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Does the Clock Matter for CAR T-Cells?

*By **Charles Gaulin, MD***

The intersection of chronobiology and oncology has long fascinated researchers, offering the possibility that the body's endogenous circadian timing system could be leveraged to maximize treatment efficacy. Circadian biology influences various aspects of antitumor immunity, including leukocyte trafficking, activation states, and the overall tumor microenvironment.[1] Correspondingly, studies in solid tumors have indicated that patients receiving immune checkpoint inhibitors earlier in the day experience better outcomes.[2]

While the data surrounding solid tumors and checkpoint inhibitors are growing, the impact of infusion timing on chimeric antigen receptor (CAR) T-cell therapy has only recently come to light. Two large retrospective studies sought to answer whether the clock on the wall matters for patients with large B-cell lymphoma (LBCL) receiving CAR T-cell therapy.

A large international, multicenter retrospective study by Luan and colleagues[3] provides compelling evidence that the timing of CAR T-cell infusion may significantly influence therapeutic efficacy.

This multicenter analysis of 1,052 adult patients with LBCL assessed the impact of infusion timing on clinical outcomes following CD19-directed CAR T-cell therapy. The cohort included patients treated with axicabtagene ciloleucel (axi-cel), tisagenlecleucel (tisa-cel), and lisocabtagene maraleucel (liso-cel), with a median infusion time of 11:48 AM.

The results demonstrated a distinct and statistically significant progression-free survival (PFS) advantage for early infusions. Each hour later in infusion time was associated with an increased risk for progression, relapse, or death, showing a hazard ratio of 1.11 after adjustment for center, product, and key clinical variables. The one-year PFS rate was 51.4% for patients infused early (before 12:00 PM). In contrast, the one-year PFS rate was only 35.2% for patients who received a late infusion (at or after 12:00 PM). This PFS benefit was primarily driven by lower relapse rates and higher complete response rates in the early infusion group. Overall survival (OS) between the early and late groups was similar.

While rates of severe immune toxicities—such as cytokine release syndrome (CRS) and immune effector cell-associated neurotoxicity syndrome (ICANS)—were comparable between groups, late infusions were correlated with a more inflammatory systemic milieu.

Standing in contrast to the study by Luan and colleagues are recently published data from the Australian CAR-T Real-World Consortium (AusCART).[4] In this study, presented at the European Hematology Association 8th European CAR T-cell Meeting, Dowling and colleagues suggest that in real-world practice, time of infusion might not impact outcomes in LBCL.

In this retrospective analysis, investigators evaluated 584 patients with LBCL treated across six Australian centers. Unlike the cohort in the study by Luan and colleagues, this study evaluated only axi-cel (68%) and tisa-cel (32%). Notably, the median infusion time in this cohort was later in the day, occurring at 1:20 PM. The investigators found no association between the time of infusion and PFS or OS, even after accounting for known confounders. Grade 3 or higher CRS and ICANS occurred at similar rates.

The authors of the Australian study concluded that any effect of infusion time on real-world effectiveness or toxicity is small relative to established clinical and disease-related predictors. They postulate that the administration of lymphodepleting chemotherapy, combined with the disrupted circadian rhythms inherently present in unwell patients with active lymphoma, may mitigate the impact of cytokine-driven chronobiology.

Comparing the Evidence: Why the Discrepancy?

A deeper look at the cohorts reveals differences that may shed light on the divergent results. Luan and colleagues included liso-cel (17.7% of the cohort),[3] whereas Dowling and colleagues exclusively evaluated axi-cel and tisa-cel.[4]

Could the specific CAR T-cell construct—particularly the use of CD28 versus 4-1BB costimulatory domains—have varying sensitivities to the patient's endogenous circadian rhythms? Dr. Edward Cliff, Haematology Registrar at the Peter MacCallum Cancer Center and author on the Australian study, notes this is a possibility. "This could be the case, although I'm not sure whether this was evaluated in the in vitro literature or not," he explained. To ensure their results weren't skewed by construct differences, the AusCART team performed a sensitivity analysis looking exclusively at the 395 patients treated with axi-cel, but again saw no difference between groups.

Timing mechanics may also play a role. Dowling and colleagues noted that most patients received their infusions in the middle of the day (from 12:00 PM to 3:00 PM), with a median time of 1:20 PM, which may have reduced the observable effect size.[4] The study by Luan and colleagues had an earlier median infusion time of 11:48 AM and a wider comparative window.[3]

When asked if the lack of patients treated at the extreme ends of the circadian cycle (such as 8:00 AM or 6:00 PM) might have masked a true chronobiological effect, Dr. Cliff acknowledged the limitation. "Absolutely, this could be the case," he said, noting that there was not even a trend toward a difference in their primary analysis. "We did two analyses to try to tease this question out further: first, we compared only patients infused prior to midday with those infused after 3:00 PM, excluding the many patients infused between 12:00 and 3:00 PM. No difference. Then we did a continuous analysis using splines... and again saw no difference. If the early bird gets the worm, who knows how early!"

As noted in both studies, "clock time" does not necessarily equate to individual "circadian phase," which can be drastically altered by hospitalization, illness severity, and prior therapies. Dr. Cliff views this shift as the next logical step. "This question is an interesting one biologically, but ultimately will come down to pragmatism, I think," he explained. "My suspicion is that the first randomized studies will have to start simple—but if there is a convincing effect, then the next step would be to nail down the optimal timing for each individual patient. It will be an interesting space to watch in the coming years."

Taken together, the uncertainty surrounding the true impact of CAR T-cell infusion timing can only be definitively resolved through well-designed, randomized, prospective clinical trials. Until such data are available, while optimizing infusion times to the morning remains a potentially low-cost consideration, physicians should continue to prioritize established clinical predictors and the logistical realities of high-quality patient care.

REFERENCES

1. Fortin BM, et al. *Nat Immunol.* 2024;25(7):1257-1269. doi:10.1038/s41590-024-01859-0
2. Huang Z, et al. *Nat Med.* Published online February 2, 2026. doi:10.1038/s41591-025-04181-w
3. Luan D, et al. *Blood.* 2026;147(12):1315-1322. doi:10.1182/blood.2025031476
4. Dowling MR, et al. *Blood.* Published online February 17, 2026. doi:10.1182/blood.2025032307

Designing Trials for the Patients We Actually Treat

Tycel Phillips, MD

When a new therapy gets approved based on the positive results of a pivotal trial, we all want to believe those outcomes will translate to our clinics. But increasingly, I find myself having difficult conversations with patients about why their response might not match the responses seen in the clinical trials. One major contributor is the differences between clinical trial populations and the patients sitting in front of us in the clinic or those that we meet in the hospital—the "real-world patients."

The issue relates to the overall patient selection. Clinical trials, by necessity, enroll healthier patients—people who meet strict lab parameters, have a good performance status, and have what trial sponsors consider "manageable" disease. These patients are more likely to complete treatment, show good responses, and contribute to clean, publishable data. I understand the rationale, but the consequence is that trial results often overestimate what we will see when treating the patients in our practices. Added to this is the reality of medication adherence. In trials, we assume or can enforce almost near-perfect adherence to treatment with the study drug. In my practice, patients miss doses for countless reasons. If a drug's efficacy depends heavily on strict adherence, the real-world results will diverge significantly from trial outcomes.

Control arm selection presents another challenge. Take the STARGLO trial as an example. This global phase 3 study compared glofitamab plus gemcitabine and oxaliplatin (GemOx) with rituximab-GemOx (R-GemOx) in patients with relapsed or refractory diffuse large B-cell lymphoma (DLBCL). Regional analyses showed substantial efficacy differences, but much of this variation traces back to what treatment options are actually available in different countries. The standard-of-care arm used R-GemOx, which has proven to be inferior for many patient populations and is of limited use in countries with access to more effective therapies. This issue particularly influenced participation in the United States, where enrollment was minimal. Why was R-GemOx used? Because it is accessible in certain countries and acceptable to certain regulatory agencies. A more meaningful comparison

PHOTO CREDIT: istock.com/Left to right: LaylaBird; Andy; PixelsEffect

would have been glofitamab-GemOx versus chimeric antigen receptor (CAR) T-cell therapy, but a study like this will likely never happen. No pharmaceutical company wants to fund a competitor's expensive therapy, no CAR T-cell manufacturer sees the upside, and no healthcare system wants to shoulder those costs for a trial that might not favor either arm.

The inclusion and exclusion criteria also create artificial barriers. Patients get excluded because they have cytopenias that are disease related and would not actually impact safety or efficacy. Patients cannot receive transfusions for arbitrary periods or use steroids before enrollment, even when these interventions would not interfere with the investigational treatment. Some trials require fresh tissue biopsy specimens when we already have confirmed diagnoses, subjecting patients to unnecessary procedures we would not do in routine practice. These restrictions exist to streamline enrollment and attract "healthier" patients who make the drug look good, not to test whether the therapy works for the sicker patients we see in day-to-day practice.

Certain patient populations have historically been excluded from trials. Patients with hepatitis B, hepatitis C, or HIV were routinely kept out of clinical studies. Thankfully, this has changed over the past several years—at least in the US—and many trials now include these patients. But the problem of exclusion has not disappeared. It has just shifted. Today, patients with cytopenias represent the group most excluded, despite cytopenia being a common problem for most of the patients I treat daily in practice.

The consequences of this trial-reality mismatch are significant. When a drug shows 10 months of progression-free survival in a trial but delivers only 4 months in my clinic, that is not a small difference: it fundamentally changes the risk-benefit discussion I have with patients. Community oncologists who do not have time to dissect every trial's details may inadvertently use treatments in populations where they were never properly studied, basing decisions on inflated expectations.

The approval of lenalidomide plus tafasitamab for relapsed or refractory DLBCL illustrates this problem perfectly. The pivotal trial demonstrated effectiveness in a specific patient population, but it was not designed to evaluate patients with refractory disease, nor was the regimen studied when more contemporary regimens were not commonly used. When we started using this regimen in the real world for third- and fourth-line patients, the response rates fell far short of published results. Only after accumulating real-world data and clinical experience did it become clear that the trial population represented a narrow niche. For most patients I see, I have had to significantly lower my expectations and adjust conversations accordingly, or look for different options altogether.

Some trials are starting to get this right. The second-line CAR T-cell studies that used lisocabtagene maraleucel allowed bridging therapy before randomization, letting us stabilize patients before they entered the study. Earlier trials did not allow bridging, which does not reflect actual practice and could limit enrollment of patients with aggressive disease. When patients experience relapse with DLBCL, the disease often progresses quickly. Allowing bridging therapy enables enrollment of patients who more closely resemble those we treat every day.

"We need trial designs that reflect clinical reality, not idealized versions of it."

When I am treating patients who do not fit trial profiles, I go back to the studies that led to the drug's approval and read carefully. I look for specific factors that might predict poor response in the patient in front of me. If nothing in the trial data suggests my patient would fare worse, I proceed. But this requires time to parse the nuances of inclusion and exclusion criteria, something not every clinician has the time to do.

