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NOT FOR SALE

*At **CancerWorld**, we don't just publish science, we publish stories. As **Alberto Costa,** reminds us: **CancerWorld** is not about papers, it's about people.*

The June, 2025 issue brings that philosophy into sharp focus. On our dual cover, we feature two individuals whose lives and leadership exemplify what it means to serve, not from the stage, but from the ground up.

*In a rare and candid interview, **Her Royal Highness Princess Adila bint Abdullah Al Saud** opens the doors to a life guided by purpose — advocating for children with cancer, championing women's empowerment, and extending dignity to the most overlooked. Her work with the Sanad Pediatric Cancer Foundation is not simply charity, it is structural compassion, and it is changing lives.*

*Across continents, in a mountain cottage on Ithaca, we find **Alberto Costa** — breast cancer surgeon, educator, ESO founder, and reluctant public figure — reflecting on medicine, mentorship, and meaning. His story is at once intensely personal and unmistakably European. He has trained generations of oncologists and quietly built institutions that outlast careers.*

But these profiles are only the beginning. This issue moves from South Africa to Santiago, from Guangzhou to Vancouver, tracing the global effort to close the gap between what science can achieve and what systems deliver.

We cover pediatric researchers in Canada using chicken eggs to test therapies in real time. We explore the inspiring story of Catharine Young from Lab to the White House. We examine the cultural and scientific negotiations of traditional Chinese medicine as it interfaces with modern oncology, and explore a pioneering hereditary cancer program in Chile, where genetic counselling and sequencing are finally reaching underserved communities — and shaping national strategy.

*We trace the microbial footprints of cancer, mapping the pathogens that drive oncogenesis across the globe and opening new fronts in prevention. Our News Editor, **Janet Fricker,** dives into a study uncovering how microbiota-derived bile acids can block androgen receptors and enhance anti-tumour immunity — revealing an unexpected axis between the gut, hormones, and immune response.*

*Then take a hard look at culture. In a special feature aligned with World No Tobacco Day, our Co-Editor-in-Chief, **Adriana Albini**, in collaboration with **Francesca Albini,** explores the quiet resurgence of smoking in cinema — and its implications for public health. In an era shaped by imagery, the fight against tobacco addiction now includes the fight for narrative accountability.*

*Finally, we celebrate a story of perseverance that began far from the spotlight. In Curious, Rejected, Accepted, **Dr. Amalya Sargsyan** reflects on her path from medical student in Armenia to practicing oncologist — and how a fellowship from the European School of Oncology (ESO) transformed her career.*

Whether through science, storytelling, or systems change, CancerWorld remains committed to elevating the human dimension of cancer care. In every issue, we ask: Who are the people shaping this field, not just in the lab, but in life?

This month, we invite you to meet them.

Yeva Margaryan, Managing Editor, CancerWorld

Princess Adila: Her Royal Highness, Her Human Mission

Inside the remarkable journey of Princess Adila bint Abdullah Al Saud

Gevorg Tamamyan

When I entered the room, it felt as though I had stepped into a page from an Arabian fairytale. The room was quiet and serene, adorned with elements of nature and refined décor that evoked calm and timeless elegance. Then Her Royal Highness arrived.

Despite a tightly packed schedule and knowing I would be leaving within a few hours, Her Royal Highness Princess Adila bint Abdullah Al Saud graciously made time for the interview. Instantly, her presence brought clarity: kind, calm, wise, and noble—her grace was unmistakable. In that moment, it was easy to see why she is a transformative figure in Saudi society: a strong advocate for women's rights, a champion for social justice, and the chairperson of the Sanad Pediatric Cancer Foundation.

No Child Left Behind

"I've always believed that healing goes far beyond medicine," says Princess Adila bint Abdullah Al Saud, reflecting on her decades-long commitment to improving the lives of children with cancer. "It's about human dignity, compassion, and making the burden of illness just a little lighter."

Her journey with Sanad, one of the Kingdom's most impactful cancer support organizations, began in 1999. But her vision for healthcare reform took root even earlier.

"I had already started the National Home Healthcare Foundation in 1997," she explains. "It focused on stable patients—helping them transition from hospital to home with proper medical support. But when friends of mine, one of whom had a child in King Fahd Center, noticed the immense social and financial hardship of many families, I knew we had to do more."

Sanad began as a personal mission. "It was just a group of women determined to help. We saw the need, we talked to the hospitals, and eventually we made it official. We got licensed and began supporting families of children with cancer more formally. But at its core, it was always about compassion."

That spirit has never changed. "Today, our mission remains: no child left behind. And that includes children who are not Saudi, who aren't eligible for free healthcare here. We help them too—because **suffering doesn't recognize nationality.**"

Princess Adila shares a particularly moving story from a recent hospital visit.

"There was this little girl—she had been admitted through the emergency room and started treatment immediately. But because of her refugee status, she couldn't be released. If she left, she wouldn't be allowed back in. So she stayed in that hospital room for nine months. Imagine that—nine months."

She pauses. "Her family had to split. The father kept working, siblings were elsewhere. Only the mother stayed with her. Eventually, we intervened and helped with the paperwork. Seeing her reunited with her family was... incredible. That moment stays with you."

The Human Cost Of Disparities

Despite her humility, it's clear Princess Adila is deeply involved. "I'm not a centralized leader," she says. "I believe in empowering others. But I visit hospitals, I meet the families. I hear their stories. And every time, I'm reminded how strong these children are. They are the heroes—not us."

Her work has taken her far beyond hospital walls. She speaks with urgency about global disparities in cancer care.

"**Eighty-five percent survival in wealthy countries. Ten to twenty percent in others. It's unacceptable.**These families are not just fighting cancer—they're fighting poverty, malnutrition, and systemic neglect."

She describes an initiative launched by King Abdullah Foundation: five floating hospitals in Bangladesh. "They go into rural areas by river, bringing care directly to those in need. It's more than healthcare—it's about building trust. Many of these families are scared of hospitals. Volunteers have to bridge that gap."

For Princess Adila, this is a matter of justice. "When we wake up, we have clean water. Food. Healthcare.

These families wake up wondering if their child will survive the day."

Her concern extends to children in conflict zones.

"I don't know what 'humanity' means anymore," she says, visibly shaken. "Hospitals are bombed. Medical workers are attacked. Children are trapped in sieges with no medicine. And the world watches. **Institutions meant to protect us are failing.**"

Still, she insists on speaking out. "Yes, it's dangerous. Yes, people are silenced. But it is our duty to speak. Especially when children are dying because someone decided their lives don't matter."

Empowering Women

She shifts to a more hopeful note when discussing her advocacy for women's rights. "I believe that if a woman is financially independent, she has power—in her home, and in society."

Her work with the Khadija bint Khuwaylid Center, under the Jeddah Chamber of Commerce, aimed to do just that.

"We weren't forcing women to work. We were giving them the option. And we helped businesses open their doors to women, especially low-income ones."

She recalls working with Batterjee Pharma. "They built a segregated workspace for women, added a nursery so mothers could bring their kids—and their profits rose 40%. These women—some of them single moms—became providers. It changed lives."

Transportation was a hurdle. "Women couldn't drive then, and half their salary went to hiring drivers. So the company sent buses to pick them up. Again—a win-win."

She smiles. "Dr. Batterjee became a role model. He took a risk. And others followed."

It's this mix of realism and quiet determination that defines Princess Adila's work. She doesn't seek the spotlight. But her fingerprints are on some of the most meaningful social reforms in the Kingdom.

"I don't want to lead for the sake of leading," she says. **"I want to serve. That's what matters."**

The Warmth Of Saudi Arabia

"There's a perception about Saudi Arabia," says Princess Adila, reflecting on how international visitors often expect something far more rigid and uninviting. "But when people come, they're surprised by the warmth. You need to be here, to feel it."

I echoed her point, sharing my own surprise: "I was expecting formality and distance. But what I found was openness, civility, and kindness."

Princess Adila smiles. "That's why it's important to visit and not just believe what you hear from afar."

"You Learn, You Build Relationships, You Grow"

While widely associated with educational progress, she humbly clarifies her role. "Education was always my husband's path—he served as Minister of Education. We worked in parallel, exchanged ideas, but I was never directly involved."

Instead, she poured her energy into cultural heritage. "I volunteered at the National Museum for ten years with a group of seven women. Our mission was to make the museum more inviting—especially for children and families who saw it as a place only for the elite."

They organized events and designed school programs that moved classroom lessons into museum exhibits. "History became tangible," she explains. "Children learned by experiencing. That had a deep impact."

Eventually, this program expanded from private schools to public ones, creating a model of educational inclusion through cultural spaces.

As for the widely praised international scholarship program, she gives credit to her late father, King Abdullah. "The scholarship program began long ago, during King Saud's time. But my father expanded it dramatically. He believed it wasn't just about academics—it was about culture."

"He used to say: **your values don't weaken by knowing others—they become richer.** You learn, you build relationships, you grow. And he practiced that. On every trip, he made time to meet with students abroad—he called them his sons and daughters."

What It Means To Be A Princess?

Asked what it means to be a Princess, she responds without hesitation: "**Responsibility.** You try to be a role model, and that's not always easy. But you try." To the younger generation, her message is equally clear: "**Never stop learning. And be confident in your identity.** I hate the dilution of cultures. I love seeing a country keep its own character—its own craft, language, rhythm."

On building teams, she emphasizes inclusion and humility. "I'm not sure what model I follow, but I believe in diverse expertise. No one person sees the whole picture. When we come together from different angles—social, financial, clinical—we make fewer mistakes."

She believes wisdom can come from anywhere. **"You learn from doctors. From drivers. From the janitor who cleans the office. If your mind is open, learning never stops."**

When asked about her favorite book, she pauses thoughtfully. "I don't have just one. But I love biographies—real lives, real stories."

One she found unforgettable was the two-volume autobiography of Jamil Hijelan.

"It spans seven Saudi kings. He wrote about his childhood, his mother raising him alone, his years in Syria and Egypt. It was vivid and deeply human. I couldn't put it down."

She's currently reading the memoir of Amr Moussa. "It's a harder read, but relevant—especially with everything happening in Gaza. It gives historical context, and foresight."

And yes, she's writing her own. "I'm nearly finished. Just in revision—but it feels like that revision may never end," she laughs.

Asked for an exclusive, she promises: "Once it's done, I'll give you the first interview. And yes—it includes a chapter about Sanad, and every initiative I've supported."

Finally, when asked whom she would recommend for the next interview, she smiles warmly.

"Princess Haifa Faisal," she says. "Not because she's my cousin—but because she has remarkable stories to share. I'm sure you'll find them inspiring."

After a few hours I was on the plane, leaving the Kingdom of Saudi Arabia—deeply inspired by the beauty of the country, the warmth of its people, and the masterclass of wisdom from my final interview with a leader whose quiet strength moves nations and whose heart remains firmly with those she serves.

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How a Chicken Egg Model Could Transform Pediatric Cancer Treatment in Canada

Researchers at UBC's faculty of medicineare using chicken eggs tostudy how childhood cancers respond to different treatments. Photo Credit: Paul Joseph, UBC

Victoria Forster

A new, innovative pipeline fusing genomics, proteomics and modelling in animals and chicken eggs is pinpointing new treatments for children with rare and relapsed cancers in Canada.

Researchers in Canada have used fertilized chicken eggs to grow tumors from children with high-risk and relapsed cancers, allowing them to test personalized therapy approaches.

The study, published in EMBO Molecular Medicine[1] combines molecular and genetic analysis techniques and several types of modelling on samples from children with difficult-to-treat cancers who are running out of treatment options.

"The pipeline is really applicable to all pediatric cancers, but of course there is a specific need for rarer cancers in children," said Philipp Lange, PhD, one of the corresponding authors of the paper and Associate Professor in the Department of Pathology and Laboratory Medicine, Faculty of Medicine, University of British Columbia in Vancouver.

The initial paper is part of an ongoing pan-Canadian study which aims to expand personalized medicine approaches particularly for children with rarer cancers in Canada.

"We are lacking standard treatments that are usually developed for bigger patient groups because there is a larger market and it's also scientifically easier to develop treatments for bigger groups of patients," Lange added.

In the study, the patient with spindle epithelial tumor with thymus-like elements (SETTLE) with metastases was ultimately prescribed antidepressant drug sertraline after the pipeline identified SHMT2 as a target for therapy.

After receiving sertraline, the patient showed decreased tumor growth rates, but with clinically progressive disease. However, the researchers stress the work is still in the early stages and proves the pipeline can be beneficial.

"Traditionally, precision oncology is done by genome sequencing, which has become reasonably standard over the last few years. But the predictions are not always right, patients don't respond or have limited or temporary responses," said Lange.

The team also looked at proteomics of the tumor,

identifying dysregulated proteins to look for functional drug targets, combining this with the genomic analyses.

"We need to understand more about individual cancers, so we added another layer to our analyses. Most drugs actually interfere with cancer cells by acting on proteins, so we hypothesized that measuring proteins as well as genetic information would be very valuable," said Lange.

The patients who are currently eligible for enrollment in the program either have high-risk disease at diagnosis which is unlikely to be successfully treated by existing treatment regimens, or they have relapsed and are running out of treatment options.

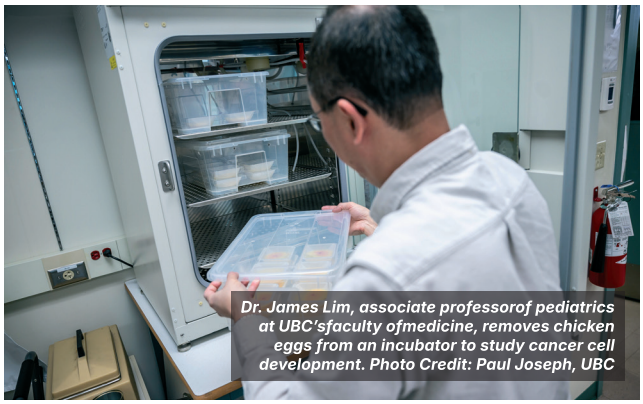
Lange explains that eligible patients undergo a biopsy, then the sample is split to send for genome analysis, proteomics and to the modelling teams for live culture. Lange describes how the process in the case described in the EMBO journal article took around 2 months but with refinement of the pipeline, he hopes this can be shortened to around a month.

"Our chicken egg model is particularly fast. We use a fertilized egg and put a piece of tumor on top, which then gets blood flow from the egg. This creates a very open and accessible model after just a couple of days with healthy tissue that our researchers can then use to test drugs," said Lange.

Conventionally, different tumor types from blood cancers to brain cancers require optimized and tightly regulated growth conditions, nutrients and sometimes "scaffolding" matrices comprised of complex cocktails of proteins to ensure they grow properly in the lab. But the chicken egg model seems to overcome many of these bespoke needs.

"This model has turned out to be quite universally applicable in supporting the growth of tumors," said Lange.

The information from the molecular analyses and modelling, which can also include more traditional mouse and zebrafish models is combined to present to a board of specialists. The board includes data analysts, oncologists and researchers to discuss whether any extra clinical tests should be done, or whether recommended drugs should be moved forward into patient care.



