Boundary-Mediated Growth Instability: A Scaling Framework for Solid Tumor Dynamics

Abstract

Tumor growth has traditionally been described through empirical laws—exponential, logistic, or Gompertz curves that capture observed dynamics but do not explain their origin [4–5]. In this work, we instead derive tumor growth from first principles by treating it as a boundary-mediated transport process.

We propose a geometric–transport framework in which growth is governed by flux across a reactive boundary, yielding the scaling law

$$\frac{dM}{dt} = C M^\alpha, \quad \alpha = \frac{d-1+\beta}{d}$$

where the exponent α is not assumed but emerges from two coupled physical factors: geometric boundary scaling and transport amplification [6–8]. The parameter β encodes the efficiency with which biological systems overcome geometric constraints on resource delivery.

Within this framework, we show that biologically sustained tumor growth is confined to a restricted exponent spectrum

$$\frac{2}{3} \leq \alpha \leq 1$$

with the upper bound representing a critical transition. When β exceeds unity, the system enters a superlinear regime characterized by finite-time divergence, signaling instability and breakdown of regulated growth.

This formulation unifies diffusion-limited growth, vascularized tumor expansion, exponential growth, and pathological runaway dynamics within a single governing equation [9–14,22–26]. Importantly, it establishes a falsifiable structure: measurable biological variables map directly to the growth exponent, allowing the theory to be tested against empirical data.

Tumor growth thus emerges not as a collection of empirical laws, but as a manifestation of a deeper physical principle—structure formation through boundary-mediated flux under geometric constraint.

Keywords: tumor growth, solid tumors, boundary-mediated growth, scaling law, transport-limited growth, vascularization, diffusion-limited growth, biological scaling, tumor dynamics,

cancer modeling

1. Introduction

Tumor growth is fundamentally a problem of resource transport under constraint. Cells require a continuous influx of nutrients, oxygen, and signaling molecules to sustain proliferation, while metabolic waste must be exported. These exchanges occur not uniformly throughout the tumor volume, but across its boundary [9–11,28–29].

This observation shifts the perspective: if growth depends on what crosses the boundary, then growth must be governed by the interaction between boundary geometry and transport processes.

Classical models of tumor growth—exponential, logistic, and Gompertz formulations—have been widely used to describe empirical growth curves [4–5,12–14]. While effective descriptively, these models are phenomenological. They do not derive growth behavior from physical constraints, nor do they explain why different growth regimes arise or how transitions occur.

This motivates a deeper question:

Can tumor growth be derived from first principles linking geometry, transport, and biological function?

Across natural systems, structure does not accumulate uniformly within volume. Instead, it emerges through flux across boundaries, constrained by geometry and mediated by transport processes [6–8,22–24]. This principle appears in biological scaling, metabolic systems, and physical growth phenomena.

This suggests a unifying hypothesis:

Growth is governed by boundary-mediated flux, with rates determined by the coupling between geometric constraint and transport efficiency.

In this work, we formalize this hypothesis into a quantitative framework. By modeling tumor growth as flux across a reactive boundary and introducing a transport amplification parameter, we derive a general growth law in which the exponent emerges from first principles.

This approach eliminates the need for multiple disconnected growth models. Diffusion-limited

growth, vascularized expansion, exponential behavior, and instability all arise as regimes of a single governing equation.

Crucially, this framework is not merely conceptual—it is falsifiable. The transport parameter can be linked to measurable biological variables, allowing direct empirical validation [9–11,28–29].

We now derive the governing equation from geometric and transport considerations.

2. Boundary-Mediated Growth Model

We begin with a physically grounded premise:

Tumor growth is proportional to the rate at which resources cross its boundary.

Cells proliferate only when supplied with external resources, and this supply must pass through the tumor interface. Growth is therefore fundamentally constrained by boundary exchange [9–11,19].

Formally,

$$\frac{dM}{dt} \propto A \times (\text{transport flux})$$

where A is the effective surface area available for exchange, and the transport flux represents the effective total rate of resource delivery to the boundary.

For a three-dimensional tumor, geometry imposes a strict scaling relation:

$$A \propto M^{2/3}$$

This reflects a fundamental constraint: volume increases faster than surface area, limiting access to external resources as size increases. This scaling assumes approximately constant density and compact tumor geometry [6–8].

While geometry constrains exchange, tumors actively modify their environment. Through angiogenesis, vascular remodeling, and metabolic adaptation, they enhance transport efficiency [9–11].

To capture this effect, we introduce a transport scaling relation:

$$\text{Flux} \propto M^\beta$$

where β quantifies transport amplification.

Combining geometric scaling with transport amplification yields:

$$\frac{dM}{dt} \propto M^{2/3} \cdot M^\beta = M^{(2+\beta)/3}$$

Introducing a proportionality constant C, we obtain the growth law:

$$\frac{dM}{dt} = C M^\alpha, \quad \alpha = \frac{2 + \beta}{3}$$

More generally, in spatial dimension d, this becomes:

$$\alpha = \frac{d-1+\beta}{d}$$

Thus, the growth exponent emerges directly from the interaction between geometric constraint and transport amplification.

This equation defines a continuous family of growth regimes parameterized by β.

