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Electromagnetic Selectivity in Membrane Transport of Metal Complexes: A Theoretical Framework Based on Thickness and Dissipation

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ABSTRACT

Metal-based complexes are widely investigated for their ability to disrupt DNA replication in cancer cells; however, achieving selective transport into malignant cells while avoiding healthy tissue remains a central challenge. In this work, we propose a theoretical model in which electromagnetic excitation facilitates the transport of metal complexes across cellular lipid membranes, with selectivity arising from differences in membrane thickness and associated electromagnetic response. The model treats membrane traversal as an energetically constrained process governed by electromagnetic flux-tube–like transport channels, where the efficiency of transmission depends sensitively on membrane geometry and dissipation. By formulating a frequency-dependent criterion linking photon energy to membrane thickness, we demonstrate that, in principle, irradiation at specific frequencies could preferentially enhance transport into cancer cells while suppressing penetration into healthy cells. The analysis is exploratory and theoretical in nature, aiming to establish a physically motivated framework rather than a clinical protocol. Experimental validation is required to assess biological feasibility, safety, and efficacy. Nonetheless, the model provides a novel perspective on membrane-selective transport mechanisms that may inform future investigations in electromagnetic drug delivery and cancer therapy.

Keywords: metal complexes; cancer cell membranes; electromagnetic transport; membrane selectivity; theoretical biophysics; frequency-dependent irradiation

INTRODUCTION

Metal-based complexes have long attracted interest in medicinal chemistry due to their ability to interact with nucleic acids and interfere with DNA replication and transcription, ultimately leading to cytotoxic effects in rapidly dividing cells. Well-known examples, such as platinum-based complexes, demonstrate that metal-centered agents can be effective anticancer therapeutics when delivered to intracellular targets. These principles form a core component of advanced medicinal chemistry and are widely discussed in graduate-level treatments of metal complexes and their biological interactions [5].

Despite their therapeutic potential, a persistent limitation of metal-based treatments is their lack of selectivity: cytotoxic agents often damage healthy cells alongside malignant ones, resulting in severe side effects that limit clinical efficacy.

A central challenge in cancer therapy is therefore the development of mechanisms that preferentially deliver cytotoxic agents into cancer cells while minimizing uptake by healthy tissue. Conventional strategies for achieving selectivity typically rely on biochemical targeting mechanisms, including ligand–receptor specificity, antibody conjugation, and nanoparticle-based delivery systems.

While these approaches have achieved varying degrees of success, they remain constrained by biological variability, immune response, and transport inefficiencies at the level of the cell membrane. These limitations motivate the exploration of alternative approaches that do not rely exclusively on biochemical specificity.

One such alternative is to exploit **physical differences** between cancerous and healthy cells. It is well established that cancer cell membranes often exhibit altered lipid composition, reduced cholesterol content, increased fluidity, and changes in mechanical and dielectric properties compared to normal cells. These differences can manifest as systematic variations in effective membrane thickness and dissipative response, even when chemical composition is broadly similar. Although membrane thickness is not a sharply defined quantity, lipid bilayers typically span only a few nanometers, and even modest variations on this scale can significantly influence transport processes that are energetically or geometrically constrained [5].

From this perspective, membrane traversal need not be governed solely by chemical affinity or receptor-mediated uptake. Instead, the membrane may be treated as a **physical and electromagnetic barrier**, whose transmission properties depend on geometry, dissipation, and the manner in which energy is supplied to a transported complex. Electromagnetic transport phenomena of this type are well studied in theoretical electrodynamics, where energy and charge propagation are often described in terms of localized conduction pathways or flux-tube–like structures rather than uniform bulk transport [1–4]. Although biological membranes differ fundamentally from metallic or plasma environments, the underlying physical concepts of localized energy transport and dissipation provide a useful theoretical framework for exploring membrane-selective mechanisms.

In this study, we explore the hypothesis that **electromagnetic excitation** can mediate the transport of metal complexes across cell membranes in a manner that is sensitive to membrane thickness. Specifically, we investigate whether irradiation at carefully selected frequencies could provide sufficient energy to enable transport into cancer cells while remaining energetically unfavorable for penetration into healthy cells. The proposed mechanism treats membrane crossing as a **dissipative electromagnetic process**, in which transport efficiency is governed by the balance between photon energy, membrane geometry, and resistive losses. In this framework, selectivity emerges not from chemical recognition but from differences in the electromagnetic response of membranes with differing thickness.