We need trial designs that reflect clinical reality, not idealized versions of it. That means relaxing lab cutoffs, allowing supportive care, comparing investigational therapies with what we actually use in practice, and enrolling patients who better reflect those we see in real-world practice. Until we close this gap between trial populations and real-world patients, we will continue to have difficult conversations about why the promising results we read about do not always materialize at the bedside.

Get to Know

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

Nihar Desai, MD

Dr. Nihar Desai reflects on his passion for medicine and wildlife photography, his personal experience with allogenic stem cell transplant, and how it inspired him to become a transplant physician and optimize GVHD prophylaxis.

*By **Melissa Badamo***

With a click of a camera, **Nihar Desai, MD,** captures the stunning greens of Scotland, golden sunrises over the Grand Canyon, and the bountiful forests of British Columbia. *Focus, zoom, capture.* It is this same vision and focus that led him to medical school to become a hematologist-oncologist.

Dr. Desai grew up in Mumbai, India, where he received his medical degree and completed an internal medicine residency. He was introduced to academia and research at the Sanjay Gandhi Postgraduate Institute of Medical Sciences, one of the largest hematology training programs in India, an experience that would later prepare him for his current role as a hematologist and stem cell transplant physician at the Princess Margaret Cancer Centre in Toronto, Canada.

For Dr. Desai, transplant hits close to home. In 2008, his father received an allogenic stem cell transplant for blast-phase chronic myeloid leukemia. Despite having a sibling donor transplant and achieving remission, he developed complications from the procedure and died of graft-versus-host disease (GVHD) around day 35.

"I experienced the journey from the perspective of a family member before I was a medical trainee," Dr. Desai reflected. "This gave me a firsthand understanding of the challenges that a caregiver faces, but also the opportunities that being a transplant physician gives you because I've seen people have successful transplants as well. That's when I realized I would love to become a transplant physician. The way the field was evolving while I was a hematology trainee further strengthened my resolve to do transplant medicine and cellular therapies."

As an early-career clinician and researcher, Dr. Desai focuses his research on improving transplant outcomes and optimizing post-transplant cyclophosphamide to mitigate early toxicities.

"The field has moved a long way. What moved the needle was the introduction of post-transplant cyclophosphamide and the fact that it's now being used across donor types," he said. "Despite its promises, we know that post-transplant cyclophosphamide is sometimes difficult to administer and has some early toxicities."

Dr. Desai and his research team observed promising outcomes in a pilot cohort of patients with acute myeloid leukemia (AML) who received matched unrelated donor allogeneic hematopoietic stem cell transplants (HSCTs) after reducing the cyclophosphamide dose by 30% from 50 mg/kg to 35 mg/kg. The lower dose was associated with a lower risk of bloodstream infections and cardiac events without compromising relapse and overall survival.[1] Dr. Desai and his team continue to expand on this pilot cohort to include more patients with AML.

In collaboration with centers across Europe and North America, Dr. Desai and his mentor **Jonas Mattsson, MD, PhD,** are also researching a next-generation sequencing–based assay to identify haplotype loss in patients with leukemia. Dr. Desai presented preliminary findings at the Cell Therapy Transplant Canada 2026 Annual Meeting in Ottawa in May, receiving the Best Research Abstract Award for his work.

"When we conducted an international survey, we identified haplotype testing as a gap," he said. "People are aware of the loss of haplotype as a mechanism, but they don't have these tests available rapidly, which can give them results so that we can act on those haplotype loss mechanisms. The goal was to provide a cohort of patients to test this assay. So far, the test has proved to be robust when compared to the other standard methods, and the major advantage is the turnaround time is around 4 to 5 days. It's giving us results in a clinically actionable time frame."

In the next 5 to 10 years, Dr. Desai hopes to improve long-term survivorship after transplant. Despite high rates of survival 10 years after HSCT, a study published in the *Journal of Clinical Oncology* found that life expectancy remains lower than expected.[2]

"By virtue of how our systems are built, there's not much

PHOTOS COURTESY OF NIHAR DESAI, MD

opportunity for longitudinally following these patients beyond the first 2 to 3 years, which is when most centers would actively follow them and look for relapse," he explained. "But a lot of patients fall through the cracks somewhere between 5 or 6 years. There should be a long-term survivorship clinic."

According to Dr. Desai, large transplant centers like Princess Margaret have resources available to follow up patients for life within the Canadian system. However, this might not be feasible for smaller transplant centers in different healthcare settings.

"My goal is to do research in the survivorship space to generate high-quality data which tells us that there is definitely a need to follow these patients," he said. "Once you have a transplant, a lot of things change. If patients are not followed up, there is a chance that these things might be missed over time."

For example, patients who received a transplant have a higher risk for secondary malignancies and cardiovascular complications after transplant. Recurrent disease, chronic GVHD, infection, organ toxicity, and second cancers are the most common causes of death after transplant.[2]

"Optimization of GVHD prophylaxis has been shown to reduce the incidence of second malignancies," said Dr. Desai. "We saw that in a recent cohort of ours that we published. We hypothesized that the reduction in GVHD, which leads to chronic inflammation, is leading to a reduction in second malignancies down the road. PTCy [post-transplant cyclophosphamide] and optimized prophylaxis don't just stop at reducing GVHD. There are downstream effects of improved GVHD prophylaxis as well."

According to Dr. Desai, there is a growing emphasis on patient-reported outcomes. "Even on Twitter, there's dialogue about this," he continued. "It's not just about the biology of the disease and making these therapies, but what happens downstream and how the patients are feeling."

As an early-career physician, Dr. Desai is heavily involved in the Twitter/X medical community to share dialogue, discuss research findings, and build connections.

"There are a lot of nuances to these discussions, which you may not gather just from reading the journal article, but from the comments that are posted under it. That is a unique feature of Twitter. You can connect with a key opinion leader in the field and have a dialogue with them one-on-one. It's a great platform to teach and learn," he said.

Dr. Desai is also a member of the European Society for Blood and Marrow Transplantation (EBMT) Equality, Diversity & Inclusion Committee, fostering connections on social media. A global community of healthcare professionals, the EBMT works to challenge health inequalities in patient care.[3]

"I've developed an interest in equitable access, which is something we need to be working on," he said. "All these amazing therapies won't make a big impact in the world until the majority of the population has access to them."

Outside of work and social media, Dr. Desai is a drummer and avid wildlife photographer, complementing his love for travel. From capturing the spry orcas in British Columbia to the majestic tigers of India, Dr. Desai first picked up the camera around the time of his father's death as a creative outlet.

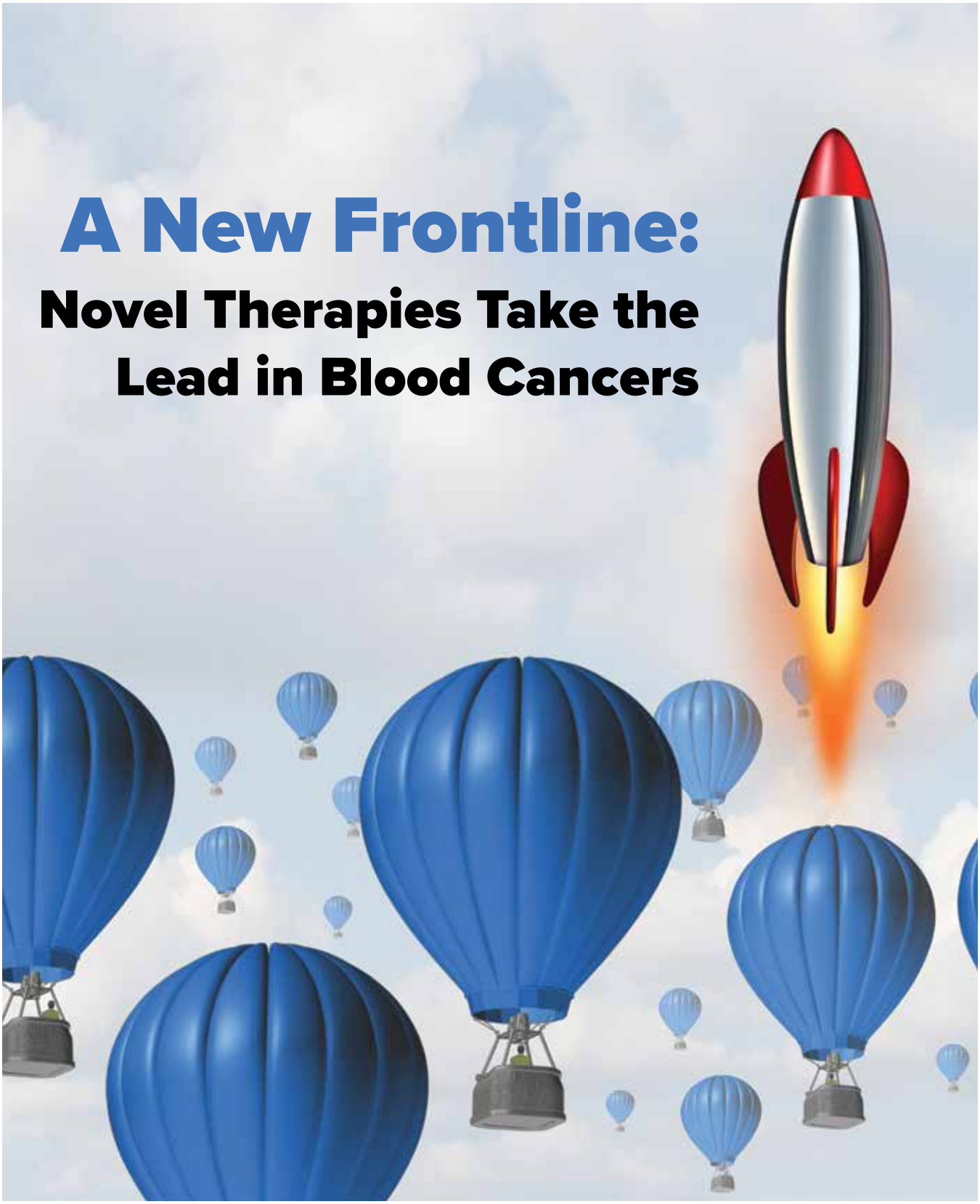
"I've been doing wildlife photography for almost 14 or 15 years now," he said. "I picked up the camera, learned a few tricks of photography from mentors, workshopped with photographers, and spent time in the forest to connect with nature. That was what I needed at the time. Recently, I hosted dear friends from the EBMT and gave them a tour of the wildlife parks in India. That's a way to build connections outside of work through hobbies. That's something I truly enjoy."

REFERENCES

1. Desai N, et al. *Br J Haematol.* 2026;208(4):1359-1368. doi:10.1111/bjh.70380
2. Wingard JR, et al. *J Clin Oncol.* 2011;29(16):2230-2239. doi:10.1200/JCO.2010.33.7212
3. EBMT. Accessed April 28, 2026. https://www.ebmt.org/ebmt/who-we-are

In Focus

Blood Cancers Today *takes an in-depth look at hot topics in hematologic oncology*



A New Frontline:

Novel Therapies Take the Lead in Blood Cancers

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By ***Sara Karlovitch***

Novel agents have proven increasingly valuable for the treatment of many hematologic malignancies, such as polatuzumab vedotin for diffuse large B-cell lymphoma (DLBCL)[1] and Bruton's tyrosine kinase (BTK) inhibitors for mantle cell lymphoma (MCL), chronic lymphocytic leukemia (CLL), and other B-cell malignancies.[2] As novel agents improve patient outcomes, there has been an effort to evaluate moving such treatments up earlier in the treatment process, potentially improving results along with reducing toxicities.