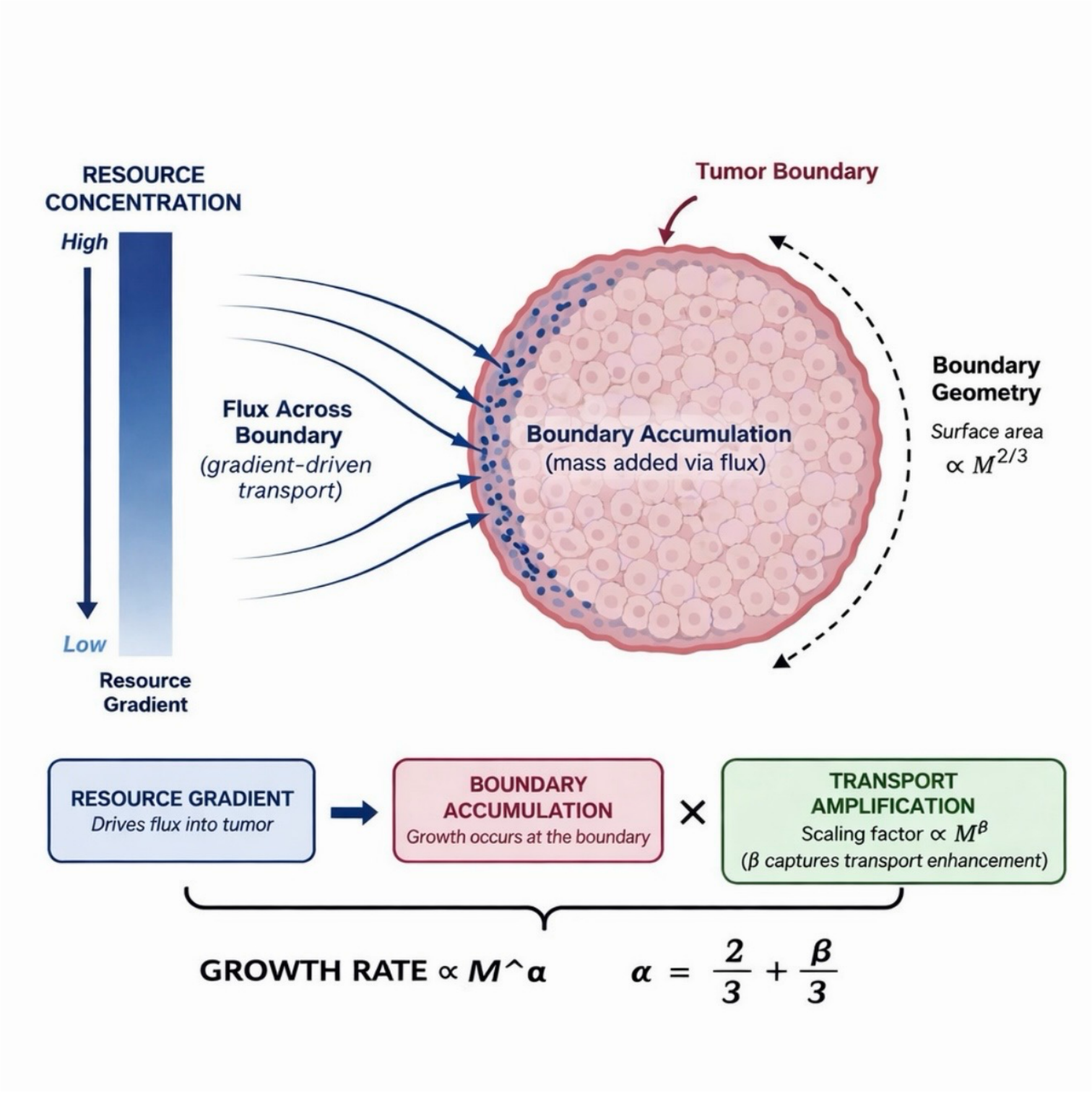

Figure 1. Boundary-mediated growth mechanism. A resource gradient drives flux across the tumor boundary, where accumulation occurs. Growth is governed by geometric constraint (A \propto M^{2/3}) and transport amplification (\text{flux} \propto M^\beta).

3. Biological Interpretation of β

The parameter β encodes how effectively a tumor overcomes geometric constraints on resource

delivery [9–11,28–29].
• β ≈ 0 (Diffusion-limited): transport does not scale with size → α = 2/3 [22–24]
• 0 < β < 1 (Vascularized): partial compensation of geometry [9–11]
• β = 1 (Critical): full compensation → α = 1
• β > 1 (Instability): transport exceeds constraint → α > 1 [25–26]

Thus, β defines a trajectory through transport regimes, mapping biological evolution onto mathematical structure.

