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ONCOLOGY COMPASS

Digest

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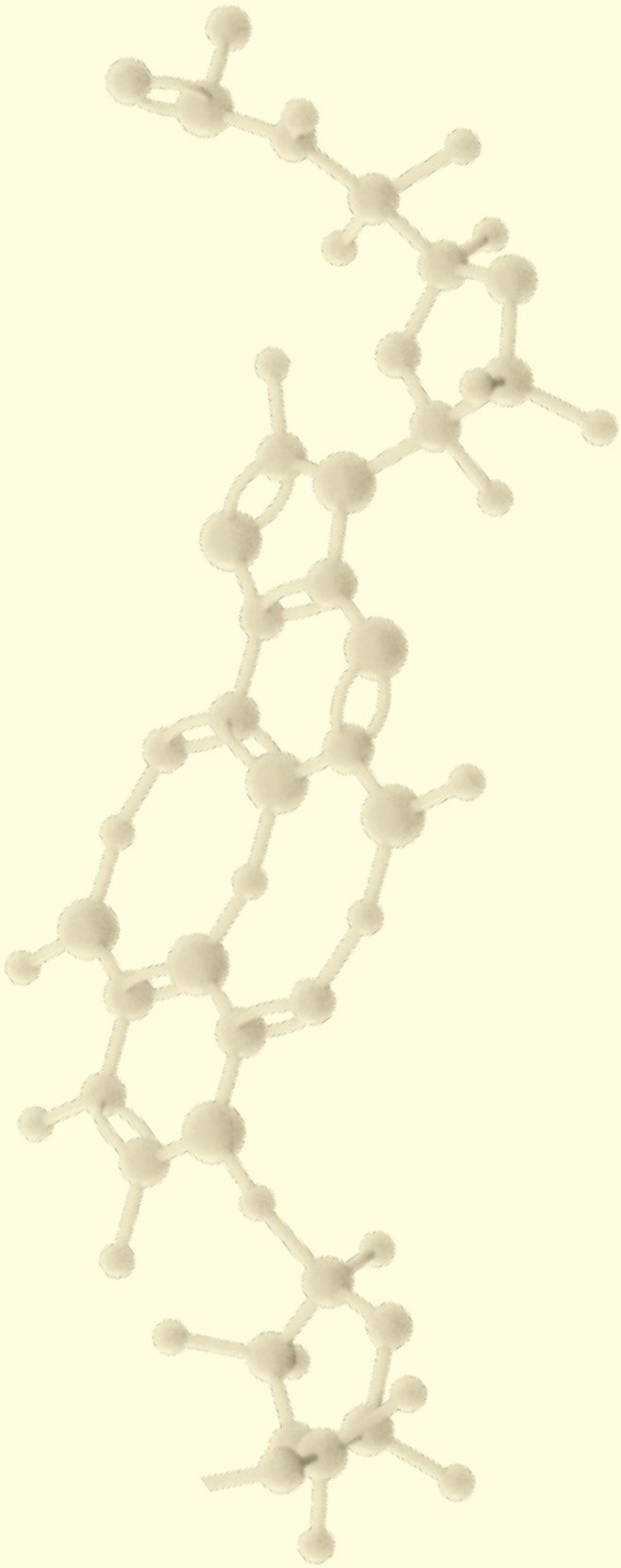


DECODING BREAST CANCER DIVERSITY

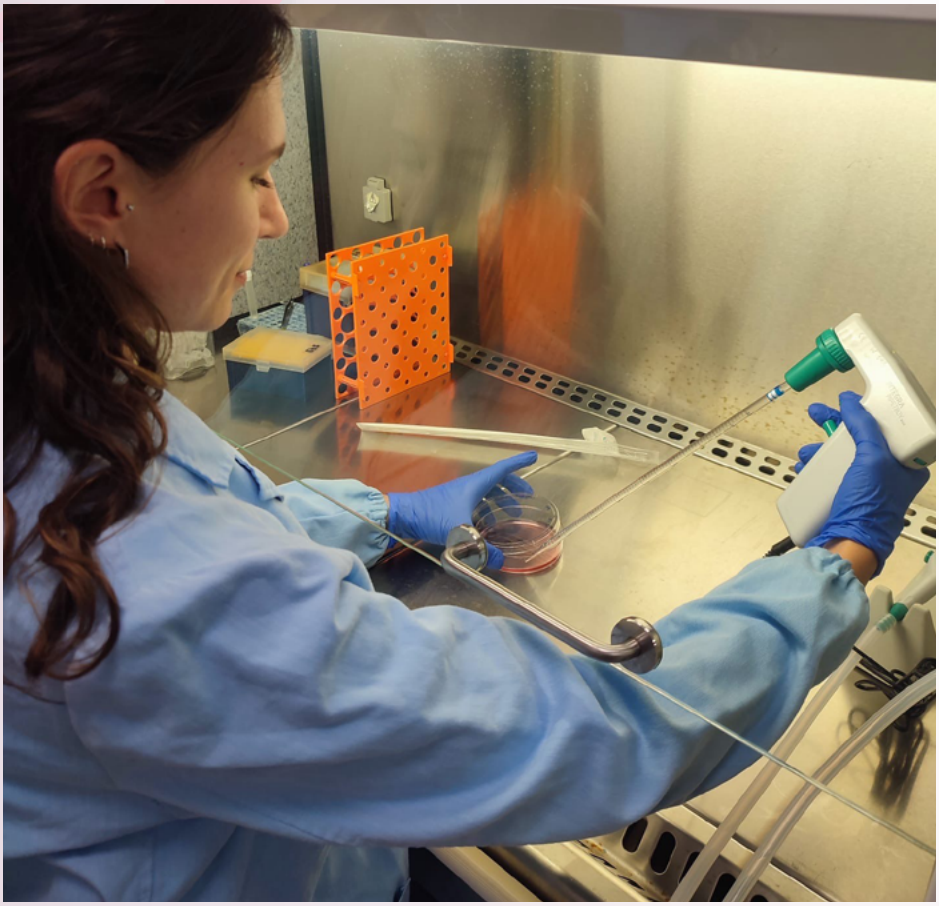
CHALLENGES AND OPPORTUNITIES IN TUMOR HETEROGENEITY

BY ANĐELIKA KALEZIĆ

Therapeutic resistance and disease relapse remain major challenges in oncology, rooted in tumor heterogeneity. Tumors are dynamic ecosystems of genetically and phenotypically diverse subclones that evolve under therapeutic pressure. This complexity drives clonal escape, adaptive resistance, and disease progression. In breast cancer, where molecular diversity is pronounced, understanding heterogeneity is critical for precision oncology. Breakthroughs in single-cell profiling, spatial genomics, and artificial intelligence now offer unprecedented resolution of this complexity, transforming how we view tumor evolution and therapeutic vulnerability.

Ana Arsenijevic, PhD in Cell and Molecular Biology
Postdoctoral Researcher at CRG Barcelona, photo courtesy of Ana Arsenijević

Dr. Ana Arsenijević is a postdoctoral researcher at the Centre for Genomic Regulation (CRG) in Barcelona, Spain. She began her scientific training at the University of Belgrade, earning



Ana Arsenijevic, PhD in Cell and Molecular Biology
Postdoctoral Researcher at CRG Barcelona, photo courtesy of Ana Arsenijević

a Master's degree in Human Molecular Genetics with a focus on breast cancer biology. Her academic journey led her into the field of epigenetics and chromatin regulation during her PhD at the University of Edinburgh, where she examined the mechanisms of heterochromatin formation.

