphoma, which occurs from a "Darwinian evolution where the CD20-negative cells grow out." As a result, Dr. Brody is focusing the microscope on patients' CD20-negative cells before they start therapy.

"We think we might be able to find some targets on those cells, which will allow us to make rational combinations," he said. "Instead of just attacking CD20, maybe we can attack CD20 and something that might be on the surface of those CD20-negative cells…It's going to help generate this next

wave of bispecific therapy rational combinations, which we're very excited for."

According to Dr. Brody, the next wave of immunotherapy consists of CD19 bispecific antibodies for B-cell lymphomas, which have shown promising preliminary efficacy.

"Those will almost certainly get some FDA approvals in the next couple of years," he said. "They will also probably be trying to work their way into second-line and frontline therapies."

In addition, combination strategies with bispecific antibodies and antibody-drug conjugates have shown early success in ongoing registrational trials, demonstrating high complete remission rates and favorable safety.

"Both the new wave of bispecifics targeting CD19 and the rational combination of bispecifics and antibody-drug conjugates will unquestionably transform the therapeutic landscape in B-cell lymphomas over the next few years," Dr. Brody said. "That's an exciting opportunity for us, our patients, and their families."

"The Brody Bunch"

In addition to researching novel immunotherapies, Dr. Brody maintains a fun, collaborative culture in his lab. Nicknamed "The Brody Brunch," Dr. Brody and his lab members coordinate group Halloween costumes each year, from Teenage Mutant Ninja Turtles to The Incredibles.

"I didn't invent the annual costume tradition, but the culture persists despite the fact that all the people who did have moved on to their own labs to become doctors. This is not what biologists would study; this is what anthropologists would study. There is a living thing called culture, and the Brody Lab culture is getting to do all this silly stuff," he said.

PHOTO CREDIT: BRODY LAB AT THE TISCH CANCER INSTITUTE

From collaborating on research papers to sharing ideas across labs, Dr. Brody credits the success of his lab to the collaborative nature of science.

"Both the new wave of bispecifics targeting CD19 and the rational combination of bispecifics and antibody-drug conjugates will unquestionably transform the therapeutic landscape in B-cell lymphomas over the next few years."

"That culture is absolutely critical. If we've had any success in our group, it's because we're all buddies and hang out together," he said. "The collaborative model is critical, and maybe the silliness in the culture is helpful towards the collaborative model."

Dr. Brody also fosters a collaborative culture through mentorship, teaching, and clear communication.

"There's a huge communication rift in humans. Most people think that science and medicine are close buddies, but they are mountains apart from each other. There's one skinny bridge that connects those two mountain peaks of science and medicine, and there's not enough people who can walk back and forth along that bridge," he said. "I'm on a floor with mostly scientists, so it behooves the doctors to explain the medical problem so we can cure a subset of cancers, which are *p53* mutated or have CD20 loss after immunotherapy. Communication is such a critical skill because it is what's missing in most collaborations. If people cannot convey the problem, they cannot convey the solution. We should all be working towards being better at that."

Lastly, Dr. Brody offered advice for junior mentees in the field of hematologic oncology.

"Most paths are not straightforward, trailblazed paths. It is very helpful to identify people who have gone down that path, identify them as mentors, and seek out their guidance because there are a lot of obstacles to that path. There are a lot of nuances of how to excel along that path, which only someone who has walked down it will tell you," he advised.

Outside of work, Dr. Brody can be found spending time with his nieces and nephews, exploring cafes, traveling, and taking bike trips around Iceland and the Adriatic coast of Croatia. He also enjoys traveling for work, even bringing his dad—his very first mentor—to professional meetings and medical conferences.

"I took my dad to talks I gave in Beijing, China, and Hokkaido, Japan. Neither of us have been to those places before, so that was very cool and exciting for us!"

Spotlight On the HemOnc Pulse

Rahul Banerjee, MD, associate professor at the Fred Hutchinson Cancer Center and host of The HemOnc Pulse, *covers the latest news in hematologic oncology with leaders in the field.*

Community-Based CAR-T Therapy: Expanding Access, Navigating Challenges

In this episode of The HemOnc Pulse, host **Rahul Banerjee, MD,** and **Gary Simmons, DO,** a hematologist-oncologist at Virginia Oncology Associates, explore the evolving role of community-based cellular therapy programs, how community practices can safely and effectively implement chimeric antigen receptor (CAR) T-cell therapy programs, and the future of patient access to innovative treatments. The discussion centers on Dr. Simmons' recent publication, *Advancing Access to CAR T-Cell Therapy: Insights and Real-World Experience from a Community Oncology Practice,* which highlights the opportunities and challenges of delivering advanced cellular therapies outside of traditional academic centers.

Rahul Banerjee, MD

Gary Simmons, DO

Dr. Banerjee: Welcome to another edition of *The HemOnc Pulse.* We'll dive into it. I am guilty as charged of living in my ivory tower, where we have the infrastructure for CAR-T. We've done it on trials for over a decade. I know it's a much heavier lift to get it down there. I'm very glad that you have it. What prompted you to go down this road of setting up CAR-T therapy at a community-based oncology center?

Dr. Simmons: My background is similar in terms of academia, and I had the opportunity after fellowship to stay in academics. I was in academics at Virginia Commonwealth University, VCU, for about 7 years and fortunate to learn a lot about the business side of medicine infrastructure, of CAR T-cell therapies. I worked in allogeneic stem cell transplants, so I had a good foundation and was looking to make a change and a calling. Virginia Oncology recruited me, and they're a forward-thinking practice, looking ahead about what's coming down the pipe and how do we better serve our population.

We're in a cell therapy desert in this area. There are about 2.5 million people, and the closest academic center could be 150 miles away. That is an obstacle that is very difficult for patients to overcome. We felt that we had the patients, the novelty, the bandwidth, and we had some folks like myself.

Dr. Banerjee: Are there any challenges you faced while setting up CAR-T therapy with fact accreditation, logistics, and safety monitoring? What is helpful to know if other community oncology practices are thinking about doing CAR-T therapy?

Dr. Simmons: For the community oncology folks out there, you need to test the water to see if this is something you want to look into. What's the landscape in terms of rheumatology and oncology down the line? Are there academic centers that you have no problem getting patients treated and getting back to your practice, or do you lose them? You might want to take on a CAR-T program to retain your patients and be more in control of the management of bispecifics pre- and post- apheresis. That was important for me. I value being the captain of the patient's team and wanted full ownership all the way through.

There are a tremendous amount of barriers, and it's very difficult to do this. You need physician buy-in and practice buy-in. This is community-based oncology, so you have to use community hospitals that may or may not allow infusions inpatient. You have to train the nurses in ICUs to look for neurotoxicity. You need to have a process of getting patients from home into the hospital directly without going through community-based ERs. Then, you have to get paid. You have to make sure you can keep the lights on, and you're competing with payers who have defined relationships with academic centers regardless of distance. It is much easier for them to deny CAR-T in the community and tell the patient to go to [Memorial] Sloan Kettering [Cancer Center] regardless of logistics. You're facing a tremendous amount of barriers, so you have to know why you're doing it, what the mission is, and whether it's worth it.

Dr. Banerjee: Agreed. You mentioned that some companies are stubborn. Can you touch a little bit more on that in terms of how that happened? How did you negotiate that with the insurance company, with the patients, and if the insurance company would allow CAR-T but not with you?

