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Beyond the Magic Bullet: Reprogramming Cancer Care Through Systems Medicine

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Dr. Jason Williams and Dr. Nathan Goodyear discuss how tumour ablation, immunotherapy, microbiome science and biological ageing are reshaping the future of oncology, while offering new perspectives on the alarming rise of early-onset cancers.

For decades, cancer treatment has largely focused on finding a single breakthrough therapy capable of eliminating disease. However, growing scientific evidence suggests that cancer is far more complex, requiring an integrated understanding of tumour biology, immune function, metabolism, the microbiome and lifestyle factors.

Among the pioneers advancing this systems-based approach is **Dr. Jason R. Williams**, Founder and Director of Interventional Oncology at the Williams Cancer Institute. More than two decades ago, he observed the *abscopal effect*—where treating one tumour triggered regression of untreated tumours elsewhere in the body—an observation that has since inspired global research into combining tumour ablation with immunotherapy.

Joining him is **Dr. Nathan Goodyear**, an integrative medicine physician, board-certified obstetrician-gynaecologist, and CEO of Goodyear Media Group. Together, they advocate a holistic, evidence-informed model that combines precision medicine with immune resilience, metabolic health and preventive strategies to improve long-term cancer outcomes.

In this exclusive interview with BioSpectrum Asia, Dr. Williams and Dr. Goodyear explore why early-onset cancers are increasing worldwide, the growing role of the gut microbiome and biological ageing in cancer risk, and how precision-integrated oncology could redefine prevention, diagnosis and treatment in the coming decade.

Colorectal cancer is increasingly being diagnosed in younger adults. What does current research suggest are the most significant factors contributing to this trend, and why is it becoming a growing public health concern?

Modern medicine continuously looks to Paul Ehrlich's magic bullet as the answer to cancer. Yet, it is modern science that dispels the magic bullet of causation and treatment of cancer with a multi system failure as the cause. As a result, a multi-

system failure approach must also meet treatment.

All aspects of medicine look to the magic bullet of cancer causation and cancer treatment. Whether natural, holistic, integrative, or conventional, this holy grail remains the focus of the cancer quest. This strategy is a **reductionist trap** across the divide.

The real divide is not between conventional and natural medicine. It is between reductionist thinking and systems thinking. Both camps repeatedly search for their own version of the magic bullet. Both look for the needle in the haystack of heterogeneity.

Heterogeneity is the concept that things are not the same. Heterogeneity is a foundational principle in biology. Patients are heterogeneous. Tumors with the same name are heterogeneous. The same tumor in two diï¬?erent patients behaves diï¬?erently—that's heterogeneity. Heterogeneity exists within the same tumor. Even diï¬?erent metastatic sites are heterogeneous. There is an old adage: the more things change, the more they are the same. Here, the more

heterogeneous the biology of cancer proves to be, the more we think all cancers are the same.

The future solutions of cancer care will not be found in either conventional medicine or natural medicine alone. It will belong to systems medicine—an approach that integrates evidence-based pharmaceuticals, nutrition, metabolism, immunology, microbiome science, lifestyle interventions, local therapies, and precision combinations, recognizing that no single intervention is likely to be suï¬?cient for most patients.

UPFs

I recently had the opportunity to travel to China. I left with many questions, but one specific to the question posed here is: is China about 20-30 years behind the U.S. China appears to be following many of the same nutrition, metabolic, and cancer trends that the United States experienced decades earlier. Could the pace and pattern diï¬?er because of cultural, dietary, and environmental diï¬?erences?

One of the greatest contrasts between modern-day America and traditional China is how food is viewed. Today, the average American adult obtains more than half of their daily calories from ultra-processed foods (UPFs). To be exact, approximately 55% of total daily calories are obtained from UPFs. A more startling statistic that predicts an increasing trend, children and adolescents consume an average of 62% of daily calorie intake from UPFs.

In China, consumption of UPFs are increasing rapidly, yet it still represents only a fraction of total calories for most adults. According to the China Health and Nutrition Survey (1997-2011), UPFs generally contribute to only 4-10% of total daily calorie intake nationally.

Despite the low percentage, China's UPF intake has increased from

< 2% of daily calorie intake to 10% today. An increase in obesity, metabolic syndrome, type II diabetes, and obesity-associated cancers in China appears to mirror to the increase in UPFs.

Take home: Americans consume roughly 6-10 times more UPFs than the average Chinese adult.

