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Beyond Tumor Starvation: Why Cancer Metabolic Therapy Must Also Strengthen the Host

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Editor's Note: This article is adapted from the author's preprint [1]:

Cheng RZ. The Biological Limits of Tumor Starvation: A Systems Physiology Perspective on Metabolic Cancer Therapy . **Preprints**. 2026;2026072145.

For more than a decade, I have **incorporated ketogenic metabolic** therapy into the care of cancer patients as part of an integrative oncology program. During that time, I have become increasingly convinced that cancer metabolism represents one of the most promising therapeutic targets in oncology.

Clinical experience, together with an expanding body of scientific literature, has reinforced my confidence that ketogenic diets and other metabolic interventions can reduce hyperinsulinemia, improve metabolic health, and create a physiological environment that is less favorable for many cancers. Used appropriately, these interventions can become valuable components of comprehensive cancer care.

I remain a strong supporter of ketogenic metabolic therapy.

Yet after treating many patients over more than ten years, I have also reached another conclusion that is equally important:

Metabolic therapy alone is not enough.

Ketogenic therapy is an important tool.

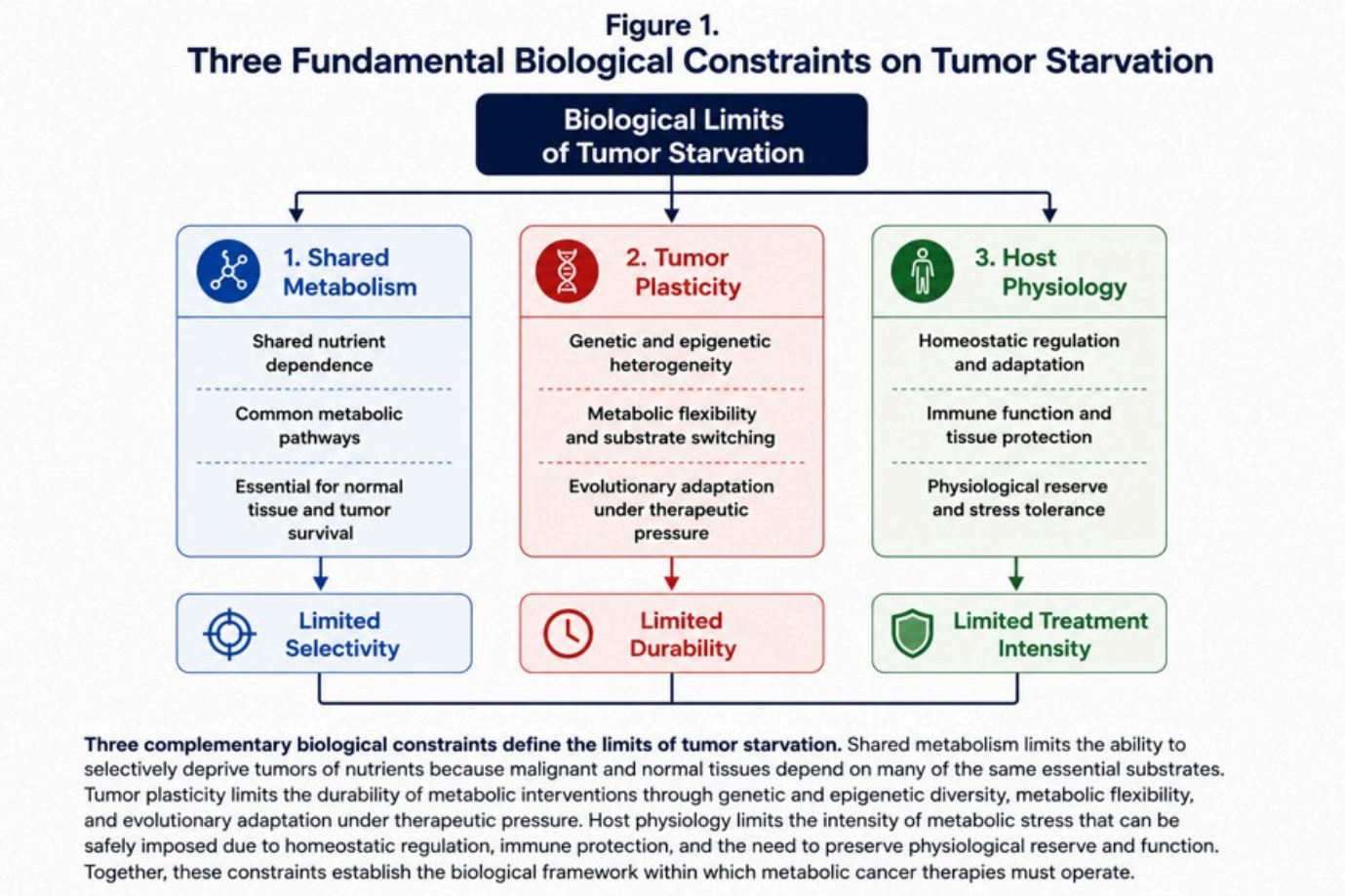
It is not the entire solution.