Dr. James Lim, associate professorof pediatrics at UBC'sfaculty ofmedicine, removes chicken eggs from an incubator to study cancer cell development. Photo Credit: Paul Joseph, UBC

"It's important to note all of this is done in a research context to provide additional information to be considered by the treating physician and family to make a decision together," said Lange.

Currently, the analysis pipeline focuses on drugs which are approved in Canada for pediatric use already, and where there is an established access pathway, a "list of 20-30" agents according to Lange. Wider drug screens are theoretically possible, but limited approvals and safety data in pediatric patients would make findings "virtually non-applicable" for children according to Lange.

"In this case, the drug was already being used in a pediatric context, for depression so it was more approachable in a short timeline," said Lange, noting that the researchers are ultimately aiming for a one-month turnaround as the timeline is still too long for patients with more aggressive tumors.

Although the pipeline has so far focused on accessible, established drugs – Lange notes that the model can be adapted to investigate how tumors might respond to newer therapies such as immune checkpoint inhibitors and CAR T cells too.

"Anything where molecular pathways or surface knockers are indicative of a sensitivity to that treatment, we can incorporate. We're currently looking at CAR T cell treatment for neuroblastoma using the chicken egg model," said Lange.

References: *1. Barnabas GD, Bhat TA, Goebeler V, et al. Proteomics and personalized PDX models identify treatment for a progressive malignancy within an actionable timeframe. EMBO Mol Med (2025)17:625-64. https://www.embopress.org/doi/full/10.1038/s44321-025-00212-8*

Photo source: China Anti-Cancer Association

Tina Jiang

Chinese Traditional Medicine: Building A Bridge To Modern Oncology

China's oncologists want to see robust evidence before putting their faith in traditional remedies. Practitioners of traditional medicines struggle with concepts of experimental medicine and animal models. But interest is growing in finding ways to bridge that gap in approaches and understanding.

Before the introduction of modern medicine to China in the 19th century, traditional Chinese medicine (TCM) played a dominant role in healing within Chinese society. Now, in the rapidly evolving landscape of cancer care, TCM is carving a niche as a complementary approach within modern oncology.

This integration is gaining attention, especially in China.

"The whole country is discussing the integration of Chinese and Western medicine, but the current integration is not so good," says Wang Guijun, a Chinese surgeon who has been treating gastric cancer for over two decades.

"Perhaps from the perspective of enhancing the body's overall health, or helping manage complications during chemotherapy or radiotherapy, maybe TCM has a role to play," he adds, "however, it often focuses on individual cases – what prescriptions were used or how the tumour regressed – without large sample analysis," he notes.

While acupuncture has been shown to alleviate chemotherapy-induced nausea and pain, and certain herbal remedies are believed to boost immune function, Wang, like many oncologists in China educated in modern medicine, believes that in general there is insufficient evidence to show that TCM is effective.

Interest in trying to develop stronger scientific evidence around TCM is attracting a growing number of researchers. Among them is Leng Jing, an immuno-oncologist at Guangxi University of Chinese Medicine. "Just because Western doctors are sceptical doesn't mean they dismiss TCM. It's more about the limited data making it hard to believe in its efficacy," she says. As a 'Western doctor' researching TCM, she recognises the many challenges to integrating the two systems.

"First, there's a language barrier. The terminology of TCM differs significantly from that of Western medicine. And you are using ancient Chinese to talk to modern people. Of course, they don't understand," she explains.

"We need to translate TCM concepts so that others can understand what we are doing"

Leng believes that individuals like her, who understand both TCM and modern medicine, can serve as vital bridges in this integration. "We need to translate TCM concepts so that others can understand what we are doing."

In recent years, her research has focused on immune recognition receptors – proteins on the outside of cells by which the immune system identifies antigenic substances – [AW1] and their roles in combating tumours. She has also worked on developing animal models to advance TCM research. A particular focus of hers is on the potential use of TCM in liver cancer, for which China accounts for more than 45% of all new cases across the globe, with particularly high incidence rates in Guangxi.

"In China, unlike other countries, more than 90% of liver cancer is caused by the hepatitis B virus. Chronic inflammation from this viral infection can lead to fibrosis, cirrhosis, and ultimately liver cancer," explains Leng. As she points out, some of the traditional Chinese herbs are known for their anti-inflammatory effects. "Can we use these herbs to intervene in the inflammation-cancer transformation process to prevent tumors?" is the question she is asking.

In 2021, Leng successfully established an animal model based on the crab-eating macaque – a type of monkey native to Southeast Asia, which she believes is ideal for studying alcoholic fatty liver and damp-heat syndrome within TCM.

Nowadays, in diagnosing illnesses, many traditional Chinese medicine practitioners still mainly rely on the 'four methods': observation, listening, inquiry, and pulse diagnosis. Leng says that, despite her advances, she faces challenges in persuading many TCM practitioners to accept her animal models as applicable to their practice.

"Some practitioners misunderstand the model. They ask how we can communicate with animals. Are we supposed to make the little mice nod?"

Leng disagrees with those who view TCM solely through a modern scientific lens, arguing that they miss its essence. While the two systems are distinct, she believes TCM practitioners cannot completely detach from modern science.

"If your Chinese herbal compounds are grounded in solid models, and if objective data from clinical practice align with TCM symptoms and characteristics, I believe the West will eventually embrace Chinese medicine."

Myriam Vidal Valero

Towards A Familial Cancer Service For Chile

A regional programme for genetic sequencing and counselling is being piloted in Chile to promote prevention and early detection in people with a hereditary high-risk of cancer.

Advances in genetic sequencing technologies are making it cheaper and easier to test people for hereditary gene mutations that confer a higher risk of certain diseases, including cancers. Integrating genetic testing services into health systems is key to identifying people at hereditary high risk, and helping them manage that risk through monitoring and/or preventive therapies. However, access to these genetic testing tools remains limited in Latin America. A new regional hereditary breast cancer programme in Chile is working to bridge these gaps.

Chile's National Cancer Plan, established by the Ministry of Health in 2022, prioritises cancer prevention, and includes genetic counselling as part of its strategy. However, like many Latin American countries, Chile still struggles to make these preventive measures widely available. Barriers include a shortage of specialised professionals, limited infrastructure, and restricted access to genetic testing services.

To address these needs, a team of experts, including oncologists, breast specialists, midwives, molecular biologists and geneticists, from different national and international institutions, launched a pilot hereditary cancer programme in Chile's Maule region. By focusing on early detection and prevention, the programme aims to shift resources from expensive treatments to proactive care, ultimately helping to reduce deaths from hereditary cancers.

Between 2022 and 2023, the programme recruited 48 patients with breast cancer from Hospital Regional de Talca, in Maule, who were suspected of having hereditary breast cancer syndrome. Genetic variants inherited from birth account for almost 20% of all cases of breast cancer, explains Ramón Pérez-Castro, biochemist at the School of Medicine of the Catholic University of Maule in Chile, and co-author of a study showcasing the project's initial findings.

The patients enrolled in the pilot programme first spoke to an oncology counsellor via telemedicine to discuss their medical and family history, mapping out their genealogy to identify both healthy and affected cancer relatives. Approximately 63% were found to have a close relative with cancer, and 77% of the enrolled patients were diagnosed before age 50. The patients then took a genetic test that analysed 162 genes linked to inherited cancer. After testing, participants received further counselling to discuss their results.

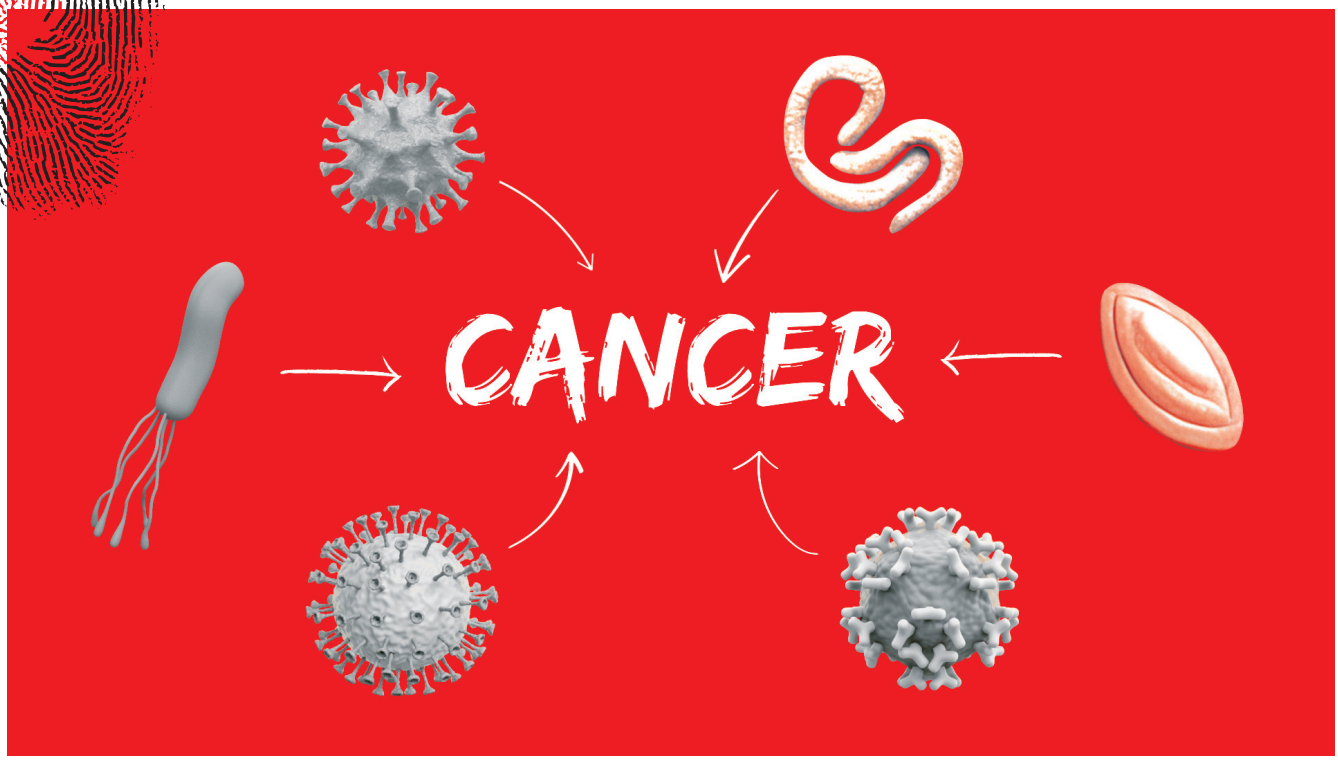
The test results revealed that 12% of the patients carried harmful changes in genes such as BRCA1, BRCA2, TP53, and PALB2 that are known to be associated with a raised risk of cancer. They also showed new genetic mutations that had never been reported before.

Relatives of patients who tested positive for genetic changes linked to cancer were invited get tested to see whether they also carry these genes. Physicians used international NCCN guidelines to offer preventive measures – such as early screening or surgery – to family members carrying specific genetic variants to reduce their cancer risk.

"Cancer treatment is much more expensive than preventing it," said Sonia Margarit, a genetic counsellor at Clínica Alemana Universidad del Desarrollo in Chile, who wasn't involved with the study. However, the country still needs to develop more strategies to address the needs of the more than 70% of patients in the public system, who have access to standard preventive medical services but not to these types of specialised services.

Since the first group, the programme has continued counselling and testing new patients. It is not only helping people with hereditary cancer but also creating a national genetic data bank. Experts hope this will improve understanding of the genetics of cancer in the country. "It is necessary to have local databases," Pérez-Castro says, adding that, as new variants appear, "we can also compare them with what is happening in other countries."

Pérez-Castro hopes this programme will inspire other regional governments in Chile to take similar action. He encourages them to understand the specific barriers patients face, and to develop local healthcare programmes and interventions, and to explore potential alliances between public and private institutions. "We have to come together to respond to the local needs of the territory."



Adriana Albini

Agents Of Mutation: Pathogens As Catalysts Of Carcinogenesis

Cancer has traditionally been viewed as a non-communicable disease—one driven by genetic predisposition, environmental exposure, and lifestyle factors. Yet a substantial and increasingly compelling body of epidemiological research has shown that a significant proportion of cancers are caused by infectious agents. The most authoritative global data on cancer incidence and mortality are compiled by the International Agency for Research on Cancer (IARC), a specialized agency of the World Health Organization. IARC's Global Cancer Observatory (GCO) hosts GLOBOCAN, an interactive platform that provides contemporary estimates of cancer burden worldwide, including incidence, mortality, and prevalence data for 36 cancer types across 185 countries. According to GLOBOCAN 2020, approximately 19.3 million new cancer cases were diagnosed globally that year (https://gco.iarc.fr/). While GLOBOCAN does not currently provide updated figures specific to

infection-attributable cancers, the most detailed global estimate remains IARC's 2018 analysis, which attributed around **2.2 million new cases to infectious agents such as human papillomavirus (HPV), Helicobacter pylori, hepatitis B virus (HBV), and hepatitis C virus (HCV).** This figure—representing roughly one in eight cancer cases worldwide—has remained consistent across multiple large-scale studies and continues to inform our understanding of the infectious contribution to cancer globally.

However, the burden of infection-related cancers varies significantly across regions.; in low- and middle-income countries, the attributable fraction can exceed 25%, while in high-income regions it is closer to 7–8%. Such disparities reflect differences in the prevalence of oncogenic pathogens, as well as in the accessibility and implementation of preventive measures, such as vaccination and antimicrobial therapies.

The principal infectious agents implicated in human carcinogenesis include a spectrum of viruses, bacteria, and parasites. Among viruses, high-risk human papillomavirus (HPV) types are causally linked to cervical, anogenital, and oropharyngeal cancers, while hepatitis B and C viruses (HBV, HCV) are major drivers of hepatocellular carcinoma. Epstein–Barr virus (EBV), Kaposi's sarcoma-associated herpesvirus (KSHV/HHV-8), human T-cell lymphotropic virus type 1 (HTLV-1), and Merkel cell polyomavirus (MCPyV) are also well-established oncogenic viruses, each associated with specific malignancies. Bacterial pathogens, most notably Helicobacter pylori, are responsible for a significant proportion of gastric cancers, with chronic infection leading to a cascade of inflammatory and genotoxic events that culminate in malignant transformation. Parasitic infections, such as those caused by Schistosoma haematobium, are linked to squamous cell carcinoma of the bladder, particularly in regions where these parasites are endemic.

Recent research has expanded the landscape of infection-associated cancers to include a potential role for fungi, with emerging evidence suggesting that mycobiome dysbiosis may contribute to carcinogenesis in certain contexts. While the mechanistic links between fungal species and cancer are less well-defined than for viruses and bacteria, this area is an active focus of investigation.

Historical Background: When Infections Were Found To Cause Cancer

The connection between infections and cancer has evolved over more than a century of scientific investigation. The earliest landmark came in 1908, when Danish researchers Vilhelm Ellerman and Oluf Bang demonstrated that leukemia in chickens could be transmitted via a cell-free extract, laying the foundation for the concept of viral oncology. In 1911, Francis Peyton Rous confirmed these findings with the discovery of Rous sarcoma virus, confirming that cancer could be caused by an infectious agent.