Dr. Simmons: For the Medicare and Medicaid folks, that's an easy bucket. But, those are difficult to get reimbursed on and it's hard to keep the doors open that way. In the commercial space, the traditional commercial payers are still reluctant to allow CAR T-cells in the physician's office. We're doing this in the community oncology practice and we're not affiliated with anyone, so that's a novel concept for them. That is why I felt very strongly that that paper had to be in print and peer-reviewed, so that these payers can review this and see that if this group can do it, other groups can do it as well.

It's still a challenge. I just got a denial yesterday. I have a patient with refractory myeloma who relapsed very quickly after transplant and needs to move fairly quickly approaching dialysis,

and it's denied. Now I have to fight that fight, and I will. For the most part, we win, especially now because of our background and we can stamp the paper to it, but it's still a challenge and it's very hard to navigate those systems that are not easy to get anyone on the phone with and navigate discussions with.

Dr. Banerjee: Fact accreditation and handling cell products is its own bucket of paperwork, logistics, and training. Cytokine release syndrome (CRS) is a big issue. I know our Fred Hutch Center probably would not run without nocturnal hospitalist fellows who take these calls overnight. How have you managed CRS and the quintessential 3:00 AM patient having a fever, let's say? Are you admitting everybody just in case? If you're doing outpatient infusion, what is your threshold to admit?

"We're in a cell therapy desert in this area. There are about 2.5 million people, and the closest academic center could be 150 miles away. That is an obstacle that is very difficult for patients to overcome."

—Gary Simmons, DO, hematologist-oncologist, Virginia Oncology Associates

Dr. Simmons: It's more of an individualized approach. I'm very grateful to have a partner, Dr. Scott Cross, who is also on the paper. He's an excellent clinician and partner. Him and I cover the service 26 weeks a year split, so we have great support staff, nurses, and advanced practice providers who help us both in the hospital and outside the hospital. We have algorithms built in, and we've learned to figure out how best to do this in the outpatient space. For example, you saw in the paper the difference of admission rates between the CD28 CARs and the 4-1BB CARs in lymphoma. We use a lot of prophylactic dexamethasone, and for patients with high disease burden, we schedule the admission on day 4. We infuse on Monday, admit on Thursday, and give prophylactic dex Monday, Tuesday, Wednesday. Usually, there are no issues.

With the 4-1BB CARs and ciltacabtagene autoleucel that are traditionally a little more toxic, we use them earlier in the second line so patients are not penta-refractory to everything and they're not so sick. The disease burden's not out of control anymore. We apherese patients, then we bridge them to get their disease burden as low as possible before cell infusion. I'm pleasantly surprised with our ciltacabtagene autoleucel data. We've had some very good outcomes with very little toxicity and reasons for admission.

So, we have dexamethasone. Patients are instructed to have dexamethasone at home, and outside of first fevers, we may have them take 12 mg of dexamethasone. We can pivot quickly. We bring them into the clinic early in the morning to get tocilizumab. These are the things that we can do easier in the community than in academia, because there are not many layers in the community. It's me, my nurses, and staff.

Dr. Banerjee: Great. This is super helpful. You touched on this and it's an interesting question, because barely a CAR-T candidate is super frail. I treat myeloma and central nervous system myeloma, and I'm happy to help triage those very difficult cases. You mentioned individualization of this. You mentioned that the axi-cel, brexu-cel, and CD28 costimulatory products may have a little bit more CRS toxicity than the 4-1BB ones, and you mentioned cilta-cel versus ide-cel. Do you triage which products you offer and whom you offer them to, or do you feel like it's worth it to try to offer all of them?

Dr. Simmons: Well, I think it's important to have an opportunity to have them all. We've onboarded all the products so that it's dealer's choice, and then we use science to guide us. Science will dictate if you have CLL, refractory ALL. Science will dictate to us what to do first and foremost. But, it's a good question what to do with some of these myeloma CARs or lymphoma CARs. I'm going to be honest with you—a lot of times, if it's dealer's choice, we have to not be ignorant to the economics of these products. The economics of these products are very important. In community oncology, we're buying these drugs at $550,000 to $560,000, there's no 340B drug pricing, they're completely outpatient, and oftentimes they're at a loss. Unless there's some negotiation, some contracting with manufacturers to work through some of this or with payers, sometimes the economics of it have to be considered in that algorithm of selection of products for patients.

Dr. Banerjee: Great. To wrap things up, what's next for your practice? Where do you see this principle of community-based CAR-T evolving in the Norfolk area for the next 3 to 5 years?

Dr. Simmons: We're going to continue to look forward to the future to serve our patients. That means what's next for us is tumor infiltrating lymphocytes (TILs). As a community oncologist, you're hamstrung by labels and giving things in-patient, so it's constantly fighting different barriers to bring these therapies to the community. But, I think we can work with the companies and develop outpatient clinical trials, and then I'm hopeful that we'll have TILs here in the next 12 months to provide for the patients here. Hopefully, that is something that is moving into other oncology and solid tumors as well. Anything to bring access and cellular therapy to the patients here is what we're trying to do.

Listen to the podcast episode on www.bloodcancerstoday.com.

Redefining Age:

The Changing Landscape of Hematologic Malignancy Treatments in Older Adults

By ***Sara Karlovitch***

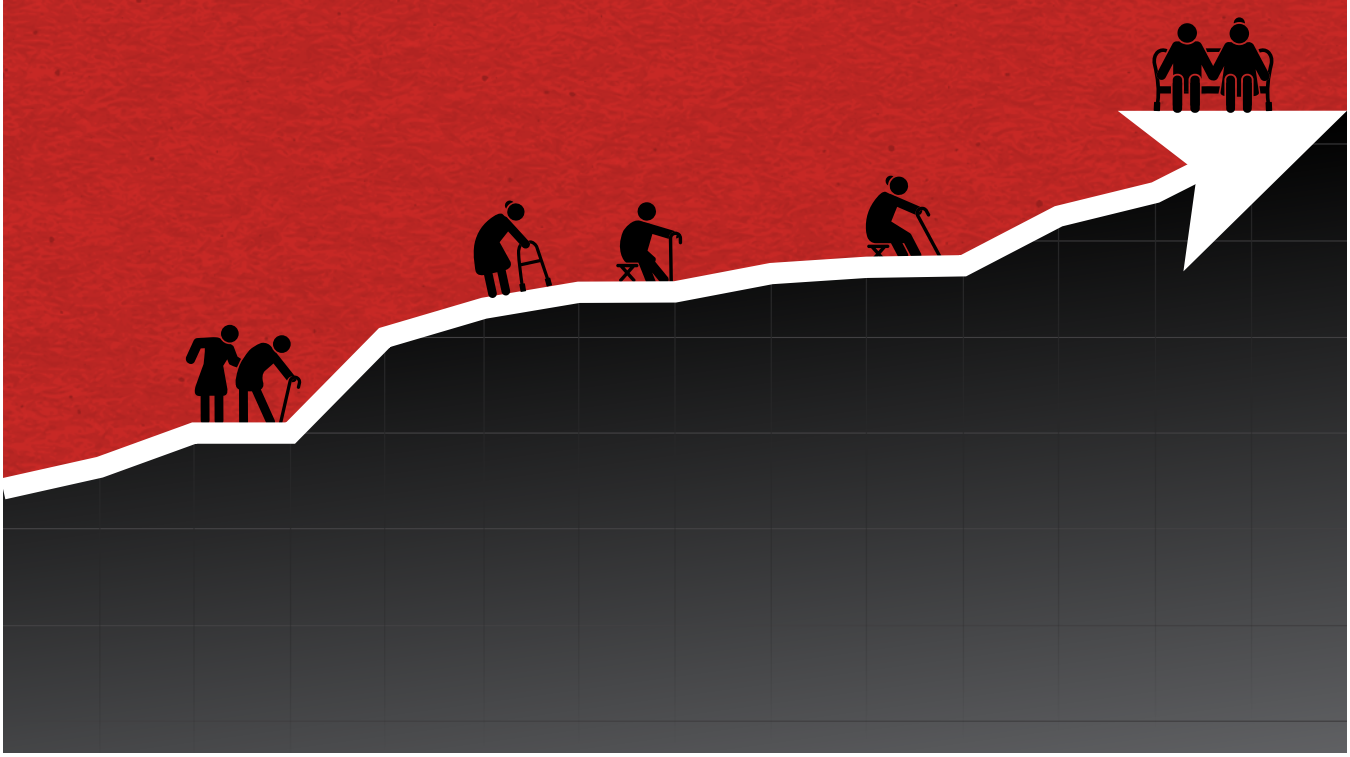

ILLUSTRATION BY JOHN SALESI

The aging United States population is presenting oncologists with a growing problem: how to treat this vulnerable population without compromising outcomes.

In the two-decade period between 2004 and 2024, the percentage of the population aged 65 and older has risen from 12.4% to 18%. This shift is largely due to the age of the Baby Boomer generation, along with a declining birth rate and rising life expectancy.[1] The 65 and older population is already the most common demographic for hematologic malignancy diagnoses. As the population ages, the rates of blood cancers are also expected to rise.

By 2030, rates of multiple myeloma (MM) are expected to jump 77% in adults aged 65 and older, compared with a 57% increase among younger patients. A similar increase is expected in non-Hodgkin's lymphoma and leukemia, with a less than 50% increase in incidences of the diseases among those under the age of 65 and a 65% increase expected in the older population.[2]

When it comes to treating older patients with hematological malignancies, there has been a shift in how oncologists approach this population. While chronologic age remains a key factor when determining patients' fitness, geriatric assessments (GAs) have become increasingly crucial tools when creating a treatment plan. However, despite making up the majority of hematologic malignancy diagnoses, the demographic is often underrepresented in clinical trials. This not only creates barriers to accessing life-saving treatment but also contributes to gaps in data for this population.[3]

"Most people will say the days where we had arbitrary chronological age cutoffs are long behind us," said **Mehdi H. Hamadani, MD,** professor of medicine, section chief of Hematological Malignancies, associate director of Clinical Research, and Cancer Center Scholar Endowed Chair at the Medical College of Wisconsin. "Biological or physiological age in determining a given therapy for the patient is here to stay. It's not a perfect system, depending on the treatment that we are considering. How you eventually evaluate a patient's physiological age, which is essentially assessing patients' fitness to tolerate a given therapy, is not clearly defined."

Redefining Fitness

Organ function is a key consideration in the tolerability of certain treatments for hematologic malignancies. However, the performance of many organs naturally decreases with age. Combined with the development of many age-related ailments and medical conditions, the recommended treatments for many hematologic malignancies may not be an option for older patients, according to **Mikkael Sekeres, MD, MS,** chief of the Division of Hematology at Sylvester Comprehensive Cancer Center at the University of Miami Health System.

"But that isn't uniform—some people age better than others and remain 'fit' over the course of their lives. So, we have to avoid falling into the trap of defining a patient and their likelihood of tolerating a treatment based purely on their chronologic age," said Dr. Sekeres.

GAs have proven to be incredibly valuable tools for determining patient fitness for certain treatment options. Assessments such as the Cancer and Aging Research Group (CARG) assessment or the American Society of Clinical Oncology (ASCO) practical geriatric assessment (PGA) can help improve outcomes for those 65 years of age and older.[4]

In 2023, ASCO updated its 2018 guidelines on the use of GAs. The guidelines were updated based on a review of randomized clinical trials, meta-analyses, and systematic reviews between January 2016 and December 2022 by a panel of experts. Twenty-six publications met the criteria and served as the evidence for the guideline updates. The updates were published in the *Journal of Clinical Oncology* in July of 2023.[5]

"Most people will say the days where we had arbitrary chronological age cutoffs are long behind us ... Biological or physiological age in determining a given therapy for the patient is here to stay."

—Mehdi H. Hamadani, MD, professor of medicine, Medical College of Wisconsin

The 2023 updates largely strengthen the 2018 guidelines and recommend streamlining the GA process based on data from several clinical trials. It strongly recommends that all patients with cancer over the age of 65 who have a GA-identified impairment have GA-guided management included in their care plans. GA-guided management should also be extended to older adults who are receiving treatments such as immunotherapy, targeted therapy, or chemotherapy. The guided management is meant to help guide treatment decisions and address impairment via the appropriate outlet.[5]

Other recommendations include prioritizing age-related conditions, such as physical and cognitive function, mental health, nutrition, social support, and comorbid conditions.[5]

"Geriatric assessments are a terrific approach to quantifying a person's functional status, particularly those that can identify a frail population who may not benefit from treatment," said Dr. Sekeres. "Pragmatically, ideally, they should be implemented in a way that minimally affects the time required to evaluate a patient and clinic flow."

The Data Gap

Despite making up a significant proportion of the population diagnosed with a hematologic malignancy, patients aged 65

and older are underrepresented in clinical trials, creating significant data gaps.

"Clinical trials usually have eligibility restrictions, and they tend to eliminate some of the risks of side effects through organ function requirements and age requirements," said **Ronald Levy, MD,** Robert K. and Helen K. Summy Professor in the School of Medicine at Stanford. "The data we have for safety and efficacy from clinical trials that are published, the data usually come from pretty healthy populations."

A 2025 analysis, titled, "Disparities in clinical trial participation among older adult Medicare beneficiaries with hematologic malignancies from 2006 to 2019: A SEER–Medicare analysis," published in *Cancer,* found that only 4.3% of older adults diagnosed with a hematologic malignancy participated in a trial within 5 years of diagnosis.[3]

"Geriatric assessments are a terrific approach to quantifying a person's functional status, particularly those that can identify a frail population who may not benefit from treatment."

—Mikkael Sekeres, MD, MS, professor of medicine, Sylvester Comprehensive Cancer Center

The retrospective study, which followed the Strengthening the Reporting of Observational Studies in Epidemiology reporting guidelines, used the Surveillance, Epidemiology, and End Results (SEER)–Medicare database. To be included in the retrospective, patients must have been diagnosed with a hematologic malignancy between 2006 and 2018, have a follow-up period through December 2019, and be 66 years of age or older. Medicare claims for clinical trial services were used to define clinical trial participation. Participation rates and adjusted hazard ratios (aHRs) were estimated using Fine-Gray models and cumulative incidences.

The cohort included 53,919 patients, 86% of whom were White and 50% of whom were female. The median age was 78 years. Clinical trial participation at 1 year after diagnosis was just 2.7% overall. The study also found that certain factors made a patient even less likely to participate in a clinical trial. Female patients had an aHR of 0.79, and Black patients had an aHR of 0.73. Comorbidities such as pulmonary and renal disease, which had aHRs of 0.76 and 0.67, respectively, also made patients less likely to participate in trials.[3]

Once the risk of death was adjusted for, older age was also associated with decreased clinical trial participation. Patients who lived farther from a National Cancer Institute center and those with dual Medicare-Medicaid eligibility were also less likely to participate.[3]

"Certain therapies that may have a very nice toxicity profile…in a clinical trial setting, because they were evaluating those strategies in fitter patients, may have a very different toxicity profile when you apply it to frailer patients outside the setting of a clinical trial," said Dr. Hamadani.

