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Unresolved Problems in Contemporary Cancer Medicine

Beyond the Breakthrough Headlines: Toward Better Cancer Outcomes Through a Disease-Host Systems Perspective

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Adapted for public education from the author's scientific preprint, [Unresolved Problems in Contemporary Cancer Medicine: Prevention Gaps, Limited Durable Benefit, Treatment-Related Harm, Quality of Life, and Financial Toxicity](#). Prepared in connection with the [International Conference on Orthomolecular Medicine](#) (ICOM-ATU 2026), Algeria.

Cancer Medicine Has Advanced. But How Much Have Patients Benefited?

Modern cancer medicine has achieved remarkable scientific and technological progress.

Targeted therapies, immunotherapy, molecular diagnostics, precision oncology, and increasingly sophisticated treatment strategies have transformed care for selected groups of patients.

Some of these breakthroughs are genuinely life-changing. Imatinib revolutionized the treatment of chronic myeloid leukemia. Immune checkpoint inhibitors have produced durable cancer control in a subset of patients with advanced melanoma. Other developments have improved survival, reduced complications, or made previously untreatable cancers more manageable.

These achievements deserve recognition.

Yet they coexist with a less celebrated reality: **many patients with cancer still face limited survival, recurrence, treatment-related suffering, declining physical function, and substantial financial hardship.**

Billions of dollars are invested in cancer research and treatment. New therapies receive regulatory approval. Promising results appear regularly in scientific journals and the media.

But patients and families deserve answers to questions that are more fundamental than whether a drug is new or technologically impressive:

- Does the treatment help people live longer?
- Does it help them live better?
- Are the benefits durable?
- What physical, psychological, and financial costs accompany treatment?
- Can more cancers be prevented in the first place?

These questions motivated my recent scientific preprint examining seven unresolved problems in contemporary cancer medicine.

They also form the foundation of my forthcoming presentation at ICOM-ATU 2026 in Aïn Témouchent, Algeria.

The central argument is straightforward: medical progress should be judged by meaningful benefits experienced by patients, not merely by the number of breakthroughs announced.

Seven Unresolved Problems in Contemporary Cancer Medicine

Despite advances in cancer diagnosis and treatment, seven major challenges warrant greater scientific and clinical attention.

1. Prevention remains inadequate

Cancer continues to impose an enormous global burden.

Although important progress has been achieved through tobacco control, vaccination, screening, and prevention of selected carcinogenic exposures, many opportunities remain incompletely implemented.

Concerning increases in certain early-onset cancers also demand careful investigation.

A comprehensive prevention strategy should consider established and potentially modifiable risk factors, including metabolic health, obesity, alcohol consumption, tobacco exposure, environmental carcinogens, selected infections, and other relevant contributors.

The relative importance of these factors differs among cancer types, and associations should not automatically be interpreted as proof of causation.

2. Durable benefits remain limited for many advanced cancers

Some modern therapies produce impressive initial responses.

However, a reduction in tumor size does not always translate into long-term disease control or improved survival.

Treatment-resistant cancer populations may persist, adapt, or expand.

Many metastatic cancers remain difficult to control durably.

3. Precision-medicine breakthroughs benefit selected populations

Targeted therapies and immunotherapies have produced major benefits in particular cancers and molecularly defined subgroups.

However, not every patient has an actionable molecular alteration, and not every patient with a relevant biomarker responds.

Even when an effective treatment exists, resistance, access, cost, and differences in clinical characteristics may limit its usefulness.

A genuine measure of progress must therefore consider not only how well a therapy works in responders, but also how many patients are eligible and how many achieve meaningful benefit.

4. Treatment-related harm remains substantial

Cancer treatments can cause both short-term and long-term complications.

These may include cardiovascular injury, neuropathy, organ dysfunction, endocrine disturbances, impaired fertility, persistent fatigue, and other forms of toxicity.

Certain therapies can also increase the risk of subsequent malignancies.

Such risks must be weighed against expected treatment benefits, especially when outcomes are uncertain.

5. Conventional endpoints do not always capture patient benefit

Tumor response, biomarker changes, and progression-free survival can provide useful clinical information.

But these outcomes are not interchangeable with overall survival, preserved function, or improved quality of life.

A favorable imaging result is meaningful. It is not the entire story.