"There has been a clear and sustained shift toward incorporating novel agents earlier in frontline therapy across many hematologic malignancies," said **Tejo Musunuru, MD,** an associate professor in the Departments of General Oncology and Leukemia within the Division of Cancer Medicine at The University of Texas MD Anderson Cancer Center. "These include targeted oral therapies, antibody-drug conjugates, bispecific antibodies, and immune-based approaches, often replacing or substantially reducing reliance on conventional cytotoxic chemotherapy."

While BTK inhibitors, chimeric antigen receptor (CAR) T-cell therapy,[3] and other novel and emerging treatments have improved patient outcomes, progress varies greatly across different hematologic malignancies. Not only are novel agents being used uniquely among different cancer types, but the associated toxicities and improvements to patients' outcomes can vary widely.

Frontline Update: MCL

In recent years, BTK inhibitors, most notably ibrutinib, emerged as the new standard of care for young and medically fit patients with MCL.[4]

"In MCL, the integration of BTK inhibitors combined with frontline therapy suggests that patients do not need to have an autologous stem cell transplant (ASCT) to consolidate their first remission," said **Catherine D. Diefenbach, MD,** associate professor at NYU Langone Health's Perlmutter Cancer Center.

The three-arm, randomized, open-label, phase 3 TRIANGLE study established the efficacy of ibrutinib for the treatment of MCL, with results published in *The Lancet* in May 2024. The study was conducted in 165 secondary or tertiary clinical centers across 13 European countries and Israel. Patients were between the ages of 18 and 65 years and were diagnosed with stage II-IV MCL. Additionally, all patients were untreated and eligible for ASCT. Enrollees were randomly assigned 1:1:1 to control group A, experimental group A+I, or group I.

Patients in group A received six alternating cycles of rituximab, cyclophosphamide, doxorubicin, vincristine, and prednisolone (R-CHOP) and rituximab, dexamethasone, cytarabine, and cisplatin (R-DHAP) before ASCT. Patients in group A+I were administered 560 mg of oral ibrutinib twice daily on days 1 through 19 of R-CHOP cycles before dropping down to 560 mg daily for 2 years after ASCT. Group I received the same ibrutinib regimen but did not undergo ASCT. The primary outcome was failure-free survival.

In total, 870 patients (662 men and 208 women) were enrolled between July 29, 2016, and December 28, 2020. Group A contained 288 patients, group A+I had 292 patients, and group I had 290 patients. Group A+I had a 3-year failure-free survival rate of 88%, and group A had a rate of 72% after a median follow-up of 31 months. Group I had a 3-year failure-free survival rate of 86%. Comparison of groups A+I and I is ongoing at the time of study publication. While those treated with R-CHOP/R-DHAP with or without ibrutinib had no relevant differences in grades 3-5 adverse events (AEs) before or after induction or ASCT, there were significantly more grade 3-5 hematologic AEs and infections after ASCT for those who received ibrutinib.[4]

Frontline Update: DLBCL

In previously untreated intermediate/high-risk DLBCL, the POLARIX study showed polatuzumab vedotin plus rituximab, cyclophosphamide, doxorubicin, and prednisone (Pola-R-CHP) significantly improved progression-free survival (PFS) versus R-CHOP alone.[5] A 5-year follow-up of the POLARIX study published in *Journal of Clinical Oncology* in September 2025 found the addition of polatuzumab continued to demonstrate improvements in PFS compared to R-CHOP alone. The study team initially enrolled a global population of 879 patients from across 22 countries, later expanding to an additional 121 patients from a Chinese extension cohort.

When looking at the expanded population, investigators placed 500 patients in the Pola-R-CHP arm and 500 in the R-CHOP arm. Each group had similar characteristics. In this population, 5-year progression-free survival (PFS) was substantially higher in patients who received Pola-R-CHP compared to those who received R-CHOP alone. In both arms, there were 197 deaths total, with 90 occurring in the Pola-R-CHP arm. Forty-six of those deaths were due to progressive disease, 25 were not related to the disease, and 19 had unknown causes. Of the 107 deaths that occurred in the R-CHOP arm, 62 were due to progressive disease, 30 were not related to the disease, and 15 had unknown causes.[5]

Frontline Update: CLL

The randomized CLL17 trial, presented at the 67th American Society of Hematology Annual Meeting & Exposition,

found that fixed-duration treatment with venetoclax plus obinutuzumab (VO) and venetoclax plus ibrutinib (VI) for the treatment of CLL is noninferior to ibrutinib alone.[6]

"[The study] showed that if you use fixed-duration therapies that are either venetoclax and BTK inhibitor–based, or venetoclax inhibitor–based, the patients had essentially comparable outcomes to those who are on BTK inhibitors continuously," said **Krish Patel, MD,** director of lymphoma research at the Sarah Cannon Research Institute. "It's really a next step forward and improving our therapies using novel agents in CLL."

In the phase 3 international trial, patients with previously untreated CLL were randomized to receive ibrutinib (I), fixed-duration venetoclax plus obinutuzumab (VO), or fixed-duration venetoclax plus ibrutinib (VI). Those in the I arm received treatment continuously until progression or intolerance occurred. In the VO arm, treatment was administered in six 28-day cycles of both drugs before six cycles of venetoclax monotherapy. Patients in the VI arm received 3 cycles of ibrutinib lead-in followed by 12 cycles of venetoclax and ibrutinib. The primary end point was PFS.

"Clinical practice is increasingly shifting away from rigid 'lines of therapy' toward a long-term disease management framework, with careful sequencing of therapies across the patient's disease course. "

—Tejo Musunuru, MD, associate professor, University of Texas MD Anderson Cancer Center

More than 900 patients were placed into the three arms, with 303 in the VO arm, 305 in the VI arm, and 301 in I. The majority of patients (67.8%) were male, the median observation time was 34.2 months, and the median age was 66. Three-year PFS was 81.1% in the VO arm, 78.4% in the VI arm, and 81% in the I arm. The overall response rate was 84.2% in the VO arm, 88.5% in the VI arm, and 86% in the I arm. The overall survival (OS) rate was highest in the VI arm at 96%, followed by 95.7% in the I arm, and 91.5% in the VO arm.[6]

Frontline Update: AML

In acute myeloid leukemia (AML), the use of novel agents has largely been to help improve outcomes and reduce toxicities in older or less fit patients. This benefit was established in a 2018 *Blood* study titled "Venetoclax combined with decitabine or azacitidine in treatment-naive, elderly patients with acute myeloid leukemia."

"It has offered real benefit to patients who otherwise couldn't tolerate standard treatment for AML, and there are trials that suggest azacitidine and venetoclax may be as good as seven plus three, even for younger patients with AML," said **Richard A. Larson, MD,** clinical professor of medicine and director of the Hematologic Malignancies Program at the University of Chicago.

The study, which enrolled 145 patients with treatment-naive AML aged 65 years and older who were ineligible for intensive chemotherapy, found the combination was well tolerated. The median duration for the entire study population was 11.3 months with a median OS of 17.5 months. The agents are well tolerated in the patient population, which has a median age of 74. Poor-risk cytogenetics was present in 49% of patients.[7]

The Evolving Treatment Algorithm

Novel agents have transformed frontline care across hematologic malignancies. Many of these treatments have led to later relapse and longer disease control. This is changing the way oncologists are thinking about effective patient care, according to Dr. Musunuru.

"Clinical practice is increasingly shifting away from rigid 'lines of therapy' toward a long-term disease management framework, with careful sequencing of therapies across the patient's disease course," said Dr. Musunuru.

As the treatment landscape continues to change quite rapidly, practitioners need to keep their knowledge up-to-date. New therapies come with new risks and considerations, which oncologists need to consider as they look to implement novel agents into patient care.

"The rapid evolution of blood cancer therapies highlights the importance of continuous education, collaboration, and adaptable care models, particularly as treatments become more immune-based and less forgiving of delayed toxicity recognition," said Dr. Musunuru. "While long-term disease control is now increasingly achievable, it comes with added responsibilities related to monitoring, survivorship, and thoughtful treatment sequencing."

REFERENCES

1. Camus V, et al. *Future Oncol.* 2021 Jan;17(2):127-135. doi:10.2217/fon-2020-0675
2. Tam C, et al. *Blood Adv.* 2024;8(9):2300-2309. doi:10.1182/bloodadvances.2023012221
3. Zugasti I, et al. *Signal Transduct Target Ther.* 2025;10(1):210. doi:10.1038/s41392-025-02269-w
4. Dreyling M, et al. *Lancet.* 2024;403(10441):2293-2306. doi:10.1016/S0140-6736(24)00184-3
5. Morschhauser F, et al. *J Clin Oncol.* 2025;43(35):3698-3705. doi:10.1200/JCO-25-00925
6. Al-Sawaf O, et al. *Blood 2025*; 146(1):1. doi:10.1182/blood-2025-1
7. DiNardo CD, et al. *Blood.* 2019;133(1):7-17. doi:10.1182/blood-2018-08-868752

NIH NATIONAL CANCER INSTITUTE

NCI: The Next Era of Cancer Research

*By **Anthony Letai, MD, PhD***

Over more than two decades as a medical oncologist and laboratory scientist, I had the privilege of working on a therapy that ultimately became venetoclax—an advance in leukemia treatment that would not have been possible without sustained support from the National Cancer Institute (NCI). My vantage point has changed, but my purpose has not. The mission of the NCI remains what it has always been: to reduce the burden of cancer and, when possible, to prevent it.

As the NCI director, my priorities are both practical and strategic. In the near term, I believe we need to reinforce confidence in the cancer research enterprise by clearly communicating what is actually happening. There has been understandable concern about funding and stability. Some of that reflects real changes in how funding to support cancer research is administered, but much of it reflects a gap between perception and reality. The facts matter here. The overall "tide" of NCI funding—our investment in the research that takes place at academic centers across the country—is at an all-time high, and we expect to fund more grants for the extramural community in 2026 than in 2025.

At the same time, I recognize that the "waves"—the year-to-year variability in how funding is delivered—can feel turbulent for investigators. Factors such as the transition to multiyear funding and the visibility of administrative delays—particularly after disruptions like last year's government shutdown—can create a real sense of uncertainty. But these dynamics do not reflect a reduction in commitment. NCI's responsibility is to ensure that the strength of the underlying system is strong and that we continue to support the best science across the country.

Beyond that, we are focusing on several scientific and operational priorities that I believe will shape progress over the next 12 to 24 months. Immuno-oncology remains central, and within that, I think it is time for NCI to place some deliberate bets.