More than fifty years later, in 1964, Epstein and Barr identified the Epstein-Barr virus (EBV) in a sample of Burkitt lymphoma, marking the first time a human cancer was linked to a virus.

In the 1970s and early 1980s, hepatitis B virus (HBV) was strongly linked to liver cancer, and by the mid-1980s, human papillomavirus (HPV) types 16 and 18 were discovered in cervical cancer tissues. These findings culminated in the 1994 classification of Helicobacter pylori as a Group I carcinogen by IARC, firmly placing bacteria among the infectious causes of cancer.

2008 marked a milestone when Harald zur Hausen received the Nobel Prize for linking HPV to cervical cancer, solidifying viruses as central players in carcinogenesis. This era also saw the discovery of Merkel cell polyomavirus (MCV) by Yuan Chang and Patrick Moore, which was directly implicated in Merkel cell carcinoma through its disruption of tumor suppressor pathways.

By 2012, the Cancer Genome Atlas (TCGA) revealed microbial signatures in tumors, including Fusobacterium nucleatum enrichment in colorectal cancer (CRC). Subsequent studies demonstrated that F. nucleatum promoted CRC progression via FadA adhesin-mediated Wnt/β-catenin activation and immune suppression through myeloid-derived suppressor cell recruitment.

2013 ushered in a new era with the FDA approval of ipilimumab, a CTLA-4 checkpoint inhibitor for melanoma, highlighting how immune evasion

tactics used by pathogens like HPV could be therapeutically targeted. By 2015, groundbreaking studies revealed the gut microbiome's role in modulating immunotherapy efficacy. For example, Bacteroides fragilis and Bifidobacterium species were found to enhance anti-PD-1 responses in melanoma by activating dendritic cells, while antibiotic use correlated with reduced treatment success.

In 2016, the WHO's Global Burden of Disease Study reaffirmed infections as responsible for ~15% of cancers worldwide, with Helicobacter pylori, HPV, HBV, and HCV dominating this landscape. H. pylori was reclassified as a Group I carcinogen due to its role in gastric cancer via CagA-induced genomic instability. By 2017, PD-1/PD-L1 inhibitors like pembrolizumab gained traction, with studies showing better responses in tumors linked to pathogens such as EBV+ gastric cancer. 2018 GLOBOCAN data highlighted stark disparities: infections caused 26% of cancers in sub-Saharan Africa (vs. 4% in high-income nations), driven by HPV, HBV, and Schistosoma haematobium.

2019 brought a paradigm shift with Bülent Aykut's Nature study, which identified Malassezia globosa as a key fungal contributor to pancreatic cancer. The research demonstrated that Malassezia activated the complement cascade (C3/MBL pathway), driving IL-33–mediated immunosuppression and tumor progression. Antifungal therapy improved survival in preclinical models, cementing the mycobiome's role in oncogenesis and opening new avenues for microbiome-targeted therapies.

Building on these findings, Avinash Alam and collaborators further elucidated the downstream immunological effects in a 2022 Cancer Cell study. They showed that Malassezia globosa triggers the Dectin-1–Src–Syk–CARD9–NF-κB signaling pathway in pancreatic cancer cells, leading to the secretion of the cytokine IL-33. The release of IL-33 was found to recruit immunosuppressive Th2 cells and innate lymphoid cells (ILC2s) to the tumor microenvironment, fostering a state of immunosuppression that facilitates tumor progression. Notably, antifungal treatment or genetic deletion of IL-33 in mouse models resulted in reduced tumor burden and improved survival, underscoring the clinical relevance of targeting the mycobiome and its associated immune pathways in pancreatic cancer.

In the early 2020s, research into fungal oncogenesis gained significant momentum. Notably, the discovery that Candida albicans can promote oral squamous cell carcinoma progression via upregulation of IL-17A/IL-17RA signalling and modulation of the tumour immune microenvironment was reported by a research team led by Xiaoyan Wang and colleagues. Their study, published in 2023, demonstrated that C. albicans infection increased oral cancer incidence in mouse models and promoted tumor progression through activation of the IL-17A/IL-17RA pathway, which in turn attracted and polarized tumor-associated macrophages toward an immunosuppressive phenotype. The study further showed that neutralization of IL-17A or depletion of macrophages alleviated the tumor-promoting effects of C. albicans infection, providing a mechanistic link between chronic candidiasis, inflammatory pathway dysregulation, and malignant progression in oral cancer.

Around the same time, the discovery that 66.8% of Schistosoma haematobium-associated bladder squamous cell carcinomas (BSCC) express PD-L1 was reported in a 2022 study conducted in Egypt, as detailed in the mini-review by Anselmo Mucavele published in Frontiers in Immunology. This study analysed tumor microarrays from Egyptian patients with pure squamous cell carcinoma (pSCC), of whom 81.2% had clinical indications of schistosomiasis. This discovery suggested that immune evasion is a central feature of schistosomiasis-related cancers and pointed to the potential for anti-PD-1/PD-L1 immunotherapies in these settings.

Concurrently, the discovery that KRAS p.G12D mutations serve as a genetic determinant for Fusobacterium nucleatum enrichment in colorectal cancer (CRC) was reported in a 2024 study published in Nature Communications. This research, led by a team investigating the interplay between somatic mutations and gut microbiota, demonstrated that F. nucleatum preferentially colonizes CRC tumors harboring the KRAS p.G12D mutation compared to wild-type or other KRAS variants (e.g., p.G13D). The study utilized both patient-derived CRC tissues and Villin-Cre/Kras

G12D+/- mouse models to establish this genotype-microbiome link.

The discovery that triple-negative breast cancer (TNBC) exhibits mycobiome shifts toward Candida dominance, which correlates with CD8+ T-cell exhaustion, was first reported in a 2021 preclinical study led by Shiao et al. (PMC8830498). Their work demonstrated that intestinal fungi, including Candida, modulate the tumor microenvironment (TME) by promoting immunosuppressive macrophage polarization (CD206+F4/80+) and PD-1 upregulation on CD8+ T cells. Depleting fungi with antifungals (e.g., amphotericin B) reduced PD-1+ T-cell populations and enhanced Granzyme B expression, restoring cytotoxic T-cell activity.

Subsequent studies in 2023–2024 extended these findings to TNBC-specific models. Researchers observed that Candida enrichment in TNBC tumours exacerbated CD8+ T-cell dysfunction via IL-10/IL-4-driven suppression and TCF1/TOX transcriptional reprogramming. Combining antifungals with anti-PD-1 immunotherapy reversed these effects, achieving 2.3-fold tumour regression in murine models compared to monotherapy. This synergy was attributed to fungal depletion restoring MHC-I antigen presentation and reducing myeloid-derived suppressor cell infiltration.

The discovery that high-risk HPV/EBV co-infection induces super-enhancer remodelling in head and neck cancers (HNCs) was reported in a 2024 study led by Almoghrabi et al. (Front. Oncol., 2020). While their work focused on HPV/EBV co-presence correlating with high-grade tumours and epithelial-mesenchymal transition (EMT), subsequent mechanistic studies in 2023–2024 revealed that HPV16 E6/E7 and EBV LMP1 synergistically remodel super-enhancers near oncogenes like MYC and NOTCH1. This remodelling amplifies their expression, accelerating tumor progression by 2.3-fold compared to single infections. These findings were validated in in vitro models of oropharyngeal squamous cell carcinoma (OPSCC), where dual infection increased metastatic potential via Erk/β-catenin pathway activation.

These scientific advances have begun to translate into clinical practice. In 2024, the National Comprehensive Cancer Network (NCCN) updated its guidelines to include 16S rRNA and ITS sequencing for colorectal cancer patients, enabling the stratification of Fusobacterium-high tumours. Patients identified through this approach showed a 34% better response to FOLFOX combined with ceftazidime regimens. Meanwhile, further research into Schistosoma haematobium revealed that its eggs secrete the ω-1 ribonuclease, which upregulates PD-L1 on myeloid-derived suppressor cells. Targeted anthelmintic therapy was shown in clinical trials to reduce PD-L1 levels by 72%, providing a new strategy for immunomodulation in infection-associated cancers.

Mechanisms Of Pathogen-Induced Carcinogenesis

The mechanisms by which infectious agents induce cancer are diverse and complex, encompassing direct genomic integration and alteration, chronic inflammation, immune evasion, and modulation of host cell signalling pathways. For example, viral oncoproteins can disrupt cell cycle regulation and inhibit tumour suppressor functions, while bacterial toxins and inflammatory mediators can induce DNA damage and promote a pro-tumorigenic microenvironment. Understanding these pathogen-specific and host-mediated processes is critical for the development of targeted preventive strategies—such as vaccines and antimicrobial interventions—as well as for informing novel therapeutic approaches.

Chronic inflammation is one of the most extensively documented mechanisms by which pathogens initiate tumorigenesis. Persistent microbial presence leads to sustained tissue damage, increased oxidative stress, and dysregulated repair processes. For instance, Helicobacter pylori disrupts the gastric epithelium and activates inflammatory pathways such as NF-κB, increasing levels of COX-2 and pro-inflammatory cytokines like IL-1β. The resulting environment, rich in reactive oxygen and nitrogen species (ROS/RNS), contributes to DNA damage and progressive genetic instability, particularly mutations in TP53. In the liver, hepatitis B virus (HBV) induces chronic inflammation through its surface antigen (HBsAg), which activates immune cells like Kupffer cells to release IL-6. This cytokine, in turn, stimulates the oncogenic STAT3 pathway, driving hepatocyte proliferation and promoting hepatocellular

carcinoma (HCC). HBV-related oxidative stress is further exacerbated by environmental carcinogens such as aflatoxins, with combined exposure known to induce specific TP53 mutations.

Direct Oncogenic Activity: certain pathogens contribute to cancer by introducing viral or bacterial proteins that directly disrupt cell cycle control and apoptotic pathways. Human papillomavirus (HPV), for example, expresses E6 and E7 oncoproteins. E6 targets the tumour suppressor p53 for degradation, while E7 binds and inactivates retinoblastoma (Rb) proteins, releasing E2F transcription factors that drive uncontrolled cell proliferation. Epstein-Barr virus (EBV) exemplifies oncogenic mimicry through its latent membrane protein 1 (LMP1), which acts as a constitutively active CD40 receptor. This activates signalling pathways including NF-κB and PI3K/AKT, enhancing cell survival and immune evasion in B cells, as observed in lymphomas and nasopharyngeal carcinoma.

Immunosuppression: pathogens also promote tumour development by suppressing or evading immune surveillance. HIV significantly increases the risk of Kaposi sarcoma by depleting CD4+ T-cells and enabling unchecked replication of Kaposi's sarcoma-associated herpesvirus (KSHV). KSHV produces viral interleukins and G-protein-coupled receptors (vGPCRs) that stimulate angiogenesis and cellular transformation.

EBV also employs immune evasion tactics, such as expressing the BZLF1 protein to downregulate MHC class I molecules, thereby reducing recognition by CD8+ cytotoxic T-cells. EBV-encoded microRNAs suppress stress-induced ligands like MICB, enabling infected cells to persist undetected.

Epigenetic and Genomic Alterations: beyond direct gene disruption, pathogens modulate host gene expression through epigenetic reprogramming. EBV has been shown to induce DNA methyltransferase activity (e.g., DNMT3B), silencing tumour suppressors such as PTEN and CDKN2A. In nasopharyngeal carcinoma, EBV non-coding RNAs influence histone modifications, removing repressive marks from oncogenic loci. HBV integrates into host DNA at sites such as the TERT promoter, activating telomerase and promoting cellular immortality. Integration events often occur near DNA repair genes like MLH1, facilitated by the HBx protein, which induces double-strand breaks in host DNA.

Many pathogens act through multiple converging mechanisms. HPV, for example, not only disables tumour suppressors but also rewires metabolic pathways via PI3K/mTOR activation. HBV's HBx protein simultaneously promotes oxidative stress and inhibits DNA repair, creating a self-reinforcing cycle of genomic instability.

These mechanistic insights are informing targeted therapies. Immune checkpoint inhibitors like PD-1 antagonists show promise in treating HPV-related cancers, while antioxidant nanoparticles and CRISPR-based tools are being investigated for their potential to reverse pathogen-induced genomic damage.

How Pathogens Shape Cancer Landscapes

Pathogens that contribute to cancer development span all major microbial categories—viruses, bacteria, parasites, and fungi—each with distinct biological traits and oncogenic strategies.

Viruses

Viruses represent the most well-established category of oncogenic pathogens and are the most extensively characterized oncogenic pathogens, leveraging genomic integration, oncoprotein expression, and immune evasion to drive malignancy. Human papillomavirus (HPV), particularly high-risk strains HPV-16 and HPV-18, is responsible for over 90% of cervical cancers and a significant proportion of anogenital and oropharyngeal malignancies. HPV oncogenesis centres on the E6 and E7 proteins: E6 recruits the E6AP ubiquitin ligase to degrade p53, disabling apoptosis and genomic surveillance, while E7 binds and inactivates retinoblastoma protein (pRb), disrupting cell cycle control (Zur Hausen, 2008; Moody & Laimins, 2010). These actions, combined with E6/E7-mediated activation of Wnt/β-catenin signaling, create a permissive environment for malignant transformation (White et al., 2010).

Hepatitis B virus (HBV) and hepatitis C virus (HCV) collectively account for ~80% of hepatocellular

carcinoma (HCC) cases. HBV integrates into host DNA, causing insertional mutagenesis (e.g., disrupting TERT or MLL4) and expressing the HBx protein, which dysregulates NF-κB, Wnt/β-catenin, and DNA repair pathways (Levrero & Zucman-Rossi, 2016). In contrast, HCV, an RNA virus, induces oxidative stress via ROS/RNS production, activating pro-fibrotic TGF-β and STAT3 signaling while impairing antioxidant defenses (Machida et al., 2006). Both viruses synergize with aflatoxin B1 exposure, multiplying HCC risk in endemic regions (Kew, 2004).

Epstein-Barr virus (EBV) is implicated in Burkitt lymphoma, Hodgkin lymphoma, and 10% of gastric cancers. Its oncogenic latency proteins, notably LMP1, mimic CD40 signaling to constitutively activate NF-κB and MAPK pathways, driving proliferation and immune evasion (Young & Rickinson, 2004). EBV also induces epigenetic silencing of tumor suppressors like PTEN through viral miRNAs, as demonstrated in nasopharyngeal carcinoma (Tsao et al., 2014).

Kaposi's sarcoma herpesvirus (KSHV) and Merkel cell polyomavirus (MCV) exemplify niche-specific oncogenesis. KSHV's viral IL-6 and latency-associated nuclear antigen (LANA) promote angiogenesis and inhibit p53 in endothelial cells, facilitating Kaposi sarcoma (Mesri et al., 2010). MCV, integrated into 80% of Merkel cell carcinomas, expresses truncated large T antigens that sequester Rb and induce helicase-mediated DNA damage (Feng et al., 2008). Human T-cell lymphotropic virus type 1 (HTLV-1) drives adult T-cell leukemia through the Tax protein, which activates CREB and NF-κB to immortalize CD4+ T cells, and the antisense-encoded HBZ, which sustains proliferation via E2F1 dysregulation (Matsuoka & Jeang, 2007).