Today, her work combines computational and experimental tools to investigate tumor heterogeneity in breast cancer, aiming to uncover more effective and personalized therapeutic strategies.

In this interview, Oncology Compass Digest discusses the biological underpinnings of tumor heterogeneity with Dr. Arsenijević, the implications for treatment resistance, and how new technologies, such as single-cell sequencing and AI, are reshaping the field.

Can you explain what tumor heterogeneity means and why it's particularly significant in breast cancer?

Tumor heterogeneity can be divided into two broad types. Inter-tumor heterogeneity refers to differences between tumors in different patients, even within the same subtype.

For example, breast cancer is categorized into subtypes based on molecular markers, but tumors within the same subtype can vary widely in aggressiveness, marker expression, and response to therapy.

Intra-tumor heterogeneity, which may be even more interesting and important, refers to the diversity of cell populations within a single tumor. This variation often arises from epigenetic changes and microenvironmental influences, making diagnosis and treatment significantly more complex.

How does tumor heterogeneity influence the effectiveness of treatment strategies?

Since no two tumors are identical, even within the same subtype, responses to therapy can vary significantly. While first-line treatments are well-established for common breast cancer types, individual outcomes can still differ. Intra-tumor heterogeneity presents additional challenges: targeted therapies may eliminate only part of the tumor, allowing resistant cell populations to survive and drive relapse.

Cells that lack the molecular target of a given therapy may initially exist as a minor subpopulation within the tumor. However, once more sensitive cells are eliminated, these resistant cells can expand and ultimately lead to disease progression and relapse.. This process, known as clonal escape, is a major mechanism behind treatment resistance.

What are the main challenges in designing therapies that can overcome tumor heterogeneity?

The key challenge lies in the presence of minor clonal subpopulations that do not express the target of a particular therapy. In this context, the treatment acts as a selective pressure, allowing resistant clonal subpopulations to survive and eventually dominate the tumor landscape.

Although broad-spectrum therapies exist that target cell division or key metabolic pathways, they often damage healthy cells as well, resulting in more severe side effects.

Designing therapies that can effectively target all tumor cell populations without harming healthy tissue remains a significant obstacle.

How does the tumor microenvironment contribute to this heterogeneity and affect treatment outcomes?

The tumor microenvironment is a complex ecosystem made up of immune and stromal cells, nutrients, cytokines, and extracellular components.

It plays a significant role in shaping intra-tumor heterogeneity.

For instance, regions of a tumor with limited oxygen or nutrient access often undergo metabolic reprogramming, leading to further phenotypic divergence.

These subpopulations may respond differently to treatment, contributing to resistance and disease progression.

What advances in single-cell sequencing have helped deepen our understanding of tumor heterogeneity?

Single-cell sequencing has transformed the field by enabling the profiling of gene expression at the level of individual cells. It has uncovered previously unrecognized subclonal populations that may be crucial drivers of relapse.

Technologies such as single-cell ATAC-sequencing provide insight into the epigenetic states of these cells, while spatial transcriptomics adds another layer by demonstrating where specific gene expression patterns occur within the tumor.

Together, these tools are aiding us in mapping tumor progression in terms of intra-tumor heterogeneity and clonal evolution with unprecedented detail.

How does this complexity affect the use of immunotherapy in breast cancer?

Immunotherapies depend on recognizing specific antigens on tumor cells. When these antigens are expressed only in certain subpopulations, or when tumor cells can alter or lose these markers, the treatment becomes less effective.

Under selective pressure, tumor cells may gradually lose antigen expression over time, resulting in relapse even in patients who initially respond well to therapy.

Moreover, structural barriers within the tumor, such as fibrotic microniches, can obstruct immune cells, limiting the effectiveness of the treatment.

How can a better understanding of tumor heterogeneity improve personalized treatment plans?

A more detailed understanding of tumor heterogeneity enables us to move beyond one-size-fits-all approaches.

By identifying clonal diversity and driver mutations, we can tailor therapies more precisely, often through combinatorial strategies.

With the help of bioinformatics and machine learning, we can better predict treatment outcomes and stratify patients based on their tumor's unique profile.

Are there biomarkers that can help predict treatment response or prognosis in the context of tumor heterogeneity?

Yes. Mutations in genes such as p53, which play a role in maintaining genome stability, are associated with increased heterogeneity and poorer prognosis.

We also utilize tools like the MATH score (Mutant Allele Tumor Heterogeneity), derived from sequencing data, to quantify clonal diversity. The MATH score reflects the number of distinct alleles present within tumor DNA; a higher score indicates greater genetic heterogeneity in the tumor.

Single-cell RNA sequencing provides even deeper insight, revealing specific cell profiles that can inform treatment planning.

What are the most promising research directions in addressing tumor heterogeneity today?

The combination of single-cell and spatial sequencing with organoid models is incredibly promising.These tools allow us to model tumors ex vivo and test potential therapies in a patient-specific way.

AI is increasingly playing a larger role in identifying patterns and predicting therapeutic responses, enhancing clinical decision-making.

How can these research findings be brought into clinical practice to improve outcomes?

We're not quite there yet, but the potential is clear. Insights from heterogeneity profiling could be used to stratify patients more effectively and personalize treatment strategies, including surgery and radiation. This will require significant investment in infrastructure and data analysis capabilities, but it's a promising direction for the near future.

How do you see the role of AI in managing tumor heterogeneity and designing more adaptive therapies?

AI is becoming essential for making sense of the vast and complex datasets we now generate. It can uncover patterns we might otherwise miss and help predict treatment outcomes by analyzing millions of data points.

Many studies have already demonstrated the use of image-based deep learning models in cancer diagnostics, and it likely won't be long before these approaches are adapted to predict tumor responses to specific therapies.
As AI tools evolve, I believe they will become central to both diagnostics and therapy design—not just in oncology but across all areas of medicine.

FINANCIAL DIGNITY FOR CANCER PATIENTS

GIVING YOUNG ADULTS WITH CANCER A VOICE

BY ANNE JÄKEL

Cancer is often perceived as a disease of older adults, but thousands of young people face this diagnosis every year. In Germany alone, approximately 15,000 young adults between the ages of 15 and 39 are diagnosed with cancer annually, representing about 3% of all new cancer cases.[1]

Photo credit: Freepik

While this percentage may seem small, the impact is profound. These are individuals at the start of their adult lives, building careers, relationships, and families. A cancer diagnosis during this formative period disrupts not only health but also education, employment, and future plans.

Despite these unique challenges, young adults remain a largely invisible group in oncology.

They fall into a gap between pediatric and adult cancer care: too old for the comprehensive pediatric oncology networks, yet with needs that differ significantly from older adults.

This lack of visibility has serious consequences, from inadequate psychosocial services to insufficient research funding for age-specific treatment and survivorship issues.[2-4]

Addressing these gaps requires not only clinical innovation, but it also demands political advocacy, public awareness, and a strong voice to ensure that young adults are represented in healthcare systems and policy decisions.

At the forefront of these efforts is the German Foundation for Young Adults with Cancer (Deutsche Stiftung für junge Erwachsene mit Krebs), which works to close these critical gaps in care and research.

A foundation to fill a void

The German Foundation for Young Adults with Cancer was founded in July 2014 by the German Society for Hematology and Medical Oncology (DGHO) to create a dedicated platform for this underserved patient group.

From the outset, the foundation's mission has been twofold: [5]
1. To advance research into cancers affecting young adults, and
2. To improve care structures, long-term follow-up, and quality of life for those navigating survivorship.
Its charter outlines a broad set of goals:

Supporting scientific projects and young researchers through scholarships and awards.