What are UPFs? Ultra-processed foods are industrialized formulations manufactured largely from refined substances extracted from foods or synthesized ingredients, often containing little intact whole food categorized as NOVA group 4.

What is NOVA? NOVA is not an acronym, but actually is the name of the grouping system of processed foods. It is a food classification system developed by researchers at the University of São Paulo that categorizes foods based on the extent and purpose of processing, rather than their nutrient content or carcinogenicity alone. It is widely used in nutrition and public health research to study dietary patterns and chronic disease. The four NOVA groups of processed foods includes:

- **NOVA Group 1 – Unprocessed or Minimally Processed Foods (cleaning, washing, trimming, chilling, freezing)**
- **NOVA Group 2 – Processed Culinary Ingredients (pressing, milling, refining, grinding)**
- **NOVA Group 3 – Processed Foods (canning, bottling, smoking, baking, fermentation)**
- **NOVA Group 4 – Ultra-Processed Foods (UPFs)**

Ultra-processed food intake in U.S. parallels the obesity risk and obesity-associated cancers. I think it is safe to question if UPFs are even food at all. Clearly, the changing demographics of cancer is multi-factorial. The magic-bullet approach need not apply.

A few UPFs are labeled as carcinogenic, but it is the cumulative composite exposure of emulsifiers, artificial sweeteners, flavor enhancers, food colorings, packaging-derived chemicals, modified starches, refined carbohydrates, and others that lead to cumulative damage, cumulated failure, and loss of resilience.. As a whole, UPFs are not labeled as carcinogens. Numerous prospective cohort studies and meta-analyses have reported an association between higher UPF consumption and increased risk of:

- **Colorectal cancer**
    - Breast cancer
    - Pancreatic cancer
    - Overall cancer incidence and mortality

Ultra-processed food frequently contain flavor enhancers, artificial colors, emulsifiers, stabilizers, preservatives, sweeteners, modified starches, protein isolates, industrial seed oils—all the foods groups we never knew existed. The problem is UPFs are refined, manufactured, extracted, synthesized, and do not resemble food at any level, but they rarely exist in isolation. In fact, UPFs often contain 10-30 diï¬?erent UPF ingredients.

Dysbiosis

Most look to obesity as a root cause of disease. Yet, it is a biomarker of root dysfunction. It is a canary in the coal mine that pivots from a biomarker of dysfunction to a contributor to dysfunction and disease. It is the gut-microbiome axis that funnels together the immune system and metabolic impact:

- Reduced gut microbial diversity
- Dysbiosis
- Increased Pathobionts
- Decreased SCFA production (butyrate, propionate, acetate)
- Altered bile acid signaling
- Breakdown of gut epithelial barrier
- Increased leaky gut
- Kynurenine/IDO1 (Indoleamine 2,3-dioxygenase 1)-mediated immune suppression
- Increased LPS ? TLR4 activation
- Increased chronic systemic inflammation
- Increased inflammaging
- Increased senescence
- Promoting a Senescence Associated Secretory Phenotype
- Increased chronic systemic inflammation
- Accelerated cell aging

The result is an alteration in the immune axis with significant tumor-immune microenvironment consequences. An increase in myeloid-derived suppressor cells (MDSCs), tumor-associated macrophages (TAMs), and regulatory T cells (Tregs) promotes immunosuppression. As a result, the activity of CD8+ T cells and natural killer (NK) cells is decreased. This promotes increased immune-desert and immune-excluded phenotypes in cancer. This impact is separate from the impact of chemotherapy and radiation on the immune system—separate, yet stacked.

The end result, no pun intended, is a change in the demographics in cancer, here colorectal cancer.

Obesity

The immune system originates in the gut. Moreover, the gut metabolome, functional or dysfunctional, is more important than the taxa. Any intervention or therapy that modulates the immune system must take into consideration the environment within the gut. Early-onset cancers and obesity-associated cancers are clear evidence of the disruption of the microbiome-immune-metabolic axis.