Bacteria

Bacterial carcinogenesis, though historically underappreciated, is increasingly recognized. Helicobacter pylori, a Class I carcinogen, induces gastric adenocarcinoma and MALT lymphoma through chronic inflammation and direct genomic sabotage. The CagA oncoprotein, injected via a type IV secretion system, binds SHP2 phosphatase to hyperactivate ERK/MAPK signalling and disrupts apical-junctional complexes, causing epithelial polarity loss (Hatakeyama, 2014). Chronic CagA exposure also inactivates tumor suppressors like RUNX3 and induces AID-mediated somatic mutations (Ohnishi et al., 2008).

Another bacterium of growing interest is Fusobacterium nucleatum, an anaerobic microbe enriched in colorectal cancer (CRC) tissues. It promotes tumorigenesis through immune modulation and Wnt pathway activation. Its FadA adhesin binds E-cadherin, upregulating Annexin A1 to stabilize β-catenin and drive Cyclin D1 expression (Rubinstein et al., 2019). F. nucleatum also recruits myeloid-derived suppressor cells (MDSCs) via TLR4/IL-17 signaling, creating an immunosuppressive niche that correlates with microsatellite instability and poor prognosis (Mima et al., 2016).

Parasites

Several helminths have been classified as carcinogenic due to their role in inducing chronic inflammation in specific tissues. Schistosoma haematobium, endemic in sub-Saharan Africa, elevates bladder squamous cell carcinoma risk 5-fold. Embedded eggs secrete omega-1 ribonuclease, inducing IL-4/IL-13-driven fibrosis and upregulating PD-L1 on tumour cells to evade immune surveillance (Mucavele et al., 2022).
Liver flukes such as Opisthorchis viverrini and Clonorchis sinensis, prevalent in East and Southeast Asia, provoke cholangiocarcinoma via biliary epithelium injury and nitrosamine exposure. O. viverrini excretory-secretory products activate ERK/STAT3 signalling, while co-exposure to dietary nitrosamines induces TP53 mutations (Sripa et al., 2012). Clonorchiasis-associated tumours exhibit KRAS mutations and COX-2 overexpression, linking fluke-induced hyperplasia to malignant progression (Kim et al., 2009).
Fungi

Although less commonly implicated, fungi have also emerged as contributors to cancer. Fungal contributions to cancer involve toxin production and immune modulation.

Aspergillus flavus synthesizes aflatoxin B1, a potent mutagen that induces TP53 R249S mutations and synergizes with HBV to accelerate HCC (Kew, 2004). Candida albicans promotes oral and esophageal carcinogenesis through acetaldehyde production, which crosslinks DNA, and biofilm formation that

sustains NF-κB-driven inflammation (Alnuaimi et al., 2016). In pancreatic cancer, Malassezia globosa activates complement C3 via mannose-binding lectin, fostering IL-33-mediated immunosuppression and metastasis (Aykut et al., 2019).

Global Burden And Regional Differences

The global impact of pathogen-induced cancers is profound and disproportionately affects low- and middle-income countries. On a worldwide scale, infections account for an estimated 13–20% of all cancer cases. However, this figure masks substantial regional variation driven by differences in pathogen prevalence, public health infrastructure, socioeconomic conditions, and access to preventive measures.

Sub-Saharan Africa bears the highest burden, where over 50% of cancers are linked to infections. High rates of HIV significantly increase the incidence of Kaposi sarcoma and non-Hodgkin lymphomas, while widespread infection with hepatitis B and C contributes to the prevalence of hepatocellular carcinoma. In parts of East Asia and Southeast Asia, liver flukes such as Opisthorchis viverrini and Clonorchis sinensis are endemic and directly linked to high rates of bile duct cancer. Similarly, high H. pylori prevalence contributes to the incidence of gastric cancer in East Asia and Latin America.

By contrast, in high-income countries, the proportion of cancers attributable to infections is typically less than 5%. This is largely due to comprehensive vaccination programs, stringent food safety standards, widespread use of antimicrobials, and improved water and sanitation infrastructure. However, disparities persist within these countries, particularly among immigrant populations and underserved communities with lower access to healthcare.

The interplay between infectious agents and other risk factors can significantly increase cancer risk because infections often interact with environmental and behavioural exposures to amplify carcinogenic processes. For example, infection with human papillomavirus (HPV) is the primary cause of cervical cancer, but the risk is further increased in individuals who also smoke tobacco, as tobacco-related carcinogens can impair immune responses and promote persistent HPV infection. Chronic infection with hepatitis B or C viruses is a major cause of liver cancer, and this risk is heightened by co-exposure to aflatoxins, toxic compounds found in contaminated food, which synergistically increase DNA damage in liver cells. Infection with Helicobacter

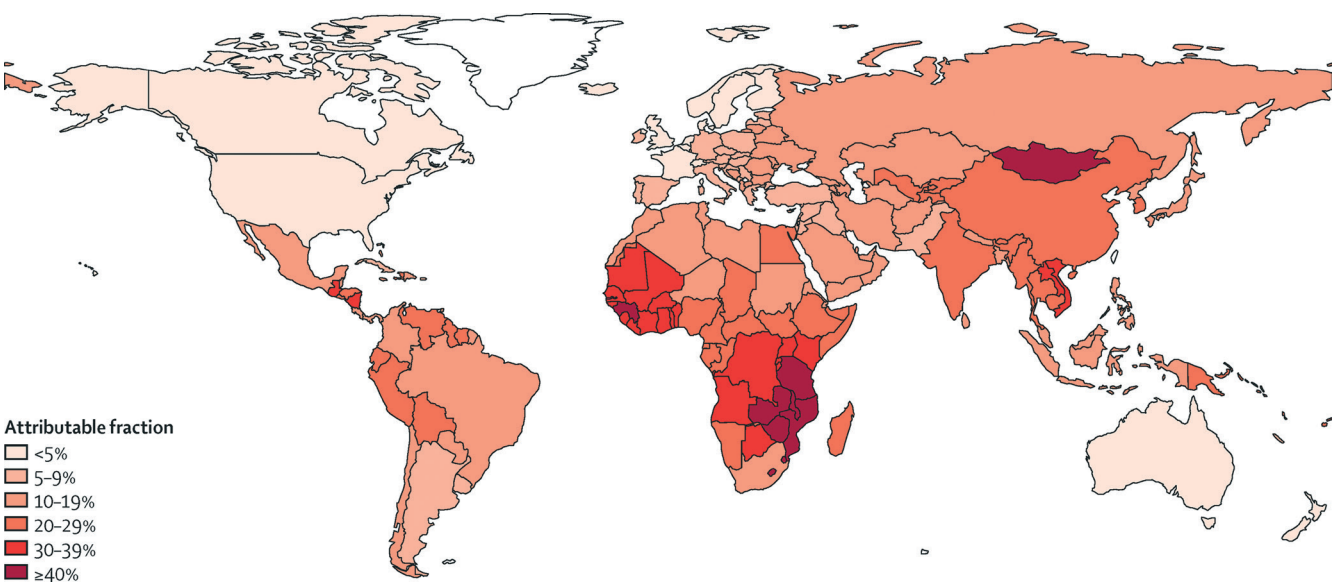


Image source: Plummer M, de Martel C, Vignat J, Ferlay J, Bray F, Franceschi S. Global burden of cancers attributable to infections in 2012: a synthetic analysis. Lancet Glob Health. 2016;4(9):e609-e616. doi:10.1016/S2214-109X(16)30143-7

pylori is a well-established cause of gastric cancer, and the risk is exacerbated in populations with high dietary salt intake, which aggravates gastric mucosal injury and inflammation. Additionally, infection with Schistosoma haematobium increases the risk of bladder cancer, particularly in regions where exposure to contaminated water and chronic inflammation are common.

Prevention Strategies

Efforts to prevent pathogen-related cancers span multiple domains, including vaccination, antimicrobial therapy, public health infrastructure, and behavioural interventions.

Vaccination represents one of the most effective tools for cancer prevention. The HPV vaccine, introduced in the early 2000s, was originally developed to prevent cervical cancer, however it has also proven effective against a broader spectrum of HPV-related cancers, including those of the head and neck. The bivalent vaccine, Cervarix, targets HPV types 16 and 18, which account for about 70% of cervical cancers. The quadrivalent vaccine, Gardasil, adds protection against types 6 and 11, which are responsible for genital warts. Gardasil 9, the most comprehensive option, covers nine HPV types-including additional high-risk strains like 31, 33, 45, 52, and 58-offering protection against approximately 90% of cervical cancers and a significant share of other HPV-related cancers.

Recognizing HPV's role in cancers beyond the cervix, vaccination programs have expanded to include boys. This not only protects males from cancers of the penis, anus, and oropharynx but also enhances herd immunity, reducing overall virus circulation. HPV is now linked to a rising incidence of head and neck cancers, especially oropharyngeal cancers in men. Studies have shown that HPV vaccination can significantly reduce oral HPV infections, a key risk factor for these cancers. For example, vaccinated individuals have been found to have an 83–93% lower prevalence of oral HPV infections compared to those unvaccinated. A large U.S. study by Jefferson DeKloe in 2024 found that men who received the HPV vaccine had less than half the risk of developing head and neck cancers compared to unvaccinated men, with only 2.8 cases per 100,000 vaccinated patients versus 6.3 per 100,000 unvaccinated. Hepatitis B vaccination, particularly when administered at birth, has had a profound impact on liver cancer incidence, especially in high-prevalence regions such as East Asia. While no vaccine yet exists for HCV, ongoing antiviral therapies have curbed disease progression and reduced cancer risk in chronic carriers.

Targeted antimicrobial treatment can significantly reduce cancer risk. Eradication of H. pylori with antibiotics has been shown to decrease gastric cancer incidence, particularly when administered before the onset of pre-cancerous lesions. Similarly, antiviral therapies for HBV and HCV reduce hepatic inflammation and delay or prevent progression to hepatocellular carcinoma.

In immunocompromised patients, antiretroviral therapy (ART) has substantially decreased the incidence of HIV-associated malignancies, including Kaposi sarcoma and certain lymphomas.

Screening programs are vital for detecting pathogen-related precancerous lesions. Routine Pap smears and HPV testing remain gold standards for cervical cancer prevention. Colonoscopies, which can detect polyps and microbial dysbiosis in the colon, play an important role in reducing colorectal cancer linked to Fusobacterium nucleatum.

Serologic tests for HBV and HCV can help identify chronic carriers who may benefit from monitoring or antiviral intervention. Ultrasound and alpha-fetoprotein (AFP) testing are increasingly used in liver cancer surveillance for at-risk populations.

Basic public health infrastructure—including access to clean water, adequate sanitation, and food safety—is critical in preventing parasitic and fungal infections linked to cancer. Educational campaigns about sexually transmitted infections and the importance of completing vaccination schedules can also reduce infection rates and improve outcomes.

Global control of infection-related cancers will require sustained investment in healthcare systems, international collaboration, and community-based initiatives tailored to regional risks and needs. The integration of pathogen-specific strategies into national cancer control programs offers a path toward meaningful reductions in cancer incidence worldwide.

I like to thank Francesca Albini for Historical Background

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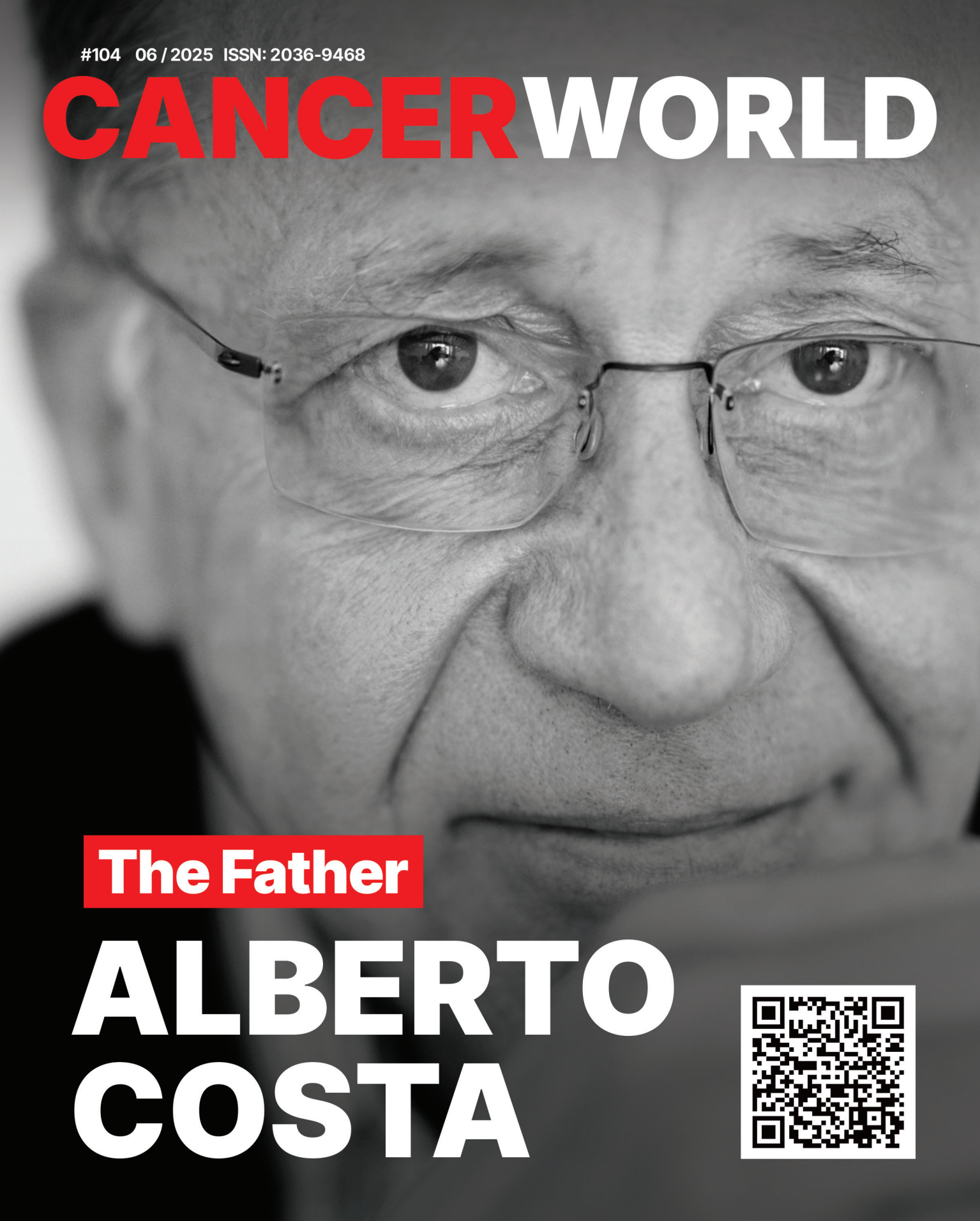
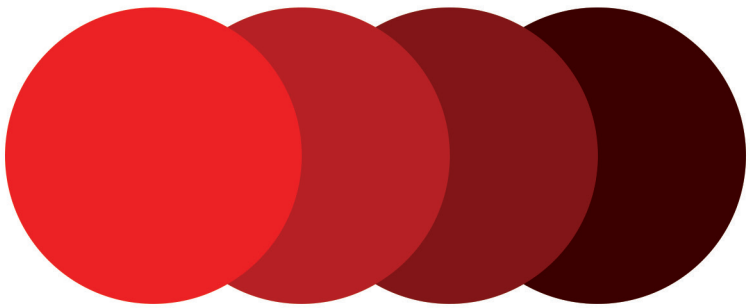


The Father

ALBERTO COSTA



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ALBERTO COSTA

The Father of Seven Children

"I mean, while I was reading the book you gave me about Umberto Veronesi," I said, "there's this expression: As you set out for Ithaca, hope your road is a long one—full of adventure, full of discovery. You wrote this about Umberto Veronesi, but I'm sure it's also about you. And it happens that today, while we're doing this interview, you're in Ithaca. What would you comment about this?"