One example is cancer therapeutic vaccines. This is an area that has generated interest for decades, often with mixed results. What is different now is the underlying technology and our ability to identify relevant tumor targets more precisely. We are seeing early signals—still preliminary—that in some high-risk settings, therapeutic vaccines may reduce recurrence, which is a major unmet need. The appropriate next step is not to assume success, but to test these approaches rigorously. Through a partnership with the Foundation for the National Institutes of Health, we are supporting a coordinated clinical trial effort to generate clear answers—what works, what doesn't, and in which patients. That is the role NCI can play: helping the field move efficiently from intriguing signal to definitive evidence.

PHOTO CREDIT: National Cancer Institute

"Improving access to screening, prevention, and treatment must be part of how we define progress— not an afterthought."

Also important is functional precision medicine. This builds directly on principles that many of us in hematologic oncology have been thinking about for years: if you want to know whether a drug will work, test it directly on the patient's living cancer cells. These approaches are now far more advanced than many appreciate and have the potential to guide therapy selection, particularly for patients who have exhausted standard options.

A central question for NCI is how we accelerate the translation of discoveries into treatments. Here, the issue is often less about generating ideas and more about how efficiently we test them. We need to shorten the path from discovery to first-in-human trials. That means addressing practical barriers—trial activation timelines, regulatory coordination, and infrastructure—and working closely with cancer centers, industry, and federal partners to streamline the process without compromising scientific rigor.

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We are also evolving how we make funding decisions to better support this goal. Moving away from strict paylines allows us to take a more strategic approach—still grounded in peer review, but with the flexibility to build a portfolio that balances foundational science, translational work, and high-risk, high-reward ideas. The challenge we face is not a lack of strong science, but an abundance of it. Our responsibility is to steward those opportunities in a way that maximizes patient impact.

Another priority is ensuring that advances reach patients wherever they receive care. Most patients with blood cancers are treated in community settings, not academic centers. Programs like the NCI Community Oncology Research Program are essential for extending clinical trials into those settings, allowing patients to participate closer to home and ensuring that new therapies are tested in real-world populations. Improving access to screening, prevention, and treatment must be part of how we define progress—not an afterthought.

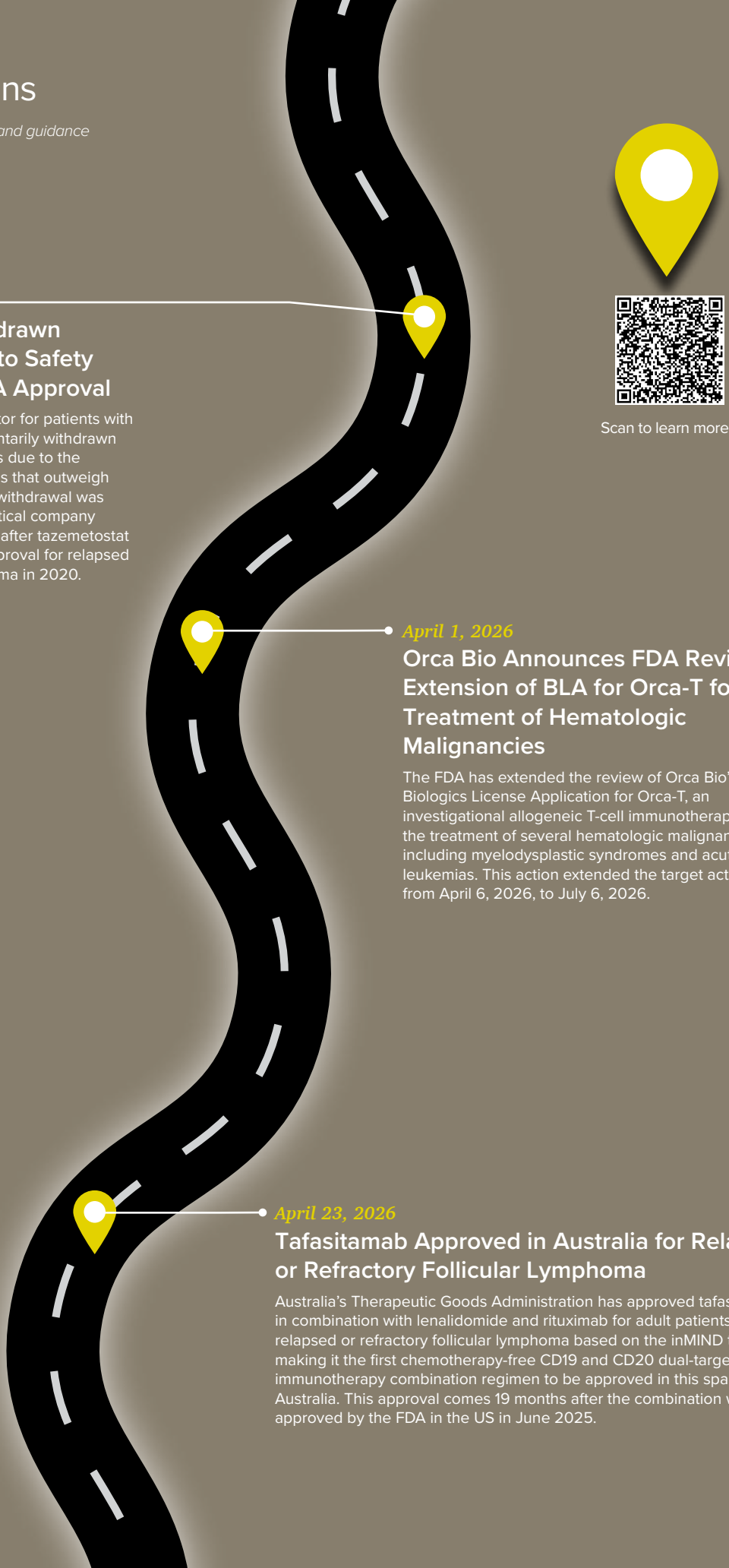
Data, technology, and artificial intelligence (AI) will support all of these efforts in practical ways. There are immediate opportunities to reduce administrative burden, harmonize data across electronic health records, and standardize clinical trial processes. Over time, more integrated datasets will help us better understand response and resistance, particularly in diseases like leukemia and lymphoma. These tools will not replace careful clinical investigation, but they can make that investigation more efficient and informative. I hope the NCI can provide an organizing point, sharing AI solutions that can operate across institutions.

Finally, I want to speak directly to clinicians who are caring for patients who have limited or no remaining options. I have been in that position, and I understand the urgency. What I would emphasize is that the progress we are seeing today is built on decades of work that often began as basic science. Venetoclax is one example, but there are many others. The same pipeline exists today. In immunotherapy, in cellular therapies, and in precision approaches, we are seeing real signals—even in refractory disease—that point to where the next advances will come from.

Not every idea will succeed. But enough will that the standard of care will continue to improve. Our role at NCI is to ensure that the best ideas are tested rigorously, efficiently, and in the right patients—and that the benefits of those discoveries reach every community. If we do that well, then over time the number of patients for whom we have no effective options will continue to decline. That is the goal that unites all of us across hematologic oncology, and it is the standard by which we should measure our progress.

Regulatory Actions

Regulatory approvals, designations, and guidance in the field of hematologic oncology



Scan to learn more.

March 9, 2026

Tazemetostat Withdrawn From Markets Due to Safety Concerns After FDA Approval

Tazemetostat, an EZH2 inhibitor for patients with B-cell malignancies, was voluntarily withdrawn from clinical trials and markets due to the risk of secondary malignancies that outweigh the benefits of the drug. The withdrawal was announced by biopharmaceutical company Ipsen in March 2026, 6 years after tazemetostat received FDA accelerated approval for relapsed or refractory follicular lymphoma in 2020.

April 1, 2026

Orca Bio Announces FDA Review Extension of BLA for Orca-T for the Treatment of Hematologic Malignancies

The FDA has extended the review of Orca Bio's Biologics License Application for Orca-T, an investigational allogeneic T-cell immunotherapy for the treatment of several hematologic malignancies including myelodysplastic syndromes and acute leukemias. This action extended the target action date from April 6, 2026, to July 6, 2026.

April 23, 2026

Tafasitamab Approved in Australia for Relapsed or Refractory Follicular Lymphoma

Australia's Therapeutic Goods Administration has approved tafasitamab in combination with lenalidomide and rituximab for adult patients with relapsed or refractory follicular lymphoma based on the inMIND trial, making it the first chemotherapy-free CD19 and CD20 dual-targeted immunotherapy combination regimen to be approved in this space in Australia. This approval comes 19 months after the combination was approved by the FDA in the US in June 2025.

Highlights from the **AMERICAN ASSOCIATION FOR CANCER RESEARCH (AACR) ANNUAL MEETING**
April 17–22, 2026, in San Diego, California

AI Improves Quality, Efficiency of Evidence-Based Cancer Care at a Resource-Limited Institution

*By **Andrew Moreno***

Artificial intelligence (AI) can improve efficiency and adherence to clinical guidelines for cancer care provided in resource-limited healthcare settings, according to results from a prospective trial on use of Navya Earthshot, a clinician decision-making AI assistance platform, at a safety-net hospital.

The results were published online for the 2026 American Association for Cancer Research (AACR) Annual Meeting and presented at the on-site event in San Diego, California.

"Cancer care guidelines are complex and frequently evolving, and timely multidisciplinary case-review is often infeasible. This makes it challenging for clinicians to consistently provide evidence-based care, particularly in resource-constrained settings," **Naresh Ramarajan, MD,** cofounder and chief medical officer of Navya Network Inc in Cambridge, Massachusetts, explained in written comments forwarded to *Blood Cancers Today*.

Naresh Ramarajan, MD

Board certified in clinical informatics and a member of the trial team, Dr. Ramarajan continued, "AI platforms such as Navya Earthshot can improve clinical capabilities and capacity to provide personalized, guideline-based treatment plans at the point of care."

In this pre-post observational study, Navya Earthshot was incorporated into the routine care provided at a cancer center within the public health system in India. There, the AI platform processed patient-specific information toward producing treatment recommendations in keeping with National Cancer Grid (NCG) standard-of-care clinical guidelines. It also identified instances of nonessential diagnostic testing that could be jettisoned to streamline care.

During the 6-month trial, 892 patients came to the cancer center for a first visit, and 537 required active treatment decisions. Ultimately, care could be evaluated for 411 of these patients, and among them a marked increase in management adherence to NCG guidelines was observed, from 67% according to baseline data obtained before integration of the AI system to 87% after integration.

"The Navya Earthshot AI model translates complex clinical guidelines into a usable, real-time decision-support tool for clinicians. Its implementation has shown a 15-20% absolute increase in guideline concordant care and received a 100% NPS [net promoter score] score from physicians at safety-net hospitals in India," Dr. Ramarajan wrote.

The researchers also reported a decrease in patients' number of visits before their care was to begin, from an average of 3.45 to 2.5 visits, a decrease that they attributed to Navya Earthshot's identification of nonessential report-review visits. After AI platform integration, patients also initiated cancer care sooner after the first visit—at 4 weeks compared with 6 weeks before integration.

REFERENCE

2026 American Association for Cancer Research Annual Meeting. Abstract No. 6352/7.

PHOTO CREDIT: istock.com/SeanPavonePhoto

Is CDK4/6 Pairing the Answer to JAK Inhibitor Limits in Myelofibrosis?