Two core convergent epidemiological signs must be considered. First, early-onset cancers (ages 20-49) are rising across multiple tumor types. According to a recent British Medical Journal article, eleven tumors are rising disproportionately in young adults:

- **Colorectal**

- Breast
- Ovarian
- Endometrial
- Pancreatic
- Liver
- Kidney
- Gallbladder
- Thyroid
- Multiple myeloma
- Oral

In parallel, the Centers for Disease Control (CDC) in the U.S. has shown that thirteen obesity-associated cancers are increasing in conjunction with increasing age-adjusted mortality rates. The thirteen obesity-associated cancers include the following:

- **Colorectal**

- Endometrial
- Esophageal adenocarcinoma
- Gastric cardia
- Liver
- Gallbladder
- Pancreatic
- Kidney
- Thyroid
- Ovarian
- Multiple myeloma
- Meningioma

The alarming mortality rates highlighted by the CDC point to an alarming change in demographics in cancer. Age-adjusted mortality rates in obesity-associated cancers have increased from 3.73 per million in 1999 to 13.52 per million in 2020. That is a 3.6-fold increase in mortality in obesity-associated cancers in one generation.

Independent data from the CDC and BMJ demonstrate a parallel

trend. We ignore them at our own peril. A lack of curiosity will allow this peril to become reality.

An analysis of overlap provides key insights. Ten of the eleven cancers between the BMJ and CDC directly overlap:

- **Colorectal**

- Breast
- Ovarian
- Endometrial
- Pancreatic
- Liver
- Kidney
- Gallbladder

- Thyroid
- Multiple myeloma

Ninety-one percent of early-onset cancers are obesity-associated. Currently, 74% of U.S. adults are either obese or overweight. The future trend provides no reason for optimism, as 21.2% of those aged 2-19 are obese, with greater than 70% maintaining obesity beyond the age of 30. The adolescent obesity rate cannot be properly framed without proper context. Pre-pandemic, the adolescent obesity rate was at 19.3%. The rise in childhood obesity from 19.3% to 21.2% represents more than excess weight—it reflects large-scale disruption of the gut microbiome, immune programming, metabolism, obesity, and future disease potential, particularly the aforementioned 10 cancer risks.

These parallel data points cannot be interpreted through the prism of cancer incidence alone. It reflects more aggressive tumor biology.

Moreover, it reflects an altered human metabolism, tumor metabolism, and reduced tumor immune competence.

Ultimately, a compromised immune system is at the root of the changing demographics of cancer, yet the root is not single, but the roots are numerous.

The gut microbiome is receiving increasing attention in cancer research. How does microbial health influence inflammation, immune surveillance, and potentially long-term cancer risk?

The gut is the origin of the immune system. The immune system priming begins with pregnancy, birth, and breast feeding. Alterations of birth, caesarean versus vaginal, and breast feeding versus formula feeding, set up diï¬?erent gut microbiome taxa and diversity. The origin of immune priming in the gut initiates the immune surveillance capacity and immune tolerance potential. Impaired immune priming at origin stunts immune maturity which compromises immune function through the life of the eï¬?ected individual.

I propose we have not even began to understand the tip of the iceberg what the gut microbiome can do for health or for disease propagation. Don't think of the gut microbiome as merely a collection of bacteria. The gut microbiome is a complex ecosystem—an organ. Beyond the gut microbiome and metabolome, the oral microbiome and the tumor microbiome are less understood. The future of cancer may have more to do with the microbiome, microbiomics, than the named cancer itself.

In general, chronic inflammation is the bed that cancer lies in. Yet, not all inflammation is bad. Inflammation is a normal part of the healing process. Just look at a sprained ankle: the accompanied pain and swelling are a part of the reparative process. Likewise, inflammation that supports anti-tumor competence is equally healing.

Some researchers suggest that biological ageing can occur independently of chronological age. How do immune dysfunction, chronic inflammation, and metabolic health contribute to this process, and what implications does this have for cancer risk?

A 2026 article, *Steatosis shapes prognosis-defining liver metastasis heterogeneity in colorectal cancer,* published in the journal Nature 1, highlights the distinction between chronological aging and biological aging.

The seed and soil hypothesis is relevant to this conversation of aging. Stephen Paget first proposed this theory in 1889. The metaphor is clear. The cancer cell is the "seed," and the organs are the "soil." Successful metastasis requires fertile soil for the circulating seed to grow. Tumor genetics, cellular plasticity, immune evasion, and metabolism are bound in the concept of the "seed." The soil encompasses organ biological age, metabolic health, immune competence, fibrosis, extracellular matrix, microbiome, and vascular biology.

Metastasis cannot be reduced to a simple seed and soil, but is actually the interaction between tumor biology and host organ biology. The hallmarks of cancer, originally published by Hannahan and Weinberg in 2000 must include the hallmarks of a metastatic organ. The process of metastasis is equally important as the carcinogenic processes described in the hallmarks of cancer because 90% of morbidity and mortality is when cancer spreads—metastasis.