6. Quality of life remains an essential unmet need

Patients may experience fatigue, pain, muscle loss, reduced mobility, psychological distress, and loss of independence during and after treatment.

Successful cancer care should include prevention and management of these problems.

Living longer matters. So does maintaining the capacity to live well.

7. Financial toxicity threatens patients and healthcare systems

Modern cancer care can impose severe economic burdens.

Drug prices, hospital expenses, repeated investigations, travel, caregiving requirements, and loss of income can affect patients and families long after treatment begins.

These burdens differ across healthcare systems but are relevant internationally, including in countries with substantial public insurance coverage.

Together, these seven challenges reveal a critical gap between scientific progress and the broader outcomes that matter to patients.

The task is not to dismiss modern oncology, but to build upon its achievements while confronting its remaining limitations.

1. Tumor Shrinkage Is Not the Same as Durable Cancer Control

Cancer is rarely biologically uniform.

Different regions of the same tumor may contain cells with distinct genetic characteristics, metabolic behaviors, and treatment sensitivities.

This phenomenon, known as *intratumor heterogeneity*, can contribute to treatment resistance.

A therapy may eliminate sensitive cancer cells while less-sensitive or resistant populations survive.

Over time, these populations can expand and cause recurrence or progression.

This distinction suggests three important dimensions of treatment success:

Tumor response: What happened to the malignant disease?

Durability: How long did cancer control last?

Patient outcomes: How long and how well did the patient live?

Each dimension provides useful information, but none should automatically substitute for the others.

The most clinically meaningful evaluation integrates tumor outcomes, overall survival, treatment harms, and the patient's physical and functional condition.

2. Cancer Is Not an Isolated Tumor

Cancer cells do not exist independently of the body.

They interact with blood vessels, immune cells, connective tissues, metabolic substrates, inflammatory mediators, and numerous physiological signals.

The tumor influences its host, and the host influences the tumor.

Cancer can disturb systemic metabolism, impair appetite, provoke inflammatory responses, contribute to muscle wasting, and reduce physiological resilience.

Meanwhile, anticancer treatments can affect both malignant and normal tissues.

These interactions suggest that successful cancer management requires attention to more than tumor characteristics alone.

They also motivate greater interest in cancer metabolism and the patient's overall physiological condition.

3. Cancer Metabolism: From Warburg to Modern Systems Biology

Otto Warburg's pioneering research identified a distinctive feature of many cancers: increased glucose fermentation even when oxygen is available.

This phenomenon became known as the **Warburg effect**.

Later investigators, including Thomas Seyfried, emphasized the possible importance of mitochondrial and metabolic abnormalities in cancer biology.

These contributions helped stimulate interest in metabolic cancer therapies.

However, contemporary evidence reveals that cancer metabolism is considerably more complex than a simple dependence on glucose fermentation.

Many cancers retain functional mitochondria.

Glycolysis and mitochondrial oxidative metabolism can coexist.

Depending on the malignancy and biological environment, cancer cells may utilize glucose, glutamine, lactate, fatty acids, and other metabolic substrates.

Metabolic characteristics can differ among tumor types, among patients, and even among cancer-cell populations within the same tumor.

This leads to an essential conclusion:

Cancer metabolism creates potentially important therapeutic opportunities, but no single metabolic vulnerability is shared uniformly by all cancers.

A scientifically credible metabolic approach must account for this diversity.

4. Why Starving Cancer Has Important Biological Limits

An appealing therapeutic proposal is to deprive malignant cells of the nutrients they need.

If many cancers consume substantial glucose, why not restrict glucose?

If certain tumors depend on glutamine, why not limit glutamine?

These questions are biologically reasonable, and selected metabolic interventions deserve continued

investigation.

But generalized nutrient deprivation faces three major physiological challenges.

Normal tissues require many of the same nutrients

Healthy cells and malignant cells share essential metabolic substrates.

Glucose supports important normal physiological functions.

Glutamine contributes to intestinal, immune, and other cellular processes.

Moreover, the body maintains circulating nutrients through regulated endogenous metabolism.

Consequently, dietary restriction does not necessarily eliminate a nutrient from the tumor environment.

Cancer cells can adapt

Some cancer cells possess considerable metabolic flexibility.

When one pathway becomes less available, malignant cells may increase their use of another.

Such adaptation can undermine therapies directed at a single metabolic dependency.