Alberto Costa smiled. "Well, first of all, those are lines from the Greek poet Cavafy, as you know. Cavafy was born in Alexandria, spent little time in Greece, but managed to summarize this concept of Ithaca—meaning that at a certain point in life, you must find a place to get ready to leave the world. The more intense your life has been, the less you need in the end."

He gazed out the window. "Ithaca is poor, simple. A small island of just 3,000 inhabitants. In the Odyssey, when Ulysses' son visits Sparta, they try to gift him beautiful horses. But he declines—'We cannot have horses in Ithaca,' he says. 'It's all rock. We only have goats.' That's the essence. When you grow older, you need less. You become simpler."

"Is that why you chose Ithaca?" I asked.

"I chose Ithaca on purely intellectual grounds," he said. "I'd never even been here. But when I retired from clinical practice at 65, after burnout from years of breast cancer stories—I needed something else. Ithaca had always been in my mind. I studied classical Greek and Latin in Milan—five years of Greek, eight of Latin. I had a fantastic teacher. And for me, The Odyssey became the book. I'm not religious—I don't read the Bible or the Torah. For me, The Odyssey is my sacred text."

"The gods in it—they help, but they also try to kill you. They're jealous. They interfere. But ultimately, man or woman decides their life—with the gods as background characters. So I asked myself: Where is Ithaca?"

He laughed. "I did the most modern thing imaginable. I went on Booking.com. Found there was just one hotel and a lot of B&Bs. I came. Rented a scooter. Explored. And I found friends. Ithaca was Venetian for 300 years. The older generation still speaks some Italian. They feel linked to Venice. Suddenly—I felt at home. That was seven years ago. Now I return regularly. I have a little cottage up on the mountain."

"Did you ever think of doing a training or ESO course here?"

"No," he replied, shaking his head. "It takes one full day to arrive here. No airport. And that's one of the reasons it's beautiful—no mass tourism. You have to want to come. Athens, then Kefalonia, then the boat. It's not practical."

Shifting gears, I asked: "Can you tell your story from the beginning? Where were you born?"

"I was born in northwest Italy, nearly on the border with France, in a town—not a city—called Biella. Look it up on Wikipedia, it's known as the City of

Wool. All the big Italian wool brands—Zegna, Loro Piana, Piacenza—they come from Biella. That's why I have this link to wool."

"My father worked at Olivetti—famous for typewriters. He was later transferred to Milan, where I studied medicine. But we kept the connection to Biella. I had two sisters. I grew up in a family of women—even my cousins were girls. Summers were spent with seven women: my mother, two sisters, aunt, two cousins, grandmother, and the maid. I was the only boy. They killed me—but they also taught me everything."

"I can cook, wash, iron. My wives never had to look after my things—I always did them myself."

"And medicine?" I asked. "Why medicine?"

"Because of my girlfriend of the time," he said, laughing. "We still call each other every year for birthdays. I wanted to study literature, be a teacher. I was very socialist at the time. You know that expression, 'A young man who isn't a socialist has no heart; an old man who is still a socialist has no brain.' It may be true."

"Those were the years of May 1968, the Paris revolts. On the world stage: Indira Gandhi, Fidel Castro, Marshal Tito. The Beatles. It was pure luck to grow up then. My kids say it's unfair—we had the best years; they have iPhones."

"She convinced me I needed to do something concrete for society. Back then, medicine was seen as a philanthropic activity. Hospitals in Italy—Florence's Misericordia—were for the poor, for the plague. Medicine was solidarity. Not just a job. A mission."

"And I don't regret it. I've had a great professional life. But I retired without hesitation. Medicine today isn't what it was. It's about DRGs, costs, guidelines. Less clinical responsibility. You follow protocols—rules made by managers. It's not the same mission."

"If not medicine today—what would you choose?" I asked.

He paused. "Wool. The environment. Circular economy. Did you know 98% of clothes today are not sheep wool? Only 2%. The rest—cotton and synthetics. Textile companies sell plastic. That's why their cardigan costs 15 euros, and a real wool cardigan costs 60."

"But people still want sheep for meat and cheese. So Europe has 70 million sheep. Each sheep produces 1.5 kilos of wool annually. That's over 100 million kilos of wool every year—thrown away. A mountain of waste."

"I've had a little foundation for 12 years now, trying to do something. We're testing ways to use wool as fertilizer—especially here in Greece, where rain is rare. Wool retains water and releases nitrogen. If you bury wool under a plant, it grows better. That's another thing I could've done—because it's research, and it's for the common good."

"Or maybe," I suggested, "you could start a new wool brand—Biella meets Milan. A rival to Loro Piana."

He smiled. "Exactly. But I have no regrets. I loved being a doctor. Breast surgery is delicate, aesthetic—but not dangerous. You rarely make catastrophic mistakes. In 40 years, I can remember only two or three significant errors. I loved teaching. That's why I have pupils all over the world. They came to Pavia. Learned. And I loved that."

Mentorship And Legacy

"Mentorship is special to you," I noted. "Can you tell me about your mentees?"

Alberto Costa lit up. "Just yesterday, I was speaking with Iskra Daskalova—a brilliant Bulgarian breast surgeon. Do you know her?"

"Yes," I replied. "We've met."

"She's the perfect example," he continued. "She came to Pavia, absorbed the philosophy. I encouraged her to go to Dublin to improve her English and learn about screening with my friend James Geraghty. She worked there, and now she's at the European Medical Center in Moscow. Watching her grow—like watching your own children mature. I told her, 'You should chair the course now. You're ready. Go.' And she is."

He paused. “I worked in three places as head of department—Milan, Pavia, and Lugano. In each one, they chose my deputy as my successor. One woman, two men. They still call me on Sundays when they have a problem. That’s fulfillment. That’s legacy.”

“When I retired, I added a note to my website: I don’t see patients anymore. Please contact one of these three doctors. Patients still call to thank me and say, ‘You’ve trained them well.’ That’s the best compliment.”

On Surgery And The Power Of Action

I turned to his surgical roots. “You’ve explained why you chose surgery—and breast surgery—but surgery also shapes character. What role did surgery play in your life?”

“I probably ended up in surgery,” Costa replied, “because as a student and intern I started in orthopedics—and liked the man I worked with. It’s often about people. Breast surgery, of course, came from Veronesi. Had he been an ophthalmologist, I’d probably have gone into that.”

“But I was never a ‘classic’ surgeon. I avoided surgery congresses—couldn’t stand endless debates about sutures and needles. My focus was always on pathology, biology. I said: Breast cancer is more cancer than breast. Prostate cancer? It’s not about the prostate—it’s about cancer. The disease matters more than the organ.”

“I trained in general surgery—one gastrectomy, two thyroidectomies, scared every time. But then I focused entirely on breast. I was always pushing innovation. I was among the first to question why we discarded the nipple in mastectomy. The nipple is very rarely affected by cancer. Surgeons removed it because it made surgery easier. But preserving it—yes, the surgery took longer—but the psychological impact was immense.”

“In Pavia, we preserved the nipple, and word spread. More women came. Our results improved—not in survival, but in quality of life. I insisted on reshaping the opposite breast for symmetry after quadrantectomy. Then came pushback: Reshaping isn’t reimbursed. Who pays? I said: I don’t know. But we do it because it’s right. Now, they’ve stopped reshaping. They discourage nipple-sparing—it takes too long.”

“And it’s nearly all business now.”

“Exactly. In surgery. In oncology. I retired just in time.”

Building Departments And Institutions

“Tell me about building departments—in Milan, Switzerland, Pavia,” I asked. “What were the challenges?

“I built from scratch four times. Partly because of my complex relationship with Veronesi. He was the alpha; I was the beta. He had the ideas. I wrote the articles. Our partnership worked—but eventually, I realized I’d never be head of a department while standing in his shadow.”

“Our first experiment was in Pavia. The Maugeri family wanted a breast surgery service in their hospital. We helped. Pavia is 20 minutes from Milan. I agreed to lead. Patients were already waiting a month for surgery in Milan. So we said: ‘If you want, go to Pavia.’ It worked.”

“In Pavia, I became head for the first time. I had to choose machines, hire nurses, recruit a pathologist—start everything. It was a great experience. And when it ran well, the Swiss approached Veronesi to help create a breast center in Lugano. He asked me again. And there was a personal reason too.”

“I had remarried. My second wife, Kathy—she’s Irish—didn’t like Milan. Missed the green, the nature, the dogs. She loved Lugano. I said, ‘This solves both our problems.’ We moved. My kids grew up there. And professionally, it was another challenge—another country, another system. But Ticino is a hybrid: too Italian to be Swiss, too Swiss to be Italian. They don’t know where they are.”

The Other Half: Global Vision And Advocacy

"Besides clinical work, you were deeply involved internationally—EORTC, FECS, Europa Donna, Europa Uomo, ESO," I said. "Was that also because of Veronesi?"

"Yes. He didn't have other people for that. I was lucky—my father made me learn English. At the time, everyone learned French. But he said: 'The future is English.' I wanted a motorbike. He said: 'You'll get it—if you enroll in evening English school.' I was desperate for the motorbike. So I learned."

"Later, I worked summers at the Venice Biennale. My friend got me in. I knew how to run press conferences, write press releases. That's how I became Veronesi's man for international affairs. He never sent me to Rome—he said I'd lose or start fights. But abroad? That was me."

"At EORTC, he became president. I was secretary general. I must've done well—because subsequent presidents kept me. Twelve years, back and forth to Brussels."

"In 1982, he sent me to New York—on paper, a fellowship at Memorial. But the real goal was to establish the American Italian Cancer Foundation. Why? Because we were collecting lira to send Italian fellows to the US—and losing money on exchange rates. His idea: collect dollars instead. So I went, supported the opening of the office, found right contacts, helped to organize fundraising."

"Each year now, our Chairman, Daniele Bodini, holds a charity dinner in New York—raise $1.5–2 million in one night. Awards, auctions, dinner. It still works and we have money for fellowships."

"In Europe, I also helped build the Federation of Cancer Societies. Don't forget—Veronesi and Maurice Tubiana were tasked by Mitterrand to lead the Europe Against Cancer program. And I traveled nearly every week."

"That's the beauty of breast surgery. One day for consults. One day for surgery. One for follow-up. The rest—for international work."

"And ESO?"

"I've been director, not president. Never been president of anything," he said.

"Really?"

"Never. Always the secretary. The number two. That was our pattern. I'm not good at pushing myself—but I'll fight for a cause until the end. I value freedom. That's why I'm here now. Some want to be Trump—feel powerful. But I'd hate that life."

"And Veronesi—he needed the spotlight. I didn't. I hate long dinners. When I was young, the only thing that kept me up late was not policy meetings—it was a new love story."

He smiled. "It's character. Just being different."

European School of Oncology

Founding Eso: A School Rooted In Purpose

"How did you start ESO?" I asked.

He leaned back, smiling. "That's a long one. Let's get all the details in."

In the late 1970s and early 1980s, Costa recalled a growing unease. Europe was on the move. Everyone was creating pan-European entities—the French and Benelux countries had launched EORTC, headquartered in Brussels. Soon after, the British founded the European Journal of Cancer. The Germans established EMBO in Heidelberg, where Harald zur Hausen would eventually win the Nobel Prize.

"And we asked ourselves—what is Italy doing?" he said. "We were being cut out."

In their characteristic symbiosis, Costa and Veronesi brainstormed. "I don't remember who had the idea," he admitted. "People used to mix our names—Alberto Veronesi, Umberto Costa—we were that close."

But they knew one thing: Italy's strength was education. With universities like Bologna and Padua, they had the historical authority to lead in learning.

"We were also annoyed with American sometimes excessive ambition," he added. "In 1973, Nixon signed the Cancer Act and said, 'We've landed on the moon—we can conquer cancer too.' That attitude—we didn't like it."

Then came serendipity. Veronesi was called to see a wealthy Milanese industrialist suffering from bone pain. Other doctors had misdiagnosed it as arthritis. Veronesi recognized metastatic prostate cancer. Grateful, the patient said, "I wasn't failed by science—I was failed by ignorance."

He offered to fund a school. "If you want to educate Europe, I'll support you." And just like that, the European School of Oncology was born.

"He had no heirs," Costa explained. "He and his wife created an endowment. Every year, we receive profits from that capital. We've sustained ESO now for 45 years."

The foundation was signed in 1982. That was the beginning. ESO became Italy's major contribution to European oncology, alongside institutions like EORTC and EONS.

"I must say," he added, "ESO also shaped your career too, no?"

I nodded. "Yes—it gave me a scholarship to Ulm University in 2013."

"Ah! You got that one?" he smiled. "Very lucky. That's how ESO works—quietly shaping people."

Eastern Roots, Unconventional Paths

ESO also stood out for its commitment to underserved regions.

"I've always felt a link with Eastern Europe," Costa said. "In the Cold War, Yugoslavia looked like the solution—socialist, but not soviet."

That worldview shaped ESO's mission. "We didn't host courses in Amsterdam or Brussels—we went to Sarajevo after the war. To Belfast after the peace. ESO always had a political line. And my board always supported me." After the aggression to Ukraine we have stopped organizing courses in Russia, but we support good Russian young doctors with fellowships and distance learning tools.

Birth Of Cancerworld: Science Beyond The Journals

"What about CancerWorld?" I asked.

"It started in Kathy's kitchen—my wife, in Dublin. She was a lecturer in cancer nursing. She said: 'Not everyone has time to read The Lancet on their commute. Find a way to make science more accessible.'"

The idea stuck. They launched a publication called Cancer Futures with Springer, then eventually parted ways and created CancerWorld under ESO's banner.

"The principle was simple: I don't have time—or the skill—to read The New England Journal. But I'll happily read an interview with the author. It wasn't papers—it was people. And people have always been ESO's obsession."