The **aim of this research** is therefore not to propose a clinical treatment protocol, but to establish a **self-consistent theoretical framework** that links electromagnetic frequency, membrane thickness, and transport probability. Drawing on concepts from quantum electrodynamics and electromagnetic transport theory [1–4], the model formulates a frequency-dependent criterion under which selective membrane traversal can, in principle, occur. By explicitly identifying the physical conditions required for selectivity, the analysis provides a foundation for evaluating whether electromagnetic-assisted transport of metal complexes is physically plausible.

The present work is intentionally theoretical and exploratory. No claim is made regarding immediate biological feasibility or therapeutic safety, and experimental validation would be required to assess these aspects. Nevertheless, by demonstrating that selective membrane penetration is not forbidden by fundamental physical principles, the framework offers a novel perspective on drug delivery and cancer treatment strategies based on **physical selectivity** rather than biochemical targeting alone. This perspective may inform future experimental investigations into electromagnetic-assisted transport mechanisms and their potential biomedical applications.

PHYSICAL AND BIOLOGICAL BACKGROUND

Metal-based complexes have a well-established role in medicinal chemistry due to their ability to bind to nucleic acids and interfere with DNA replication and transcription processes. Such interactions can prevent cell division and ultimately lead to apoptosis, particularly in rapidly proliferating cells such as cancer cells. These principles are extensively discussed in advanced treatments of metal complexes and their biological activity [5]. From a therapeutic standpoint, however, the principal difficulty associated with these compounds is not their intrinsic cytotoxicity, but rather the absence of a reliable mechanism to ensure preferential uptake by malignant cells while avoiding comparable damage to healthy tissue. This limitation motivates the search for selective transport mechanisms that operate at the level of the cell membrane.

The cell membrane is a lipid bilayer whose physical properties are governed by lipid composition, cholesterol content, and molecular organization. These parameters collectively influence membrane thickness, elasticity, dielectric response, and fluidity. Cancer cells are known to exhibit altered lipid metabolism, reduced cholesterol concentration, and increased membrane fluidity relative to normal cells, leading to systematic differences in membrane structure and mechanical response [5]. Although membrane thickness is not a sharply defined or universal quantity, lipid bilayers typically occupy a scale of several nanometers, and even modest variations in this thickness can significantly influence transport processes that are energetically or geometrically constrained. Such sensitivity becomes particularly important when transport is mediated by localized energy-transfer mechanisms rather than bulk diffusion.

Traditional descriptions of membrane transport emphasize biochemical pathways, including passive diffusion, facilitated diffusion through membrane proteins, and active transport driven by metabolic energy. While these mechanisms are essential for cellular function, they do not exhaust the possible modes of interaction between external agents and biological membranes. In addition to its biochemical role, the membrane can also be regarded as a **physical barrier**, whose transmission properties depend on electromagnetic, mechanical, and dissipative characteristics. The present work adopts this latter perspective, treating membrane traversal as a physically constrained process influenced by electromagnetic excitation rather than solely by chemical affinity or receptor-mediated uptake [1].

Electromagnetic transport phenomena are well studied in condensed matter physics, plasma physics, and electrical engineering, where energy and charge propagation are frequently described in terms of **localized conduction pathways** or **flux-tube–like structures** rather than uniform bulk transport [1–3,6].

In such descriptions, transport occurs along preferred channels defined by electromagnetic fields, boundary conditions, and material properties. These channels typically support both surface-associated propagation modes and axial transport modes, with energy dissipation governed by resistive and inductive elements of the medium [1–3]. Although biological membranes differ fundamentally from metallic lattices or plasma environments, the conceptual framework of localized electromagnetic transport provides a useful theoretical abstraction that can be heuristically extended to nanoscale environments embedded within lipid bilayers.

Within this framework, membrane traversal is modeled as a **dissipative electromagnetic process**. Energy supplied by irradiation may be partially converted into heat unless transport conditions are tuned appropriately. The likelihood that a metal complex successfully crosses a membrane is therefore assumed to depend on the balance between electromagnetic energy input and resistive losses associated with the membrane.