Aggressive restriction may weaken the patient

Many patients with advanced cancer experience reduced appetite, unintended weight loss, muscle wasting, and metabolic dysfunction.

Restrictive nutritional interventions may worsen these problems, especially when they reduce energy or protein intake.

This creates an important paradox:

A strategy intended to starve the cancer may also weaken the person living with cancer.

This does not mean that metabolic strategies, including ketogenic diets or other carefully selected interventions, should be dismissed.

It means that their benefits, risks, patient selection, and effects on nutritional adequacy and clinical outcomes must be evaluated rigorously.

Metabolic therapy should not be equated with indiscriminate caloric deprivation.

These challenges are discussed in greater detail in my related preprint, [The Biological Limits of Tumor Starvation: A Systems Physiology Perspective on Metabolic Cancer Therapy](#).

5. The Missing Half of Cancer Treatment: Disease Pressure and Host Capacity

Why do two patients with apparently similar cancers sometimes experience different clinical outcomes?

Differences in tumor biology, stage, molecular features, and treatment response provide part of the explanation.

But another important dimension is the physiological condition of the patient.

Patients vary in organ reserve, muscle mass, metabolic health, nutritional status, functional capacity, and ability to tolerate treatment.

This observation motivates a proposed systems-medicine framework that I refer to as the **Disease-Host Dual-System Framework**.

It considers two interacting dimensions.

Disease Pressure

Disease Pressure represents the biological burden imposed by the malignancy.

Potential components include tumor burden, invasiveness, metastatic activity, treatment resistance, metabolic disturbances, inflammation, and other disease-associated effects.

Host Capacity

Host Capacity represents the patient's ability to maintain physiological function, tolerate stress, adapt, repair damage, and recover.

Potential contributors include nutritional status, metabolic regulation, organ function, immune competence, muscle mass, and physical capacity.

The framework proposes that patient outcomes depend on the dynamic interaction between these dimensions.

As disease burden increases, patients may become increasingly vulnerable.

As effective treatment reduces disease burden and evidence-based supportive care helps maintain physiological function, the overall clinical condition may improve.

The proposed therapeutic principle is:

Reduce Disease Pressure. Preserve and strengthen Host Capacity.

Disease Pressure and Host Capacity are presently conceptual constructs, not validated numerical clinical indices.

Their usefulness must ultimately be evaluated through research demonstrating whether they improve assessment, prediction, or treatment planning beyond existing clinical methods.

Read more: [The Disease-Host Dual-System Framework - OMNS Global](#).

6. Therapeutic Window Engineering: Protecting the Patient While Treating the Cancer

Cancer therapy often requires imposing substantial biological pressure on malignant cells.

Unfortunately, many treatments also affect normal tissues.

This creates a fundamental clinical challenge: maximizing anticancer benefit while minimizing harm.

The proposed I-OM concept of **Therapeutic Window Engineering** expands this question.

Rather than focusing exclusively on increasing tumor sensitivity, it also examines whether systematic preservation of host physiology may improve treatment tolerance, recovery, and meaningful outcomes.

Two complementary objectives are involved.

Disease-directed treatment seeks to eliminate cancer, suppress malignant growth, and reduce tumor burden using effective oncologic interventions.

Host-directed optimization seeks to preserve nutritional adequacy, physical function, organ reserve, and the physiological capacity to tolerate treatment and recover.

Several elements already have established roles in supportive oncology, including appropriate nutrition care, exercise and rehabilitation when feasible, symptom management, prevention of complications, and correction of clinically significant deficiencies.

Other proposed interventions remain investigational.

An important research question is whether a more systematic combination of disease control and host optimization can improve treatment completion, reduce complications, preserve function, and potentially improve survival.

The objective is not simply to deliver more intensive therapy.

It is to improve the balance between treatment effectiveness and patient well-being.

7. Beyond Nutrient Starvation: Targeting Differences Between Cancer and Normal Cells

The limitations of generalized tumor starvation suggest another promising scientific direction.

Instead of asking only how to deprive cancer cells of nutrients, researchers can investigate whether malignant cells possess vulnerabilities that differ sufficiently from those of normal tissues to permit selective treatment.