The European Ideal

"Many organizations you've led have 'Europe' in the name," I noted. "You're a true advocate for the European cause."

Costa didn't hesitate. "Absolutely. The words I heard most growing up were: 'Never again.' My father went to jail during the war—he was in artillery, ordered to fire on Nice. But he had lived there as a boy. He refused. He sabotaged the coordinates. He was court-martialed."

"Europe means peace," he continued. "Italy was a founding member of the EU. The Erasmus program created a new generation—still proudly Spanish, Italian, or Greek, but first and foremost, European."

"In oncology, too, it meant difference. The EU's plan under Commissioner Kyriakides emphasized quality of life, financial toxicity, universal care. That's Europe."

He looked reflective. "ESMO is becoming global. Only 52% of its members are European now."

"But ECO survived. Forty-two societies. All European. That's still something worth fighting for."

Medicine, Empathy, And Advice To The Young

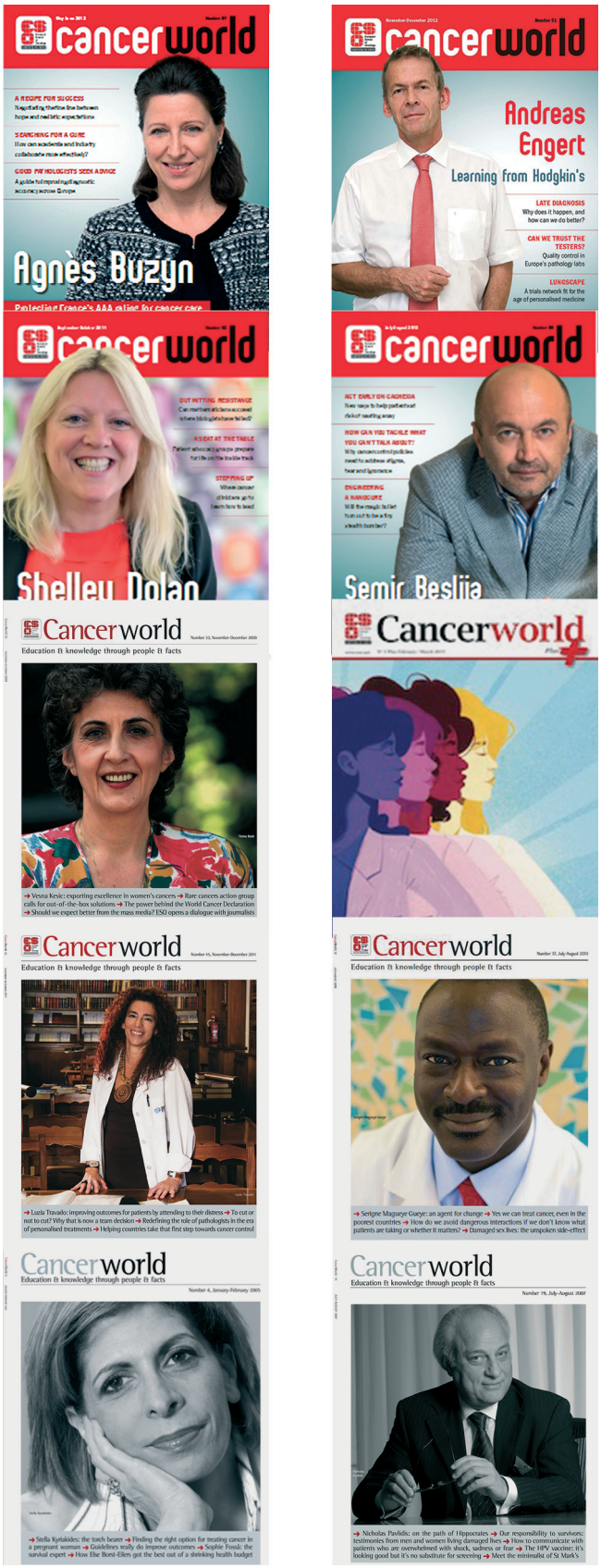
"What would be your advice to the younger generation?" I asked.

Costa didn't hesitate.

"They must resist the transformation of medicine into just another profession," he said. "People say: a doctor is like an architect—you fix a problem, you get paid. But no. You are working on another human being. Especially in surgery. You don't cut someone's skin if it's only for a bill. There has to be something deeper."

"If you don't have that sense of mission," he added, "do another job."

He paused and smiled. "Same in breast surgery.



If you don't like women—don't do breast. If you don't care about aesthetics—don't do breast. Be a pediatrician, be a radiologist. But if you don't feel the human side, don't go into oncology."

He reflected for a moment. "I must say, the ESO young people—our students—they are different. They do care. It's not just about issuing an invoice. That gives me hope."

Books That Shape An Oncologist's Soul

We turned to literature. "The Odyssey is clearly your book," I said, "but what else?"

"One Hundred Years of Solitude," he answered instantly. "It's about destiny and free will. How much of life is our doing, and how much is just meant to be. And what happens when you try to act for the greater good—but the world turns against you."

He added, "And Blindness by José Saramago. Every doctor should read it. It's about how society collapses when people lose their sight—literal and moral."

"And then of course, the Italians," he smiled. "Calvino, Leopardi, Dante. Though Dante's Paradise is boring. Inferno is much better. Nobody reads Paradise."

Who Is Alberto Costa?

"If you had to describe yourself in one sentence," I asked, "what would you say?"

He grinned. "The father of seven children."

He counted them: "Five biological, from three mothers. One—my stepson—was raised with me. And one more—a girl from Sarajevo. An orphan. Her parents were killed during the siege."

He explained how he joined Italy's campaign to support war orphans. He picked her from a list—liked her name. Started sending money. Met her when she turned nine. Now she's 35 and working in Brussels.

"These seven kids and their mothers," he said, "they've been the center of my life. The rest—my work—came from luck, from hard work, from traveling all the time. But the family came first."
None of them became doctors. "They think I'm crazy," he laughed. "One translates Japanese manga, one's a professor in Scotland, another's in Salzburg with Red Bull. One's a lawyer in New York. The elder is an IT expert. They all do their own thing. Maybe they're right."

A European Personal Life

He didn't shy away from the personal.

"I've had three great women in my life," he said. "And I've been European about it: one French, one Italian, one Irish. Three languages, three cultures."
"We rarely fought. No lawyers. The children grew up together. They're still close."

"Maybe that's why I never wanted to be president of anything," he said. "I had too much to do at home. I like being number two. I like my freedom."
He chuckled. "Poor Trump—he thinks he's on top of the world. But to me, that's the worst life imaginable."

And Who Should We Interview Next?

I asked him the question I ask everyone at the end: "Who should I interview next?"

He thought for a second. "I like original stories. Here's one. I'm going to Zagreb in two days. Look up Steven Pavletić."

"He was the assistant to Boris Labar—they did the first bone marrow transplant in Europe. Americans brought Steven to Bethesda. He's retiring now, coming back to Croatia. A man who quietly did a lot for oncology."

Then he smiled again. "It's the ones you've never heard of that change the world."

Janet Fricker

Microbiota-Derived Bile Acids As Androgen Receptor Antagonists Enhance Anti-Tumour Immunity

Gut microbiota can transform cholesterol-derived bile acids into metabolites capable of blocking androgen receptors and strengthening anti-cancer immunity. The study, published in Cell, April 15, demonstrates how one of the microbiota-derived secondary bile acids is capable of suppressing tumour progression and boosting efficacy of anti-PD1 treatment.

"The unexpected crosstalk between microbiota BAs [bile acids], AR [androgen receptors], and CD8+ cells could provide previously unknown insights into how microbiota metabolites are sensed and recognised by host receptors to exert their biological functions. Our results suggest an unrecognised route through which microbiota secondary BAs are sensed by host AR that likely play an important

role in modulating host physiology/ disease," conclude the authors, led by Chun-Jun Guo, from the Jill Roberts Institute for Research in IBD and the Division of Gastroenterology & Hepatology at Weill Cornell Medicine, New York.

Primary bile acids are produced by the liver and released into the small intestine where they are potent 'digestive surfactants', breaking down dietary fats and helping absorption of fat-soluble vitamins (like A, D, E and K). While studies have shown that gut microbiota can transform primary bile acids into secondary bile acids that are associated with host biology/ disease, the molecular mechanisms whereby they interact with human signalling pathways have been largely undefined.

In the current study, Guo and colleagues set out to integrate microbial genetics and metabolomics to explore how secondary bile acids affect host biological function. First, they expressed candidate bile acid-modifying genes in E-coli that were exposed to bile acids in vitro leading to identification of a series of 56 new bile acids that had not been previously characterised.

Given that the secondary bile acids share the same steroid backbone as sex hormone (like testosterone and oestrogen), the team explored whether they interacted with commercially available cell lines expressing a range of different receptors in a cell-based luciferase reporter assay. "Put simply, a bile acid that was an agonist of the receptor led to luminescence going up and a bile acid that was an antagonist to luminescence going down when tested in the presence of an agonist," Guo explains to Cancerworld. To their surprise, the team discovered that one of the secondary bile acids, 3-oxo-Δ5-LCA -was a potent human androgen receptor antagonist. The finding led the investigators to test an additional 44 microbiota-derived bile acids that had previously been identified in their cell-based luciferase reporter assay leading to the discovery of three additional androgen receptor antagonists.

Recently, a study published in Immunity in 2022, showed that blocking androgen receptor signalling led to enhanced CD8+ T cell stemness and a study in Nature ,also published in 2022, showed that it enhanced immune check point inhibitor activity. Such findings inspired the team to explore the idea that modified bile acids might also be able to enhance CD8+ cell stem-like properties through their ability to block androgen receptors. As a proof of principle, in a mouse model of bladder cancer the team were able to demonstrate that one of the microbiota derived bile acids, 3-oxo-Δ4,6-LCA, suppressed tumour progression and potentiated the efficacy of anti-PD1 in an androgen receptor (-dependent manner. However, in a different mouse model with the androgen receptors 'knocked out; no such beneficial effects were found.

"Our results suggest that these secondary bile acids help shrink tumours by enhancing T cells' ability to survive within the tumour and destroy cancer cells," explains Nicholas Collins, the co-senior author of the study.

The findings, says Guo, suggest a promising new field of investigation, where a wide number of secondary bile acids may interact with a variety of host receptors. "Our findings likely mark just the beginning of understanding these complex interactions. Individuals differ in their gut microbiome composition and the levels of gut microbial enzymes that modify the bile acids, leading to distinct bile acid profiles. The interactions between microbiota-derived secondary bile acids and host signalling pathways would have a range of different biological functions, including influencing cancer onset, progression, and treatment response," he says.

Next, the team is looking to test the identified secondary bile acids in different mouse models of cancer (such as colorectal and prostate cancer), to see if blocking the androgen receptors has similar anti-tumour effects.

As the lab specialises in microbiota engineering, they hope to explore the possibility of precisely regulating production of these secondary bile acids to make cancer therapies more effective.

Another plan is to investigate whether differences in modified bile acids are found between cancer patients and healthy controls. "We hope to establish a panel, including microbiota-derived secondary bile acids along with their producing microbes and enzymes, that could be used as biomarkers to provide useful information about cancer onset, progression, and treatment," explains Guo.

June 2018, ESO-ESTRO-ESSO Course for Medical Students, Turin, Italy

Amalya Sargsyan

Curious, Rejected, Accepted: An ESO Fellow's Road to Becoming an Oncologist

It was 2018, and I was a fifth-year medical student at Yerevan State Medical University. Word spread that our new oncology professor was someone extraordinary: Gevorg Tamamyan - a Harvard-trained, Nature-published pioneer of pediatric oncology in Armenia and president of POEM. Naturally, we Googled him. We read the papers, scrolled through posts, and quickly realized he wasn't just teaching oncology - he was shaping it.

During our brief rotation, Professor Tamamyan brought oncology to life with real patient stories, global guidelines, and relentless encouragement to apply for opportunities. When he shared two student programs—one from ESMO and the other from the European School of Oncology (ESO)—I chose and applied to the ***ESO-ESTRO-ESSO Course for Medical Students,*** a two-week summer school in Torino, Italy. Back then, I was set on neurology and

arrived in Torino unsure of what oncology could offer.

Landing in Italy, I didn't expect a seismic shift. But it happened. At Molinette Hospital, surrounded by 25 students worldwide, I saw a version of oncology I'd never imagined—where research, compassion, and community collided. Immunotherapies. CAR-T cells. Cancer vaccines. Concepts I'd never heard before. And just like that, I knew: ***I wanted to be part of this. I had found my home.***

Fast forward to 2020, I found myself confidently choosing oncology residency at Yeolyan Oncology and Hematology Center in Armenia.

Under the guidance of Armenia's first ESO Ambassador, Jemma Arakelyan, and with incredible mentors like Dr. Liana Safaryan and Dr. Davit Zohrabyan, I was constantly encouraged to think bigger and push harder.

Just two months into residency, the ***ESO Masterclass in Clinical Oncology*** opened for applications. I jumped at the chance, applied, and got accepted. Then the pandemic hit. The masterclass went virtual. Yet, it became another essential milestone for a young oncologist—hearing directly from the people who shape the global standards we study every day.

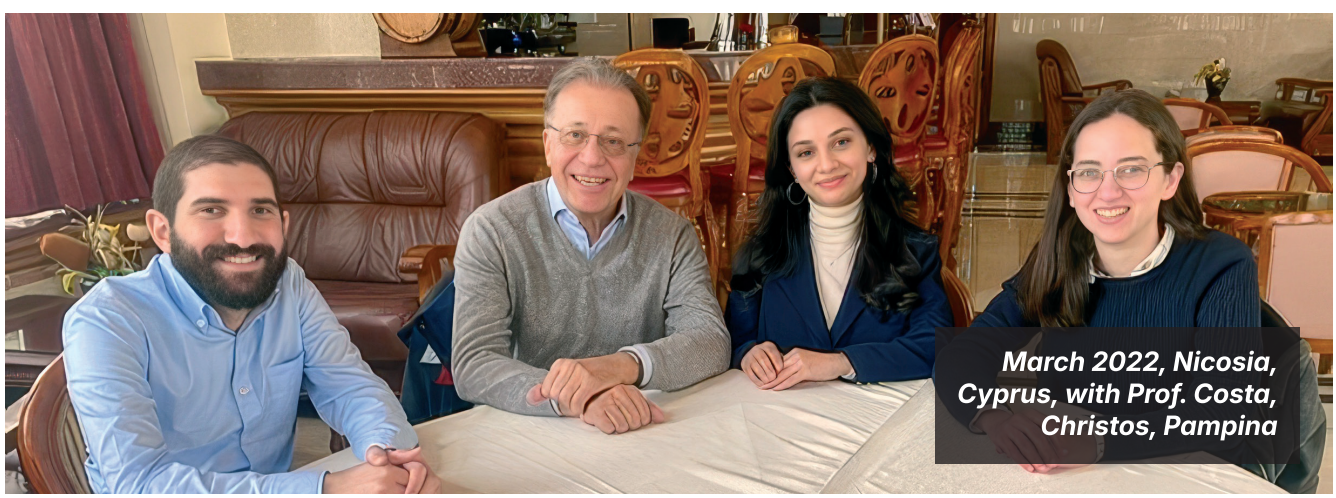
But my dream of studying abroad was still alive. Years earlier, I had been accepted to Cambridge Medical School, but couldn't attend. So when I saw ESO offering a single funded place for the ***Master's in Precision Oncology*** at the University of Cyprus, I knew I had to try. The process felt like an exam in itself: TOEFL, endless paperwork, recommendation letters, three visa rejections, countless embassy calls, and stretched nerves. At the same time, I had another offer through the ERASMUS program in Porto. It's true. ***Life is about choices, and every choice you make makes you.***

So I did what I always do—I opened Excel, listed pros and cons, measured long-term impact, and chose the less easy, but more promising road: Cyprus, with ESO.