Tailoring Therapy Without Compromising Efficacy

Like most patients with cancer, older adults with hematologic malignancies are not treated with a one-size-fits-all approach. Some older patients may be well suited to the usual treatment intensity, while others may need more tailored treatment for the best outcomes.

"In an older adult with a great functional status and limited or absent comorbidities, we may not want to adjust the dose or intensity of therapy in an initial treatment cycle at all and instead adjust these parameters if side effects are substantial," said Dr. Sekeres. "In an older adult with a compromised functional status and/or comorbidities, we may start with a less intensive regimen and then adjust the dose and intensity up if that person tolerates the initial treatment dose."

However, many of the best treatment options available for patients with blood cancers have already become safer and less toxic overall. Hematopoietic stem cell transplantation (HCT) is already considered the standard of care for the majority of patients aged 75 years and younger, while many centers have no age limit on HCT at all. This increase in accessibility for older patients has largely been due to improvements in key factors, such as supportive care and more effective prevention of graft-versus-host disease (GVHD). GAs have proven to be one of the best ways to predict HCT survival and toxicity, aside from a comorbidity assessment.[4]

Acute myeloid leukemia (AML) induction options have also become easier to tolerate for many older patients. Azacitidine in combination with venetoclax in elderly patients, or those otherwise ineligible for intensive chemotherapy, had a median overall survival of 14.7 months, an improvement over the 9.6 months seen with azacitidine alone. The complete remission rate was also twice as high for the combination group, at 66.4%.[6]

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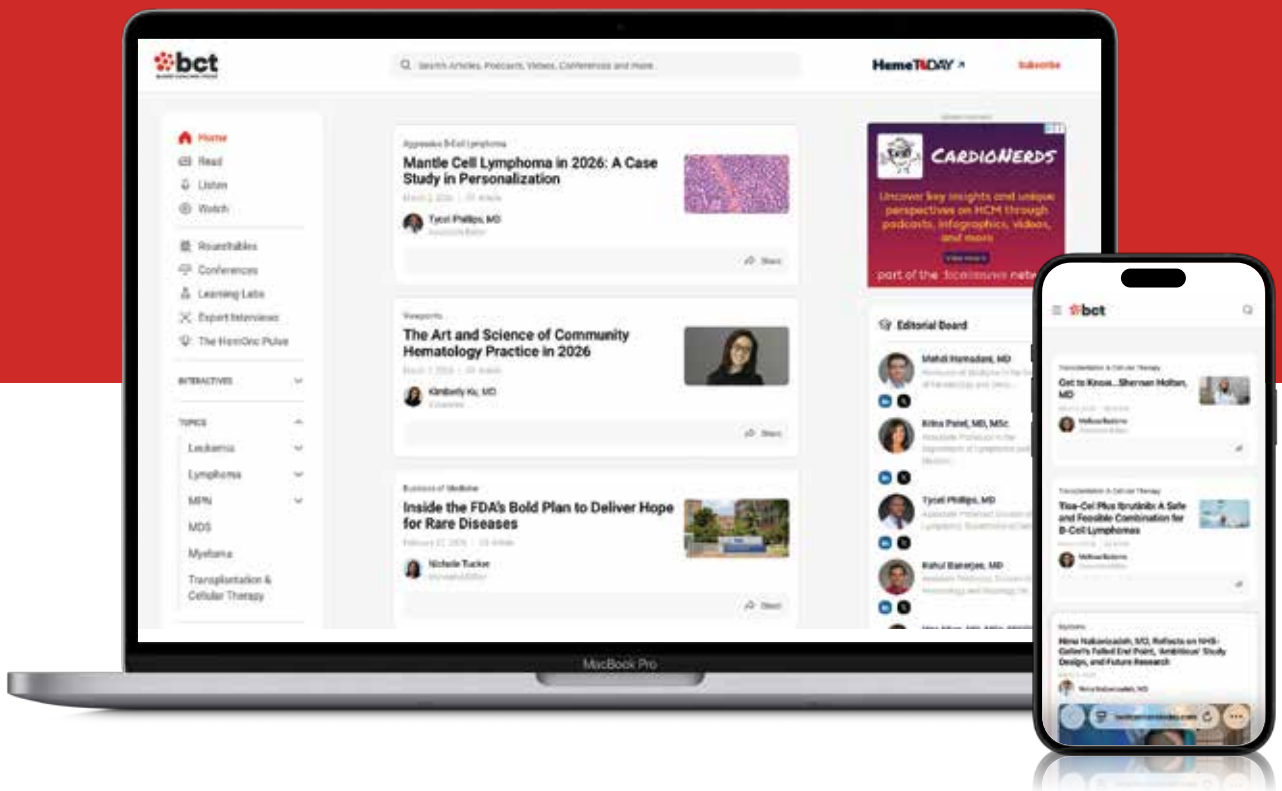

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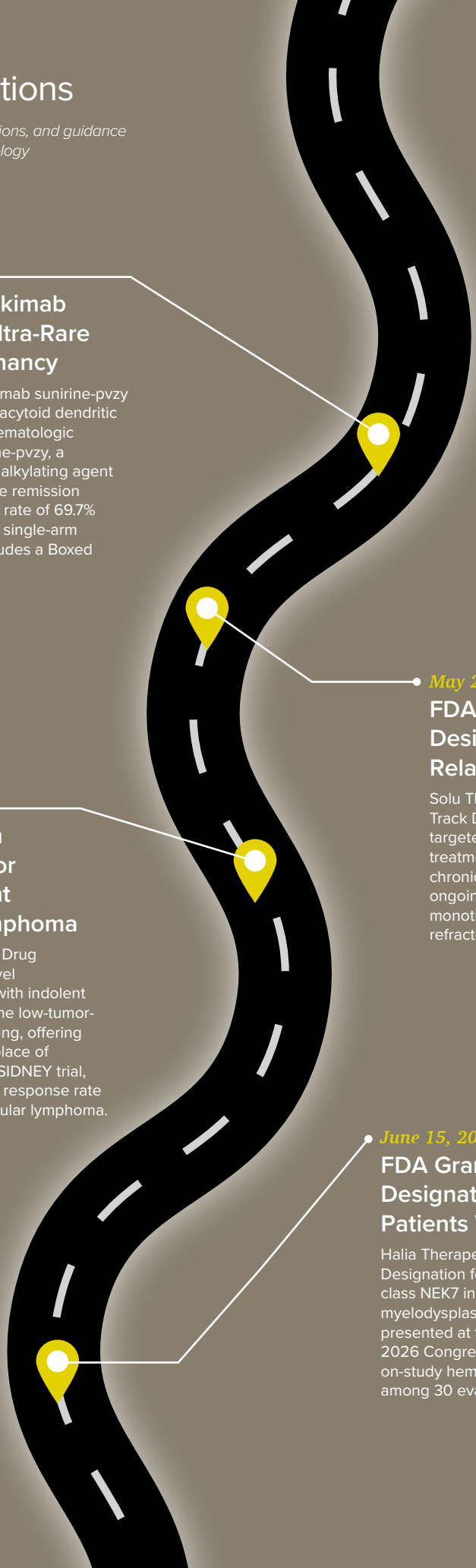

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May 27, 2026

FDA Approves Pivekimab Sunirine-Pvzy for Ultra-Rare Hematologic Malignancy

The FDA has approved pivekimab sunirine-pvzy for patients with blastic plasmacytoid dendritic cell neoplasm, an ultra-rare hematologic malignancy. Pivekimab sunirine-pvzy, a CD123-directed antibody and alkylating agent conjugate, showed a complete remission or clinical complete remission rate of 69.7% in the multicenter, open-label, single-arm CADENZA trial. The label includes a Boxed Warning for hepatotoxicity.