Potential research areas include:

- Metabolic pathway dependencies
- Redox regulation and oxidative stress responses
- Cellular repair mechanisms
- Stress-response signaling
- Treatment timing and recovery dynamics

The central question becomes:

Can we preferentially damage malignant cells while preserving the capacity of normal tissues to function and recover?

One area relevant to orthomolecular medicine is pharmacological intravenous vitamin C.

At sufficiently high concentrations, intravenous ascorbate has been investigated for potential pro-oxidant effects in experimental cancer systems.

Certain laboratory studies have identified differential responses between malignant and nonmalignant cells.

However, cancer responses vary, and clinical efficacy has not been established broadly across cancer types.

High-dose intravenous vitamin C also carries potential risks, and its suitability requires careful clinical assessment.

It should not be presented as a proven substitute for established anticancer therapy.

The scientific opportunity is to investigate potentially measurable differences between malignant and normal-tissue responses rather than assume that such differences necessarily translate into better clinical outcomes.

8. Dual-Pulse High-Dose Intravenous Vitamin C: An Investigational Research Hypothesis

A proposed extension of this research direction is **Dual-Pulse High-Dose Intravenous Vitamin C (DP-HDIVC)**.

The concept has been described in the scientific preprint [*High-Frequency Pharmacologic Ascorbate Therapy for Cancer: A Dual-Pulse Oxidative Stress Strategy \(DP-HDIVC\) Targeting Tumor Redox Vulnerability*](#) and discussed in OMNS materials.

The hypothesis examines whether repeated pharmacological ascorbate exposures, separated by an appropriately investigated interval, might exploit differences between malignant-cell stress responses and normal-tissue recovery.

Its conceptual sequence is:

First exposure → Investigational recovery interval → Second exposure

The underlying research question is whether malignant and healthy tissues respond differently to

sequential redox-related stress.

Several questions remain unanswered:

- Do clinically relevant differences in tumor and normal-tissue recovery occur in humans?
- Can these responses be measured reliably?
- Does exposure timing influence therapeutic selectivity?
- Can repeated exposures improve meaningful clinical outcomes?
- How might the approach interact with chemotherapy, radiation, or immunotherapy?
- Could cumulative toxicity offset any proposed benefit?

At present, **no validated DP-HDIVC dose, pulse interval, recovery protocol, or clinical efficacy has been established.**

DP-HDIVC remains a research hypothesis, not a validated cancer-treatment regimen or clinical dosing recommendation.

Its relevance to the Disease-Host framework lies in the possibility of studying tumor vulnerability and host protection together.

Clinical investigation must precede therapeutic claims.

9. A New Systems-Medicine Perspective: Tumor → Metabolism → Host → System

The scientific progression discussed in the forthcoming Algeria presentation can be summarized in four interconnected dimensions.

TUMOR

Understand malignant disease, tumor heterogeneity, molecular targets, treatment resistance, and effective oncologic interventions.

METABOLISM

Investigate cancer-cell metabolism, metabolic flexibility, and potential vulnerabilities while recognizing the physiological limitations of generalized nutrient deprivation.

HOST

Assess nutritional status, metabolic regulation, organ reserve, immune function, muscle mass, physical resilience, and treatment tolerance.

SYSTEM

Integrate effective disease-directed care with appropriate host assessment, supportive interventions, longitudinal monitoring, and patient-centered outcomes.

The framework is not intended to replace conventional oncology.

It seeks to extend the scientific discussion toward a more comprehensive understanding of cancer as a disease occurring within-and interacting with-a living human system.

Weaken the disease. Preserve and strengthen the host.

10. How Should Cancer Progress Be Measured?

Returning to the seven unresolved problems, a fundamental question emerges:

How should we measure genuine progress in cancer medicine?

My proposed **Cancer Progress Scorecard** encourages more comprehensive evaluation across seven domains:

Domain	Central question
Prevention	How much cancer burden can be prevented?
Durable benefit	Do treatments extend survival or provide lasting disease control?
Reach of innovation	How many patients actually benefit?
Treatment harm	What acute and long-term complications occur?
Meaningful outcomes	Are survival and functional outcomes improved?
Quality of life	Are independence, physical capacity, and well-being preserved?
Financial burden	Is care accessible and economically sustainable?

This scorecard is an organizing framework, not a validated numerical scoring instrument.

Its purpose is to make benefits, harms, patient populations, time horizons, and uncertainties more transparent.

The same evidence standards should apply to all medical approaches.