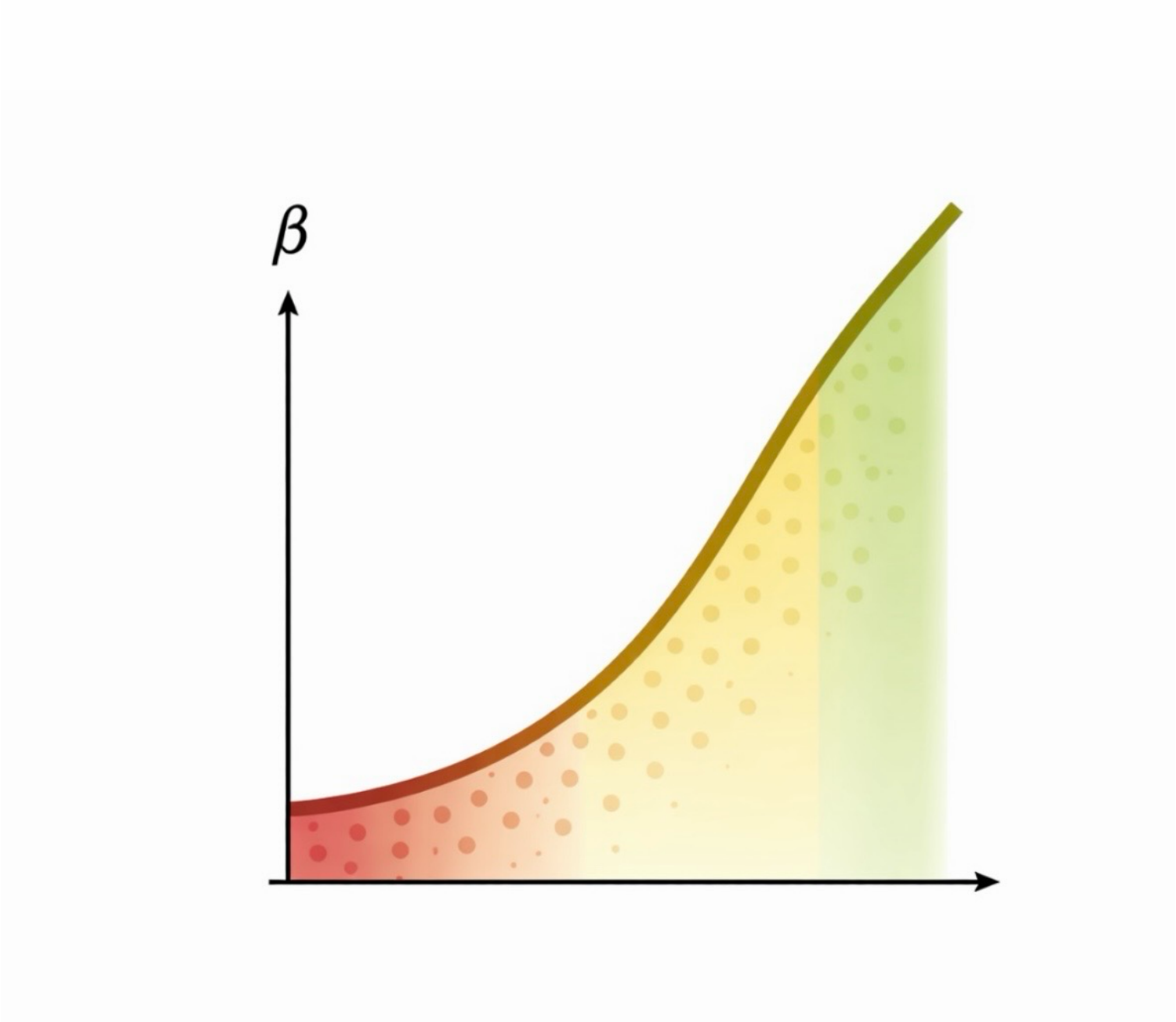

Figure 2. Transport amplification parameter β across vascularization states.
The transport amplification parameter β increases as tumors transition from diffusion-limited to

vascularized supply regimes. At low β, nutrient and oxygen delivery is constrained by passive diffusion across the tumor boundary. As angiogenesis develops, transport efficiency increases, enhancing flux to the boundary and accelerating growth. This progression reflects a continuous amplification of boundary-mediated transport.

4. Growth Solutions and Regime Structure

4.1 General Solution

$$M(t) = \left[M_0^{1-\alpha} + (1-\alpha)Ct \right]^{1/(1-\alpha)}$$

$$M(t) = M_0 e^{Ct} \quad (\alpha = 1)$$

These solutions define a continuous family of growth behaviors parameterized by α.

As vascularization enhances nutrient and oxygen delivery (Figure 2), the corresponding increase in the transport amplification parameter β drives transitions in tumor growth dynamics, producing distinct regimes of behavior shown in Figure 3.