That realization gradually evolved into a series of publications that ultimately became the foundation of what we now call I-OM (**Integrative Orthomolecular Systems Medicine**).

The first paper, **Why Targeting Glutaminolysis Fails** [2], examined a specific example of why targeting an essential metabolic pathway inevitably affects both tumor and normal physiology.

The second paper, **The Disease-Host Dual-System Framework**, proposed that successful cancer therapy should optimize both the disease and the host rather than focusing exclusively on malignant cells.

The third paper, ***The Biological Limits of Tumor Starvation***, provides the physiological explanation for why this dual-system approach is necessary. It argues that the principal limitations of tumor starvation are not technological but biological.



Adapted from [1].

Why Tumor Starvation Has Biological Limits

The logic behind tumor starvation appears simple.

Cancer cells require nutrients.

Reduce nutrient availability. Slow tumor growth.

Indeed, decades of research have demonstrated that many cancers exhibit increased dependence on glucose, glutamine, and other metabolic substrates. This has generated enormous interest in ketogenic diets, fasting, glutamine restriction, and numerous metabolic therapies.

These approaches have genuine scientific merit.

However, human physiology is more complicated than the simple concept of starving cancer cells.

My recent review [3] identifies three fundamental biological constraints.

1. Tumors Share the Same Metabolism as the Host

Cancer cells are not foreign organisms.

They originate from normal human cells.

Consequently, they continue to depend upon many of the same nutrients required for normal physiology.

Glucose, amino acids, fatty acids, vitamins, and micronutrients support not only tumor growth but also immune cells, skeletal muscle, endocrine function, mitochondrial energy production, wound healing, and countless normal physiological processes.

The challenge therefore is not simply depriving tumors of nutrients.

It is doing so without simultaneously weakening the patient.

2. Cancer Constantly Adapts

Cancer is remarkably adaptable.

Restrict glucose, and some tumors increase glutamine utilization.

Restrict glutamine, and other metabolic pathways become more active.

Cancer cells recycle nutrients through autophagy, exchange metabolites with surrounding stromal cells, and continuously evolve under therapeutic pressure.

Tumors are moving targets.

This evolutionary plasticity explains why therapies directed at a single metabolic pathway often produce only temporary responses.

3. The Body Protects Itself

The third limitation comes from the patient.

Human physiology evolved to survive starvation.

When nutrients become scarce, the body activates complex hormonal, metabolic, mitochondrial, and immune responses designed to preserve life.

These adaptations help us survive.

They also define the biological limits of how aggressively nutrients can be restricted without harming the host.

Beyond a certain point, further metabolic stress may compromise immune competence, skeletal muscle, treatment tolerance, and physiological reserve more than it harms the cancer itself.

A Different Therapeutic Goal

Recognizing these limitations does not diminish the importance of ketogenic metabolic therapy.