The traditional view of cancer metastasis is one primarily the property of the cancer cell (the "seed"). Clinical emphasis has focused on tumor genomics, driver mutations, immune checkpoints, targeted

1 Peng-Winkler, Y., Liu, X.-Z., Verheul, S. M. L., Girondel, C., Igelmann, S., Rotter, S. M., et al. (2026). *Steatosis shapes prognosis-defining liver metastasis heterogeneity in colorectal cancer. Nature*. Advance online publication.

https://doi.org/10.1038/s41586-026-10686-2 therapies, tumor mutational burden, and circulating tumor cells. The metastatic organ was largely a passive recipient of the circulating tumor cell.

The 2026 paper referenced above links hepatic steatosis towards a worse type of liver metastasis. Hepatic steatosis is a biomarker of accelerated liver aging. Fatty liver demonstrates many hallmarks of biological aging, including:

- Cellular senescence
- Senescent Associated Secretory Phenotype (SASP)
- Mitochondrial dysfunction
- Oxidative stress
- Chronic inflammation
- Fibrosis
- Extracellular matrix remodeling
- Stem cell dysfunction
- Telomere shortening
- Immune senescence

The steatotic liver functions as a **biologically older liver**. The result is an increase in replacement pattern metastasis (worst prognosis) versus desmoplastic and pushing pattern liver metastasis.

Aging has historically been isolated to chronological age. Modern-day aging discussions separate aging into chronological and biological aging as distinct. The 2026 article adds a new category to the age debate—organ aging. The investigators measured aging based on PhenoAge, KDM Biological Age, Metabolic clocks, and Proteomic organ clocks. To determine organ-specific biological aging, they used plasma proteomics and machine learning to estimate organ-specific age. They looked at the liver, immune system, adipose tissue, brain, kidney, heart, and muscle. Based on the findings of the study, the authors proposed that hepatic steatosis was consistent with accelerated organ aging in the liver, increasing metastatic risk. Worse, the associated accelerated aging seems to naturally select for liver metastasis with the worst prognosis.

Another 2026 article, *Biological Aging and Generational Shifts in Early-Onset Cancer Risk*, appropriately titled, investigated if accelerated aging can help explain the worldwide rise in early-onset cancers. The authors examined whether younger generations are

biologically older than previous generations at the same chronological age and hypothesized that accelerated aging may be an unrecognized contributor. They call the separation of chronological age and biological age the “generation gap.”

The cohort of 160,000 individuals used multiple aging clocks, PhenoAge, the Klemar-Doubal method, the metabolic aging clock, and the proteomic organ-specific aging clock to assess the separation gap and its connection to early-onset cancers, i.e., colorectal cancer, pancreatic cancer, endometrial cancer, and renal cancer. The authors found that the diï¬?erent organs evaluated did not age in parallel. Two organ clocks were independently associated with early-onset cancer. Specific organ aging and specific early-onset cancer types found that immune aging was associated with an 89% increased relative risk (hazard ratio of 1.89) with early-onset lung cancer, and adipose tissue was associated with a 60% increased relative risk (hazard ratio of 1.60) with early-onset colorectal cancer.

The studies’ collective findings:

- Younger generations are aging faster biologically

Out of the multiple generations evaluated, recent generations showed higher biological age, greater age acceleration, and larger age gaps. Biologically plausible drivers, though not proven causal, include ultra-processed foods, obesity, sedentary behavior, sleep disruption, circadian disruption, environmental pollutants, chronic inflammation, psychosocial stress, and microbiome alteration.

- Accelerated aging increased early-onset cancer risk

An eight percent higher risk of early-onset solid cancers was associated with greater systemic aging. The highest aging category showed a 15% greater risk compared to the lowest aging group. Is this **intergenerationally** and

transgenerationally inheritable? Are we seeing the acceleration compounding?

- The strongest associations of the separation gap were cancer-specific.

Lung cancer showed the strongest association with a hazard ratio of 1.42. Elevated biological aging increased risk after accounting for non-inheritable risk factors, and the association persisted independent of smoking. Others include colorectal cancer at a hazard ratio of 1.14 and “other” gastrointestinal malignancies with a hazard ratio of 1.25. Endometrial cancer showed one of the clearest signals between age acceleration and early-onset cancer. Overall, the hazard ratio between accelerated aging and cancers evaluated was 1.17.