Professor Anastasia Constantinidou supervised my academic training and welcomed me into her clinic at the Bank of Cyprus Oncology Center. That's where I had my first real exposure to sarcomas, clinical trials, research, and an entirely new healthcare system. I started learning the language of trials, AI, molecular profiling, and biostatistics. Back then, it all felt theoretical, far from our daily reality. But those same tools helped me coordinate Armenia's first Investigator-Initiated Clinical Trial two years later.

I even published my first paper with Dr. Constantinidou and Dr. Robin Jones from The Royal Marsden—someone I later had the chance to meet in person. ***Proof that every choice you make shapes your future*** and often finds its way back to you years later.

One afternoon in Cyprus, I got an email from everyone's favorite person—ESO's heart, Corinne Hall. If you've ever applied to an ESO program, you've probably met her kindness. She wrote that



March 2022, Nicosia, Cyprus, with Prof. Costa, Christos, Pampina

ESO's CEO, Professor Alberto Costa, would be in Cyprus and wanted to meet us. That conversation about our experience, goals, and expectations was a turning point. He listened. And most importantly, told that ESO believed in us. And sometimes, that's all a yound doctor needs.

When I returned to Armenia, I poured my energy into building what I'd learned—organizing journal clubs, sharing trial updates, and encouraging others to apply for ESO opportunities. When our Cyprus publication came out, ESO invited me to speak on their 5-Minute Science podcast—***another first.***

June 2023, Graduation Ceremony, Cyprus, with Prof. Constantidou, Pampina, Despoina, Christos

Though we see all types of tumors in Armenia, my interest in sarcoma kept growing. It's rare and complex, and our local experience was limited. I remember watching ***ESO's online sarcoma modules,*** led by Prof. Paolo Casali, one of the field's founding figures. I never imagined I'd one day walk the halls of his department.

By the end of my second residency year, I applied for ESO's CTC fellowship in sarcomas in Lyon. Unfortunately, I was rejected. I also applied to EORTC but was rejected again. It felt like my lucky star had vanished—but I didn't give up. My first mentor, Professor Tamamyan, always says, ***"Persistence is key."***

Whatever happens—or doesn't—has its reason. Try again. Try harder.

After graduating, I started working as a consultant oncologist and joined the Immune Oncology Research Institute as a research fellow. But I hadn't forgotten my earlier goals.

I applied again the following year for ***ESO's CTC fellowship*** at the Istituto Nazionale dei Tumori in Milan. I got the interview. I was nervous, but my eyes were sparkling with motivation—ready, as I love to say, to conquer the world.

November 2024, ESO CTC Fellowship in Milan with Salvatore Provenzano, Mary Ann, and Helena.

I didn't know then that the person interviewing me, Dr. Salvatore Provenzano, would soon become both a mentor and a friend.

I was accepted to Paolo Casali's department—the same name I had once seen only in webinars and paper titles. ESO CTC Fellowship in Milan was intense in the best possible way. Every morning started with an Italian lesson at 7 a.m. Then, I read a research paper on the tram. Then, it was the clinic and Italian coffee.

Most of my days were spent in the inpatient unit, where Dr. Provenzano patiently walked me through every case, every treatment, every nuance. His

kindness matched his brilliance—and that rare gift of being both an exceptional doctor and a generous teacher. For the first time, I truly began to understand sarcoma from the inside out. No question was too small. No moment wasted. Every day mattered.

Fueled by that experience - and with some spare time for academic work - I stayed as proactive as ever. I kept coming up with new ideas, sending papers, asking for feedback, pushing for more. One day, after yet another idea, Dr. Provenzano looked at me, smiled, and asked with a laugh:

"Do you ever stop, Amalya?"

To be fair, they were having incredibly busy days—back-to-back meetings, routine clinical challenges, and a new article draft on the table. ***If you're reading this... I'm sorry!***

But to every young doctor out there: if you want your idea reviewed or your paper seen, you must follow up. Gently. Repeatedly. Be respectfully pushy. Sometimes, that's how progress happens.

No, I don't stop.

Because in a world full of opportunities, there's no time to stop.

I wanted to share what I was learning, so with ESO and Dr. Provenzano, we recorded a podcast on angiosarcomas—part of what I hope becomes an ongoing ***"Rare Tumor Podcast"*** series, because knowledge should never stay in one room.

When I returned to Armenia, my department trusted me to handle all new sarcoma cases. And that's what three focused months, the right mentor, and the right opportunity can do. ***If you told me this in 2018, I would've laughed.***

I started as a junior researcher and volunteer at OncoDaily, curating papers. Now I lead our Research and Intelligence Unit with a team of twelve. ***Because it doesn't matter how small you start - what matters is how bold you dare to go,*** and as Dr. Tamamyan says: ***"Shoot for the moon. Even if you miss, you'll land among the stars."***

The journey isn't just built on luck but on sleepless nights, hard choices, and relentless curiosity. And

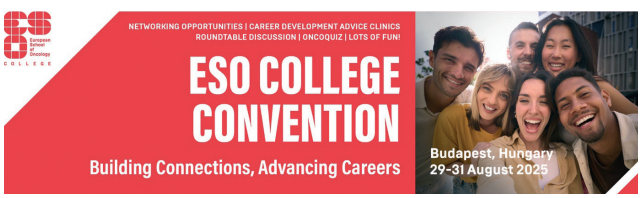
20 March, 2025, ESMO Sarcoma and rare cancer congress, discussion on stage between two ESO Fellows

it hasn't been all success - there were rejections too. ***But what matters isn't perfection—it's progress.*** Three months after ESO CTC fellowship in Milan, my abstract was accepted for a mini oral presentation at ***ESMO's Sarcoma & Rare Tumor Congress.*** Standing on that stage—among people whose names once lived only in my reference list—gave me the energy to do more and made me feel that I hadn't disappointed those who believed in me. I hope they are proud.

Just weeks ago, back in Milan, I saw Professor Costa again. Three years later, this time, I wasn't the student with questions—I was sharing results, ideas, and gratitude for believing. I told him how much ESO had shaped me—***how it became my first oncology school—and continues to be.***

Because ESO doesn't just teach, it listens. It challenges. And it believes in you before you believe in yourself.

As we prepare for ESO's first-ever Convention



in Bucharest, I hope this story inspires another young doctor to leap. Apply. Ask questions. Show up. And never underestimate the power of a single opportunity.

Dream big. Work hard. And keep checking the ESO website.

Adriana and
Francesca Albini

Back On Screen: The Return Of Smoking In Film And Its Public Health Implication

May 31 is "World No Tobacco Day" Smoking is one of the major factors for tumors, not only lung cancer. This year's World No Tobacco Day (WNTD) campaign aims "to reveal the strategies employed by the tobacco and nicotine industries to make their harmful products enticing, particularly to young people. By exposing these tactics, WHO seeks to drive awareness, advocate for stronger policies, including a ban on flavours that make tobacco and nicotine products more appealing, and protect public health".

WHO organizes the event: "Exposing lies, protecting lives: Unmask the appeal of tobacco and nicotine products".

Co-Editor-In-Chief Adriana Albini, with the Collaboration of Francesca Albini, PhD, Philosophy, describes the return of Smoke in Movies.

A Warning

In recent years, cigarette smoking has reemerged as a striking visual motif in cinema, occupying a space once thought to be fading from popular storytelling. Lit cigarettes dangle from lips in pivotal scenes, clouds of smoke curl through tightly framed shots, and tobacco becomes part of the visual language used to convey depth, rebellion, or intimacy. This resurgence is striking—not only for its aesthetic boldness, but for its contrast with the preceding decade, during which the presence of smoking in film had markedly declined in response to growing public health concerns and policy interventions.

The renewed visibility of smoking in cinema has sparked concern among health professionals and researchers. Decades of evidence suggest that tobacco imagery in media can normalise smoking, particularly among adolescents and young adults. As filmmakers revisit the iconography of tobacco, often under the guise of historical realism or character authenticity, the question arises: are we witnessing a cultural relapse with potential public health consequences?

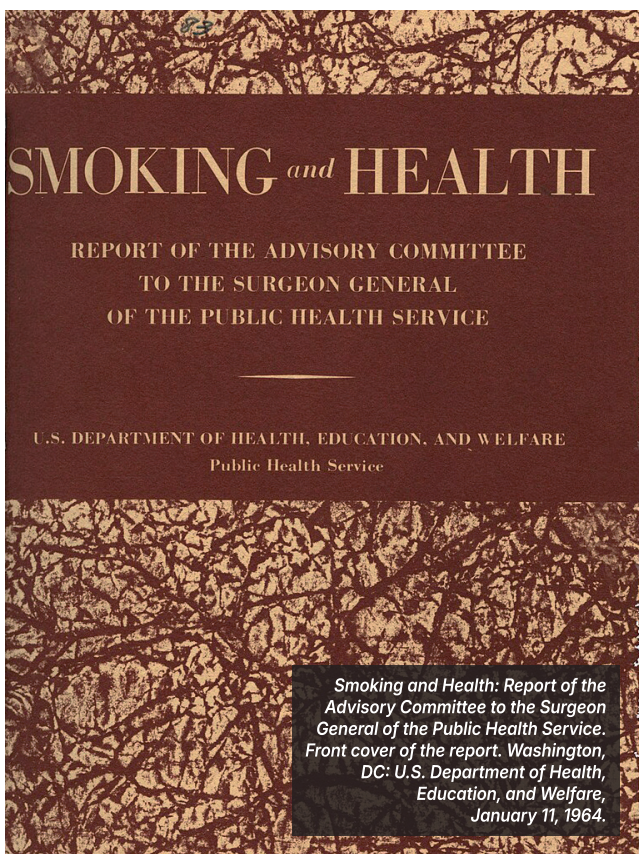
A Historical Association: Tobacco And Cancer

The relationship between tobacco and cancer is one of the most well-documented—and hard-won—public health discoveries of the 20th century.

While tobacco had been cultivated and smoked for centuries, its association with cancer emerged only gradually, and not without resistance.

By the early 1900s, physicians in Europe and North America had begun noticing a rise in lung cancer diagnoses, a disease that had previously been rare. Suspicion fell on the growing popularity of cigarette smoking, but early observations lacked the rigor to counter the formidable influence of the tobacco industry. It wasn't until the 1950s that landmark epidemiological studies provided compelling evidence of causality. In the UK, Richard Doll and Austin Bradford Hill conducted a prospective study of British doctors that established a strong link between smoking and lung cancer. Around the same time in the US, Ernst Wynder and Evarts Graham published case–control studies showing a significant correlation between cigarette use and cancer risk.

These findings culminated in a historic moment in 1964, when the US Surgeon General's report officially declared cigarette smoking a cause of lung cancer. The report galvanized public health efforts and marked the beginning of a new era in tobacco control. In subsequent decades, further research expanded the list of smoking-related diseases to include cancers of the larynx, esophagus, pancreas, bladder, kidney, cervix, and stomach, as well as myeloid leukemia.



Smoking and Health: Report of the Advisory Committee to the Surgeon General of the Public Health Service. Front cover of the report. Washington, DC: U.S. Department of Health, Education, and Welfare, January 11, 1964.

Furthermore, the link between smoking and cancer was not limited to direct users. By the 1980s and 1990s, studies began to show that second-hand smoke exposure also increased cancer risk, leading to widespread public smoking bans in workplaces, restaurants, and, eventually, film sets and studios.

The cultural response was just as significant. Smoking, once glamorised in media and film, began to lose its sheen. Public campaigns reframed it as an addictive, deadly habit rather than a symbol of sophistication. Cigarette advertising was banned from television and sports sponsorships. The tobacco industry, once deeply entwined with Hollywood, was gradually pushed out of mainstream visibility. However, the story is far from over.

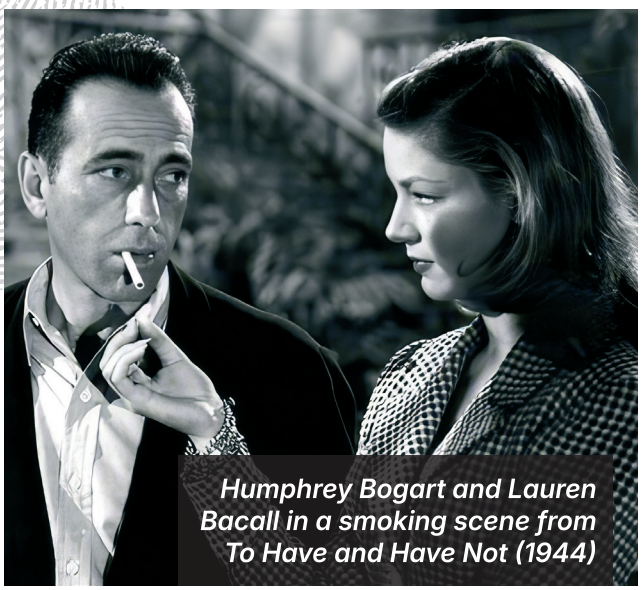
Tobacco In Hollywood: From Glamour To Backlash

For much of the 20th century, smoking was not only common in everyday life—it was a cinematic shorthand for allure, danger, introspection, and control. Hollywood's Golden Age stars, from Humphrey Bogart and Lauren Bacall to James Dean and Marlene Dietrich, lit up on screen with a cool defiance that was both aspirational and carefully constructed.

Cigarettes weren't just props, they were character cues, emotional punctuation marks, and marketing tools. Behind the scenes, the tobacco industry actively cultivated these portrayals through extensive product placement and covert sponsorship. Archival documents from the 1930s to the 1950s reveal deliberate collaborations between tobacco companies and studios. Actors were paid to endorse cigarette brands in magazines and newsreels, while screenplays were peppered with smoking scenes that reinforced brand familiarity. This wasn't coincidental, it was a strategic extension of advertising into the emotional core of film. As a result, generations of viewers grew up equating smoking with strength, sophistication, and sexual appeal.

But the cinematic glamour of tobacco began to unravel in the latter half of the century. As scientific consensus about the harms of smoking solidified, advocacy groups and public health organizations turned their attention to the screen. The juxtaposition was becoming untenable: while governments issued warnings and restricted cigarette advertising, films continued to depict smoking without context or consequence.