Strengthening psychosocial care and reintegration into education and the workforce.

Raising public awareness through campaigns and lobbying efforts.

Building a network that connects clinicians, researchers, policymakers, and most importantly, patients themselves.

By combining the credibility of a scientific organization with the

advocacy of a patient-centered NGO, the foundation has positioned itself as a unique bridge between the worlds of research and policy.

Driving change through concrete initiatives

One of the foundation's most visible roles is supporting scientific progress. It funds young researchers and doctoral candidates whose projects focus on cancers in young adults, which is a field that remains chronically underfunded.

In 2025, the foundation introduced the Mathias Freund Prize, named after one of its founding figures, to recognize exceptional contributions to patient advocacy and care innovation for young adults.

This prize symbolizes the growing recognition of this patient group within the broader oncology community. [5]

For patients, the foundation provides practical, accessible resources that directly improve daily life: [5]

The Young Cancer portal and app:

A central hub where young adults can access expert counseling through phone, chat, or in-person appointments.

It also features a "tandem counseling" system that pairs newly diagnosed individuals with trained peers who have experienced cancer themselves, offering invaluable psychosocial support.

The study portal:

A curated platform listing relevant clinical studies for young adults, empowering both patients and clinicians to identify appropriate trial opportunities.

Regional support networks:

Local self-help and peer groups create safe spaces for ongoing emotional support and community building.

Podcasts, blog posts and campaigns:

Multimedia resources help demystify complex topics like fertility preservation, returning to work, or managing treatment side effects.

Beyond providing direct support to young adults with cancer, the foundation plays an active role in the scientific and political landscape.

Through its Scientific & Politics initiatives, it works to raise awareness among policymakers, healthcare stakeholders, and the wider public about the unique needs of this underserved age group.[5]

Why this lobbying effort matters

Lobbying is often viewed with skepticism, yet in healthcare it plays a vital role.

Without organized advocacy, vulnerable groups risk being overlooked in budget allocations and policy decisions. Young adults with cancer are a textbook example of this problem.

Because they represent a relatively small percentage of total cancer patients, their specific needs are rarely prioritized. Fertility preservation, mental health services, and educational reintegration programs are frequently underfunded or absent altogether.

By amplifying these concerns through evidence-based lobbying, the foundation ensures that young adults are not just passive recipients of care but active stakeholders in shaping the oncology landscape.

The foundation's dual identity, as both a scientific and advocacy organization, gives it exceptional credibility. It is trusted by clinicians, policymakers, and patients alike, making it a powerful intermediary capable of translating research findings into actionable health policy.

Practical resources for clinicians and oncologists

For German-speaking oncologists and other healthcare professionals, the foundation is not only a lobbying body but also a practical partner in patient care.
Here are some ways clinicians can leverage its resources:

Referral to the young cancer portal and app:

Direct young patients to this platform for expert advice, psychosocial counseling, and peer support.

This can greatly reduce the burden on clinical teams while ensuring comprehensive care for patients navigating complex psychosocial challenges.

Access to the study portal:

Use the portal to identify clinical trials and studies specifically designed for young adults, which are a population often underrepresented in mainstream oncology research.

Regional self-help groups:

Inform patients about local peer networks, which can play a critical role in long-term survivorship and mental health.

Collaborative projects:

Clinics and practices can partner with the foundation to host awareness events, develop educational materials, or co-sponsor research initiatives.

Advocacy participation:

Clinicians can support campaigns aimed at improving psychosocial services, insurance coverage for fertility preservation, and funding for specialized rehabilitation.

By incorporating these resources into their practice, oncologists can directly enhance the care journey of their young adult patients.

The gaps that remain

Research in Germany underscores significant gaps in care for young adults with cancer.

A qualitative analysis at Humboldt University Berlin showed fragmented and underdeveloped support systems, particularly in psychosocial care, vocational rehabilitation, and fertility counseling.[6]

Longitudinal data reveal persistent psychological distress, fatigue, lower life satisfaction, and difficulties in return to work, even years after treatment.[7,8]

Moreover, many young survivors face information deficits: while medical aspects may be covered, broader care needs, such as emotional, social, and survivorship planning, remain insufficiently addressed, reflecting a critical need for age-specific services and resources.[6]

Conclusion & call to action

The German Foundation for Young Adults with Cancer stands as an example of what targeted advocacy can achieve.

By funding research, offering psychosocial support, and lobbying for systemic improvements, it has brought visibility to a group that too often goes unheard.

Engaging with the foundation allows oncologists and healthcare teams to go beyond referral, actively improving care pathways and long-term outcomes for young adults with cancer.

Whether by directing patients to the Young Cancer Portal, participating in research collaborations, or supporting policy campaigns, clinicians can help amplify the voices of young adults and drive meaningful change.
Cancer care is evolving rapidly, with breakthroughs in precision medicine and immunotherapy reshaping treatment paradigms. Yet progress must not be measured only in survival rates and molecular targets.

Progress also means recognizing and addressing the unique challenges of every patient group, including young adults who face a lifetime of survivorship ahead.

By working together, clinicians, researchers, policymakers, and advocates, we can ensure that young adults with cancer receive not only the best treatments but also the resources, opportunities, and dignity they deserve.

Photo credit: Freepik

MISINFORMATION IN ONCOLOGY

WHY CLEAR, TRUSTED COMMUNICATION HAS NEVER BEEN MORE CRITICAL

BY ANNE JÄKEL

In today's digital world, oncologists face not only the challenge of treating cancer but also navigating a parallel epidemic: misinformation.

Photo credit: Freepik

The World Health Organization has even coined a term for it – an infodemic – to describe the overwhelming flood of information, both accurate and false, that patients encounter every day.[1]

A recent analysis of 200 highly popular social media articles on the four most common cancers found that 32.5% contained misinformation and 30.5% contained harmful information, with a striking 76.9% of articles containing misinformation also classified as harmful; importantly, these misleading or harmful articles generated significantly higher engagement than accurate content, highlighting the potential for widespread negative impact on cancer care decisions.[2]

This growing problem poses a serious threat to patient outcomes. When misinformation drives decisions, even the most effective therapies can fail. In oncology, where every decision has potentially life-or-death consequences, the stakes could not be higher.

The complexity of modern cancer care demands not only cutting-edge drugs and diagnostics, but also clear, trusted communication that helps patients and clinicians cut through the noise.

A growing tide of misinformation

Cancer is one of the most searched health topics worldwide.[3] Social media platforms such as TikTok, Instagram, and Facebook have

become major sources of health information, but unfortunately, they are also fertile ground for misleading or outright dangerous content.

The range of misinformation in oncology is broad:

Miracle cures:

Unproven treatments such as apricot kernels, alkaline diets, or extreme fasting protocols are still promoted as alternatives to evidence-based care.

Misinterpretation of real science:

Early-phase study results are sometimes exaggerated or stripped of critical context, leading patients to believe a new drug is more effective or accessible than it actually is.

Distrust of established treatments:

Persistent myths about chemotherapy or radiation therapy being more harmful than cancer itself continue to circulate.

The consequences are profound. A 2018 retrospective study published in the Journal of the National Cancer Institute showed that patients who opted for alternative medicine instead of conventional cancer treatments were more than twice as likely to die within five years of diagnosis.[4]

Misinformation not only misleads patients, it delays treatment, fuels anxiety, and in some cases, directly impacts survival.