Dissipation plays a central role: excessive energy loss suppresses transmission, whereas sufficiently low dissipation permits traversal. A convenient measure of this balance is the **quality factor**, which characterizes the extent to which an oscillatory or transport process is underdamped and capable of sustaining energy flow with minimal loss [1–3].

The theoretical picture explored in this work assumes that electromagnetic irradiation at a specific frequency can couple energy into a metal complex, enabling a temporary transformation that facilitates membrane crossing. This coupling is treated as frequency-dependent, with photon energy determining the effective potential difference applied across the membrane. Crucially, the energy required for traversal depends on membrane thickness. Thinner membranes require lower photon energies to overcome dissipative losses, whereas thicker membranes impose greater resistance and therefore suppress transport at the same frequency [1,2].

Under these assumptions, it becomes possible in principle to select irradiation frequencies that preferentially enable transport into cancer cells while remaining insufficient to drive penetration into healthy cells.

An additional and essential feature of the proposed mechanism is the inherent **asymmetry of membrane transport**. Successful entry into a cell does not merely require penetration of the first membrane interface; it also requires that the transported complex does not immediately traverse the membrane on the opposite side of the cell.

This introduces a two-stage physical constraint: electromagnetic energy must be sufficient to overcome the entrance membrane while remaining insufficient to overcome the exit membrane, thereby trapping the complex within the intracellular region [1].

From a physical standpoint, this asymmetry transforms the problem from a simple transmission event into a **conditional trapping process** governed by dissipation and geometry.

In the present framework, the intracellular space between the two membranes is treated as a region of minimal dissipation, analogous to a zero-resistivity segment in an electrical circuit, whereas the membranes themselves constitute the dominant dissipative elements [1,6].

Energy loss and radiation are therefore assumed to occur primarily at the membrane interfaces rather than within the cell interior. This distinction is critical, as it allows membrane thickness to emerge as a controlling parameter that determines whether the supplied electromagnetic energy is dissipated or transmitted.

The physical background outlined here does not constitute an experimentally verified model of biological membrane transport, nor does it claim immediate clinical applicability. Rather, it establishes a theoretical foundation grounded in electromagnetic transport concepts, dissipation theory, and membrane geometry, drawing upon earlier work in electrodynamics, quantum theory, and flux-tube transport mechanisms [1–6]. By emphasizing physical selectivity arising from membrane properties rather than biochemical specificity, this approach offers an alternative conceptual pathway for investigating selective cancer treatment mechanisms based on controlled electromagnetic excitation.

THEORETICAL MODEL AND ASSUMPTIONS

The theoretical framework developed in this work is based on the assumption that membrane traversal by a metal complex can be modeled as an **electromagnetic transport process** occurring along localized conduction pathways embedded within the lipid bilayer. These pathways are treated conceptually as **flux-tube–like structures** that support both surface-associated and axial transport modes, as illustrated schematically in Fig. 1.

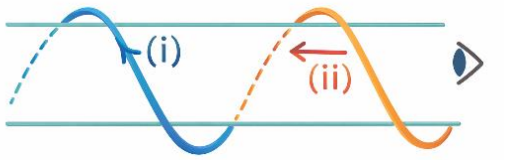

Figure 1. One-way propagation in an electromagnetic flux tube. Reverse propagation is neglected. The metal complex is transported photonically along the helical surface (i), while the ligands are transported axially within the interior (ii), treated as weak–strong gauge-boson–like carriers.

The model does not assume the existence of literal anatomical channels within the membrane; rather, flux tubes are introduced as a **physically motivated abstraction** that captures the essential features of localized electromagnetic energy transport and dissipation in a constrained nanoscale environment [1–3,6].

A central assumption of the model is that, under electromagnetic irradiation at an appropriate frequency, a metal complex can transiently undergo a transformation that allows it to participate in electromagnetic transport rather than remaining purely in its atomic–molecular state.

In this transient state, different components of the complex are assumed to couple differently to the transport mechanism. As depicted in Fig. 1, the metal center is associated with surface propagation along the helical boundary of the flux tube, while the ligands are associated with axial transport within the interior of the tube [1,2].