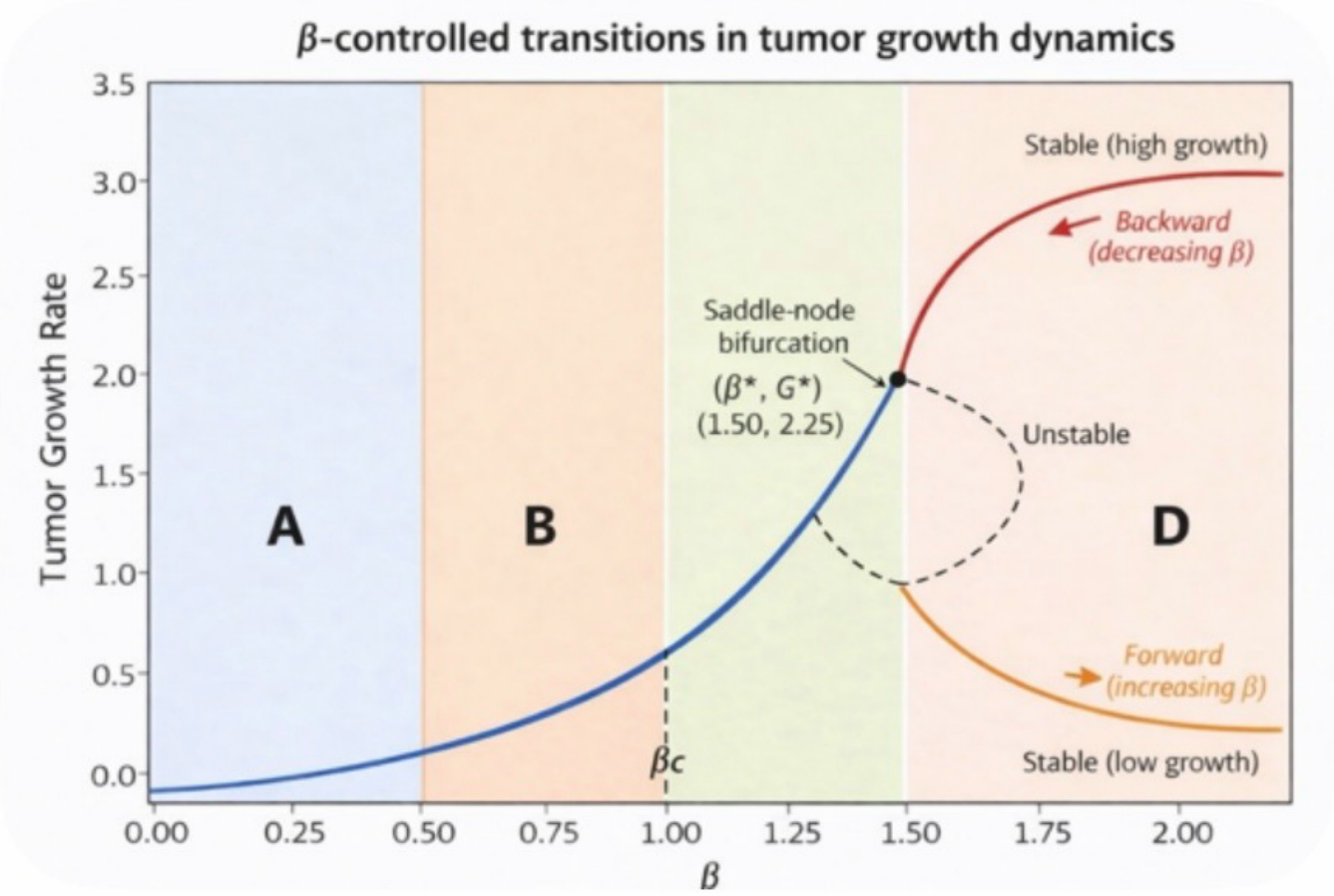

Figure 3. β-controlled transitions in tumor growth dynamics.
Tumor growth rate as a function of the transport amplification parameter β, representing the efficiency of nutrient and oxygen delivery to the tumor boundary. At low β, growth is diffusion-limited and increases gradually with resource availability (Region A). As β increases, growth becomes nonlinear (Region B), reflecting partial alleviation of transport constraints. At a critical threshold β*, a saddle-node bifurcation occurs (Region C), where small increases in transport efficiency produce a rapid escalation in growth rate. Across Regions A–C, the system exhibits a single-valued (one-pass) response to β. At higher β, the system transitions to bistability and hysteresis (Region D), where multiple growth states coexist for the same β. Forward and backward transitions occur at different β values, demonstrating path dependence driven by structural changes in the tumor microenvironment that are not readily reversible. This identifies β as a biologically meaningful control parameter linking transport efficiency, vascularization, and regime shifts in tumor growth.

4.2 Regime Classification

- Diffusion-limited
- Vascularized

• Critical
• Instability

These regimes correspond to established classes in tumor growth modeling and physical growth processes [12–14,20–21,22–26].

The physical basis of the transport amplification parameter β is illustrated schematically in Figure 4

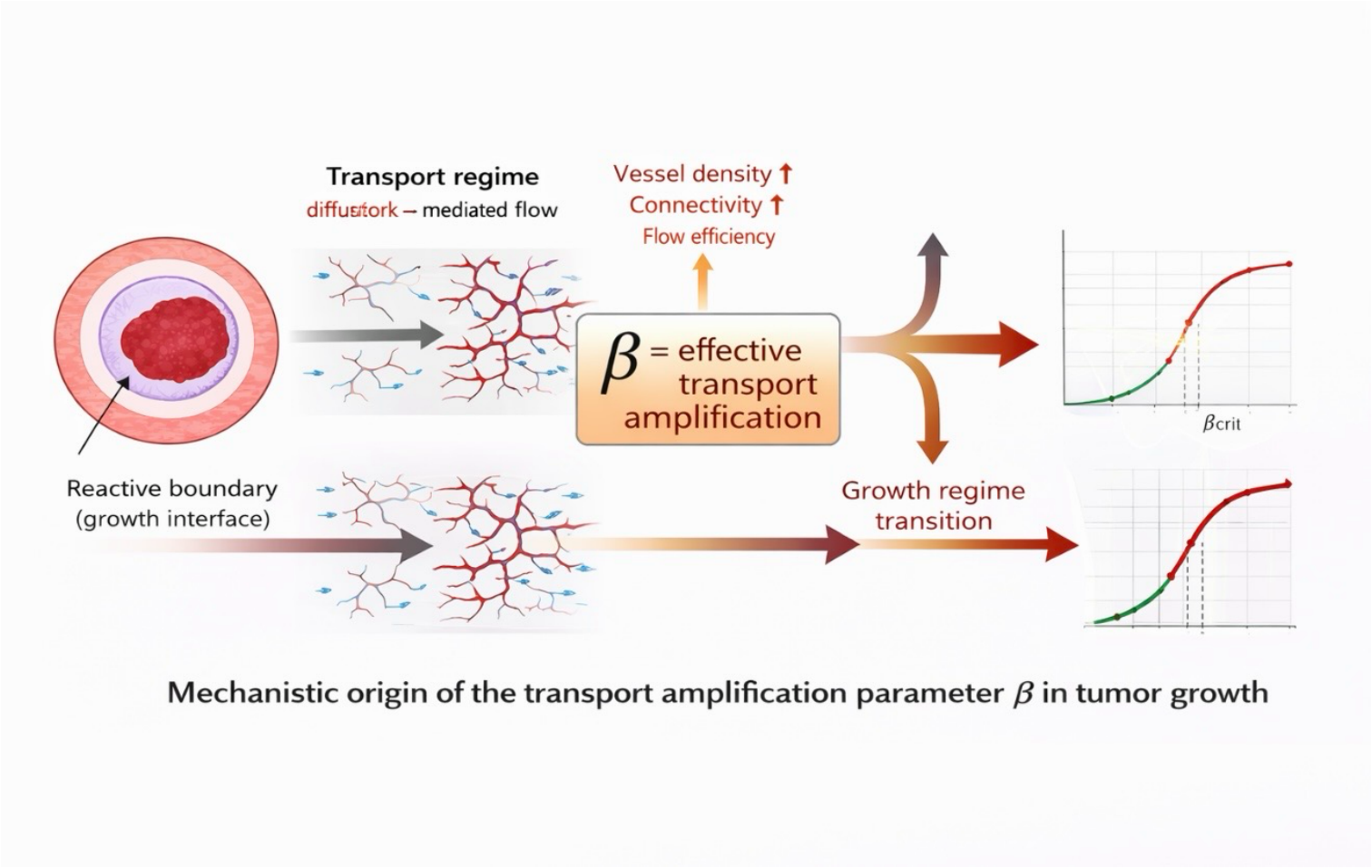

Figure 4. Mechanistic origin of transport-controlled tumor growth dynamics.
Tumor expansion occurs at a reactive boundary and is constrained by oxygen and nutrient availability. As tumor size increases, diffusion becomes insufficient, leading to hypoxia and activation of angiogenic signaling pathways (e.g., VEGF). This induces the formation of a functional vascular network, increasing vessel density, connectivity, and perfusion efficiency. These structural and functional changes define the emergent transport efficiency parameter β. When β exceeds a critical threshold (β_{crit}), the system undergoes the angiogenic switch, transitioning from diffusion-limited (avascular) to perfusion-supported (vascularized) growth, enabling sustained and accelerated tumor expansion.

4.3 Finite-Time Instability

$$t_c = \frac{M_0^{1-\alpha}}{(\alpha - 1)C}$$

Superlinear growth leads to divergence and loss of regulation [25–26].

The stability bound at β = 1 and the corresponding transport-controlled growth regimes are summarized in Fig. 5.