May 27, 2026

FDA Grants Fast Track Designation to STX-0712 for Relapsed or Refractory CMML

Solu Therapeutics has received FDA Fast Track Designation for STX-0712, a CCR2-targeted cytotoxicity targeting chimera, for the treatment of patients with relapsed or refractory chronic myelomonocytic leukemia (CMML). An ongoing phase 1 study is evaluating STX-0712 monotherapy in patients with relapsed or refractory CMML and acute myeloid leukemia.

May 28, 2026

FDA Grants Orphan Drug Designation for EO2463 for Indolent Non-Hodgkin's Lymphoma

The FDA has granted Orphan Drug Designation to EO2463, a novel peptide vaccine, for patients with indolent non-Hodgkin's lymphoma in the low-tumor-burden, "watch and wait" setting, offering a potential new treatment in place of observation. In the phase 1/2 SIDNEY trial, EO2463 showed an objective response rate of 52.6% in patients with follicular lymphoma.

June 15, 2026

FDA Grants Fast Track Designation to Ofirnoflast for Patients With Lower-Risk MDS

Halia Therapeutics has received FDA Fast Track Designation for ofirnoflast (HT-6184), a first-in-class NEK7 inhibitor, for patients with lower-risk myelodysplastic syndromes (MDS). Based on results presented at the European Hematology Association 2026 Congress, ofirnoflast achieved an overall best on-study hematological improvement rate of 67% among 30 evaluable patients.

Highlights from the **EUROPEAN HEMATOLOGY ASSOCIATION (EHA) 2026 CONGRESS,** June 11 –14, 2026, in Stockholm, Sweden

‘Mutation-Agnostic’ Triplet Combination Achieves High Response Rates in Newly Diagnosed AML

*By **Melissa Badamo***

Tuspetinib in combination with azacitidine (AZA) and venetoclax (VEN) demonstrated high response rates and safety in a genetically diverse population of patients with acute myeloid leukemia (AML), addressing an unmet need for those with adverse *FLT3-ITD, RAS,* or *TP53* mutations who have reported poor long-term outcomes with AZA-VEN alone.

“AML biology is complex and driven by multiple interconnected mechanisms, including enhanced growth signaling, resistance to apoptosis, epigenetic dysregulation, and differentiation block. While azacitidine plus venetoclax effectively target some of these pathways, additional mechanisms may continue to sustain leukemic survival and contribute to resistance,” first author **Nikolai Podoltsev, MD, PhD,** of Yale School of Medicine, told *Blood Cancers Today.* Dr. Podoltsev presented the findings at the EHA 2026 Congress.

Nikolai Podoltsev, MD, PhD

In this trial, 23 patients with previously untreated AML received oral tuspetinib, a kinase inhibitor targeting SYK, FLT3, JAK1/2, RSK1/2, and mutant KIT kinases, at four dose levels between 40 and 160 mg once daily for 28 days in 28-day cycles. AZA was administered at the standard dose of 75 mg/m^2 daily for 7 days, and VEN was administered at the standard dose of 400 mg daily for 21 or 28 days. This was an older population, aged 63 to 81 years, with predominantly *FLT3*-unmutated disease (78.2%).

The triplet combination achieved high composite complete remission (CRc) rates of 82.4% among the 17 patients eligible for response evaluation. As the best response, 76.5% of patients achieved CR or CR with partial hematologic recovery (CRh) and 70.6% achieved a CR. The 120-mg dose yielded the highest response rate, 100%, followed by 80.0% at 80 mg and 160 mg, and 75.0% at 40 mg. The measurable residual disease negativity rate, defined by more than 0.1% leukemic blasts by central flow cytometry, was 64.7%.

Notably, mutation status did not seem to impact whether patients responded to treatment. The five patients with *FLT3* mutations achieved a CRc of 100%, while the 12 patients with unmutated *FLT3* achieved a CRc of 75.0%. The four patients with *TP53* mutations achieved a CRc of 75.0%, while the 13 patients with *TP53* mutations achieved a CRc of 84.6%. Additionally, the two patients with RAS mutations also achieved a CR.

As for safety, decreased platelet count (56.5%), decreased neutrophil count (47.8%), diarrhea (47.8%), decreased white blood cell count (39.1%), anemia (34.8%), nausea (34.8%), and constipation (30.4%) were the most common adverse events reported. There were no dose-limiting toxicities, positioning tuspetinib plus AZA-VEN as a well-tolerated option that achieves objective responses in a diverse AML population.

“Adding a safe and effective inhibitor of multiple growth factor signaling pathways to the current standard-of-care backbone of azacitidine plus venetoclax may further improve outcomes in a mutation-agnostic manner,” Dr. Podoltsev said. “Based on its mechanism of action, favorable safety profile, and encouraging clinical activity observed in early-phase studies in relapsed/refractory AML, as well as findings from the TUSCANY trial presented at EHA 2026, tuspetinib appears to be a promising frontline combination partner for newly diagnosed AML.”

Despite these improved outcomes, unmet needs remain, such as finding new therapeutic options for patients without a single mutation. Patients with co-occurring mutations, or even those without actional mutations, often have incomplete responses and poor long-term outcomes, according to Dr. Podoltsev.

“Combination strategies that incorporate agents with activity across multiple growth factor signaling pathways may help overcome resistance mechanisms and improve outcomes irrespective of mutational status. Such mutation-agnostic approaches may complement existing targeted therapies and expand treatment opportunities across a broader AML population,” he concluded.

REFERENCE:

European Hematology Association 2026 Congress. Abstract #S134.

Early UCART22 Data Suggest Potential Bridge to Transplant in Relapsed/Refractory ALL

*By **Nichole Tucker***

Patients with relapsed or refractory CD22-positive acute lymphoblastic leukemia (ALL) achieved high response rates with the investigational allogeneic chimeric antigen receptor (CAR) T-cell therapy UCART22, enabling many patients in a phase 1 study to proceed to stem cell transplantation.

The findings from a phase 1 dose-finding study were presented at the EHA 2026 Congress. The protocol called for UCART22 administration following lymphodepleting therapy with fludarabine and cyclophosphamide (FC), with or without alemtuzumab. A total of 45 patients were enrolled, and 43 received UCART22.

UCART22 is an allogeneic, off-the-shelf CAR T-cell therapy targeting CD22. Unlike autologous CAR T-cell products, it is manufactured from healthy donor cells and can be administered without the delays associated with individualized production. In BALLI-01, investigators explored multiple lymphodepletion strategies, including the addition of alemtuzumab, to determine whether deeper immune suppression could improve CAR T-cell persistence and treatment outcomes. The study evaluated two versions of UCART22: an externally manufactured product used in the initial P1 cohorts and a subsequently developed product manufactured by Cellectis that was evaluated in the later P2 cohorts.