Conventional oncology, metabolic interventions, nutritional therapies, and proposed I-OM protocols should be judged by scientifically credible evidence, patient safety, and meaningful clinical outcomes.

A treatment should not be considered superior merely because it is technologically sophisticated, newly developed, natural, nutritional, or unconventional.

The ultimate measure of medical progress is the meaningful benefit experienced by patients.

11. What This Means for Orthomolecular and Integrative Systems Medicine

The unresolved problems of modern oncology present important research opportunities for orthomolecular medicine.

For decades, orthomolecular medicine has emphasized the biological importance of nutritional adequacy, vitamins, minerals, and other essential molecular factors.

Contemporary systems biology creates opportunities to examine these issues within a broader physiological context.

Relevant research questions include how nutritional status, metabolic regulation, cellular energetics, redox signaling, immune function, organ reserve, and tissue repair influence treatment tolerance and recovery.

For the I-OM Systems Medicine approach, the objective is to understand the biological interactions among disease processes, modifiable physiological factors, conventional treatment, and patient outcomes.

This can include individualized consideration of metabolic health, nutritional adequacy, physical function, clinically significant exposures, and other relevant contributors.

Such factors must be evaluated according to their specific evidence base. Their biological plausibility alone does not establish that modifying them improves cancer survival.

Likewise, the rationale for an intervention must be accompanied by an appropriate assessment of clinical benefits, safety, and possible treatment interactions.

The goal is to build upon effective cancer treatment, not to bypass it.

Cancer is not merely a tumor to attack. It is also a disease-host system to understand and

manage.

That broader perspective may help identify new research priorities while reinforcing the importance of whole-person clinical care.

12. Advancing the Discussion at ICOM Algeria 2026

These concepts form the scientific foundation of my forthcoming presentation:

Beyond Tumor Starvation: The Disease-Host System in Cancer Medicine

International Conference on Orthomolecular Medicine (ICOM-ATU 2026)

October 27-29, 2026

Aïn Témouchent, Algeria

The presentation will examine the seven unresolved problems in contemporary oncology, the metabolic diversity of cancer, the limitations of generalized tumor starvation, and the emerging Disease-Host framework.

It will also introduce proposed research directions involving therapeutic selectivity, host physiology, and time-structured pharmacological ascorbate exposure.

The aim is to encourage serious scientific discussion-not to claim that these hypotheses have already been clinically validated.

[Official ICOM-ATU 2026 Conference Website](#)

[University Conference Announcement](#)

Conclusion: Beyond Tumor Control, Toward Better Lives

Contemporary cancer medicine has made extraordinary advances.

Those achievements should be preserved, strengthened, and extended.

But the next generation of progress requires more than increasingly sophisticated technologies and new therapeutic targets.

It requires greater attention to prevention, long-term benefit, treatment-related harm, physiological resilience, patient function, quality of life, and affordability.

It also requires a willingness to explore carefully testable scientific hypotheses that integrate malignant disease with the biology of the person experiencing it.

The proposed Disease-Host Dual-System Framework offers one possible way to organize that investigation.

Its value will depend on rigorous testing and demonstrable improvement in meaningful patient outcomes.

Weaken the disease. Preserve and strengthen the host. Improve the patient's life.

Selected References and Further Reading

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About the Author

Richard Z. Cheng, MD, PhD, is Editor-in-Chief of the Orthomolecular Medicine News Service and founder of I-OM.ai. His professional interests include Integrative Orthomolecular Systems Medicine, cancer metabolism, clinical nutrition, metabolic health, disease prevention, and healthy aging. His work emphasizes root-driver investigation, scientifically grounded integration of therapeutic approaches, and meaningful patient-centered outcomes.

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This article is intended for public and scientific education. It is not individualized medical advice.

The Disease-Host Dual-System Framework, Therapeutic Window Engineering, Cancer Progress Scorecard, and DP-HDIVC are proposed research frameworks or hypotheses and are not validated cancer-treatment protocols.

Pharmacological nutritional interventions, including high-dose intravenous vitamin C, may involve significant risks, contraindications, or interactions with cancer treatments. No DP-HDIVC regimen has established clinical efficacy.

Patients should make cancer-treatment decisions with qualified oncology professionals. Nutritional and metabolic interventions should not replace or delay effective evidence-based oncologic care.