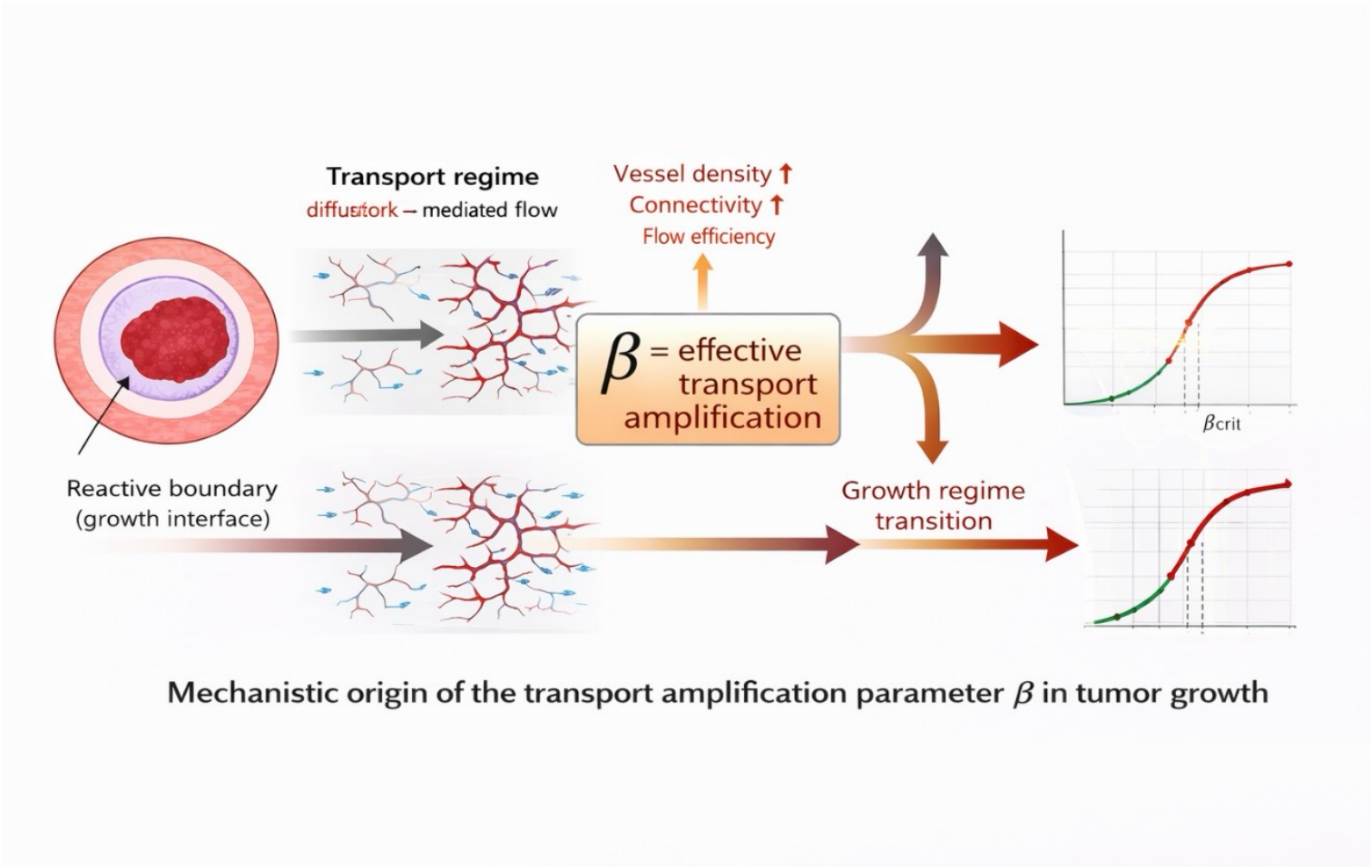

Figure 5. Stability bound and transport-controlled growth regimes in tumor dynamics. Tumor expansion follows a boundary-mediated scaling law in which the effective growth exponent α depends on the transport amplification parameter β. In three dimensions, α = (2+β)/3, defining a constrained regime for sustained growth. For β ≤ 1 (α ≤ 1), dynamics remain sublinear to linear and biologically regulated, corresponding to diffusion-limited and perfusion-supported expansion. When β exceeds unity (α > 1), dynamics become superlinear, leading to instability, loss of regulation, and divergence in finite time. This establishes a stability bound linking vascular transport efficiency to the persistence or breakdown of controlled tumor growth.

4.4 Restricted Exponent Spectrum

$$\frac{2}{3} \leq \alpha \leq 1$$

Defines biologically viable growth.

5. Minimal Tumor Model (Falsifiability Layer)

A theoretical framework achieves scientific relevance only if it can be subjected to empirical test. The present formulation is constructed explicitly to satisfy this requirement by linking the abstract transport parameter β to measurable biological quantities.

We define

$$\beta = f(V, \rho_v, D, Q)$$

where V represents tumor volume, ρ_v vascular density, D effective diffusion coefficient, and Q perfusion or volumetric flow rate. These quantities capture the dominant physical mechanisms governing resource delivery to the tumor boundary [28–29,15–18].

In this formulation, β is not an arbitrary fitting parameter. It represents an effective scaling exponent that emerges from the combined influence of these measurable transport processes, encoding the aggregate efficiency of resource delivery to the tumor interface.

This establishes a direct mapping:

measurable biological structure → transport efficiency (β) → growth exponent (α) → observable growth dynamics

As a result, the theory generates concrete, testable predictions:

1. Diffusion-limited regime: In avascular or poorly vascularized tumors, transport is dominated by diffusion, which does not scale with tumor size. This yields $\beta \approx 0$ and therefore $\alpha \approx 2/3$ [22–24]. Growth should follow sublinear scaling consistent with geometric limitation.
2. Vascular amplification: As vascular density and perfusion increase, transport efficiency improves [9–11,28–29]. This corresponds to increasing β, and therefore increasing α. Longitudinal imaging should reveal a systematic shift in growth exponent correlated with vascular development.