Why oncology is especially vulnerable

Every area of medicine faces misinformation, but oncology is particularly exposed.

The emotional weight of a cancer diagnosis makes patients and families more vulnerable to persuasive, simplistic solutions that promise hope.

The complexity of modern cancer treatments, with their scientific terminology, evolving biomarkers, and genetic testing, makes it difficult for patients to fully understand their options. Adding to this challenge, the field of oncology evolves at lightning speed. New drugs, indications, and trial data are published almost daily. Even highly trained clinicians must work hard to stay current. In this environment, misinformation easily fills knowledge gaps.

One striking example is immunotherapy. While these treatments have revolutionized care for many cancers, public narratives often portray them as "miracle cures."[5]

Without careful explanation from clinicians, some patients overestimate their chances of response and discontinue other necessary treatments prematurely, with potentially dangerous consequences.

The digital amplifier

Social media has dramatically accelerated the speed and reach of misinformation. A landmark study published in Science found that false information spreads significantly faster, farther, deeper, and more broadly on social media than truthful information.[6]

Algorithms reward engagement rather than accuracy, meaning sensational or emotionally charged content is amplified, while nuanced, evidence-based updates are drowned out.

Cancer patients often turn to online communities for support. Many of these spaces provide genuine comfort and valuable peer-to-peer insights. However, they can also become echo chambers where unverified claims are repeated until they feel like truth.

At the 2025 ASCO annual meeting, oncologists warned that cancer patients were increasingly turning to unproven "natural" treatments promoted on social media platforms like TikTok, such as coffee enemas and radical diets, placing them at serious risk of refusing life-saving conventional treatments.[7]
The digital literacy gap compounds the problem. Most patients lack the skills to critically evaluate online content.[8]

As a result, oncologists often encounter patients who arrive at appointments armed with a mixture of accurate information, half-truths, and dangerous myths.

The hidden burden on clinicians

For oncologists and their teams, misinformation is not only an abstract problem, but a daily challenge that shapes patient interactions.

Although exact figures are lacking, clinicians report that a substantial portion of consultation time is now devoted to addressing misconceptions, challenging outdated beliefs, debunking popular myths, and providing accurate, evidence-based explanations.[9]

This additional workload can lead to longer visits, delayed decision-making, and increased frustration for both clinicians and patients.

Misinformation also undermines trust. When patients doubt their clinician's recommendations due to something they read online, the therapeutic relationship suffers.

Over time, this erosion of trust can contribute to clinician burnout, adding yet another layer to an already demanding specialty.

Fighting back: practical strategies

The battle against misinformation cannot be won through fact-checking alone.

Oncologists and HCPs need proactive strategies to prevent misinformation from taking root in the first place. Several approaches show promise:

1. Teach digital (health) literacy – Even brief interventions have shown promise; for example, media literacy campaigns in the U.S. and India reduced perceived trustworthiness of false headlines, and higher digital health literacy equips individuals to

better assess health claims.[10,11]

2. Use targeted educational tools – In low-literacy contexts, structured education can improve misinformation detection, though nuances remain.[12]

3. Leverage digital platforms for engagement – Tools like mobile applications and telehealth platforms are linked to improved health literacy and access, and enhancing digital readiness among HCPs is actively recognized as necessary.[13,14]

4. Partner with communicators – While quantifiable data is still emerging, collaboration between clinicians, journalists, and educators is widely seen as vital for translating complex oncology research into trusted, patient-friendly information.

The role of curated knowledge

One promising strategy to combat misinformation lies in curated, trustworthy platforms that help clinicians keep up with the ever-growing volume of scientific publications.

Instead of expecting oncologists and healthcare professionals to sift through hundreds of new studies each month, these platforms summarize and highlight the most relevant, practice-changing data in an accessible format.

Oncology Compass, for example, curates and distills key findings from current oncology research. By doing so, it provides clinicians with quick, reliable updates they can integrate into patient care and also share with their teams.

This streamlined access to high-quality information supports evidence-based decision-making and helps counteract the noise of misinformation by ensuring that accurate, contextualized knowledge reaches those who need it most.[15]

Conclusion & call to action

Misinformation in oncology is a direct threat to patient outcomes and clinician well-being. The sheer volume of digital content means that even the most dedicated oncologists cannot fight this battle alone.

To protect patients, the oncology community must embrace a proactive, collaborative approach.

Clinicians, scientists, communicators, and platforms like Oncology Compass must work hand in hand to ensure that accurate, contextualized information reaches those who need it most.

Clear and compassionate communication can be as life-saving as the therapies themselves. The challenge is great, but so is the opportunity: to conduct healthcare where every cancer patient receives not just the best treatment, but the best information.

Photo credit: Freepik

LUNG CANCER

IMFORTE TRIAL ESTABLISHES NEW MAINTENANCE STANDARD IN EXTENSIVE-STAGE SMALL-CELL LUNG CANCER

BY ANNE JÄKEL

Small-cell lung cancer (SCLC) is one of the most aggressive forms of lung cancer, representing approximately 13–15% of all lung cancers.[1,2]

Among patients diagnosed with extensive-stage SCLC (ES-SCLC), the disease is typically widespread at presentation, and prognosis remains dismal.[3,4]

Historically, platinum-based chemotherapy has been the backbone of treatment, producing high initial response rates but almost universal relapse within months.[5]

Median overall survival (OS) for these patients has rarely exceeded 12 months, underscoring the urgent need for therapeutic innovation.[3]

The addition of immune checkpoint inhibitors (ICIs) to chemotherapy has marked the first meaningful advance in decades.

Landmark trials such as IMpower133 and CASPIAN demonstrated that adding PD-(L)1 inhibitors such as atezolizumab or durvalumab to standard carboplatin and etoposide improved survival, establishing chemo-immunotherapy as the global standard of care for first-line treatment.[6-8]

However, despite these gains, most patients experience disease progression shortly after completing the initial four cycles of chemo-immunotherapy.[6,8]

Attempts to extend disease control using maintenance therapy with single-agent ICIs or cytotoxic agents have been largely unsuccessful. [9,10]

Lurbinectedin, a selective inhibitor of oncogenic transcription, has shown promising activity as a second-line agent in relapsed SCLC, leading to accelerated approval by the U.S. Food and Drug Administration.[11,12]

Its unique mechanism of action, which includes modulation of the tumor microenvironment, raised the hypothesis that lurbinectedin could complement immune checkpoint blockade in earlier disease settings.[11,13]

The phase 3 IMforte trial was designed to test whether adding lurbinectedin to maintenance atezolizumab following induction chemo-immunotherapy could improve outcomes for patients with ES-SCLC. [14,15]

IMforte: study design and patient population

IMforte (NCT05091567) was a global, open-label, randomized, phase 3 trial. Treatment-naïve patients with histologically confirmed ES-SCLC were eligible if they had completed four 21-day cycles of induction therapy consisting of carboplatin, etoposide, and atezolizumab, without evidence of disease progression.

All patients had an ECOG performance status of 0 or 1 at study entry. [14,15]

After induction, eligible patients were randomized 1:1 to receive maintenance treatment every three weeks with either lurbinectedin (3.2 mg/m² IV, with G-CSF prophylaxis) plus atezolizumab (1200 mg IV) or atezolizumab alone.

Randomization was stratified by key prognostic factors, including the presence of liver metastases at baseline, use of prophylactic cranial irradiation, ECOG performance status, and lactate dehydrogenase (LDH) levels.