- Organ-specific aging matters

Organ-specific aging was one of the most novel aspects of the study. The authors assessed whether aging within specific biological systems increased the risk of early-onset cancer. This is where the specific organ systems, the immune system, and adipose tissue enter. Advancing immune aging was associated with increased risk of early-onset lung cancer, and accelerated adipose tissue aging was linked to increased early-onset colorectal cancer.

One could say we are PHARMing disease and not FARMing health, and not only are we getting more obese, but we are accelerating aging and cancer risk as a result.

- **Organ-specific aging is not uniform across all organs in the same individual**

The authors looked across multiple “organs” to assess uniformity of aging and its association with early-onset cancer. Two distinct organ systems were found to be associated with increased early-onset cancer risk: immune system and adipose tissue. The take home point is that aging is not merely chronological, biological, or uniform across all organ systems.

- **The eï¬?ects were independent of genetics**

The eï¬?ect was independent of genetics. The variables cancer genetic risk, genetic aging predisposition, and telomere length were adjusted for, yet the biological age gap remained predictive. This suggests we are moving far beyond cancer as a genetic disease towards the concept that aging itself may be a measurable and actionable cancer risk factor.

Are there warning signs or symptoms of colorectal cancer that younger adults and healthcare providers may be overlooking because of traditional assumptions about age-related risk?

The first hurdle is educating the general public and doctors. The old adage “you don’t know what you don’t know” points to the relative ignorance about the changing demographics of cancer. The result is that patients and healthcare providers are unaware of risk and need to recognize warning signs and symptoms of a disease, colorectal cancer, that has been historically delegated to decades later.

Early-onset colorectal cancer (EOCRC) is likely environmentally driven

- a canary in the coal mine. It is not simply an earlier version of late-onset CRC. A 2026 paper published in the journal Nature Medicine, “*Epigenetic fingerprints link early-onset colon and rectal cancer to pesticide exposure,”* indicates that early-onset colorectal cancer is a distinct cancer type in itself. Early-onset colorectal cancer (EOCRC) shows distinct epigenetic signatures compared to late-onset CRC. These changes are not primarily age-driven (late-onset) but rather reflect environmental imprinting in EOCRC, distinct from that in late-onset CRC. There is strong overlap of EOCRC with epigenetic patterns associated with pesticide and toxicant exposure. This supports EOCRC and the changing demographics of cancer as a shift from a one-hit, mutation-first carcinogenic paradigm to an environment-driven epigenetic reprogramming, cumulative damage, cumulative failure, and loss of resilience.

The evidence points to a structured mechanistic cascade that is relevant to identifying early warning signs and symptoms:

- Composite, cumulative environmental exposure (pesticides or toxicants)
- leads to epigenetic reprogramming (DNA methylation changes)
- Gut microbiome dysbiosis
- Leaky gut
- Impaired immune priming
- Chronic inflammation
- Altered gut metabolome

- Leads to accelerated aging at the cell level
- Increases immunosenescence
- Promotes a senescent-associated secretory phenotype
- Promotes inflammaging
- leads to altered gene expression
- leads to immune dysregulation and impaired surveillance
- leads to a cold tumor microenvironment (immune-excluded to immune-desert spectrum)
- leads to anti-tumor immune competence
- leads to tumor initiation and progression

Early-onset colorectal cancer reflects environmentally imprinted epigenetic reprogramming that drives a cold, immune-excluded or immune-desert tumor microenvironment and impairs immune surveillance. The result is a PHARMed new cancer type, consistent with the changing demographics of cancer, that is equipped to accelerate development and immune evasion.

The earliest warning signs begin in our individual history. Maternal health, maternal nutrition, cesarean versus vaginal birth, breastfeeding versus formula feeding, and antibiotic exposure warrant earlier screening secondary to elevated risk. Though relegated to “medical history,” they identify risk and the need for accelerated screening. These earliest of screenings must include the

health of the mother because maternal diet ? maternal gut microbiome ? maternal microbial metabolites ? placenta/fetal circulation ? fetal immune programming ? altered postnatal microbiome and immune trajectory. In a 2023 article, maternal diet was found to reshape the maternal gut microbiome and metabolome, and microbial metabolites from this system can reach the fetoplacental unit and influence fetal immune development, placental biology, and postnatal microbiome/immune trajectories. Here, the risk and screening predate the infant immune priming—it is the potential, the trajectory. Imagine if we intervened here?? That would be true prevention.