*By **Nichole Tucker***

At the American Association for Cancer Research (AACR) Annual Meeting 2026, researchers observed encouraging results when they paired a third-generation CDK4/6 inhibitor with a first-generation Januse kinase (JAK) inhibitor. With a limited median overall survival, patients with primary and secondary myelofibrosis are in dire need of more options beyond JAK inhibitors.

"JAK inhibitors such as ruxolitinib improve disease-related symptoms and splenomegaly in patients with myelofibrosis but do not change the underlying disease biology. In fact, life expectancy for patients with myelofibrosis at the time of JAK inhibitor discontinuation is only around 1 year. Thus, novel treatments for these patients are urgently needed," first author **Jan P. Bewersdorf, MD,** of Yale Cancer Center, told *Blood Cancers Today*.

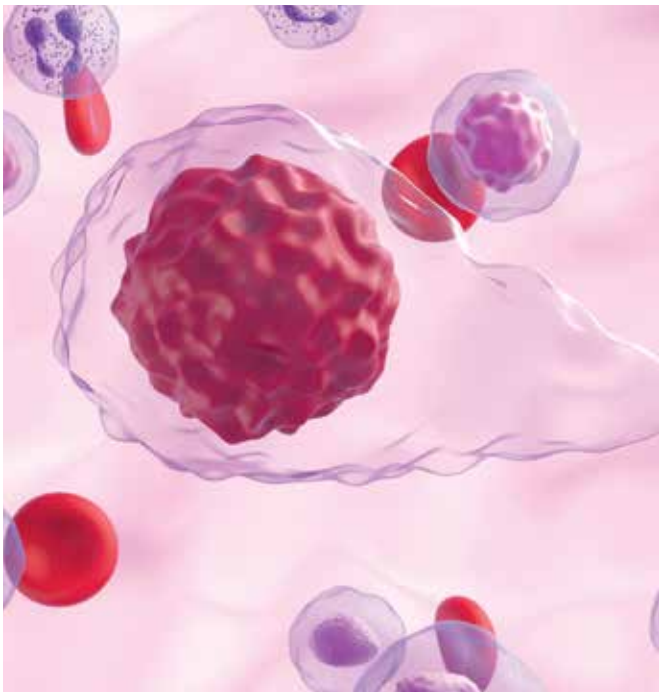
"JAK-STAT activating mutations are a hallmark of myelofibrosis and activate cell cycle progression. Preclinical studies from our group and others testing the combination of JAK inhibitors and CDK4/6 inhibitors have demonstrated disease-directed biologic effects beyond those seen with JAK inhibition alone. CDK 4/6 inhibitors have already been approved for the treatment of certain forms of breast cancer."

Findings supporting continued study of abemaciclib plus ruxolitinib come from a phase 1 dose-escalation study led by researchers at Memorial Sloan Kettering Cancer Center and Yale Cancer Center (NCT05714072). The goal was to determine the preliminary safety and efficacy of abemaciclib plus ruxolitinib in an 11-patient cohort. To determine this, investigators administered abemaciclib at 50mg to 150mg BID and ruxolitinib at 10 mg or 15 mg BID.

By cycle 2, the authors reported that six of nine patients who remained on treatment had white blood cell normalization, as did six of the seven patients who remained on treatment by cycle 6. In terms of responses, the median best spleen volume reduction (SVR) was 36.3% (range, 19.1%-48.6%). Seven of nine patients in the study reduced their total symptom score, resulting in a median absolute change of –10 (range, –17 to +4). Investigators also noted that 55.6% of patients had an SVR of 35%.

Jan P. Bewersdorf, MD

"When reviewing the latest data, we were most impressed with the duration of spleen response for patients on study. All seven patients at the two highest dose levels remain on study, with six of seven maintaining a spleen volume reduction of 25% or greater persisting more than one year in those with longer follow-up," **Brian Chernak, MD,** of Memorial Sloan Kettering Cancer Center, the presenter of the research, told *Blood Cancers Today*.



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Patients in the study received a median on 9.5 cycles of treatment (range, 3-23 cycles) and were evaluated for safety and dose-limiting toxicities. Investigators did not report any dose limiting toxicities or serious adverse events (AEs). The most frequently observed AEs included diarrhea (80%), anemia (80%), and thrombocytopenia (70%).

Overall, treatment-emergent AEs were predominantly low grade, but the study team observed grade 3 anemia in 30% of patients, while grade 3 decreased neutrophil count and grade 3 diarrhea were observed in 20% of patients each. No patients discontinued treatment as a result of AEs, although four discontinued for other reasons.

"Given these promising findings and the demonstration of tolerability, we are moving forward with a phase 2 study of RUX/ABE [ruxolitinib/abemaciclib], which we are eager to open in the coming months," Dr. Chernak said.

REFERENCE

2026 American Association for Cancer Research Annual Meeting. Abstract CT191.

ARV-393 Moves Into Phase 1 Dose-Escalation Study for Non-Hodgkin's Lymphoma and DLBCL

*By **Melissa Badamo***

With the goal of improving clinical outcomes and preventing patient relapse, researchers are launching a phase 1 dose-escalation and optimization/expansion study of ARV-393 for patients with non-Hodgkin's lymphoma (NHL) and diffuse large B-cell lymphoma (DLBCL). The PROTAC BCL6 degrader will be evaluated as monotherapy for patients with NHL and in combination with glofitamab for patients with DLBCL.[1,2]

Noah Berkowitz, MD, PhD

The trial design was presented at the 2026 American Association for Cancer Research Annual Meeting by **Martin Hutchings, MD, PhD,** of Rigshospitalet and University of Copenhagen in Copenhagen, Denmark.

The open-label, first-in-human trial will evaluate the safety, tolerability, pharmacokinetics, pharmacodynamics, and preliminary antitumor activity of ARV-393 while determining the recommended phase 2 dose. An estimated 255 patients will receive ascending doses of oral ARV-393, once a day in 28-day cycles as monotherapy, or in combination with IV glofitamab during 21-day cycles. Eligible patients have either relapsed or refractory B-cell NHL after at least two lines of prior therapy, including rituximab, or nodal T-follicular helper cell lymphoma (nTFHL) with prior standard-of-care therapy.[1,2]

Developed by Arvinas, ARV-393 works by binding to BCL6 and an E3 ubiquitin ligase to "induce ubiquitination of BCL6 and its subsequent proteasomal degradation."[1]

"BCL6 is a previously undruggable oncogenic driver regulating hundreds of genes linked to oncogenesis and therapeutic resistance," **Noah Berkowitz, MD, PhD,** chief medical officer of Arvinas, told *Blood Cancers Today.* "ARV-393 is an investigational, orally bioavailable PROTAC BCL6 degrader designed to address unmet needs in NHLs by degrading this key oncogenic driver, with the potential to be combined with current and emerging therapies."

In a preclinical study, ARV-393 (3 or 6 mg/kg) combined with glofitamab 0.15 mg/kg showed greater antitumor activity compared with ARV-393 alone, with the addition of ARV-393 enhancing CD20 expression.[3] Similarly, ARV-393 monotherapy showed "significant single-agent" activity in patient-derived xenograft models of angioimmunoblastic type nTFHL and transformed follicular lymphoma, resulting in 95% to 99% tumor growth inhibition.[4]

"ARV-393's preclinical single-agent antitumor activity across NHL subtypes suggests broad BCL6 dependency," Dr. Berkowitz said. "Together with emerging data from other BCL6 targeting degraders, we believe these findings highlight BCL6 as a promising therapeutic target in NHL and support clinical investigation of ARV-393 monotherapy, as well as its potential integration into combination strategies, including all-oral and chemotherapy-free options."

REFERENCES

1. American Association for Cancer Research Annual Meeting 2026. Abstract CT268/2.
2. ClinicalTrials.gov. ID NCT06393738. Updated February 10, 2026. Accessed April 27, 2026. https://clinicaltrials.gov/study/NCT06393738
3. Van Acker A, et al. *Blood.* 2025;146(suppl 1):1520. doi:10.1182/blood-2025-1520
4. Van Acker A, et al. *Clin Lymphoma Myeloma Leuk.* 2025;25(suppl 1):S761. doi:1016/S2152-2650(25)02305-5

American Cancer Society Report: Five-Year Cancer Survival Rate Climbs to 70%

*By **Melissa Badamo***

The latest annual report from the American Cancer Society reveals a 5-year cancer survival rate of 70% for patients diagnosed between 2015 and 2021 in the United States, an increase from 50% in the 1970s.

"This is the highest 5-year relative survival that we have seen yet," **Lore Gruenbaum, PhD,** chief scientific officer and senior vice president of Research at Blood Cancer United, told *Blood Cancers Today*. "It reflects not only tremendous advancement in novel therapies, but also our understanding in terms of providing better care for patients."

Lore Gruenbaum, PhD

The largest survival gains were observed among patients with advanced diagnoses and high-mortality cancers such as multiple myeloma. The survival rate for myeloma increased from 32% to 62% since the 1990s, attributed to the increased development of targeted immunotherapy drugs.

"We're coming to a point where many patients [with myeloma] are approaching a lifespan quite comparable to someone without this type of cancer," Dr. Gruenbaum added. "This is a big improvement."

Less Chemotherapy, More Targeted Therapies

Similarly, the 5-year survival rates for chronic myeloid leukemia (CML) tripled from 22% to 70% since the 1970s due to the development of tyrosine kinase inhibitors. Dr. Gruenbaum also noted a growing emphasis on developing time-limited therapies for blood cancers, rather than continuous therapy, to improve patients' quality of life and reduce toxicities.

"The other big change we've seen is the increasing understanding of the role that the immune system is playing in cancer therapy," she added. "We may need a combination of targeted therapies and immune therapies. Bispecific antibodies that specifically target a patient's T cells to tumor targets have the potential to be game changers for many indications."

According to Dr. Gruenbaum, it takes decades for novel therapies to advance from early research to improving patient outcomes.

"There's a continuing trend to replace chemotherapies with therapies that are more targeted and better tolerated," she said. "We're really seeing the fruits of this labor."

Leukemia and Lymphoma Among Children and Adolescents

Leukemia remains the most common childhood cancer, making up 28% of cases. Similarly, lymphoma (19%) and leukemia (13%) are the top adolescent cancers after central nervous system tumors (22%). The American Cancer Society also reports a larger increase in lymphoid leukemia among adolescents compared with children.

The cancer incidence in adolescents continued to increase by 0.9% per year, with a 1% per year increase for both Hodgkin's and non-Hodgkin's lymphoma, and the incidence of major leukemia types and stable lymphoma increased by 0.6% per year in children.

Despite the uptick in leukemia and lymphoma cases among children and adolescents, cancer mortality rates show a continuing downward trend. From 1970 to 2023, cancer mortality declined by more than two-thirds in children (6 per 100,000 to 2 per 100,000) and by more than one-half in adolescents (7 per 100,000 to 3 per 100,000). From the 1970s to the period from 2015 to 2021, the 5-year survival rate for leukemia rose from 50% to 89% in children and from 24% to 78% in adolescents.

"I'm happy to report very good outcomes, but we mostly still achieve them with chemotherapies and chemotherapy-based treatments that haven't really changed over decades," Dr. Gruenbaum said. "We have not seen the same level of advancement in terms of targeted therapies being utilized for children and adolescents that we have seen for adult patients."