Crossover between study arms was not allowed to preserve the integrity of overall survival data. [14,15]

The trial's co-primary endpoints were progression-free survival (PFS), assessed by an independent review facility (IRF) according to RECIST v1.1 criteria, and overall survival (OS) from the time of randomization into the maintenance phase. Secondary endpoints included safety and

tolerability. [14,15]

Between study initiation and data cutoff (July 29, 2024), a total of 660 patients were enrolled, with 483 randomized into the maintenance phase (242 to lurbinectedin plus atezolizumab and 241 to atezolizumab alone).

Baseline characteristics were generally well balanced between groups. [14,15]

Efficacy results

At a median follow-up of 15.0 months, the IMforte trial met both of its primary endpoints, demonstrating a statistically significant improvement in PFS and OS with the addition of lurbinectedin. [14,15]

Median PFS was 5.4 months (95% CI: 4.2–5.8) in the lurbinectedin plus atezolizumab group compared with 2.1 months (95% CI: 1.6–2.7) in the atezolizumab-only group, representing a 46% reduction in the risk of progression or death (HR 0.54, 95% CI: 0.43–0.67; p<0.0001). [14,15]

For OS, the combination arm achieved a median survival of 13.2 months (95% CI: 11.9–16.4), compared with 10.6 months (95% CI: 9.5–12.2) with atezolizumab alone. This translated to a 27% reduction in the risk of death (HR 0.73, 95% CI: 0.57–0.95; p=0.0174). [14,15]

These results represent the first time a maintenance strategy has been shown to improve both PFS and OS in the extensive-stage setting.

The absolute gains, an extension of PFS by more than three months and OS by 2.6 months, are clinically meaningful in this aggressive disease, where incremental progress has been difficult to achieve. [14,15]

Safety profile

The addition of lurbinectedin was associated with higher rates of treatment-related adverse events (TRAEs), but no new or unexpected safety concerns emerged. [14,15]

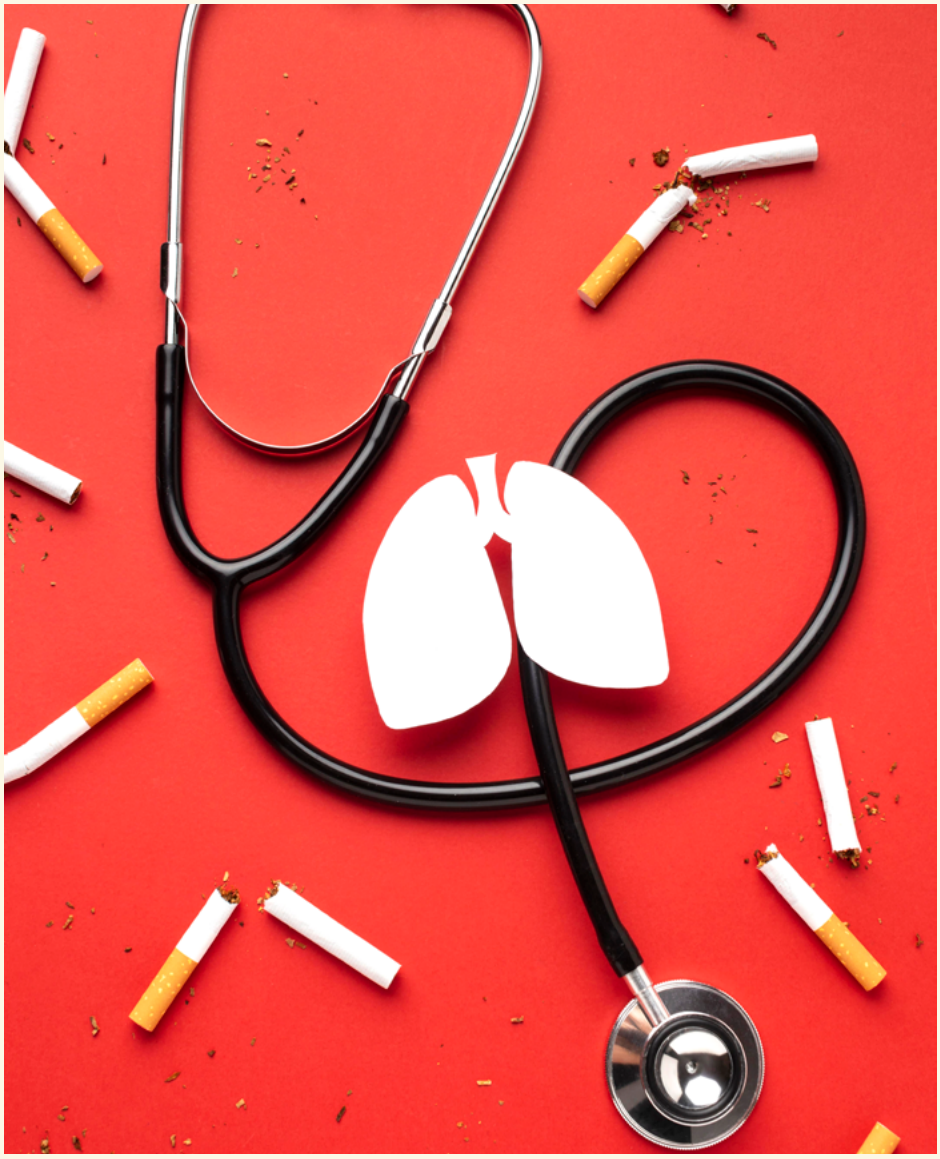


Photo credit: Freepik

TRAEs of any grade occurred in 83.5% of patients receiving the combination versus 40.0% of those receiving atezolizumab alone. Grade 3–4 TRAEs were more frequent in the combination arm (38% vs. 22%).

The most common high-grade events were hematologic, consistent with lurbinectedin's known toxicity profile. [14,15]

Grade 5 events occurred in 5% of patients in the combination arm and 3% of patients in the monotherapy arm.

Treatment discontinuation due to adverse events occurred in 6.2% of patients receiving the combination versus 3.3% of those receiving atezolizumab alone. [14,15]

Clinical context

The IMforte trial addresses a long-standing challenge in SCLC: how to sustain disease control after initial response to therapy.

In earlier landmark studies such as IMpower133 and CASPIAN, the benefit of PD-(L)1 inhibitors was largely concentrated during the induction phase, with single-agent maintenance offering limited additional durability. [6-8]

Previous maintenance strategies, including cytotoxic agents and other immunotherapy combinations, failed

to produce meaningful improvements in survival. [9,10]

Lurbinectedin's distinct mechanism of action, by selectively inhibiting oncogenic transcription while also modulating the tumor microenvironment, appears to complement immune checkpoint blockade. [11,13]

This synergy likely underlies the dual improvement in PFS and OS observed in IMforte.

By establishing the first evidence-based maintenance strategy for ES-SCLC, IMforte may shift the treatment paradigm.

The results suggest that patients who respond or achieve stable disease after induction therapy could benefit from the continued combination of lurbinectedin and atezolizumab, rather than atezolizumab alone.

Limitations and future directions

Although the IMforte results are highly promising, several limitations should be acknowledged.

The trial's open-label design introduces potential bias, though the use of independent radiologic review helps mitigate this risk.

Median follow-up was 15 months, and longer-term data will be needed to fully assess the durability of the OS benefit.

Additionally, the study population consisted of relatively fit patients (ECOG 0–1), leaving questions about the generalizability of results to more frail individuals.

The increased toxicity associated with the combination also underscores the need for careful patient selection and proactive management of hematologic side effects.

Photo credit: Freepik

Future research will focus on biomarker analyses to identify subgroups most likely to benefit from this approach, as well as studies integrating newer therapeutic modalities such as novel checkpoint inhibitors.