This division reflects earlier theoretical work in which surface and axial modes are treated as **energetically equivalent but**

dynamically distinct forms of transport, enabling a unified description of energy flow along the flux tube [2,3].

Transport along the surface of the flux tube is assumed to occur in a **helical fashion**, characterized by an angular frequency and associated angular momentum, whereas axial transport proceeds rectilinearly along the tube axis. These two modes are taken to be **energetically coupled**, such that the power dissipated by surface processes equals the power dissipated by axial processes. This equivalence permits the transport process to be analyzed using standard electromagnetic power relations, where power may be expressed either in terms of rotational quantities (torque and angular velocity) or in terms of axial force and velocity [2,6]. Physically, this implies that electromagnetic energy supplied to the system is redistributed between surface and axial degrees of freedom without preferential loss to either mode.

Photon absorption plays a decisive role in initiating transport. As illustrated schematically in Fig. 2, the absorption of a photon by the metal complex is assumed to impart angular momentum to the system, aligning one of the metals–ligand axes with the direction of transport.

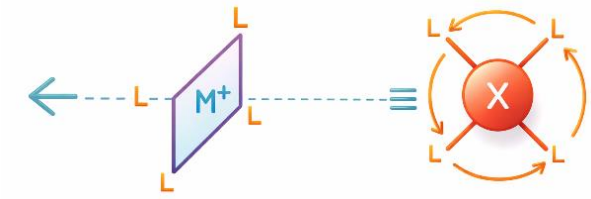

Figure 2. Absorption of a photon by the metal complex imparts angular momentum corresponding to the photon spin, assuming outcomes $s = \pm 1$only. Angular momentum is transferred along a single metal–ligand axis. Here, L denotes ligand, M^+ the metal center, and " × " indicates direction into the page.

This angular momentum provides the inductive component required for sustained helical propagation along the surface of the flux tube. The model assumes that only photon absorption events resulting in **nonzero angular momentum** contribute effectively to membrane traversal, whereas absorption events leading to zero net angular momentum do not support transport [1,2]. This selection rule ensures that transport is intrinsically tied to the angular-momentum content of the electromagnetic excitation.

Dissipation is treated as the **primary limiting factor** governing whether transport across a membrane is successful. The membrane is modeled as a resistive element whose effective resistance increases with membrane thickness. Transport is therefore characterized by a competition between supplied electromagnetic energy and resistive losses. This competition is quantified using a **quality factor**, which measures the extent to which the transport process is underdamped. High-quality-factor conditions correspond to minimal dissipation and favor successful membrane traversal, whereas low-quality-factor conditions correspond to excessive energy loss and transport failure [2,3].

A crucial assumption of the model is that **inductive effects decrease with increasing membrane thickness**. As the effective length of the transport pathway increases, the helical winding becomes less tightly constrained, leading to a reduction in inductance and a corresponding increase in resistance.

As a result, the quality factor decreases rapidly with increasing membrane thickness, implying that thicker membranes suppress transport more strongly than thinner ones [1,2].

This dependence forms the physical basis for selectivity between cancer cells and healthy cells, whose membranes may differ systematically in thickness and dissipative response.

The model further incorporates the assumption that membrane traversal is inherently **asymmetric**. Successful entry into a cell requires penetration of the first membrane interface, but successful retention within the cell requires failure to penetrate the second membrane on the opposite side.

This two-stage constraint transforms membrane crossing into a **conditional trapping process** rather than a simple transmission event. The mechanism is represented schematically in Fig. 6, where the acceptance and rejection of photons by diseased and healthy cells are depicted as frequency-dependent response curves [1]. The operating frequency is assumed to lie at an intersection point where transport into cancer cells is energetically permitted while transport through healthy cells remains suppressed.

To analyze frequency selectivity quantitatively, the model draws on an analogy with **spectral-area conservation** developed in earlier theoretical work on photon wave packets. As illustrated in Fig. 3, transport efficiency is associated not with peak amplitude alone, but with the integrated balance between power dissipation and quality factor across a frequency range [2,3].