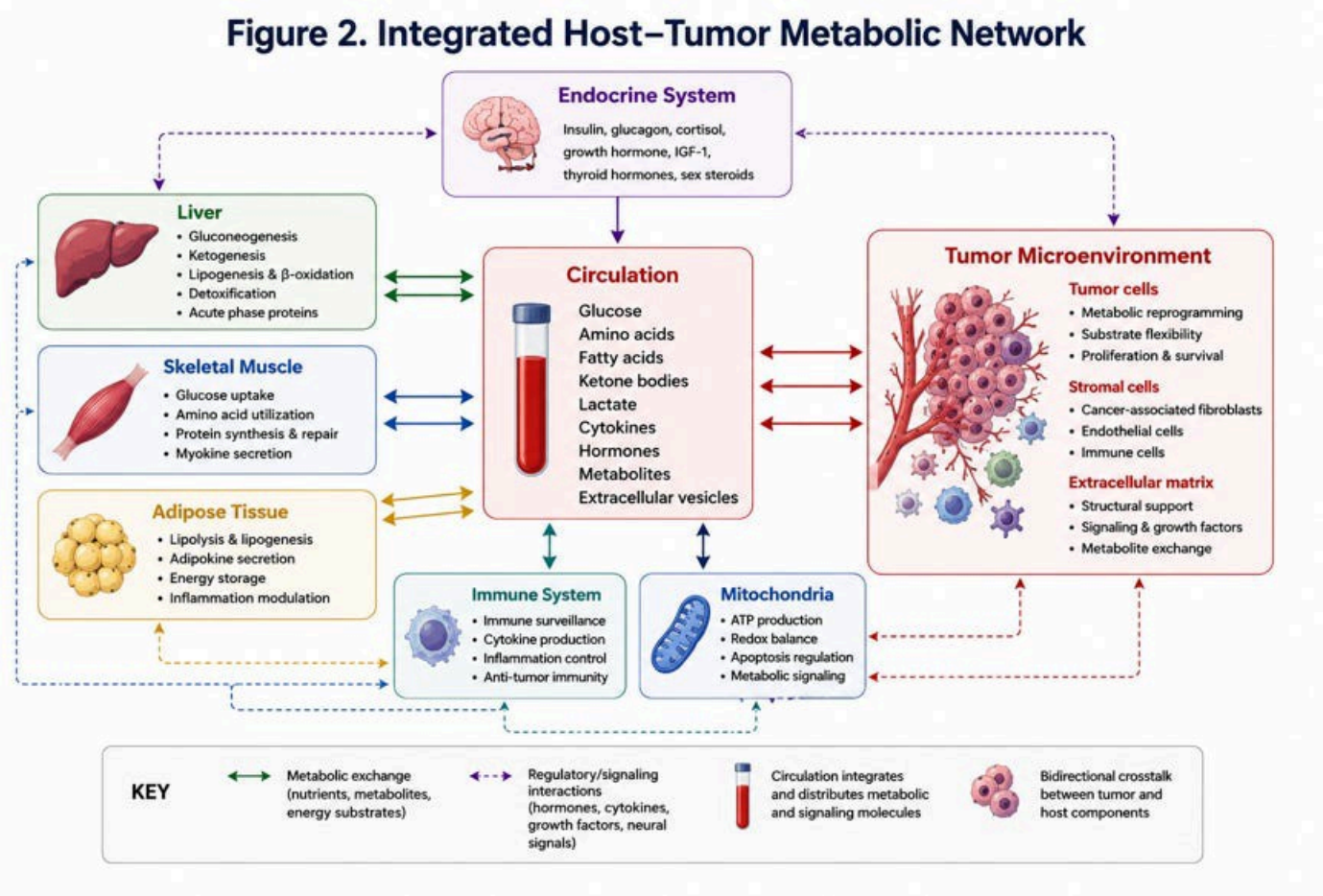
On the contrary, recognizing these limitations clarifies its proper role.

Rather than attempting to maximize tumor starvation, we should maximize the **therapeutic differential**-placing greater metabolic stress on cancer while preserving or strengthening the host.

This represents an important shift in perspective.