Implications for clinical practice

IMforte provides a strong rationale for changing the current treatment algorithm for ES-SCLC.

For patients who do not progress after induction chemo-immunotherapy, maintenance with lurbinectedin plus atezolizumab should be considered a new standard of care.

The combination's ability to significantly extend both PFS and OS represents an important advance in a disease that has historically been resistant to therapeutic progress.

Clinicians will need to balance these benefits against the higher rates of toxicity, ensuring that patients receive appropriate supportive care and monitoring throughout treatment.

Conclusion

The IMforte trial is the first global phase 3 study to demonstrate that maintenance therapy can meaningfully improve survival outcomes in extensive-stage small-cell lung cancer.

By combining lurbinectedin with atezolizumab after standard induction chemo-immunotherapy, investigators achieved significant gains in both progression-free and overall survival.

These results herald a new era for ES-SCLC, offering hope to patients and clinicians confronted with one of the most aggressive forms of lung cancer.

With appropriate patient selection and toxicity management, maintenance lurbinectedin plus atezolizumab has the potential to become a cornerstone of first-line treatment and set the stage for future innovations in this challenging disease.

NOVEL IMAGING TECHNIQUES

HYBRID INNOVATION: EXPLORER AND THE FUTURE OF IMAGING IN ONCOLOGY

BY: ANĐELIKA KALEZIĆ

Positron emission tomography (PET) and computed tomography (CT) are essential imaging techniques in oncology. PET scans show metabolic activity in tumors by detecting radiotracer uptake,[1] while CT offers detailed anatomical information to locate and measure lesions.[2] Combined, PET/CT allows clinicians to stage cancer, evaluate treatment response, and detect recurrence and metastasis. [3,4] However, traditional modalities have certain limitations in clinical practice: limited axial coverage requiring multiple consecutive scans, which increases time and patient discomfort; sensitivity limits that may miss small or low-uptake lesions; and repeated scans that add to cumulative radiation exposure. [5,6]

Several years ago, the U.S. Food and Drug Administration granted 510(k) clearance to the uEXPLORER total-body combined PET/CT scanner, developed by United Imaging Healthcare in collaboration with the EXPLORER Consortium at the University of California, Davis.[7]

This marked the arrival of the first FDA-approved scanner capable of imaging the entire human body simultaneously.

By early 2019, the system was operational, offering oncologists an unprecedented tool for molecular and anatomical imaging with significantly shorter acquisition times and the ability to improve diagnostics, assess disease progression, and research new therapies. [8]

The EXPLORER platform was born from a $15.5 million NIH Transformative Research Award in 2015 to develop the first whole-body PET scanner.

It increased sensitivity and reduced radiation exposure by a factor of 40, with significantly shorter scanning time of approximately 15-30 seconds instead of 15-20 minutes.[9] Initially conceived for research, EXPLORER quickly demonstrated potential for clinical transformation—particularly in oncology.

How EXPLORER works

Traditional PET scanners capture metabolic activity using radiotracers; however, their limited axial field of view requires sequential bed positions to cover the body, leading to longer scans and higher cumulative radiation exposure. With 194-cm axial field of view, the EXPLORER scanner overcomes these constraints by covering the entire body in a single pass. This enables ultra-fast acquisitions—less than 1 minute in some cases—while improving sensitivity and reducing signal-to-noise ratio.[10]

The scanner integrates PET with CT, providing both functional and structural information in the same session. This dual-modality approach is critical in oncology, where detecting and characterizing lesions relies on understanding both their metabolic activity and anatomical context. [11]

Landmark first human imaging study

The first human imaging studies with EXPLORER, published in 2019 in The Journal of Nuclear Medicine, confirmed the transformative potential of total-body PET.

Conducted at UC Davis, these trials demonstrated that the 194-cm axial field of view allowed complete adult body coverage in a single acquisition in more than 99% of the population.

The system enabled whole-body dynamic imaging with frame durations as short as one second, making real-time pharmacokinetic studies feasible for the first time. High-quality images were obtained with ultra-low tracer doses of

approximately 25 MBq (0.7 mCi) or in scan times of around 1 minute.[10]

These findings established that EXPLORER could detect small lesions, capture detailed tracer kinetics, and operate with flexibility to prioritize speed, dose reduction, or extended imaging windows—laying the groundwork for new diagnostic and research applications in oncology.

Recent clinical insights

A notable example of clinical utility came from a 2025 UC Davis study focusing on parametric cancer imaging. This advanced PET analysis method generates maps of tracer kinetics to accurately quantify biological parameters of interest, such as blood flow, glucose metabolism, and receptor binding, over time.[12] This approach provides more detailed physiological information than static PET images, supporting precise assessment of tumor aggressiveness and therapy response.

The study used a relative Patlak plot method—a mathematical modeling technique for analyzing irreversible tracer uptake, particularly relevant for FDG-PET in oncology. By incorporating AI-driven noise reduction, researchers were able to reduce acquisition time for kinetic imaging to just 20 minutes without compromising accuracy.[13]

This work, published in the Journal of Nuclear Medicine, demonstrated the feasibility of integrating rapid, quantitative imaging protocols into routine oncology practice, potentially expanding their use for treatment monitoring and drug development, including in pediatric patients who cannot tolerate prolonged scanning time.

Advantages for cancer detection and management

EXPLORER's strengths translate into tangible benefits for cancer care:

Earlier detection and whole-body mapping:

The scanner's high sensitivity and resolution reconstruction improve the visualization of small features, which could lead to earlier detection of tumors or metastases, previously undetectable by conventional PET/CT.

Single-pass imaging provides a comprehensive full-body assessment, which could be particularly beneficial in staging cancers with widespread metastatic disease.[10]

Reduced radiation:

The ability to operate with lower tracer doses reduces cumulative exposure, which could be of particular importance for pediatric oncology and serial imaging in treatment monitoring.[14, 15]

Dynamic imaging:

High temporal resolution enables whole-body kinetic studies, which could significantly contribute to advancing diagnostics and therapy assessment, as well as tracer development.[16, 17]

The next step: hybrid PET-enabled dual-energy CT

In August 2025, UC Davis secured a new $2.5 million NIH grant to integrate EXPLORER's PET capability with dual-energy CT technology.[18]

The innovation leverages PET data to generate a secondary high-energy CT dataset, enhancing tissue differentiation without additional radiation exposure or costly hardware modifications.[19]

This approach could improve the ability to distinguish between malignant and benign tissue, refine staging, and guide biopsies more accurately.

If successful, PET-enabled dual-energy CT could become a powerful tool in oncology imaging, adding another layer of precision to cancer detection and characterization.

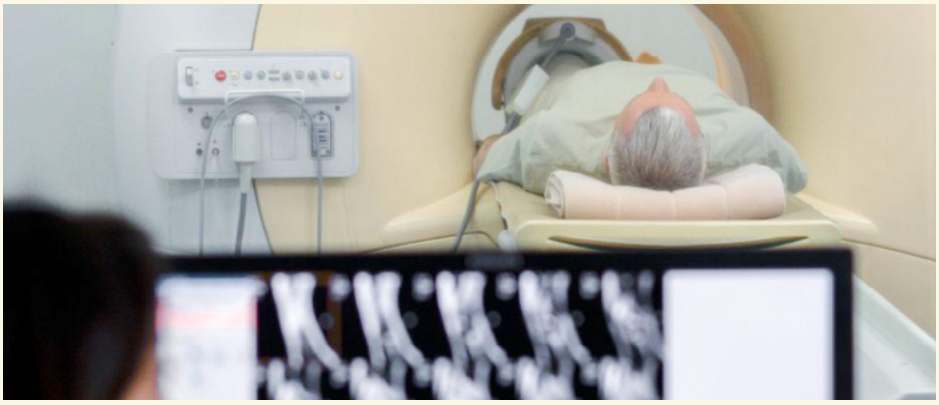
Looking ahead

Several trials are currently in progress to evaluate uEXPLORER's performance in specific cancers.