Racial Disparities in Cancer Incidence and Survival

The report also examined cancer incidence and mortality rates among five racial and ethnic groups: White, Black, American Indian/Alaska Native, Asian American/Pacific Islander, and Hispanic/Latino. Cancer incidence and mortality are highest among American Indian and Alaska Native people, and cancer survival is lower among Black people than White people.

From 2018 to 2022, the cancer incidence was 78% higher among Black men (541 per 100,000) than among Native Hawaiian or other Pacific Islander men (304 per 100,000).

"We continue to see very persistent differences in outcomes for patients with different races and ethnicities," Dr. Gruenbaum emphasized. "This is certainly something that requires a lot more effort."

These differences are influenced by a lack of representation in clinical trials, unconscious bias, treatment inequality, and less access to high-quality care, according to the report.

"This reflects disparities that can be linked to differences in

socioeconomic factors and access to healthcare, but there's also an emerging and growing field that recognizes that we do see true biological differences at times," Dr. Gruenbaum said. "It is important to do research and clinical development work across all cancer patient populations so that we recognize all the contributing mechanisms and develop therapies that benefit all patients, regardless of their ancestral and ethnic origin."

Remaining Unmet Needs for Rare Cancers

Despite the improvement in survival rates, treatment options, and quality-of-life outcomes among adult patients with CML and myeloma, unmet needs remain for other blood cancers. According to Dr. Gruenbaum, drug development is particularly challenging for patients with rare and heterogeneous cancers.

"We haven't seen this type of improvement in acute myeloid leukemia," she emphasized. "We also haven't seen it in the many rare cancers, such as certain types of T-cell lymphomas and T-cell leukemias. We still need to find new treatment algorithms, new targets, and new combinations that allow us to address those cancers where we haven't seen this type of success yet."

"Every new treatment that we develop will change the biology of the cancer that we are treating," she added. "Patients will sadly relapse even from some of the latest therapies. We are starting to understand [that] the most successful way to treat cancer is to not just look at the tumor cells, but to look at the immune cells and the entire environment in the context of a particular tumor. That's something that we really need and that's being actively worked on."

REFERENCES

1. American Cancer Society. Accessed March 25, 2026. https://pressroom.cancer.org/cancer-statistics-report-2026
2. Siegel RL, et al. *CA Cancer J Clin.* 2026;e70043. doi:10.3322/caac.70043

Real-World Data Reveal Persistent Toxicity Challenges With Brexu-Cel in Adult B-ALL

*By **Nichole Tucker***

Brexucabtagene autoleucel (brexu-cel) was approved in 2021 as a result of the pivotal ZUMA-3 study, which showed a toxicity profile in line with other CD19-directed chimeric antigen receptor (CAR) T-cell therapies. The trial clearly mapped the expected patterns of cytokine release syndrome (CRS) and immune effector cell–associated neurotoxicity syndrome (ICANS). But real-world experience paints a slightly more complicated picture. Toxicities in routine practice appear more variable, and in some cases, more severe than those captured in the controlled trial setting.

To better understand these risks, researchers analyzed outcomes from a multicenter cohort of 292 adults with relapsed or refractory B-cell acute lymphoblastic leukemia (B-ALL) treated with brexu-cel between October 2021 and June 2024. They found that rates of grade 3 or higher ICANS (28.8%) were similar to those seen in ZUMA-3, whereas grade 3 or higher CRS occurred less frequently (9.8%). Still, 20.5% of patients required ICU-level support, and the mortality rate at 28 days after CAR T-cell therapy was 5.2%. Severe CRS was linked to an increased risk of death (hazard ratio, 2.24; 95% CI, 1.27-3.94; P=0.005), and patients with a history of allogeneic hematopoietic cell transplantation (HCT) had a lower likelihood of developing severe CRS (odds ratio, 0.35; 95% CI, 0.14-0.89; P=0.03). These data show that even outside the controlled environment of a trial, high-grade toxicities remain a serious concern, particularly for those with high disease burden.

Clinicians find these real-world data informative but acknowledge a need for caution. In an interview with *Blood Cancers Today,* **Ryan Cassaday, MD,** of Fred Hutchinson Cancer Center and the University of Washington School of Medicine, explained: "The toxicity profile with brexu-cel is a legitimate concern. Products such as obecabtagene autoleucel [obe-cel] and, for patients aged 25 and under, tisagenlecleucel [tisa-cel], show lower rates of severe toxicity, particularly ICANS. At the same time, studies like ours provide clinicians with detailed evidence about what to expect with brexu-cel. The pivotal trial reported that 25% of patients experienced grade 3 or higher ICANS, which aligns with what we observed in real-world practice."

Ryan Cassaday, MD

Dr. Cassaday noted the implications for patient counseling and treatment selection: "The association between higher disease burden and grade 3 or higher CRS is clear. Interestingly, prior allogeneic HCT was linked to reduced risk of severe CRS, though we cannot determine causality. ICANS remains unpredictable and should be considered for all patients. In certain cases, such as patients with high disease burden or limited tolerance for severe neurotoxicity, I might favor using obe-cel."

The real-world findings offer valuable insight, but they also serve as a warning for clinicians. They illustrate that although brexu-cel is highly effective, its toxicities cannot be overlooked. Clinicians must balance the therapy's benefits against the potential risks, tailoring treatment decisions to each patient's clinical profile.

REFERENCE

Kopmar NE, et al. *Blood Immunol Cell Ther.* 2026;2(1):100028. doi:10.1016/j.bict.2025.100028

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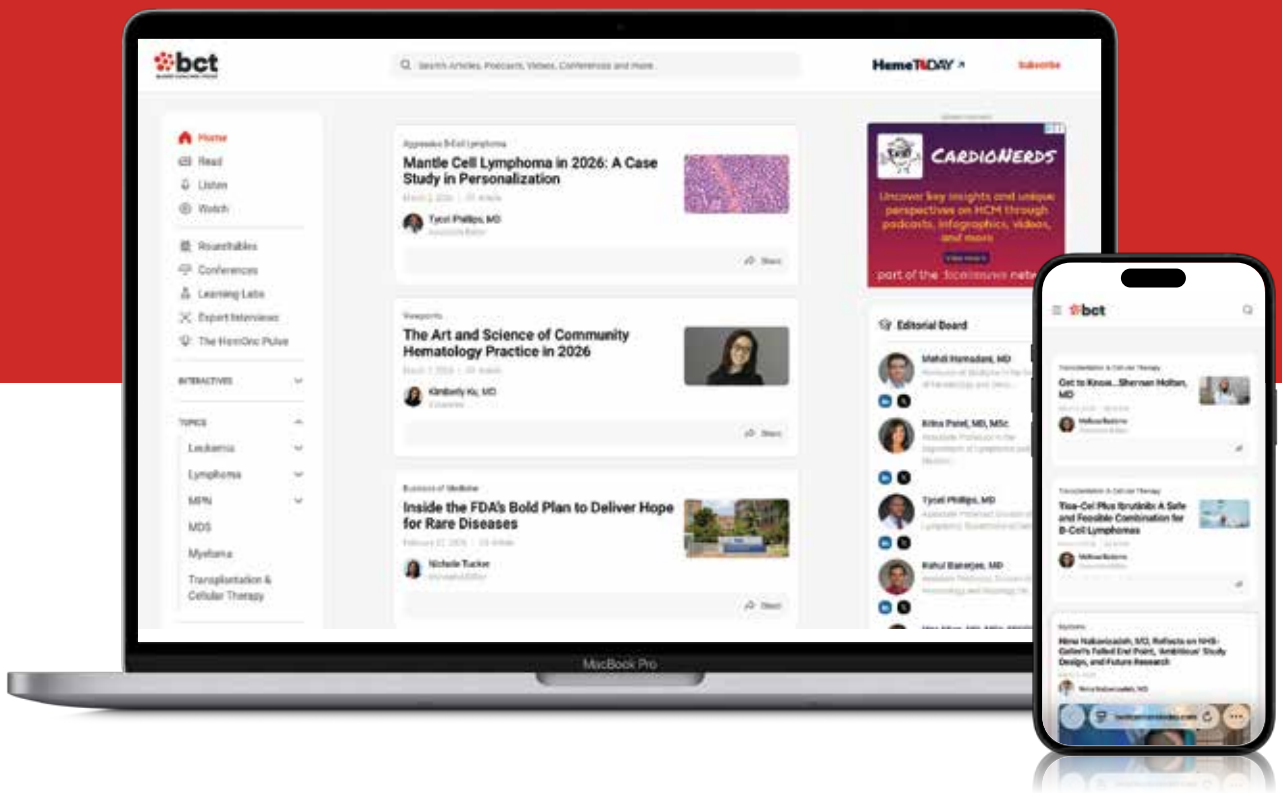

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– Dr. Anthony Petrillo

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DREAMM-8 Follow-Up Supports BPd in First-Relapse Myeloma

*By **Dean Patterson***

Longer follow-up from the phase 3 DREAMM-8 study is sharpening the case for belantamab mafodotin, pomalidomide, and dexamethasone (BPd) as an early-relapse treatment option for relapsed or refractory multiple myeloma (RRMM). **Suzanne Trudel, MD,** of Princess Margaret Cancer Centre, Toronto, Ontario, Canada, and colleagues reported updated efficacy and safety data from DREAMM-8, a phase 3, open-label, randomized trial comparing BPd with pomalidomide, bortezomib, and dexamethasone (PVd) in patients with RRMM after at least one prior line of therapy that included lenalidomide. The updated analysis had a data cutoff of July 7, 2025, and a median follow-up of 35.8 months.

Suzanne Trudel, MD

In this setting, clinicians now have a practical question: Can an outpatient, off-the-shelf anti-BCMA regimen deliver not just depth of response but durability that matters in everyday relapse care? DREAMM-8 suggests it can. As Dr. Trudel told *Blood Cancers Today,* "In this analysis with a longer median follow-up of 36 months, a substantial progression-free survival benefit was observed with BPd, with a median 32.6 months versus 12.5 months with PVd." Those numbers corresponded to a hazard ratio of 0.49 (95% CI, 0.36-0.67). At 24 months, 55% of patients in the BPd arm remained alive and progression free versus 31% in the PVd arm.

The study enrolled 302 patients. Most had lenalidomide-refractory disease, and about one-quarter in each arm had disease refractory to prior treatment with anti-CD38 monoclonal antibodies. In the BPd arm, 53% had received one prior line of therapy, 34% had received two or three prior lines, and 14% had received four or more. Triple-class exposure was also common. That makes the magnitude of benefit harder to dismiss as a function of unusually favorable biology.

Depth of response also favored BPd. Overall response rates were 76% with BPd and 72% with PVd, but the more meaningful separation came at higher-quality responses: at least a very good partial response was achieved in 63% versus 39%, and complete response (CR) or better was achieved in 43% versus 17%. Duration of response was not reached with BPd, compared with 16.4 months with PVd. A second question follows naturally from that: How often does this regimen translate deep response into measurable disease clearance? Here again, BPd looked stronger. Among patients achieving a CR or better, measurable residual disease (MRD) negativity at 10^{-5} was 64% with BPd versus 36% with PVd. In the intent-to-treat population, CR-based MRD negativity was 28% with BPd and 6% with PVd, and among those with a very good partial response, MRD negativity was 35% and 7%, respectively. Sustained CR-based MRD negativity for at least 12 months was seen in 15% versus 3%.