Richard Larson, MD

"Among the several cohorts studied in this phase 1 trial, we saw clinically meaningful anti-leukemia responses when the lymphocyte-depleting regimen included fludarabine, cyclophosphamide, and alemtuzumab. This conditioning regimen led to the greatest persistence of UCART22 cells in patients and the greatest likelihood of remission," **Richard Larson, MD,** of UChicago Medicine, told *Blood Cancers Today.*

The objective response rate (ORR) among patients treated with the externally manufactured UCART22 product in the P1 cohorts was 33%, including a 28% complete response (CR) and CR with incomplete count recovery (CRi) rate. Among those treated with the Cellectis UCART22 product in the P2 cohorts, the ORR was 64%, and the CR/CRi rate was 36%.

Notably, the highest response rates were observed in patients treated with 5×10^6 UCART22 cells/kg, which was later selected as the recommended phase 2 dose. Among patients who received FC in combination with alemtuzumab, the ORR was 73%, and the CR/CRi rate was 40%. Investigators also reported that five of the six patients who achieved a CR/CRi were measurable residual disease-negative by flow cytometry.

REFERENCE:

European Hematology Association 2026 Congress. Abstract #S114.

High Response and Manageable Safety in Post-Transplant Teclistamab and Lenalidomide: Multiple Myeloma Data

*By **Andrew Moreno***

The three-cohort, phase 3 EMN30/MAJESTEC-4 clinical trial compared teclistamab with or without lenalidomide against lenalidomide monotherapy for maintenance following autologous stem cell transplantation (ASCT) in newly diagnosed multiple myeloma (MM).

Niels van de Donk, MD, PhD

Newly available safety run-in data from the study, compiled over a median of 3 years of follow-up, were presented at the EHA 2026 Congress. In these data, the trial team led by **Niels van de Donk, MD, PhD,** of the Amsterdam University Medical Center in the Netherlands, observed encouraging efficacy findings. The percentage of patients with a complete response (CR) or better after ASCT approached 100% from teclistamab with or without lenalidomide.

Regarding safety findings, Dr. van de Donk and colleagues concluded in the abstract that "Tec ± Len [teclistamab with or without lenalidomide] can be safely administered as maintenance therapy post ASCT in NDMM [newly diagnosed multiple myeloma], with monthly Tec dosing as early as C2 [cycle 2] in responders."

EMN30/MAJESTEC-4 comprised 94 patients with at least partial response to 4 to 6 cycles of 3- or 4-drug induction therapy, along with ASCT with or without consolidation, and were divided into three cohorts. The first cohort included 32 patients who received teclistamab dosed at 1.5 mg/kg once weekly, then 3 mg/kg every 2 weeks, then 3 mg/kg every 4 weeks, and lenalidomide dosed at 10 mg once daily, then 15 mg once daily. The second cohort consisted of 32 patients who received teclistamab at 1.5 mg/kg, followed by 3 mg/kg every 4 weeks, and lenalidomide at 10 mg once daily, then 15 mg once daily. The 30 patients in the third cohort received teclistamab at a 1.5 mg/kg dose, followed by 3 mg/kg every 4 weeks.

For efficacy data, the respective median follow-ups for Cohorts 1, 2, and 3 were 35.3, 23.7, and 23.7 months. The rates of CR or better following ASCT until data cutoff increased in all three cohorts, rising in Cohort 1 from 46.9% to 100%, in Cohort 2 from 40.6% to 96.9%, and in Cohort 3 from 60.0% to 96.7%. The rates of CR or better in measurable residual disease (MRD) at 12 months following ASCT also increased in evaluable patients, from 33.3% to 100% in Cohort 1, from 33.3% to 100% in Cohort 2, and from 44.0% to 90.0% in Cohort 3. The investigators also estimated a 24-month progression-free survival (PFS) rate of 96.6% in Cohort 1 and 18-month PFS rates of 93.8% and 96.4% in Cohorts 2 and 3, respectively.

For safety data, the respective median follow-ups for Cohorts 1, 2, and 3 were 32.7, 21.2, and 21.2 months. The prevalence of grade 3 or 4 treatment-emergent adverse events (TEAEs) was 100% in Cohort 1, 96.9% in Cohort 2, and 66.7% in Cohort 3, with neutropenia the most common grade 3 or 4 TEAE in all three cohorts. Hypogammaglobulinemia was present in 96.9%, 87.5%, and 93.3% of patients in Cohorts 1, 2, and 3, respectively.

"CRS [cytokine release syndrome] and infections were manageable with established protocols, making this regimen suitable for use across practice settings," Dr. van de Donk and colleagues wrote in the abstract. They also noted there were no instances of immune effector cell–associated neurotoxicity syndrome.

Nine patients discontinued treatment due to TEAEs. Two patients in Cohort 2 died due to TEAEs, and two other patients in the study died from non-TEAE causes.

The EMN30/MAJESTEC-4 trial is currently enrolling for its randomized component.

REFERENCE

European Hematology Association 2026 Congress. Abstract #EHA-3444 Short: S197.

Pelabresib Added to Ruxolitinib Shows 'Meaningful Clinical Benefit' in Myelofibrosis

*By **Melissa Badamo***

New MANIFEST trial data show that adding pelabresib to ongoing ruxolitinib treatment can improve outcomes in patients with myelofibrosis, overcoming suboptimal or short-lived responses associated with standard-of-care ruxolitinib alone.

Marina Kremyanskaya, MD, PhD

The phase 2, open-label study was presented at the EHA 2026 Congress by **Marina Kremyanskaya, MD, PhD,** of the Icahn School of Medicine at Mount Sinai, showing the synergistic benefits of combining a BET inhibitor with a Janus kinase (JAK) inhibitor.

"There is preclinical data showing synergy with combination therapy of JAK inhibitor and BET inhibitor," Dr. Kremyanskaya told *Blood Cancers Today*. "This is believed to be due in part to the BET inhibitor effect on inflammatory pathways involved in MF pathogenesis such as NF-kB pathway."

To evaluate this effect, arm 2 of MANIFEST enrolled 87 patients (median age, 69 years) assigned to two cohorts: transfusion dependent (TD; Cohort 2A; n=59) or transfusion independent (TI; Cohort 2B; n=28). This was a mostly intermediate- or high-risk population, with 8% of patients classified as Intermediate-1, 63% as Intermediate-2, and 29% as high-risk.

All patients received oral pelabresib 125 mg once daily added to ongoing ruxolitinib, administered at a range of 5 to 25 mg twice daily. Patients had already been receiving ruxolitinib for a median of 30.2 months before the study's start.

Primary end points varied by cohort: Cohort 2A measured the transition from transfusion dependence to transfusion independence, while Cohort 2B measured spleen volume reduction ≥35% (SVR35) at week 24. Twelve of 46 (26.1%) evaluable TD patients achieved TI, with a median duration of 68.0 weeks and a median time to TI of 12.4 weeks.

Fifteen of 83 (18.1%) evaluable patients achieved SVR35 at week 24, including 16.4% of TD patients and 21.4% of TI patients. SVR35 at any time was higher at 28.8%, and durable SVRs with a median spleen response duration of 180.9 weeks were reported.

Additionally, the authors investigated total symptom score reduction less than or equal to 50% (TSS50), bone marrow fibrosis, hemoglobin improvement, and safety as secondary end points. TSS50 at week 24 was achieved by 37.6% evaluable patients overall, 36.2% of TD patients, and 40.7% of TI patients. This percentage climbed to 57% when TSS50 was measured at any time point.