These include a UC Davis–led trial (NCT04478318) on using uEXPLORER in comparison to conventional PET/CT in patients with lung cancer, lymphoma, and melanoma to determine the minimum scan duration time that results in non-inferior image quality.[20]

Another is a pilot study (NCT05160480) led by UC Davis to improve the clinical use of uEXPLORER by collecting PET scan data using FDA-approved imaging agents from patients with prostate, neuroendocrine, or breast cancer.[21]

As these technologies develop and become better integrated into clinical workflows, hybrid imaging platforms like EXPLORER could redefine the standards for cancer detection and treatment—bringing in a new era of precision oncology.



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FDA APPROVALS IN Q2 2025

EXPANDING THE FRONTIERS OF CANCER THERAPY

BY ANĐELIKA KALEZIĆ

Between April and June 2025, the U.S. Food and Drug Administration granted 13 oncology approvals, including eight novel therapies across solid tumors and hematologic malignancies.[1] These decisions highlight the agency's dedication to diversifying immunotherapy approaches, broadening the use of antibody-based therapeutics, and approving precision therapies for rare oncogenic drivers. Overall, these approvals represent not just incremental progress but also significant advances in addressing unmet clinical needs.

Checkpoint inhibitors: expanding the PD-1 landscape

Checkpoint inhibitors continue to be a key part of modern cancer treatment. In Q2 2025, two new agents were added to the PD-1 inhibitor class: Retifanlimab-dlwr and Penpulimab-kcqx. Both act by blocking the PD-1 receptor on T cells, restoring immune responses that tumors suppress in the microenvironment.[2, 3]

Promise and advantages of mRNA cancer vaccines:

Retifanlimab was approved in combination with carboplatin and paclitaxel for inoperable locally recurrent or metastatic squamous cell carcinoma of the anal canal (SCAC). This represents the first PD-1–based regimen for SCAC, supported by pivotal trial data from the phase III POD1UM-303/ InterAACT 2 trial (NCT04472429), which demonstrated an improved progression-free survival of 9.3 months (95% CI: 7.5, 11.3) in the retifanlimab-dlwr arm and 7.4 months (95% CI: 7.1, 7.7) in the placebo arm (hazard ratio 0.63 [95% CI: 0.47, 0.84] p-value 0.0006)) and an overall response rate of 56% (95% CI: 48, 64) and 44% (95% CI: 36, 52) in the retifanlimab-dlwr and placebo arms, respectively.[4]

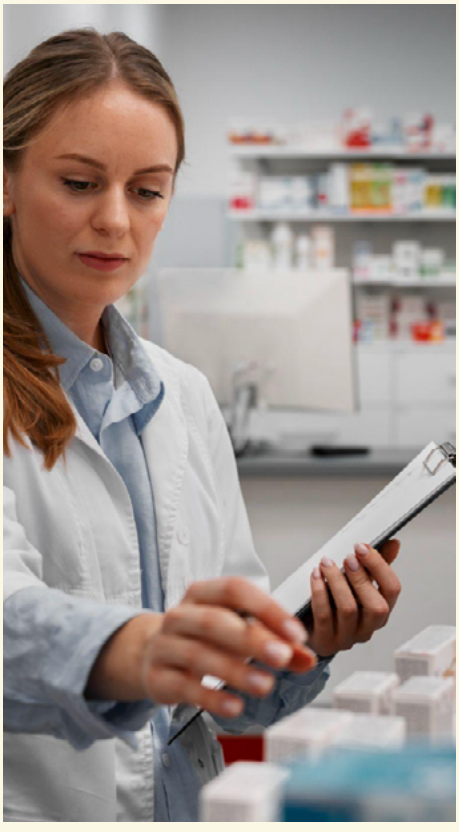

Photo credit: Freepik

Penpulimab received approval with cisplatin or carboplatin and gemcitabine for recurrent or metastatic non-keratinizing nasopharyngeal carcinoma (NPC), supported by clinical evidence from the phase III AK105-304 trial (NCT04974398), adding another option for a disease where immune checkpoint inhibition has shown particular promise.

The primary efficacy outcome measure was progression-free survival, as assessed by BICR according to RECIST v1.1. Median progression-free survival was 9.6 months (95% CI: 7.1, 12.5) in the penpulimab-kcqx arm and 7.0 months (95% CI: 6.9, 7.3) in the placebo arm (hazard ratio [HR] 0.45 [95% CI: 0.33, 0.62], two-sided p-value <0.0001).[5]

Antibody-based therapies: precision immuno-oncology

Antibody-based therapies continue to advance, with both antibody–drug conjugates (ADCs) and monoclonal antibody combinations gaining traction. ADCs deliver toxic agents directly to tumor cells using antigen-specific antibodies, while monoclonal antibodies influence immune pathways or directly target tumor-associated antigens to stimulate anti-tumor immune responses.[6]

Telisotuzumab vedotin-tllv is a c-Met–targeted ADC approved for locally advanced or metastatic, non-squamous non-small cell lung cancer (NSCLC) with high c-Met protein overexpression. By combining tumor-specific targeting with a potent cytotoxic payload, telisotuzumab vedotin demonstrated meaningful activity in patients with limited options, as shown in the phase II

LUMINOSITY trial (NCT03539536).

The major efficacy outcomes were overall response rate and duration of response, determined by BICR according to RECIST 1.1. The overall response rate was 35% (95% CI: 24, 46) and median duration of response was 7.2 months (95% CI: 4.2, 12).[7]

Tafasitamab-cxix, in combination with lenalidomide and rituximab, was approved for relapsed or refractory follicular lymphoma (FL). Tafasitamab targets CD19 on B cells, enhancing immune-mediated cytotoxicity.

The triplet regimen leverages complementary mechanisms of action to improve outcomes in patients who have exhausted standard therapies, as supported by the phase III inMIND trial (NCT04680052).

After a median follow-up of 14.1 months, progression-free survival was statistically significantly longer in the tafasitamab-cxix arm (hazard ratio 0.43 [95% CI: 0.32, 0.58]; p-value <0.0001). The estimated median progression-free survival was 22.4 months (95% CI: 19.2, not evaluable) in the tafasitamab-cxix arm and 13.9 months (95% CI: 11.5, 16.4) in the placebo arm.[8]

Small-molecule targeted therapies: new generations of precision agents

Beyond immunotherapy, Q2 2025 approvals showcased the diversity of small-molecule targeted therapies. These agents reflect a growing trend in oncology toward rational drug design aimed at intercepting resistance mechanisms and exploiting tumor-specific molecular dependencies.[9]

Taletrectinib, a next-generation tyrosine kinase inhibitor, was approved for locally advanced or metastatic ROS1-positive non-small cell lung cancer (NSCLC).

It offers improved activity against acquired resistance mutations compared to earlier ROS1-directed agents, as demonstrated in the phase II TRUST-I trial (NCT04395677). The major efficacy outcome measures were overall response rate and duration of response, determined by BICR according to RECIST v1.1.