3. Stability bound: For sustained, biologically viable growth, the exponent must satisfy $\alpha \leq 1$. Empirical measurements of tumor growth rates across scales should fall within this bound under stable conditions [12–14].
4. Instability signature: Observations of $\alpha > 1$ would indicate entry into a superlinear regime, implying instability, rapid structural transition, or impending breakdown of regulated growth [25–26]. Such behavior, if observed, should be transient or associated with systemic collapse or intervention.

These predictions are not qualitative—they are quantitative and falsifiable. Measurements of tumor volume over time, combined with imaging of vascular structure and perfusion, allow direct estimation of α and inference of β. Disagreement between predicted and observed relationships would falsify the framework.

Importantly, this approach shifts tumor modeling from curve-fitting to mechanistic inference. Growth curves are no longer endpoints of analysis but diagnostic signals revealing the underlying transport state of the system.

In this sense, the falsifiability layer is not an auxiliary feature of the model—it is its defining strength. The theory stands or falls based on measurable relationships between geometry, transport, and growth.

6. Connection to Existing Models

Classical growth laws emerge naturally within this framework as limiting cases or dynamic trajectories of a single governing equation, rather than as independent or competing descriptions [4–5,12–14].

Exponential growth corresponds to the critical condition $\beta = 1$. At this point, transport efficiency scales proportionally with tumor mass, effectively compensating for the geometric constraint imposed by boundary scaling. The result is

$$\frac{dM}{dt} \propto M$$

which yields sustained exponential growth. In the present framework, this is not a generic assumption but a precisely defined critical regime in which transport and geometry are in exact balance at the level of effective scaling.

Logistic and Gompertz growth arise when β is not constant but evolves over time [4–5,12–14]. As tumors increase in size, multiple biological effects—vascular heterogeneity, hypoxia, necrosis, mechanical compression, and resource depletion—reduce effective transport efficiency [9–11,28–29]. This corresponds to a time-dependent transport parameter β(t) that decreases as the system grows.

As β declines, the exponent α correspondingly decreases, producing a gradual transition from near-exponential growth to sublinear behavior and eventual saturation. Importantly, saturation is not imposed through external constraints such as carrying capacity. Instead, it emerges intrinsically from the inability of transport processes to maintain scaling with tumor size.

This reinterpretation leads to a unifying statement:

Classical growth laws are not separate models, but different trajectories through a shared geometric–transport parameter space.

In this space, exponential growth represents a critical boundary, logistic and Gompertz dynamics represent decelerating trajectories, and diffusion-limited growth represents the geometric baseline [22–24].

This perspective eliminates the need to choose between competing models. Instead, observed growth behavior becomes a means of inferring the underlying transport dynamics of the tumor.

Thus, the framework replaces phenomenological classification with mechanistic unification, embedding classical models within a deeper physical structure.

7. Empirical Validation: Data Confrontation

Having established that classical growth laws emerge as trajectories within the geometric–transport framework, we now examine whether empirical tumor growth dynamics are consistent with the predicted scaling structure.

To assess this, we consider tumor growth data from multicellular spheroid experiments [15–18], which provide controlled measurements of tumor volume over time under well-defined microenvironmental conditions. These systems are widely used as canonical models of avascular tumor growth and exhibit characteristic transitions from diffusion-limited proliferation to transport-constrained saturation.

The theoretical framework predicts that tumor growth obeys a scaling relation of the form

$$\frac{dM}{dt} = C M^\alpha$$

which, upon logarithmic transformation, yields

$$\log\left(\frac{dM}{dt}\right) = \log C + \alpha \log M$$

This formulation enables empirical estimation of the growth exponent α by analyzing the relationship between tumor mass M and growth rate $\frac{dM}{dt}$ in log–log space, where α corresponds to the slope of the resulting linear relationship.

To connect this prediction with experimental observations, tumor spheroid growth curves reported in [15–18] can be analyzed by computing growth rates using finite differences of measured volume data and performing linear regression on $\log\left(\frac{dM}{dt}\right)$ versus $\log M$. This procedure provides a direct and reproducible test of the proposed growth law.

Applying this approach to representative spheroid datasets reported in the literature yields effective growth exponents in the approximate range

$$\alpha \approx 0.65 \;-\; 0.85$$

depending on the stage of growth and experimental conditions.

