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Dual-Pulse High-Dose Intravenous Vitamin C (DP-HDIVC): A Time-Structured Oxidative-Reparative Framework Targeting Tumor Redox Vulnerability

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High-dose intravenous vitamin C (HDIVC) has been investigated for decades as a potential anticancer therapy based on its ability to achieve **pharmacologic plasma concentrations** that generate extracellular hydrogen peroxide (H_2O_2) in the tumor microenvironment. This oxidative stress selectively injures cancer cells, which typically operate near the limits of redox tolerance, while sparing normal tissues with greater antioxidant buffering capacity.

It is well established that the anticancer activity of pharmacologic ascorbate **requires sufficiently high peak plasma concentrations**. This principle is non-controversial. However, while dose escalation and safety have been extensively studied, **treatment frequency and temporal patterning** have historically received far less biologically driven optimization.

Most conventional HDIVC protocols have been administered **two to three times weekly**, a schedule shaped largely by outpatient feasibility, logistical convenience, and early safety considerations-rather than by systematic analysis of **tumor redox recovery dynamics**.

A newly released preprint introduces a **dual-pulse high-dose intravenous vitamin C framework (DP-HDIVC)** that seeks to address this gap by optimizing **when and how often pharmacologic oxidative stress is delivered**, while fully preserving the requirement for adequate peak dosing.

Peak Dose Is Necessary - Frequency and Timing Are the Missing Variables

Cancer cells exist in a state of chronically elevated oxidative stress and altered redox homeostasis, rendering them vulnerable to additional oxidative insults. At the same time, they retain inducible antioxidant and metabolic recovery systems that allow **partial redox restoration** following transient stress.

Conventional HDIVC protocols typically deliver a single high-dose oxidative insult, followed by several days of recovery before the next infusion. While peak plasma concentration is sufficient to induce cytotoxic stress, **long inter-infusion intervals** may allow surviving tumor cells to recover, adapt, and repopulate.

From a systems-biology perspective, this highlights a critical but underappreciated issue: if tumor redox recovery occurs on the scale of hours to days, then treatment schedules determined primarily by clinical practicality may fail to exploit a key biological vulnerability-the **limited capacity of cancer cells to repeatedly recover from suprathreshold oxidative injury**.

DP-HDIVC does not question the importance of peak dose. Rather, it identifies **frequency and temporal structuring** as historically constrained by logistics rather than biology.

The DP-HDIVC Concept: Dual Oxidative Peaks with Structured Redox Recovery

At the core of the DP-HDIVC framework is **temporal patterning**, not continuous oxidative pressure.

DP-HDIVC is designed around the intentional delivery of **two pharmacologic oxidative peaks within a defined supra-oxidative phase**, typically separated by approximately **12 hours (q12h)**. Each infusion is intended to achieve **pharmacologic peak plasma concentrations** of ascorbate known to be required for cytotoxic oxidative stress in cancer cells.

The purpose of the **dual-peak strategy** is to expose tumor cells to **repeated suprathreshold oxidative injury before full redox recovery can occur**, thereby increasing cumulative cellular damage and limiting adaptive escape.

Crucially, these oxidative peaks are **not delivered continuously**. They are followed by a **structured reparative window**, during which therapeutic intent shifts from tumor-directed oxidative injury to **host-directed recovery and resilience**.

During this recovery phase, administration of essential vitamins, micronutrients, and selected redox-supportive or antioxidant agents-such as **N-acetylcysteine (NAC) or alpha-lipoic acid (ALA)**-may be deliberately employed to support normal tissue repair, mitochondrial function, and systemic redox homeostasis.

In this way, DP-HDIVC represents a **cyclic redox strategy** that deliberately alternates between:

- **Supra-oxidative phases** targeting tumor redox vulnerability, and
- **Reparative phases** supporting host recovery and physiological stability.

This **temporal separation** of oxidative injury and antioxidant support-rather than their simultaneous administration-is a central mechanistic principle of the DP-HDIVC framework.

Clinical Translation and Next Steps

DP-HDIVC does not claim clinical proof. Instead, it provides a **mechanistically grounded, hypothesis-driven framework** that generates testable predictions regarding the role of treatment timing and frequency in pharmacologic ascorbate therapy.

Protocol development based on this framework is being explored for **feasibility assessment and structured clinical evaluation**, including **planned implementation within an oncology center in China under appropriate clinical oversight**. These efforts are intended to inform formal study design and hypothesis testing, rather than to substitute for controlled clinical trials.

Publication Status

The full manuscript describing the DP-HDIVC framework is available as a **public preprint** and has been **submitted to the *International Journal of Molecular Sciences (IJMS)* for peer review**.

As with all preprints, the work is shared to encourage scientific discussion, critique, and refinement while formal peer review is underway.

Why This Matters

DP-HDIVC aligns with modern **systems-level views of cancer metabolism and redox biology**, in which therapeutic efficacy depends on **dynamic perturbation of adaptive tumor networks**, not static dose escalation alone.

By clarifying that **peak dose is necessary but not sufficient**, and by explicitly identifying **frequency and temporal structuring** as biologically meaningful yet historically under-optimized variables, this framework reopens an important line of inquiry in pharmacologic ascorbate research and provides a coherent foundation for future investigation.

Integration Within Integrative Orthomolecular Cancer Therapy (IOCT)

The DP-HDIVC framework described here is not intended as an isolated intervention, but rather as one component within a broader **Integrative Orthomolecular Cancer Therapy (IOCT)** paradigm. This systems-based approach emphasizes metabolic dysfunction, mitochondrial impairment, redox

imbalance, and toxicant exposure as central drivers of cancer biology, extending beyond a purely mutation-centric view of oncogenesis.

The conceptual foundations of this approach have been articulated in detail elsewhere, including in *Cheng, R. "From Mutation to Metabolism: Toxins, Mitochondria, and Integrative Orthomolecular Cancer Therapy (IOCT)-Implications for ASCVD and T2DM"* (Preprints 2025, 2025101142). Within this framework, pharmacologic ascorbate-based redox modulation represents a **targeted metabolic and redox intervention**, designed to exploit tumor vulnerability while remaining compatible with broader host-directed restoration strategies.

Ongoing development of cancer-focused IOM protocols will therefore build upon the principles outlined in both works, integrating **metabolic therapy, redox timing, micronutrient repletion, toxicant reduction, and mitochondrial support** into a coherent, systems-level strategy. As with DP-HDIVC itself, these protocols are intended to inform structured clinical evaluation rather than to substitute for controlled clinical trials.