The objective is no longer simply starving tumors.

The objective is optimizing the host-tumor system.



Adapted from [1].

Three Pillars of the I-OM Cancer Framework

This systems perspective has gradually evolved into what we now call the **I-OM cancer framework**.

Rather than focusing exclusively on malignant cells, I-OM seeks simultaneously to reduce tumor fitness while improving host resilience.

Three foundational pillars support this approach.

Ketogenic Metabolic Therapy

Ketogenic therapy remains the cornerstone of metabolic intervention.

Reducing hyperinsulinemia, improving metabolic flexibility, and lowering systemic metabolic dysfunction can create a physiological environment less favorable for many cancers.

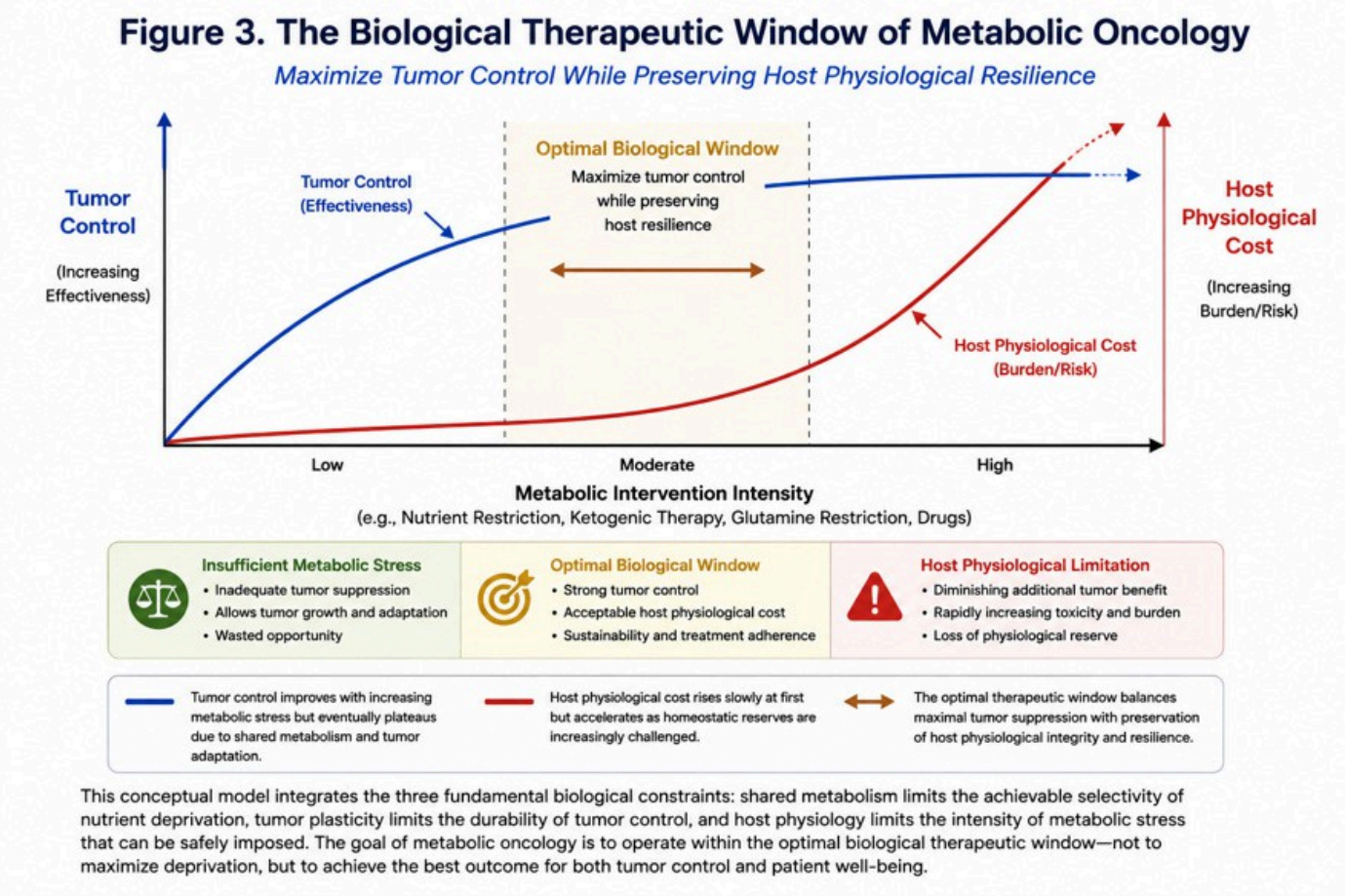
After more than ten years of clinical application, I continue to regard ketogenic metabolic therapy as an essential component of integrative cancer management.