Dr. Trudel emphasized that signal directly, saying, "MRD negativity rates in patients achieving a complete response or better were 64% with BVd versus 36% with PVd. In the intent-to-treat population, BPd led to a fivefold increase in MRD negativity and sustained MRD negativity versus PVd in patients with RRMM, most of whom had lenalidomide-refractory disease and a quarter of whom had disease refractory to anti-CD38 monoclonal antibodies."

Importantly, benefit extended beyond first progression. Median PFS2 (ie, time from randomization to disease progression after initiation of new antimyeloma therapy or death from any cause, whichever occurred first) was 47.1 months with BPd versus 21.7 months with PVd, with a hazard ratio of 0.52 (95% CI, 0.38-0.70), suggesting that earlier disease control did not come at the expense of subsequent therapy sequencing.

> **"These findings support BPd as an accessible, outpatient anti-BCMA agent for treatment of MM at first relapse across all sites of care."**
>
> *—Suzanne Trudel, MD, professor of medicine, Princess Margaret Cancer Centre*

Safety, at least according to these updated findings, did not appear to drift in an unexpected direction. "The safety profile of BPd remained generally consistent with the findings from the primary analysis," Dr. Trudel said. Any grade 3 or 4 adverse event was reported in 91% of BPd-treated patients, treatment-related adverse events in 96%, permanent discontinuation in 22%, dose reduction in 63%, and dose interruption or delay in 91%.

The findings from the long-term follow-up strengthen the clinical relevance of treatment with BPd at first relapse. As Dr. Trudel put it, "These findings support BPd as an accessible, outpatient anti-BCMA agent for treatment of MM at first relapse across all sites of care."

REFERENCE

Presented at Hematology/Oncology Pharmacy Association (HOPA) 2026 Annual Conference.

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 lymphoma division at Dana-Farber Cancer Institute and an associate professor at Harvard Medical School, highlights recent research in chronic lymphocytic leukemia.*

Visit bloodcancerstoday.com to stay up to date on the latest news in each area of hematologic oncology.

Matthew Davids, MD

CHRONIC LYMPHOCYTIC LEUKEMIA

Pirtobrutinib Dominates in CLL Trial, Beating Standard Regimen

By ***Nichole Tucker***

In patients with chronic lymphocytic leukemia (CLL) and small lymphocytic lymphoma treated for up to 2 years, the combination of pirtobrutinib plus venetoclax and rituximab resulted in better reduction in rates of disease progression or death compared with venetoclax and rituximab alone, meeting the primary end point of the phase 3 BRUIN CLL-322 clinical trial.[1]

The progression-free survival (PFS) advantage of the pirtobrutinib combination was consistent in the relevant CLL subgroups in the trial, according to topline results announced by Eli Lilly and Company. Notably, prior receipt of a covalent Bruton's tyrosine kinase (BTK) inhibitor did not interfere with the PFS advantage of the addition of pirtobrutinib.

Pirtobrutinib stands out among other covalent BTK inhibitors because of its ability to inhibit both wild-type and C481-mutant BTK with equal low nanomolar potency, according to a study led by **Toby A. Eyre, MD**, of Oxford University Hospitals NHS Foundation Trust, Churchill Cancer Center.[2] This ability was made clearer in the phase 1/2 BRUIN study, in which pirtobrutinib elicited durable responses.[3]

Topline results from BRUIN CLL-322 come on the heels of other positive announcements from the BRUIN program, including the phase 3 BRUIN CLL-321 study. At the time results were announced, investigator **Jeff Sharman, MD,** of Sarah Cannon Research Institute told *Blood Cancers Today,* "BRUIN-321 is the first prospective randomized phase 3 trial among patients with CLL previously treated with a covalent BTK inhibitor. In this study we showed that pirtobrutinib extends progression-free survival and time-to-next treatment with an improved safety profile relative to the control arm. In this difficult-to-treat population, pirtobrutinib is an effective and well tolerated therapy." Now, BRUIN CLL-322 adds to the excitement about pirtobrutinib in the research community.

In a news release,[1] **Jacob Van Naarden**, executive vice president and president of Lilly Oncology explained the impact of BRUIN CLL-322 further: "BRUIN CLL-322 was an ambitious trial, building on an effective regimen, and these results outperformed our expectations."

The news release also stated that detailed findings from BRUIN CLL-322 are expected to be presented at a medical congress and submitted for publication in a peer-reviewed journal. Lilly plans to share these results with regulatory bodies later in 2026.

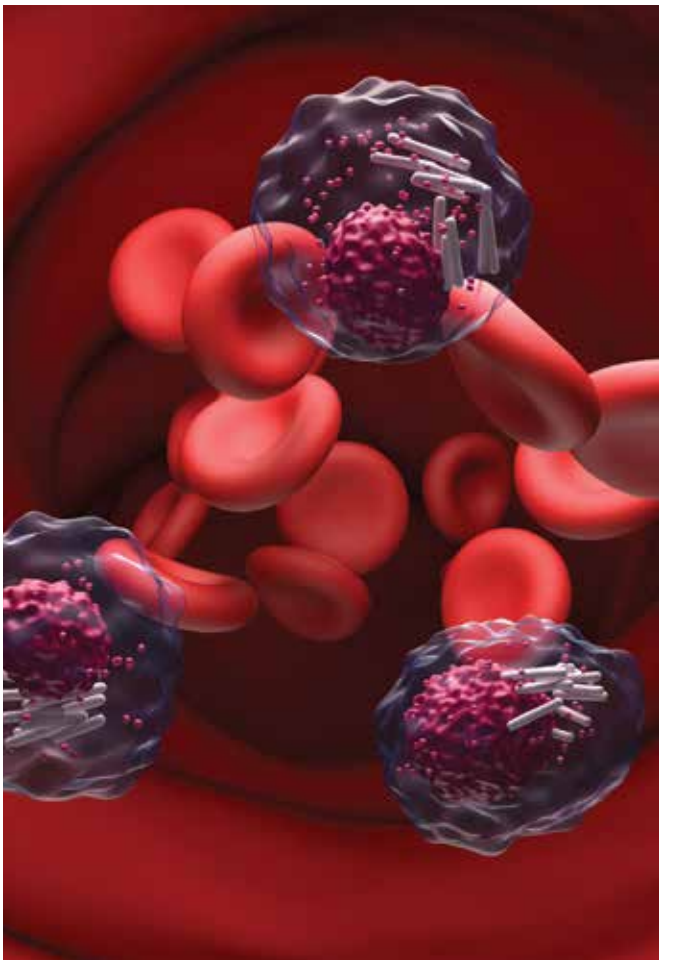

PHOTO CREDIT: istock.com/Nemes Laszlo

REFERENCES

1. Lilly. Accessed April 14, 2026. https://investor.lilly.com/news-releases/news-release-details/lillys-jaypirca-pirtobrutinib-significantly-extended-progression
2. Erye TA, et al. *J Clin Oncol.* 2023;41(16):TPS7583. doi:1200/JCO.2023.41.16_suppl.TPS758
3. Mato AR, et al. *N Engl J Med.* 2023;389(1):33-44. doi:1056/NEJMoa2300696
4. Sharman JP, et al. *J Clin Oncol.* 2025;43(22):2538-2549. doi:1200/JCO-25-00166

Comparison Study Affirms Zanubrutinib's Safety and Efficacy Over Ibrutinib in CLL

*By **Melissa Badamo***

In the real-world setting, zanubrutinib exhibited lower rates of treatment discontinuation and toxicities than ibrutinib in patients with chronic lymphocytic leukemia (CLL), according to a multicenter, retrospective, comparative study published in *Blood Advances*.[1]

Lead author **Enrica Antonia Martino, MD,** of the Azienda Ospedaliera Annunziata in Italy, and colleagues extracted data from patients in a large, real-world Italian cohort from the CLL-ZANU2024 study. The goal was to compare the two Bruton's tyrosine kinase inhibitors (BTKis) outside of a traditional clinical trial setting. Of the 934 patients, 541 received the first-generation BTKi ibrutinib and 393 received the second-generation BTKi zanubrutinib. The researchers evaluated time to treatment discontinuation (TTD) and time to next treatment or death (TTNTD) in two populations: the overall cohort and a propensity score–matched population.[1]

The 1-year TTD rates were lower with zanubrutinib versus ibrutinib among both the overall cohort (12.6% vs 21.4%, respectively) and the matched cohort (12.4% vs 20.2%, respectively). Similarly, zanubrutinib also exhibited higher 1-year TTNTD rates than ibrutinib in both the overall (91.9% vs 83.0%) and matched cohorts (93.2% vs 83.4%). According to multivariable analyses, zanubrutinib was confirmed to be an independent predictor of longer TTD and TTNTD.[1]

Multivariable analyses also determined high-risk features associated with worse outcomes. These include age, relapsed or refractory disease, Binet stage C, *TP53* disruption, Eastern Cooperative Oncology Group status of 2 to 3, and congestive heart failure. Adverse events (AEs) leading to treatment discontinuation, such as atrial fibrillation, bleeding, and infections, were also less frequent with zanubrutinib.[1] These results reflect an expected safety profile, as zanubrutinib was developed to avoid AEs associated with ibrutinib.[2]

This study affirms zanubrutinib's efficacy and favorable safety from two pivotal phase 3 trials, ALPINE[2,3] and SEQUOIA.[4] In the final comparative analysis of ALPINE, zanubrutinib showed a sustained progression-free survival (PFS) benefit versus ibrutinib in patients with relapsed or refractory CLL or small lymphocytic leukemia (SLL) while maintaining a favorable safety profile. At 3.5 years, zanubrutinib also exhibited a higher overall response rate of 85.6% versus 75.4% with ibrutinib. The ALPINE investigators noted that responses even deepened over time, with zanubrutinib showing a complete response (CR)/CR with incomplete bone marrow recovery rate of 11.6% versus 7.7% with ibrutinib.[3]

The ALPINE trial also reported an improved safety profile with zanubrutinib versus ibrutinib. Notably, both the incidence of cardiac events (25.9% vs 35.5%, respectively) and discontinuations due to cardiac events (0.9% vs 4.9%) were "significantly lower" with zanubrutinib versus ibrutinib. The most common hematologic AE of any grade was neutropenia (31.5% vs 29.6%, respectively), and the most common nonhematologic AEs were infections related to COVID-19 (46.0% vs 33.3%), upper respiratory tract infection (29.3% vs 19.8%), diarrhea (18.8% vs 25.6%), and hypertension (27.2% vs 25.3%).[3]