A similar percentage of patients reported grade 1 or higher improvements in bone marrow fibrosis at both week 24 (31.3%) and week 48 (37.1%), while 21.4% of TI patients achieved a hemoglobin response.

The safety profile was consistent with expected adverse events associated with both treatments, including grade 3 or higher thrombocytopenia in 25.3% of patients and anemia in 23% of patients.

"In patients with myelofibrosis who have suboptimal response to ruxolitinib, the addition of pelabresib has the potential to affect all aspects of myelofibrosis: improve splenomegaly, improve symptom score, decrease transfusion burden, improve anemia, and improve bone marrow fibrosis. This is typically a difficult-to-treat patient population, and additional agents beyond JAK inhibitors are desperately needed," Dr. Kremyanskaya told *Blood Cancers Today*.

Next, researchers are studying the optimal timing when combining pelabresib with ruxolitinib in both the upfront and add-on settings.

"This combination has been shown to be effective, and the optimal patient population as well as optimal timing are being investigated in ongoing and future trials," Dr. Kremyanskaya said.

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Editor's Picks

In each issue of Blood Cancers Today, *we will take a closer look at a particular topic in hematologic malignancies. This month, section editor* ***Matthew Davids, MD,*** *director of clinical research for the LLymphoma Division at Dana-Farber Cancer Institute and an associate professor at Harvard Medical School, highlights recent research in chronic lymphocytic leukemia.*

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Matthew Davids, MD

CHRONIC LYMPHOCYTIC LEUKEMIA

Double-Exposed CLL: From Dead End to a Field of Choices

By ***Dean Patterson***

Susan O'Brien, MD

For years, patients with chronic lymphocytic leukemia (CLL) who experienced disease progression after treatment with a Bruton's tyrosine kinase (BTK) inhibitor and venetoclax had limited treatment options. Now, those days are ending.

At a recent session during the American Society of Clinical Oncology Annual Meeting, **Susan O'Brien, MD,** of the Chao Family Comprehensive Cancer Center at the University of California, Irvine, walked through a field of treatments that now includes one approved noncovalent BTK inhibitor, an approved chimeric antigen receptor (CAR) T-cell product, and two oral degraders that "look almost interchangeably good."

Patients are considered double-refractory if they have progressive disease during treatment with a BTK inhibitor (not intolerance) plus progression during treatment with venetoclax. However, fixed-duration venetoclax therapy has muddied the field. "If you have a median PFS [progression-free survival] with venetoclax/obinutuzumab of 5 years, does progression at 2 years define relative refractoriness to venetoclax? What about 3 years? I think the jury is still out on that," Dr. O'Brien said. Hence, the term *double-exposed* has been used to refer to disease progression during treatment with a BTKi, followed by relapse after treatment with fixed-duration venetoclax therapy.

Pirtobrutinib was the first drug approved in this space in the US and Europe. Because of its noncovalent binding, pirtobrutinib stays active against *C481*-mutant BTK, the dominant resistance mutation after covalent inhibitors have been used to treat CLL. Patients in the BRUIN CLL-321 study were heavily pretreated, with a median of four to five prior regimens; roughly half had del(17p) or a *TP53* mutation, and complex karyotypes were common. Response held up.

"It is uncommon to see a patient who does not have some response to pirtobrutinib," Dr. O'Brien noted. The overall response rate (ORR) was 82%, with mostly partial responses. Progression-free survival was about 2 years for venetoclax-naive patients and roughly 16 months in the BCL2i-exposed group. Atrial fibrillation or flutter showed up in 4.6%. Accelerated approval was granted by the FDA in December 2023. Results of the randomized BRUIN CLL-321 trial (14.0 vs 8.7 months PFS against idelalisib-rituximab or bendamustine-rituximab) led to full approval for relapsed CLL or small lymphocytic lymphoma after treatment with a covalent BTKi in December 2025.

Then there's lisocabtagene maraleucel, the CD19-directed CAR T-cell therapy with a defined CD4:CD8 ratio. In the TRANSCEND CLL 004 BTKi-progression/venetoclax-failure subset at the 100×10^6 dose, the rate of complete response (CR)/CR with incomplete hematologic recovery (CRi) was 20%, with ORR around 44%. "The CR rate was not that impressive, frankly, at 20%," Dr. O'Brien said. The complete and partial responses, however, carried meaningful PFS, and undetectable measurable residual disease rates were high. The cost is toxicity: cytokine release syndrome occurred in 85% of patients (mostly grade 1 or 2), and neurologic events in about 45%. FDA accelerated approval followed in March 2024.

The conversation shifted to two investigational degraders, BGB-16673 and bexobrutideg (NX-5948). BGB-16673, a chimeric degradation-activating compound that drags BTK to the proteasome, posted an 85.3% ORR across doses (94.4% at the 200-mg dose moving forward) and a 65.9% PFS rate at 18 months, with the median not yet reached. Fatigue led the toxicity list; atrial fibrillation appeared in three patients, two during infection. Bexobrutideg delivered an ORR of 83%, median duration of response of 20.1 months, and median PFS of 22.1 months. Both work across *BTK* mutations, including some that emerge after pirtobrutinib. Neither is approved anywhere yet.

Dr. O'Brien noted how alike the degraders look. "I sometimes have trouble keeping the data separate because the data look so similar: very high efficacy rates, similar PFS, and excellent toxicity profiles with both agents," she said.

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Obinutuzumab and Acalabrutinib Yields Faster, Deeper Remissions in Frontline CLL

By ***Leslie Feldman***

Patients with treatment-naive chronic lymphocytic leukemia (CLL) who received acalabrutinib in combination with obinutuzumab achieved deeper and more rapid responses than those treated with acalabrutinib alone, according to findings from a real-world retrospective study presented in abstract CLO26-111.

The single-center study published in the *Journal of the National Comprehensive Cancer Network* included 117 adults with previously untreated CLL who initiated acalabrutinib between January 2018 and August 2025. Investigators compared outcomes among 50 patients treated with acalabrutinib monotherapy and 67 patients who received acalabrutinib plus obinutuzumab.

The results addressed a common question faced by clinicians when selecting frontline therapy for patients with CLL.

"The study was intended to address a practical question that comes up often in clinic: when starting acalabrutinib for treatment-naive CLL, what is gained by adding obinutuzumab, and what additional burden does that add for patients?" **Truman Koh, MD,** of the University of Texas Southwestern Medical Center told *Blood Cancers Today*.

Truman Koh, MD

Patients who received the combination experienced substantially deeper responses. Seventy percent of patients treated with acalabrutinib plus obinutuzumab achieved a complete response, complete response with incomplete marrow recovery, or complete response with lymphocytosis, compared with just 12% of those receiving acalabrutinib alone. The overall response rate was also higher with combination therapy, at 89% versus 67%.

Responses also occurred much sooner in the combination arm. The median time to first response was 76.5 days for patients receiving acalabrutinib plus obinutuzumab, compared with 576 days among those treated with acalabrutinib alone. Most patients completed the planned six cycles of obinutuzumab within approximately 5 months.

Although measurable residual disease (MRD) testing was only performed in a subset of patients, the available data suggested a trend toward deeper molecular responses with the addition of obinutuzumab. One of eight evaluable patients in the combination group achieved MRD negativity, while none of seven evaluable patients receiving monotherapy did so.