High-Dose Intravenous Vitamin C

The second pillar is **high-dose intravenous vitamin C (HDIVC)**.

HDIVC has accumulated decades of clinical experience and growing scientific evidence supporting its use as an adjunctive therapy.

Within the I-OM framework, HDIVC complements metabolic therapy by helping support normal physiology while contributing additional metabolic stress to cancer cells.



Adapted from [1].

Host Optimization

The third pillar distinguishes I-OM from many tumor-centered approaches.

Cancer develops within a living host.

The patient's immune system, endocrine balance, mitochondrial function, nutritional status, inflammatory burden, vascular health, skeletal muscle, microbiome, sleep, stress physiology, and

metabolic resilience all influence outcomes.

Our objective is therefore not simply to attack cancer.

It is equally important to strengthen the patient.

Host optimization seeks to preserve physiological reserve, support immune competence, improve treatment tolerance, and enhance long-term resilience.

Beyond Tumor-Centered Oncology

Traditional oncology understandably concentrates on eliminating malignant cells.

Metabolic oncology has largely concentrated on depriving malignant cells of nutrients.

The next stage of metabolic oncology may require a broader systems perspective.

Cancer should be viewed as an interaction between tumor biology and host physiology.

Effective therapy depends on both suppressing malignant metabolism and preserving the adaptive capacity of the patient.

That philosophy forms the foundation of I-OM Systems Medicine.

Looking Forward

Cancer metabolism remains one of the most exciting frontiers in oncology.

I believe ketogenic metabolic therapy will become an increasingly important component of future cancer care. At the same time, our goal should not be ever more aggressive attempts to starve tumors.

This perspective is intended to complement-not replace-appropriately selected surgery, radiotherapy, systemic therapy, and other evidence-based cancer treatments.

Instead, future cancer care should integrate ketogenic metabolic therapy, high-dose intravenous vitamin C (HDIVC), host optimization, conventional oncology, and other evidence-informed approaches into comprehensive treatment programs that maximize tumor control while preserving the patient's biological integrity.

Ultimately, the future of cancer metabolic therapy lies not in starving the tumor alone, but in simultaneously weakening the cancer and strengthening the host.

That is the central message of my recent preprint, the underlying philosophy of the Disease-Host Dual-System Framework, and the guiding principle behind the I-OM approach to integrative cancer management.

Related Reading

This article is part of an ongoing series describing the development of the **I-OM** (**Integrative Orthomolecular Systems Medicine**) framework for cancer and chronic disease.

1. Cheng RZ. **The Biological Limits of Tumor Starvation: A Systems Physiology Perspective on Metabolic Cancer Therapy.** *Preprints*. 2026;2026072145. https://doi.org/10.20944/preprints202607.2145.v1

2. Cheng RZ. **Why Targeting Glutaminolysis Fails: The Cost of Disrupting Essential Physiology.** *Orthomolecular Medicine News Service (OMNS)*. 2025;21(68). Available at: https://orthomolecular.org/resources/omns/v21n68.shtml

3. Cheng RZ. **The Disease-Host Dual-System Framework: A Systems Architecture for Cancer and Chronic Disease.** *Orthomolecular Medicine News Service (OMNS)*. Available at: https://orthomolecular.org/resources/omns/v22n37.shtml

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