Screening for obesity in children and adolescents is a priority. Age-adjusted mortality rates in obesity-associated cancers have increased from 3.73 to 13.52 per million, according to the CDC 2. Worse, according to the most recent data, 21.1% of children and adolescents, ages 2-19, are obese, including 7% with severe obesity, increasing the future adult cancer risk pool in real time 3. Obesity is showing up earlier, lasting longer, and often with greater severity. A large meta-analysis found that about 80% of obese adolescents remain obese in adulthood, and about 70% remain obese beyond age 30 4. The U.S. estimates that about 42.9 million people ages 10–19 in 2023 are obese; that implies roughly 9.1 million adolescents are currently obese, and on the order of 7.3 million could carry obesity

2 Ullah A, Haider R, Ahmed F, Fayyaz A, Ali R, Aman K, Zahid H, Abid H, Rasheed Y, Nayyab I, Gohar N, mohamed Kamel O, Mirza T, Abid M, Bakr M, Eltawansy S. SUN-583 Obesity-Associated Cancer Mortality in the United States (1999-2020): A National Epidemiological Analysis. J Endocr Soc. 2025 Oct 22;9(Suppl 1):bvaf149.1234. doi: 10.1210/jendso/ bvaf149.1234.

3 Noiman, A. N., Fryar, C. D., Saif, N. T., & Aï¬?ul, J. (2025). Prevalence of overweight, obesity, and severe obesity among children and adolescents ages 2–19 years: United States, 1963–1965 through August 2021–August 2023. *NCHS Health E-Stat, 112*, 1–7. https://doi.org/ 10.15620/cdc/174645

4 Simmonds, M., Llewellyn, A., Owen, C. G., & Woolacott, N. (2016). Predicting adult obesity from childhood obesity: A systematic review and meta-analysis. *Obesity Reviews, 17*(2), 95–

107. https://doi.org/10.1111/obr.12334 into adulthood if nothing changes 5. This is not a cancer forecast by itself, but it is a very large “at-risk pipeline.” In a meta-analysis focused on early-onset colorectal cancer, overweight was associated with a 32% higher risk and obesity with an 88% higher risk, compared with normal weight6.

Early screening should prioritize ecosystems that promote an environment conducive to EOCRC: assessment of dysbiosis. Dysbiosis assessment is not just about individual taxa or species, but about patterns, durability, impact on the metabolome, and the eï¬?ect on immune priming in a critical window

Looking ahead, what emerging areas of research do you believe will have the greatest impact on our understanding of early-onset cancers, and how might these insights shape future prevention and screening

strategies?

I believe the emerging areas of research that will provide the biggest breakthroughs in understanding early-onset cancers won't come from one “hit” causation, one concept, one pathway, one categorization, one revolutionary drug, or one treatment.

Understanding will come from integrating multiple ecosystems into a smarter biological strategies. One that programs, reprograms, and engineers. I believe the emerging areas of research that will drive the innovation of the future in

5 U.S. Census Bureau. (2026). Annual estimates of the resident population by single year of age and sex for the United States: April 1, 2020 to July 1, 2025. Population Estimates Program.

6 Li, H., Boakye, D., Chen, X., Hoï¬?meister, M., & Brenner, H. (2021). Association of body mass index with risk of early-onset colorectal cancer: Systematic review and meta-analysis. *The American Journal of Gastroenterology, 116*(11), 2173–2183. https://doi.org/10.14309/ ajg.0000000000001393 understanding, preventing, and treating early-onset cancers include:

- Microbiome
- Biology as bi-directional
- Spatial biology
- Tumor microenvironment
- Tumor immune microenvironment
- Intratumoral therapy
- Immune resilience
- Immune reprogramming
- Ageing biology
- Artificial Intelligence and disease modeling
- Precision-integrative therapies
- Remembering our past

I believe the future of oncology is moving from fighting cancer to restoring and reprogramming biology resilience to prevent cancer and to restore health when cancer is present.

Cancer develops when the body's communication networks and biological systems lose resilience. The more precisely we understand these communication networks—across the genome, immune system, metabolism, microbiome, nervous system, and tumor microenvironment—the more eï¬?ectively we can help patients restore control over the disease.

The next decade will not simply be defined by better drugs. It will be defined by better integration in prevention and treatment.

Precision, combination, and integration are not competing ideas—they are the three pillars of the next era of cancer care. Precision tells us who and what to target. Combination determines how to intervene across multiple biological systems. Integration ensures we treat not only the tumor, but also the patient as a whole. That is the direction I believe oncology must take if we want to improve both survival and quality of life—enter that next phase of precision.