By the late 1990s and early 2000s, mounting pressure led to change. In 2003, the World Health Organization adopted the Framework Convention on Tobacco Control (FCTC), whose article 13 called for restrictions on tobacco advertising, promotion,



Humphrey Bogart and Lauren Bacall in a smoking scene from To Have and Have Not (1944)

and sponsorship, including indirect forms such as product placement. Around the same time, research emerged showing that adolescents exposed to smoking in films were significantly more likely to try smoking themselves. A study published in The Lancet in 2003 estimated that smoking in films could be responsible for 52% of smoking initiation among young people in the United States.

In response, major studios began to revise their internal policies. The Motion Picture Association of America (MPAA) introduced guidelines flagging smoking as a content consideration in film ratings. Several studios pledged to eliminate smoking in youth-rated films unless historically justified. The number of tobacco depictions in top-grossing movies began to fall, especially in films aimed at younger audiences. By 2010, the depiction of smoking in mainstream cinema had declined sharply, and it seemed that the once-glorified habit had been relegated to the margins of popular storytelling.

A Quiet Comeback: Smoking In Contemporary Cinema

Despite a clear decline in tobacco imagery through the 2000s and early 2010s, recent years have seen a notable—if understated—resurgence. Smoking is reappearing in arthouse dramas, independent films, and prestige productions. Titles such as Lee (2023), The Brutalist (2024), Oppenheimer (2023), and Blonde (2022) feature cigarette use prominently, not only as a historical or stylistic detail, but often as part of the emotional or visual identity of central characters. This resurgence is not confined to adult-rated films. Analyses by groups such as the Truth Initiative and the University of California, San Francisco have shown that smoking has also returned to streaming platforms, especially in series popular with adolescents.

Several factors may help explain this quiet return. One is the shift from traditional studios to streaming platforms, which has loosened regulatory oversight. Unlike theatrical releases, streaming content is not held to consistent content rating standards across countries. Guidelines tend to vary between platforms, and decisions about showing tobacco often come down to individual directors or producers. Another reason frequently cited is the pursuit of realism. Period dramas, in particular, rarely avoid cigarettes, with many filmmakers arguing that omitting tobacco from a mid-20th-century setting would compromise historical credibility.

Smoking is also still used as a visual shorthand for mood or personality: to convey defiance, introspection, or instability, especially in stories about artists, outsiders, or troubled lives. But aesthetic realism may also be masking a deeper trend. In a media landscape increasingly shaped by risk-aversion and algorithmic content delivery, smoking may be regaining traction as a visually arresting, emotionally loaded, and narratively

efficient tool. It requires no dialogue to convey tension or rebellion. It signals interiority and danger. And in a post-pandemic, post-truth world, smoking—ironically—can feel transgressive again.

Whatever the reason, the reappearance of smoking on screen matters. Studies consistently show that visual exposure to smoking—especially when portrayed by protagonists or romanticized characters—increases the likelihood that young viewers will try smoking themselves. Even when the character is morally ambiguous or the context is historical, the act of smoking on screen remains a behavioural cue. The risk is not just theoretical: tobacco use among adolescents in several countries has plateaued or increased in recent years, with media exposure identified as a contributing factor.

Visual Exposure And Cancer Risk

The relationship between onscreen smoking and public health is not merely associative—it is causative, cumulative, and of direct concern to oncology. Repeated exposure to tobacco use in media, particularly film and streaming platforms, has been shown to increase smoking initiation among adolescents and young adults, a pathway that directly contributes to long-term cancer risk.
A landmark 2012 report from the US Surgeon General concluded that "there is a causal relationship between depictions of smoking in the movies and the initiation of smoking among young people." Since then, multiple large-scale studies have quantified this effect: adolescents with the highest exposure to tobacco use in films are roughly two to three times more likely to begin smoking compared to their least-exposed peers. A 2020 meta-analysis found that for every 500 tobacco impressions viewed in movies, the odds of adolescent smoking initiation increased by 39%.

Tobacco remains the leading preventable cause of cancer worldwide. It is implicated in at least 15 types of malignancy. Globally, smoking is responsible for over 1.8 million cancer deaths per year, according to the most recent IARC estimates. Among these, lung cancer remains the most lethal, accounting for more than 80% of tobacco-related cancer mortality.

What makes onscreen exposure especially potent is its ability to normalize smoking without showing its consequences. In most films where smoking is depicted, there is no mention of addiction, disease, or death. Characters smoke without coughing, without diagnosis, without repercussion. This discrepancy reinforces the myth of the "social smoker," the illusion of control, and the separation of smoking from its long-term health outcomes.

From a public health perspective, exposure to tobacco imagery in entertainment is now widely recognized as a modifiable cancer risk factor. Both the World Health Organization and the Centers for Disease Control and Prevention (CDC) have issued recommendations urging the film and streaming industries to reduce or eliminate tobacco portrayals, especially in content accessible to youth. Suggested measures include:

- ***Assigning adult ratings to films and series with smoking***
- ***Including anti-smoking PSAs before content with tobacco imagery***
- ***Requiring clear labelling of tobacco use in content advisories***
- ***Ending public subsidies for productions that depict smoking***

While voluntary adoption has been uneven, the rationale is clear: limiting onscreen tobacco exposure is a form of cancer prevention.

Smoking scenes in recent films

Vaping And The Visual Shift

While vaping has surged among adolescents and young adults, its depiction in film and television remains relatively limited. Unlike cigarettes, vaping devices lack the cinematic weight that once made tobacco a storytelling tool. When e-cigarettes do appear, it is often in the margins: a futuristic flourish in science fiction, a visual shorthand for tech-savviness, or a throwaway joke in teen comedies.

Rarely are they framed with the same emotional or aesthetic significance once reserved for cigarettes. This absence is notable given the scale of the vaping epidemic. In many countries, particularly the United States and the United Kingdom, e-cigarette use among adolescents has surged over the past decade. According to the CDC, more than 2 million middle and high school students reported current e-cigarette use in 2021. In the UK, similar trends have been observed, with a notable rise in underage vaping, often facilitated by flavoured disposable devices.

The oncological concerns around vaping are twofold. First, while e-cigarettes may contain fewer carcinogens than combustible tobacco, they are not harmless. Heating elements in some devices produce formaldehyde, acetaldehyde, and acrolein—compounds with known mutagenic and cytotoxic properties. Moreover, flavouring agents such as diacetyl have been implicated in chronic respiratory conditions, and emerging studies suggest that chronic e-cigarette exposure may lead to DNA damage, impaired repair mechanisms, and oxidative stress—hallmarks of carcinogenesis.

Second, and perhaps more critically, vaping often serves as a gateway to smoking, particularly among youth. Longitudinal studies have shown that adolescents who begin with e-cigarettes are significantly more likely to transition to combustible tobacco products, a progression that reopens pathways to the very cancer risks that decades of tobacco control sought to close. The World Health Organization and major cancer research agencies have therefore taken a cautious stance, warning against the normalization of vaping, particularly in media accessible to young audiences.

In response to these concerns, the European Union has implemented comprehensive regulations on the marketing and sale of vaping products. The Tobacco Products Directive (2014/40/EU), effective since May 2016, prohibits cross-border advertising and promotion of e-cigarettes in various media, including television, radio, print, and online platforms. It also mandates health warnings on packaging, restricts nicotine content, and requires child-resistant packaging for e-cigarette products.

Building upon these measures, Belgium has taken a pioneering step by becoming the first EU country to ban the sale of disposable e-cigarettes, effective January 1, 2025. This decision, approved by the European Commission, is part of Belgium's broader anti-tobacco strategy aimed at protecting public health and the environment. Health Minister Frank Vandenbroucke emphasized that disposable e-cigarettes are particularly attractive to young people due to their low cost and appealing flavours, leading to increased nicotine addiction among youth. Additionally, the environmental impact of these single-use devices, which contain plastics and lithium batteries, contributes to hazardous waste. Following Belgium's lead, France enacted a similar ban on February 26, 2025, prohibiting the sale and distribution of disposable e-cigarettes, commonly known as "puffs." This legislation aims to protect adolescents from nicotine addiction and address environmental concerns associated with these devices. In Italy, while a complete ban on disposable e-cigarettes has not been implemented, the government introduced stricter regulations in January 2025. These include increased taxation on nicotine-containing e-liquids and a ban on the online sale of nicotine products, measures intended to curb youth access and align with traditional tobacco product regulations.

The United Kingdom has also announced a ban on the sale and supply of single-use vapes, set to take effect on June 1, 2025. This measure, part of the broader Tobacco and Vapes Bill, aims to address the environmental impact of disposable vapes and reduce their appeal to young people. The legislation prohibits the sale of single-use vapes across England, Scotland, Wales, and Northern Ireland, with businesses required to deplete their stocks before the enforcement date.

While cinematic depictions of vaping remain limited, social media platforms such as TikTok, Instagram, and YouTube have filled the visual void. Vaping tutorials, lifestyle content, and influencer marketing have created a parallel universe of visual exposure, often devoid of regulatory oversight. The narrative may have shifted from the silver screen to the smartphone, but the implications for public health and cancer prevention remain pressing.

Whether the film and television industry will begin to reflect the ubiquity of vaping in real life, or consciously avoid it as part of a harm reduction effort, remains to be seen. In the meantime, the muted presence of e-cigarettes on screen may represent a rare window of opportunity: to prevent the glamorization of a product whose long-term health consequences are still unfolding.

Policy, Prevention, And Public Responsibility

The return of smoking to film and television is not just a stylistic or narrative development—it raises important questions for public health, particularly in the context of cancer prevention. For oncology professionals and researchers, how tobacco and vaping are portrayed in popular media remains a relevant and unresolved part of the broader effort to reduce avoidable cancer risk. Public health bodies have long recognized the media's role in shaping behaviour. The World Health Organization's Framework Convention on Tobacco Control (FCTC) explicitly calls for a ban on all forms of tobacco advertising, promotion, and sponsorship, including indirect promotion through film and streaming content. Despite this, enforcement has been uneven, particularly in the digital age, where content transcends borders and platform accountability remains limited.

In the European Union, the Tobacco Products Directive remains the cornerstone of e-cigarette regulation. While it addresses advertising and labelling, it does not yet cover the nuanced depictions of vaping and tobacco use in entertainment media. Health advocacy groups, such as the European Cancer Leagues and the European Respiratory Society, are now calling for the inclusion of visual tobacco exposure within broader cancer prevention strategies.

The European Beating Cancer Plan, launched in 2021, recognizes tobacco control as a key pillar of cancer prevention, but its implementation in media policy is still evolving.

Tobacco remains the leading preventable cause of cancer in Europe, responsible for approximately 700,000 deaths annually. Every measure that reduces smoking initiation, particularly among young people, directly translates to future reductions in cancer incidence and mortality. Reducing visual exposure is one such measure.

Several recommendations have emerged from public health agencies, including:

- ***Assigning adult ratings to films and series that depict smoking or vaping, except when used to portray its harmful effects or for historical accuracy.***
- ***Requiring disclosure statements that certify no tobacco industry influence or funding.***
- ***Including anti-smoking messages or disclaimers before films containing tobacco imagery.***
- ***Prohibiting public subsidies for productions that include smoking without public health justification.***
- ***Expanding media literacy campaigns to educate young viewers on how smoking and vaping are used as narrative devices.***

As the cultural pendulum swings, the window to prevent a new generation of tobacco-related cancers may narrow. The cinematic comeback of smoking is not yet a crisis, but it is a cautionary signal. In the face of evolving media habits, shifting aesthetics, and emerging nicotine products, cancer prevention must extend beyond the clinic and into the cultural spaces where risk is quietly rehearsed, and behaviour takes root.

Shushan Hovespyan

Equity: The Word That Shaped Her Career From The Lab To The White House: The Story Of Catharine Young

When Catharine Young talks about inequity, she isn't referring to it in abstract terms. She grew up in South Africa at the end of apartheid, when systemic injustice was not just visible, it was part of daily life. That early exposure to imbalance and transformation would go on to define her career: one rooted in science, but driven by policy. One committed to making breakthroughs reach those who need them most.

"It all began with me growing up in South Africa, where I was born and raised, during a time of incredible transformation," she recalls. "I witnessed a system that was completely inequitable...

That understanding—that big problems require united, systemic solutions—has guided Catharine through every stage of her career. From earning a PhD in biomedical sciences with a focus on neuroscience, to transitioning into policy leadership roles, she has consistently moved toward greater impact.

"I quickly realized that scientific breakthroughs, while amazing, are not enough. They have to reach people who need them the most."

Her shift from academia into policy was more than a career pivot—it was a values-driven decision. For the past decade, she's been applying her scientific training to some of the world's biggest health challenges, culminating in her most high-profile role yet: Assistant Director for Policy and International Engagement for the Biden Cancer Moonshot.

Inside the Cancer Moonshot

"It was one of the greatest privileges of my life to be able to serve in that role," she says.

She worked at the White House Office of Science and Technology Policy, helping to implement the ambitious vision set by President Biden and First Lady Dr. Jill Biden. The initiative's core objectives were twofold: reduce the U.S. cancer death rate by 50% in 25 years and improve the experience of people living with and surviving cancer.

"My role evolved over my time at the White House to think about how we take the mission and the vision of the cancer moonshot globally."

For Catharine, global equity was non-negotiable. While the U.S. faces its cancer burden, most cancer deaths occur in low- and middle-income countries, where access to diagnostics, treatments, and technologies remains far out of reach for many.

"We saw so many amazing things happen during that time and... the beauty of what happens when you have even at the country levels, countries coming together to decide that something was not acceptable anymore."

Her work led to collaborations that transcended borders—unifying governments, public health institutions, and advocates in a shared goal of reducing disparities in cancer care.

The Human Element

Though Catharine has worked with some of the most powerful leaders in the world, she remains deeply grounded in the stories of patients and scientists.

"I will say, though, that many of the greatest teachers that I've had and motivators are those—the patients who have turned their immense pain into advocacy."

She adds: "I am inspired every day by the scientists who turn their incredible curiosity about the world and their motivation to really see impactful change happen around the world... and to work tirelessly for breakthroughs for people that they will never meet."

It's that combination of empathy and systems-level thinking that makes her a unique voice in cancer policy. She believes the greatest change comes not from lone individuals but from sustained collaboration.

"Everybody that I have had the privilege of crossing paths with in my career has underscored that change as a whole is certainly a collective effort."

On Resilience

When asked about resilience—a theme she's written about publicly—she doesn't hesitate.

"I think my whole career has been a test of resilience." Working in cancer, she notes, means grappling not just with biology, but with complex systems, fragmented infrastructures, and emotionally heavy realities. "There are so many aspects that need to be improved for cancer patients that resilience is a trait that is required to push change forward."

A Legacy of Shared Purpose

As her time at the White House came to a close, Young was reflective—not about her achievements, but about the larger movement she had been part of.

"Truly, the legacy that we're leaving behind belongs to the President Biden and First Lady, who were the true leaders in this space... They were the ones who galvanized an entire community, an entire country, an entire world behind this one singular mission."

For Catharine, the legacy isn't a single project or policy—it's the ongoing, global fight to ensure no one is left behind in cancer care.

"It is the work that was done, and it will be the work that continues to improve the lives of patients around the world."

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