Overall response rate was 90% (95% CI: 83, 95) with 72% of responders having a duration of response (DOR) ≥12 months in treatment-naïve patients, compared to pretreated patients where the overall response rate was 52% (95% CI: 39, 64), with 74% of responders having a DOR ≥6 months.[10]

Combination of Avutometinib and Defactinib was approved for KRAS-mutated recurrent low-grade serous ovarian cancer (LGSOC) in patients who have received prior systemic therapy.

Avutometinib inhibits the RAF/MEK pathway, while defactinib targets focal adhesion kinase (FAK), together providing dual blockade of critical tumor survival pathways, as demonstrated in the phase II RAMP 201 trial (NCT04625270) where the overall response rate assessed by BICR according to RECIST v1.1. was 44% (95% CI: 31, 58).[11]

Belzutifan received approval for locally advanced, unresectable, or metastatic pheochromocytoma and paraganglioma (PPGL) in adult and pediatric patients. As a first-in-class inhibitor of hypoxia-inducible factor 2-alpha, belzutifan disrupts tumor adaptation to hypoxia.

Its approval was supported by clinical evidence from the phase II LITESPARK-015 trial (NCT04924075), which demonstrated an overall response rate of 26% (95% CI: 17, 38) and a median duration of response of 20.4 months (95% CI: 8.3, NR).[12]

Reformulated classics: novel delivery approaches

Not all innovation comes from new molecular targets. **Mitomycin intravesical solution** was approved for recurrent low-grade intermediate-risk non-muscle invasive bladder cancer. Though mitomycin is a decades-old chemotherapy, its novel formulation and intravesical delivery offer patients a bladder-sparing alternative to more invasive treatments, as demonstrated in the phase III ENVISION trial (NCT05243550), showing that 78% (95% CI: 72, 83) of patients had a complete response defined as no detectable disease in the bladder by cystoscopy and urine cytology at 3 months.[13]

Current trends and future outlook

The Q2 2025 approvals reinforce key trends in oncology. These approvals highlight the ongoing expansion of PD-1 inhibition into rare and difficult-to-treat cancers beyond the established indications in melanoma and lung cancer. Additionally, antibody-based approaches, both ADCs and monoclonal antibodies, are proving versatile tools for tackling resistant disease.

These approvals emphasize how antibody platforms can be tailored either toward precise delivery of cytotoxic drugs or immune modulation through synergistic combinations. Third, small-molecule therapies increasingly aim to target previously intractable pathways.

These agents demonstrate how rational drug design and pathway targeting continue to produce effective therapies. Finally, innovations in delivery remain crucial in transforming how established drugs are deployed. Confirmatory trials, typically phase III studies required by the FDA to verify the clinical benefit of therapies granted accelerated approval, play a critical role in establishing the durability and reproducibility of early findings.

As confirmatory trials mature, these therapies will determine whether accelerated approvals result in durable, practice-changing standards. For now, the oncology community enters the second half of 2025 with an expanded arsenal of tools to personalize treatment, address unmet needs, and improve patient outcomes.

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OPINION PIECE

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UPCOMING ONCOLOGY CONFERENCES THIS AUTUMN

Oncology Compass Digest presents a selection of medical conferences happening this autumn. The Oncology Compass Calendar is the most comprehensive calendar of global oncology conferences.

Be sure to check out the whole calendar on www.oncologycompass.com/calendar and find more conferences.

NOVEMBER 2025

ESMO AI & Digital Oncology Congress 2025

Location:
Berlin, Germany

Date:
12 Nov -14 Nov

Cancer Indication:
General

www.oncologycompass.com/calendar/esmo-ai-digital-oncology-congress-2025

NOVEMBER 2025

IGCS 2025 Annual Global Meeting

Location:
Cape Town, South Africa

Date:
5 Nov - 7 Nov

Cancer Indication:
Hematologic malignancies

www.oncologycompass.com/calendar/igcs-2025-annual-global-meeting

NOVEMBER 2025

International Kidney Cancer Symposium (IKCS) 2025 North America

Location:
Denver, Colorado

Date:
13 Nov - 15 Nov

Cancer Indication:
Genitourinary cancers

www.oncologycompass.com/calendar/international-kidney-cancer-symposium-ikcs-2025-north-america

NOVEMBER 2025

Annual Scientific Meeting of the Society for Neuro-Oncology (SNO) 2025

Location:
Honolulu, Hawaii

Date:
19 Nov - 23 Nov

Cancer Indication:
Neurological cancers

www.oncologycompass.com/calendar/annual-scientific-meeting-of-the-society-for-neuro-oncology-sno-2025

DECEMBER 2025

ESMO Congress Asia 2025

Location:
Singapore

Date:
5 Dec - 7 Dec

Cancer Indication:
General

www.oncologycompass.com/calendar/esmo-congress-asia-2025

DECEMBER 2025

ESMO Immuno-Oncology Congress 2025

Location:
London, United Kingdom

Date:
10 Dec - 12 Dec

Cancer Indication:
General

www.oncologycompass.com/calendar/esmo-immuno-oncology-congress-2025

INSIGHTS FOR Q3 2025

ONCOLOGY COMPASS IS GLOBALLY BECOMING AN INCREASINGLY IMPORTANT PLATFORM FOR ONCOLOGISTS

17 SCIENTIFIC LEADERS

selected practice-relevant publications for lung, renal, gastro-esophageal, melanoma, multiple myeloma, prostate cancer and triple-negative breast cancer

892 PRACTICE RELEVANT ARTICLES PUBLISHED

- 253 NSCLC
- 146 ESOPHAGEAL & GASTRIC CANCER
- 28 MPM
- 122 MELANOMA
- 45 TNBC
- 82 SCLC
- 99 RENAL CELL CARCINOMA
- 85 MULTIPLE MYELOMA
- 31 PROSTATE CANCER

473 ACTIVE USERS

TOP 3 FILTER CRITERIA:

- NSCLC 418
- MELANOMA 285
- RCC 283

*Total number of clicks on filter criteria over time

WEBSITE VISITORS	6,373
PAGEVIEWS	9,812
SESSIONS	7,055
AVG. SESSION	01:20
PAGES PER SESSION	1.39

VISITORS BY DEVICES

DEVICE CATEGORY	TOTAL VISITORS	PAGEVIEWS
Mobile	3,733	4,054
Desktop	2,026	2,388
Tablet	570	607

VISITORS BY GENDER

Total female	64%
Total male	36%

TOP 10 COUNTRIES WHERE VISITORS COME FROM:

COUNTRY	VISITORS	SESSIONS
1. Ireland	1,156	1,266
2. United States	1,120	1,250
3. Poland	493	515
4. Germany	486	502
5. Sweden	263	274
6. United Kingdom	205	248
7. Malta	203	226
8. Switzerland	186	228
9. France	159	166
10. Netherlands	150	149

The number of Visitors represents all visitors to Oncology Compass, both registered and non-registered users. The metrics for Users relate to the Registered Users data who have full access to the Oncology Compass platform.

VISITORS BY AGE / GENDER

AGE	VISITORS	PAGEVIEWS
1. 65+	474	545
2. 45-54	256	290
3. 25-34	236	281
4. 55-64	203	227
5. 35-44	170	193
6. 18-24	118	164

MOST READ BLOG ARTICLES:

mRNA vaccines: A new frontier in cancer immunotherapy

Need specific audience data?

Our data analysts will gather it at your request.

Contact oncologycompass@capptoo.com for more info.

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Digest

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Please share your opinion and your story ideas by using the QR code below:

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