Despite the improved depth and speed of response, the addition of obinutuzumab was associated with greater treatment complexity and a higher frequency of adverse events.

"In our real-world cohort, the combination was associated with deeper and faster responses, but it also came with more infusion-related logistics, temporary treatment holds, and low-grade toxicities such as bruising, bleeding, and hypertension," Dr. Koh said.

Investigators reported that most adverse events in both treatment groups were grade 1 or 2. Serious grade 3 or 4 toxicities were relatively uncommon, occurring in 9% of events in the combination group and 7% in the monotherapy group. Rates of atrial fibrillation remained low in both cohorts.

However, bleeding and bruising events were more frequently observed among patients receiving obinutuzumab, as were cases of hypertension. Temporary interruptions of acalabrutinib treatment were also more common with combination therapy, affecting 47% of patients compared with 30% of those receiving acalabrutinib alone. These interruptions were generally related to bleeding events, elevated blood pressure or planned procedures.

At the time of analysis, neither median progression-free survival nor overall survival had been reached. Investigators found no statistically significant differences between treatment groups for either end point, suggesting that longer follow-up will be needed to determine whether deeper responses ultimately translate into improved long-term outcomes.

According to Dr. Koh, the findings highlight the importance of individualizing frontline treatment decisions rather than favoring one approach universally.

"Both approaches remain reasonable, but they may fit different patients," he said. "For some patients, the priority may be achieving the deepest response possible. For others, especially those with comorbidities, bleeding risk, or difficulty coming in for infusions, acalabrutinib alone may still be the better choice."

The study's retrospective design and single-center setting limit the ability to draw definitive conclusions regarding comparative efficacy. Nevertheless, the data provide insight into how the two approaches perform outside of clinical trials and may help guide discussions between physicians and patients.

"These data help frame that discussion more clearly and support a shared decision-making approach between patients and clinicians," Dr. Koh added.

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European Hematology Association Awards Excellence at Annual Meeting

*By **Melissa Badamo***

As the sun set on the European Hematology Association (EHA) 2026 Congress in Stockholm, Sweden, held June 11-14, six leading hematologists were recognized for their commitment to clinical and research excellence, education and mentoring, and diversity and inclusion.[1]

EHA Lifetime Achievement Award

Claire Harrison, MD, a professor of hematology and deputy chief medical officer at Guy's and St. Thomas' NHS Foundation Trust in London, England, received the EHA Lifetime Achievement Award for her sustained contributions to hematology over more than 20 years.[1] An expert in myeloproliferative neoplasms (MPNs), Dr. Harrison has led clinical trials on Janus kinase inhibitors in myelofibrosis and polycythemia vera and is the cofounder of MPN Voice, a nonprofit organization that provides resources and information to patients and their families.[2]

Claire Harrison, MD

"I consider my career to date to have spanned a period of great change in hematology, and especially in MPNs, as we have traveled on a journey from the original descriptions from [William] Dameshek and others through molecular discovery, new therapies, and now to the era of disease modification," Dr. Harrison said. "I am grateful to continue to work with this community and for the incredible support of my local team and, most importantly, my family."

EHA Clinical Excellence Award

Josep-Maria Ribera, MD, PhD, director of the Stem Cell Transplantation Unit and head of the Clinical Hematology Department for the Catalan Institute of Oncology at the Hospital Universitari Germans Trias i Pujol in Spain, received the EHA Clinical Excellence Award for his significant advancements in the field of hematology-oncology and his "pivotal role" in European cooperative groups and international scientific committees.[1] Dr. Ribera's research focuses on novel treatment approaches to adult acute lymphoblastic leukemia and HIV-related lymphomas.[3]

Josep-Maria Ribera, MD, PhD

"This award highlights my role in national and European cooperative groups to develop diagnostic and therapeutic protocols for hematologic malignancies, together with my work in training and mentoring new generations of specialists," Dr. Ribera said. "However, this award is in fact shared by multiple colleagues that have been involved in these tasks."

EHA Research Excellence Award

George Vassiliou, MD, a professor of hematological medicine at the University of Cambridge and consultant hematologist at Cambridge University Hospitals in England, is the winner of this year's EHA Research Excellence Award for his leadership and pioneering research in the biology, pathogenesis, and treatment of myeloid malignancies.[1] With a focus on cancer genomics, Dr. Vassiliou studies somatic mutation drivers in acute myeloid leukemia to develop novel approaches for detection and prevention.[4]

George Vassiliou, MD

"Whatever progress we have achieved reflects the efforts of a remarkable community united by the shared goal of improving the lives of people affected by blood cancers," Dr. Vassiliou said. "I hope this recognition inspires more young scientists and clinicians to devote their talents to preventing and treating blood cancers."

EHA Education and Mentoring Award

Raúl Córdoba Mascuñano, MD, PhD, an attending physician in the Department of Hematology at the MD Anderson Cancer Center Madrid, received the Education and Mentoring Award for his commitment to guiding young professionals to success. He is credited with fostering collaborative learning, contributing to EHA's educational initiatives and international programs, and inspiring critical thinking through formal teaching.[1]

Raúl Córdoba Mascuñano, MD, PhD

"What makes this recognition especially meaningful is that it comes in the middle of my career rather than at its end," said Dr. Córdoba Mascuñano. "To me, it is much more than an acknowledgment of past achievements—it is a powerful encouragement to continue doing what I love most: educating, mentoring, and helping shape the next generation of hematologists."

EHA Diversity, Equity, and Inclusion Award

Gianluca Gaidano, MD, PhD, a professor of hematology and deputy rector at the University of Eastern Piedmont in Italy, received the EHA Diversity, Equity, and Inclusion Award. As Chair of the EHA Global Outreach Program Committee, Dr. Gaidano fosters international collaborations to promote equitable access to research and patient care in hematology.[1] His research focuses on precision medicine and molecular predictors of lymphoid malignancies such as non-Hodgkin's lymphoma and chronic lymphocytic leukemia.[5]

Gianluca Gaidano, MD, PhD

Young EHA Award

Nico Gagelmann, MD

Nico Gagelmann, MD, a hematologist oncologist in the Department of Stem Cell Transplantation at the University Medical Center Hamburg-Eppendorf in Germany, received the Young EHA Award for his research in stem cell transplantation and cellular therapy, leadership and mentorship, and efforts towards building global collaborative networks among young hematologists.[1]

"To me, one of the most rewarding aspects of science is being part of a generation of young researchers who openly exchange ideas, support one another, and build collaborations across institutions and countries," Dr. Gagelmann told *Blood Cancers Today*. "Creating an environment where early-career scientists have the freedom to think independently while learning from each other is essential for driving innovation and improving patient care in a time of unprecedented opportunities but also dangers to freedom."

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Calendar

September 9-12
Society of Hematologic Oncology (SOHO) Fourteenth Annual Meeting
Houston, TX

September 11-13
Association of VA Hematology/Oncology (AVAHO) 21st Annual Meeting
Austin, TX

September 18-19
8th Annual The Leadership, Empowerment, And Development (LEAD) Conference
Washington, DC

September 18-19
NCCN 2026 Annual Congress: Hematologic Malignancies
New York, NY

October 13-16
Lymphoma, Leukemia & Myeloma Congress
New York, NY

October 16-17
2026 National Summit on Hematologic Cancers
Nashville, TN

October 23-27
European Society for Medical Oncology Congress 2026
Madrid, Spain

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figure1

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