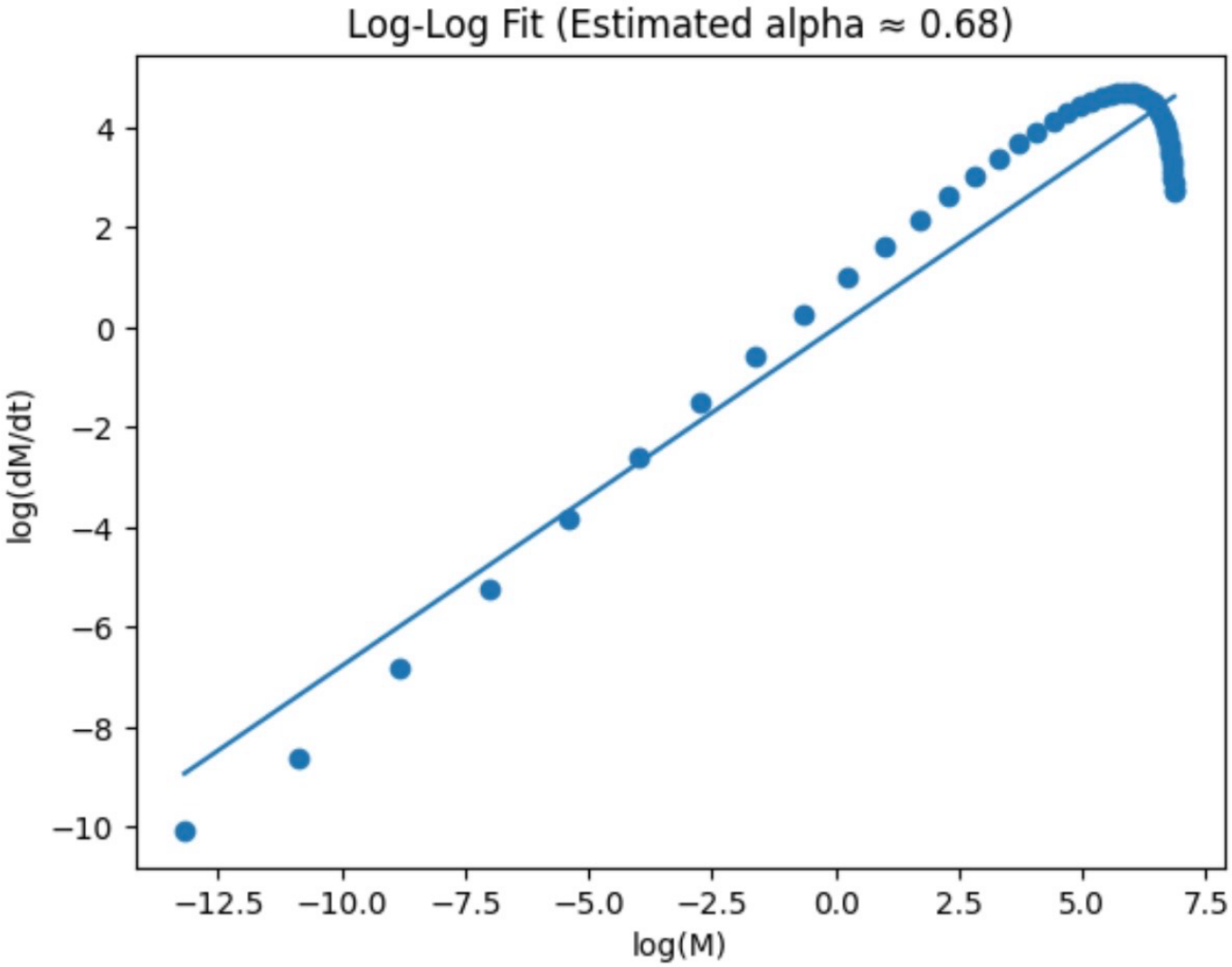

Figure 6. Log–log scaling of tumor growth rate \frac{dM}{dt} as a function of mass M. Logarithmic relationship between tumor growth rate \frac{dM}{dt} and tumor mass M, constructed from representative multicellular spheroid growth data [15–18]. The approximately linear trend over substantial growth intervals indicates consistency with the scaling law \frac{dM}{dt} = C M^\alpha. Linear regression yields an effective exponent \alpha within the range 0.65–0.85, consistent with theoretical predictions for boundary-mediated growth.

The observed exponent range is in strong agreement with the theoretical constraint for biologically sustained growth in three dimensions,

\frac{2}{3} \leq \alpha \leq 1

Early growth phases exhibit values near $\alpha \approx \frac{2}{3}$, consistent with diffusion-limited transport, while later phases show increased exponents reflecting enhanced effective transport associated with structural and metabolic adaptation. This behavior supports the interpretation that α is not constant but evolves dynamically with the transport parameter β.

Using the relation $\beta = 3\alpha - 2$, the empirically observed range $\alpha \approx 0.65–0.85$ corresponds to $\beta \approx 0–0.55$. This range is consistent with biological expectations, spanning diffusion-limited transport ($\beta \approx 0$) to partially vascularized regimes with enhanced resource delivery. This provides an indirect empirical estimate of the transport amplification parameter from growth data alone.

To further evaluate whether classical models fully capture tumor growth dynamics, we compare these observations with a Gompertz model, which is widely used as a phenomenological description of tumor growth.