In a 5-year update of the SEQUOIA trial, zanubrutinib continued to show a superior PFS of not reached versus 44.1 months with bendamustine plus rituximab in patients with previously untreated CLL and SLL. The most common treatment-emergent AEs for both zanubrutinib and bendamustine-rituximab were infection (79.6% vs 65.6%, respectively), bleeding (52.1% vs 13.2%), hypertension (22.9% vs 13.7%), neutropenia (17.1% vs 56.8%), anemia (9.6% vs 21.1%), thrombocytopenia (7.1% vs 18.5%), and atrial fibrillation/flutter (7.1% vs 3.5%).[4]

Zanubrutinib has consistently shown superior efficacy to other BTKis, such as acalabrutinib. Although the two BTKis haven't been compared in a head-to-head randomized trial, a matching-adjusted indirect comparison found that zanubrutinib was associated with higher PFS and CR compared with acalabrutinib. However, safety was not evaluated due to the various treatment exposure times across the ASCEND or ALPINE trials.[5]

Despite the improved efficacy and safety of zanubrutinib across the board, ongoing research is needed. According to Dr. Martino and colleagues, "These findings provide real-world evidence that zanubrutinib offers more durable disease control and improved persistence compared with ibrutinib, reinforcing its clinical value as a preferred second-generation BTKi. Nevertheless, the relatively short follow-up for zanubrutinib warrants cautious interpretation of long-term outcomes, and underscores the need for ongoing observation to fully characterize its durability and safety."[1]

REFERENCES

1. Martino EA, et al. *Blood Adv.* 2026;10(5):1687-1699. doi:10.1182/bloodadvances.2025018757
2. Brown JR, et al. *N Engl J Med.* 2023;388(4):319-332. doi:10.1056/NEJMoa2211582
3. Brown JR, et al. *Blood.* 2024;144(26):2706-2717. doi:10.1182/blood.2024024667
4. Shadman M, et al. *J Clin Oncol.* 2025;43(7):780-787. doi:10.1200/JCO-24-02265
5. Shadman M, et al. *Ther Adv Med Oncol.* 2025;17:17588359251340554. doi:10.1177/17588359251340554

HemOnc Happenings

Reporting on recent announcements, awards, and appointments in the hematology/oncology sphere

From the Lab to Frontline Care: Dr. Shadman Begins Deputy CMO Role at Fred Hutch Cancer Center

*By **Katie Kosko***

Mazyar Shadman, MD, MPH

For **Mazyar Shadman, MD, MPH,** ensuring that clinical research and operational efficiency are integrated into patient care at Fred Hutch Cancer Center in Seattle, Washington, is crucial. In his new leadership role as Deputy Chief Medical Officer (CMO) at Fred Hutch, he will focus on improving the patient experience.

He will work closely with CMO **Thomas Purcell, MD, MBA,** and recently appointed Deputy CMO **Vyshak Venur, MBBS, MSCI,** to lead hematologic, solid tumor, acute care, and inpatient/outpatient services at Fred Hutch.

"Our priorities at Fred Hutch are to deliver the best available investigational or standard-of-care treatments to patients," Dr. Shadman said in an interview with *Blood Cancers Today*. "There is a significant overlap between the clinical trial performance and clinical operation. Our goal is to provide timely and universal service to all our patients, and my job is to make sure that patients in need of treatment get to us immediately."

Building a Career in Hematology

In 2011, Dr. Shadman began his hematology and medical oncology fellowship at the University of Washington Fred Hutch Cancer Center. Upon completion in 2014, he became a faculty member with a focus on treating high-risk lymphoma and chronic lymphocytic leukemia. He was named recipient of the Innovators Network Endowed Chair in July 2023 and has served as Medical Director of Cellular Immunotherapy for the past 2 years.

Dr. Shadman emphasized the importance of timely care, particularly in immunotherapy and transplant programs. "Part of my job is to make sure the coordination of care across disease groups is seamless," he said.

While his focus is on hematology, stem cell transplant, and immunotherapy, Dr. Shadman highlighted Fred Hutch's multidisciplinary, disease-focused approach to delivering personalized cancer care. "Because care could include any number of modalities, multidisciplinary care allows for picking the right treatment, quickly and efficiently moving between modalities as needed," he said.

Operational Challenges in Oncology Care

One of the biggest clinical challenges remains access to oncology care, Dr. Shadman said. Treatments such as chimeric antigen receptor (CAR) T-cell therapy have demonstrated significant benefits for patients with leukemia, lymphoma, and multiple myeloma, yet many eligible patients are not receiving them.

Dr. Shadman shared how CAR-T is now curing some patients with diffuse large B-cell lymphoma. However, he estimated that roughly 20% of eligible patients nationally receive it.

Efforts to bridge the gap include patient and physician education. At Fred Hutch, small continuing education meetings and collaboration with community oncologists aim to ensure patients receive appropriate treatment throughout the course of their care journey.

Logistical challenges also play a significant role, according to Dr. Shadman. Barriers such as travel, lodging, and the need for caregiver support can delay treatment, often requiring coordination with social workers to help patients navigate access to care.

Addressing the 'Good Problems'

As Dr. Shadman steps into this role, he is motivated to address what he described as the "good problems," such as optimizing treatment sequencing and resource allocation.

With a background as a clinical investigator, Dr. Shadman is excited about the rapid advancements in blood cancer treatments and what they may mean for those who turn to Fred Hutch Cancer Center for treatment.

"Patient satisfaction and clinical outcomes are key. If you bring in more patients and keep the quality of care as great as it is, then that's a great success," Dr. Shadman said.

University of Iowa Appoints New Director of Hematology, Oncology and Blood & Marrow Transplantation as the State's Cancer Rates Rise

*By **Sara Karlovitch***

Umar Farooq, MD, has been appointed director for the Division of Hematology, Oncology, and Blood & Marrow Transplantation at the Holden Comprehensive Cancer Center (HCCC), according to an announcement from the Department of Internal Medicine at University of Iowa Health Care.[1] Prior to his appointment, Dr. Farooq served as the department's interim director for the past year.

Umar Farooq, MD

Dr. Farooq's area of study is centered around chimeric antigen receptor (CAR) T-cell therapy and other areas of immunotherapy. Recently, he coauthored a study evaluating

the effectiveness of LYL314, an autologous dual-targeting CD19/CD20 CAR T-cell candidate intended for CD621+-naïve and central memory T cells, for the treatment of relapsed or refractory large B-cell lymphoma. Results showed that LYL314 had a manageable safety profile and both a high overall response rate and complete response rate.[2]

The appointment comes as cancer rates in Iowa continue to grow, according to the press release. Iowa Cancer Registry's Cancer in Iowa Report estimates that 21,700 people in the state will be diagnosed with cancer and 6,400 will die of cancer in 2026. In addition, the cancer incidence rate for young adults in Iowa increased "significantly" from 2008-2012 to 2018-2022, ranking second highest in the US and exceeding the national average.[3]

According to the announcement, Dr. Farooq was chosen for the position due to his clinical and academic expertise, as well as his ability to command multidisciplinary teams.[1] In addition to his new role of director, Dr. Farooq is the Gary D. Arthur Clinical Professor in Adult Bone Marrow Transplantation, Clinical Professor of Internal Medicine-Hematology, Oncology, and Blood & Marrow Transplantation, and Associate Director for Clinical Research at the HCCC.[4]

"I am excited about the next few years in cancer care in Iowa," Dr. Farooq said in the press release.[1] "The research and clinical activities within the division and our connections with the Holden Comprehensive Cancer Center have always had a huge impact. With the addition of Mission + Blood, coordination is more complex and our reach even broader. I look forward to making it easier for our faculty and staff to bring their expertise to more Iowans and to continue doing the good work that has brought us all here."

Dr. Farooq is also a full member of the Experimental and Redox Therapeutics (ERT) program at HCCC, which aims to target redox pathways while taking advantage of tumor vulnerability and preserving normal tissue function. Other areas of focus of the program include using model systems to test drug delivery, theranostic strategies, and new therapies. The ERT program also helps to guide the pipeline of preclinical discovery to clinical trials.[5]

REFERENCES

1. University of Iowa Department of Internal Medicine. Accessed April 14, 2026. https://internalmedicineiowa.org/2026/02/20/farooq-named-hobmt-division-director/
2. Merchant A, et al. International Conference on Malignant Lymphoma; June 17-21, 2025; Lugano, Switzerland. Presentation No. 106.
3. Iowa Cancer Registry. Accessed April 14, 2026. https://shri.public-health.uiowa.edu/wp-content/uploads/2026/03/2026-Cancer-in-Iowa.pdf
4. University of Iowa Department of Internal Medicine. Accessed April 14, 2026. https://internalmedicine.medicine.uiowa.edu/profile/umar-farooq
5. Holden Comprehensive Cancer Center. Accessed April 14, 2026. https://holden.uihealthcare.org/research/areas-cancer-research/experimental-and-redox-therapeutics

Ulrich G. Steidl, MD, PhD, Named Director of Montefiore Einstein Comprehensive Cancer Center

*By **Melissa Badamo***

Ulrich G. Steidl, MD, PhD

Ulrich G. Steidl, MD, PhD, a blood cancer and stem cell researcher, has been named the director of Montefiore Einstein Comprehensive Cancer Center (MECCC) in the Bronx, New York, and vice president of cancer medicine at Montefiore Einstein, where he has served as interim director since 2025. In these leadership roles, Dr. Steidl will work with a team of clinicians, researchers, and administrators to deliver innovative therapies to patients with cancer.[1]

"I am deeply honored to accept the leadership of MECCC," Dr. Steidl said in a press release.[1] "I look forward to working together as we advance our mission to reduce the burden of cancer in the Bronx and beyond and drive innovation that improves the lives and well-being of people in our borough and around the world."

Dr. Steidl joined Montefiore Einstein in 2008, where he leads basic and translational research at the Steidl Lab. His research focuses on the molecular mechanisms of pre-leukemic stem cells in hematopoiesis and developing novel therapies to prevent progression to leukemia stem cells.[1] In 2018, he co-published a paper in *Nature* that paved the way for understanding how myelodysplastic syndromes progress from defective blood-forming stem cells.[1,2]

Dr. Steidl has received numerous accolades for his work, including the Scholar Achievement Award from Blood Cancer United in 2019 and the Outstanding Investigator Award from the National Cancer Institute in 2021.[1]

REFERENCES

1. Montefiore Einstein. Accessed April 10, 2026. https://montefioreeinstein.org/news/2026/04/07/ulrich-steidl-md-phd-named-director-montefiore-einstein-comprehensive-cancer-center
2. Chen J, et al. *Nat Med.* 2019;25(1):103-110. doi:10.1038/s41591-018-0267-4

Do you know of a clinician or researcher who has been the recipient of a recent award?

Send the details to editor@bloodcancerstoday.com.

Calendar

May 29–June 2
2026 American Society of Clinical Oncology (ASCO) Annual Meeting
Chicago, IL

June 8–9
22nd Global Summit on Hematology and Blood Disorders
Dubai, UAE

June 11–14
European Hematology Association (EHA) 2026 Congress
Stockholm, Sweden

June 24–27
AACR Advances in Malignant Lymphoma
Philadelphia, PA

June 26–27
Mayo Clinic Updates in Hematology and Oncology
Montreal, Canada

July 23–26
Debates and Didactics in Hematology and Oncology
Sea Island, GA

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

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

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figure1

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