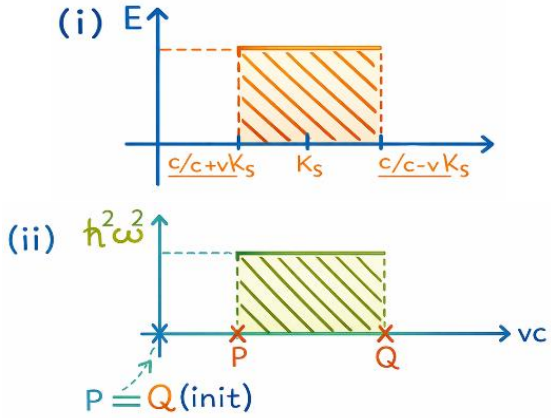

Figure 3. Spectral-area analogy. (i) The QTE spectrum [2,3] and (ii) the corresponding $P - Q$spectrum. In both cases, the spectra are constrained to have equal integrated area, forming the basis for the frequency–geometry relationship.

By enforcing a constant-area condition, a relationship is obtained linking photon frequency to membrane thickness, leading to a **fourth-power dependence** of angular frequency on geometric parameters. This dependence is treated as a structural outcome of the transport model rather than as an empirical fitting relation [1–3].

Throughout this theoretical construction, the intracellular region between the two membranes is assumed to behave as a **low-dissipation domain**. In electromagnetic terms, this region is analogous to a zero-resistivity segment of a circuit, in which

energy is transported without loss. Dissipation and radiation are therefore assumed to occur primarily at the membrane interfaces rather than within the cell interior [2,6]. This assumption allows the analysis to focus on membrane properties as the dominant determinants of selectivity and transport outcome.

The theoretical model presented here is exploratory and rests on a series of **explicit simplifying assumptions** intended to isolate the physical principles underlying selective electromagnetic transport. It does not claim to provide a complete or experimentally validated description of biological membrane behavior. Rather, it establishes a coherent physical framework linking electromagnetic excitation, membrane geometry, dissipation, and selectivity, thereby preparing the ground for the mathematical formulation and quantitative analysis that follow [1–6].

MATHEMATICAL FORMULATION OF THE TRANSPORT MODEL

The transport mechanism proposed in this work is formulated as an **electromagnetic energy–transfer process** constrained by membrane geometry and dissipation.

The central quantity governing transport is the electromagnetic energy supplied by irradiation, which is treated as a **discrete photon energy** $E = h\nu = \hbar\omega$.

This energy is assumed to couple into a localized transport pathway embedded within the membrane, represented schematically by the flux-tube geometry shown in Fig. 1. Successful membrane traversal occurs when the supplied electromagnetic energy exceeds dissipative losses associated with the membrane interface, while remaining insufficient to drive traversal of the membrane on the opposite side of the cell. This dual constraint encodes both entry selectivity and intracellular trapping within a single energetic framework.

Power Dissipation and Photon Energy

The power dissipated during transport is modeled using standard electromagnetic relations. For a localized transport channel characterized by an effective resistance R, the dissipated power is written as

$$P = \frac{V^2}{R} \tag{1}$$

where the effective potential difference V is identified with the photon energy, $V = \hbar\omega$.

This identification emphasizes that the transport process is treated as a **voltage-driven event**, reflecting the discrete nature of photon absorption rather than a continuous current flow. The formulation therefore differs from classical conduction models and is instead appropriate for localized, quantum-mediated energy transfer processes [1–3].

Coupled Surface and Axial Transport Modes

The transport pathway supports two coupled modes of energy propagation: a **surface-associated helical mode** and an **axial mode**, as illustrated in Fig. 1. These modes are assumed to carry equal current and dissipate equal power, allowing the total dissipation to be expressed equivalently in terms of rotational or axial quantities. In particular, the dissipated power may be written as

$$P = \tau\omega = Fv \tag{2}$$

where τ and ω denote the torque and angular frequency associated with helical transport, and F and v denote the force and velocity associated with axial transport [2,6]. This equivalence expresses the conservation of energy flow between surface and axial degrees of freedom and implies that electromagnetic energy supplied to the system is redistributed between these modes without preferential loss. Within this framework, angular frequency may be linked to electromagnetic field quantities through the relation

$$\hbar\omega = | \mathbf{v} \times \mathbf{B} |, \tag{3}$$

which emerges naturally in earlier formulations of electromagnetic flux-tube dynamics and provides a physical connection between photon energy, transport velocity, and magnetic confinement [2,5,6].