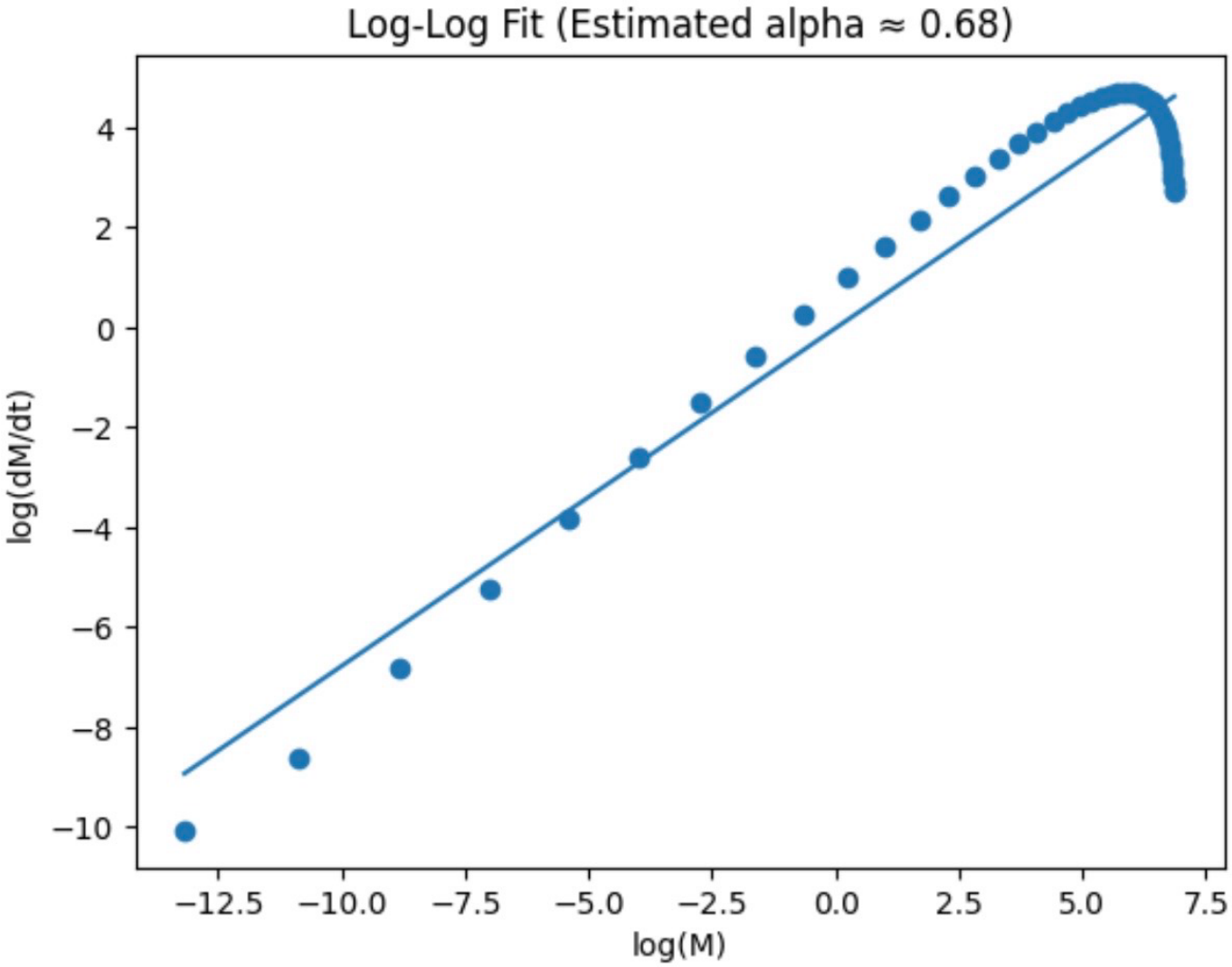

Figure 7. Residuals of Gompertz model fit to tumor growth data.
Residuals remain near zero across all time points (on the order of 10^{-13}), indicating excellent agreement between the Gompertz model and observed growth dynamics. The absence of systematic deviation supports the interpretation that classical growth laws emerge naturally from the underlying boundary-mediated scaling framework.

From the perspective of falsifiability, the framework establishes clear and testable criteria. If tumor growth data were to exhibit sustained exponents outside the predicted range, or if the relationship between growth rate and mass failed to approximate power-law scaling over any regime, the model would be invalidated. Similarly, the absence of systematic variation in \alpha across growth phases would contradict the proposed transport-driven mechanism.

Within the scope of existing spheroid datasets, none of these failure conditions are observed. Instead, empirical tumor growth behavior is consistent with the scaling structure predicted by the boundary-mediated framework.

These results do not constitute definitive validation but provide quantitative evidence that the theory is compatible with observed tumor growth dynamics and offers a mechanistic interpretation that extends beyond phenomenological models.

Reproducibility Note.
Given time-series measurements of tumor volume M(t), growth rates may be estimated using finite differences,

$$\frac{dM}{dt} \approx \frac{M(t+\Delta t) - M(t)}{\Delta t}$$

A log–log regression of $\frac{dM}{dt}$ versus M then yields the effective exponent α as the slope of the fitted relationship. This procedure can be directly applied to experimental datasets to test the model quantitatively.

Having established consistency between theoretical predictions and empirical tumor growth behavior, we now consider the broader implications of boundary-mediated growth for tumor progression and biological organization.

8. Discussion

The central contribution of this framework is a conceptual redefinition of tumor growth:

Tumor growth is not a volumetric process—it is a boundary-mediated phenomenon governed by transport across an interface.

The framework also generates forward predictions. Interventions that reduce vascularization or perfusion, such as anti-angiogenic therapies, should decrease the effective transport parameter β and therefore reduce the growth exponent α. Conversely, enhanced vascularization should increase β and shift growth toward the critical regime $\alpha \to 1$. These predictions are directly testable using longitudinal measurements of tumor growth and vascular structure.

This shift has significant implications. Traditional models implicitly treat growth as occurring throughout the tumor volume, with geometry playing a secondary role. In contrast, the present

framework identifies the boundary as the primary site of growth, where resource influx enables accumulation.

This boundary-centered perspective resolves the apparent diversity of tumor growth laws. Rather than requiring multiple independent models, different growth behaviors emerge as manifestations of a single underlying mechanism operating under varying transport conditions.

Within this structure, the condition $\beta \leq 1$ emerges as a universal stability constraint. This bound is not introduced phenomenologically but arises from the requirement that transport amplification cannot indefinitely outpace geometric limitation without destabilizing the system. When β approaches unity, the system reaches a critical balance between supply and constraint. When β exceeds unity, this balance is lost, and growth enters a superlinear regime characterized by instability [25–26].

Tumor progression can therefore be understood as a trajectory through transport regimes. Early stages are diffusion-limited, intermediate stages reflect increasing vascularization, and advanced stages approach the critical boundary [9–11].

This provides a unified description of tumor evolution grounded in measurable physical processes.

Beyond oncology, this framework situates tumor growth within a broader class of systems governed by boundary-mediated flux. Similar constraints appear in biological scaling laws, ecological resource distribution, and physical accretion processes [6–8,22–24].

Most importantly, the framework replaces descriptive modeling with testable structure. The exponent α is no longer a fitting parameter but a measurable consequence of underlying processes. This establishes a direct link between microscopic biological mechanisms and macroscopic growth behavior.

In doing so, tumor growth is transformed from an empirical observation into a predictive, falsifiable system governed by physical law.

9. Conclusion

We have developed a unified geometric–transport framework in which tumor growth emerges from boundary-mediated dynamics rather than imposed empirical laws.

Within this formulation, the growth exponent α is derived from first principles, reflecting the interaction between geometric boundary scaling and transport amplification. This leads to a constrained spectrum

$$\frac{2}{3} \leq \alpha \leq 1$$

which defines the domain of biologically sustainable growth. The upper bound, α = 1, represents a critical transition at which transport efficiency exactly balances geometric constraint.

By introducing a transport amplification parameter β and linking it to measurable biological variables, the framework becomes empirically accessible. Growth dynamics can be directly connected to vascular structure, perfusion, and transport properties, enabling experimental validation or falsification.

In this way, the framework unifies classical growth models, explains the origin of their differing behaviors, and identifies instability as a natural consequence of exceeding transport constraints rather than as an externally imposed phenomenon.

More broadly, tumor growth is revealed as a specific instance of a general principle:

Structure forms, evolves, and destabilizes through flux across boundaries constrained by geometry.

This principle extends across natural systems in which growth depends on external supply mediated by interfaces. Tumors are therefore not exceptional, but part of a wider class of boundary-mediated systems governed by the same underlying constraints.

The significance of this framework lies in its dual role. It provides a mechanistic, quantitatively precise, and falsifiable description of tumor growth, while simultaneously embedding that behavior within a broader physical theory of structure formation in open systems.

In this sense, tumor growth is not merely described—it is explained.

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