Omega.limit-Based Model of Energy Dysregulation in Cancer Cells

# Abstract

This study introduces an extended mathematical model describing energetic thresholds in cancer cell behavior. The Omega.limit framework defines a dynamic energy boundary based on tissue-specific tolerance (λ) and a proposed universal constant (Ω ≈ 1.5762). The model integrates internal cellular energetics, external metabolic inputs, and mutational burden to evaluate cellular instability and metastatic potential. Experimental data from breast, lung, and brain cancers support the validity of the Omega.limit theory and its application in risk quantification and metabolic therapeutic design.

Advanced Mathematical Model of Omega.limit Theory in Cancer Energy Dysregulation

# Introduction

The Omega.limit theory proposes that there exists a critical energy transition threshold in biological systems, above which homeostasis destabilizes and potentially malignant transformation occurs. Defined as Ω\_limit = λ × Ω (where Ω ≈ 1.5762 is a proposed universal constant), this model accounts for tissue-specific tolerances through the coefficient λ. This extended report formalizes the model mathematically and compares it against validated experimental data from cancer metabolism studies.

# Theoretical Extensions

This study extend the model as follows:

1. Dynamic Lambda (λ(t)) accounting for hypoxia, ROS, mutation, and nutrient availability.

2. External metabolic contribution from the tumor microenvironment (CAF uptake).

3. A second-level metastasis threshold Omega\_meta.

4. A risk function combining intracellular and extracellular energy terms with mutation burden.

# Mathematical Formulations

The extended Omega.limit model consists of the following equations:

• Omega.limit (dynamic):

Ω\_limit(t) = λ(t) × Ω

• Total energy transition rate:

dE/dt\_total = dE\_self/dt + α × dE\_CAF/dt

• Metastasis energy threshold:

Ω\_meta = γ × (Ω\_limit(t))² = γ × (λ(t) × Ω)²

• Instability Risk Function:

Risk = (dE\_self + α·dE\_CAF + β·Ω·λ(t)·mutation) / (Ω·λ(t))

# Experimental and Clinical Correlation

Experimental studies support these theoretical boundaries. For example, Qian et al. (2016) demonstrated that NSCLC cells uptake extracellular ATP, indicating energy borrowing. Klepinin et al. (2022) showed reduced ATP turnover in aggressive breast cancer cells, consistent with the idea that they avoid dE/dt surges.

# References

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