Quality Factor and Dissipation

Dissipation is quantified through the introduction of a **quality factor** Q, defined as

$$Q = \frac{1}{R}\sqrt{\frac{L}{C}}, \tag{4}$$

where L and C are the effective inductance and capacitance of the transport channel [2,3]. In the present context, inductance arises from the helical nature of surface transport, which is initiated by photon-induced angular momentum transfer to the metal complex (Fig. 2). Capacitance is associated with charge separation across the membrane thickness. High values of Q correspond to weakly damped transport with minimal energy loss, whereas low values of Q correspond to strongly dissipative transport that suppresses membrane traversal.

A key assumption of the model is that inductance decreases with increasing membrane thickness.

As the effective length of the transport pathway increases, the helical winding becomes less tightly constrained, reducing inductive coupling and increasing resistance. This behavior is captured by expressing resistance as proportional to inductance,

$$R \propto L \tag{5}$$

which leads to a quality factor that decreases rapidly with increasing resistance,

$$Q \propto R^{-3/2} \tag{6}$$

This strong dependence implies that even modest increases in membrane thickness can significantly reduce transport efficiency, providing a natural physical basis for selectivity between cancer cells and healthy cells [1,2].

Spectral-Area Constraint and Frequency Selectivity

The competition between power dissipation and transport efficiency is encapsulated in the difference $P - Q$, which serves as a measure of the net ability of the system to support membrane traversal. To analyze frequency selectivity, an analogy is drawn with earlier work on photon wavepackets, in which **conservation of spectral area** plays a central role [2,3]. As illustrated in Fig. 3, transport is associated not with peak amplitude alone but with the **integrated balance** between dissipative and inductive effects across a frequency range. By enforcing a constant-area condition on the $P - Q$spectrum, one obtains

$$\hbar^2\omega^2\,\Delta(P - Q) = \text{constant} \tag{7}$$

Substituting the expressions for $P$and Qinto this condition yields a **fourth-power dependence** of angular frequency on membrane resistance, and therefore on membrane thickness,

$$\hbar^4\omega^4 \propto \left(\frac{1}{R} - \frac{1}{R^{3/2}}\right) \tag{8}$$

This result establishes a direct quantitative link between irradiation frequency and membrane geometry, forming the mathematical core of the proposed selectivity mechanism [1–3].

Physical Interpretation and Asymmetric Transport

The physical interpretation of Eq. (8) is straightforward. If the membrane thickness of cancer cells and healthy cells were identical, no finite photon energy could distinguish between them, and selective transport would be impossible. Conversely, even small systematic differences in membrane thickness give rise to finite frequency windows in which transport into cancer cells is energetically permitted while transport into healthy cells remains suppressed. In the limiting case of vanishing membrane thickness, arbitrarily low photon energies suffice to permit transport, whereas increasing thickness shifts the required frequency upward.

The model further incorporates the **asymmetric nature of membrane traversal**. Successful intracellular accumulation requires penetration of the entrance membrane without penetration of the exit membrane. This two-stage constraint is represented schematically in Fig. 6, where frequency-dependent acceptance and rejection curves intersect at a critical operating frequency $\hbar\omega$[1]. The intracellular region between the two membranes is assumed to behave as a low-dissipation domain analogous to a zero-resistivity segment of an electrical circuit, such that energy loss occurs predominantly at the membrane interfaces rather than within the cell interior [2,6].

Throughout this formulation, momentum transport is assumed to remain constant along the flux-tube pathway, ensuring continuity between surface and axial modes. Variations in transport outcome therefore arise from changes in resistance and dissipation rather than from changes in momentum. This assumption is consistent with earlier treatments of electromagnetic transport in flux-tube systems and underlies the frequency–geometry relationship derived above [2,6].

The mathematical framework presented here is intentionally idealized. It does not attempt to capture the full biochemical complexity of real cell membranes, nor does it claim quantitative predictive accuracy without experimental calibration. Instead, it provides a **self-consistent theoretical structure** linking electromagnetic frequency, dissipation, and membrane thickness, thereby enabling explicit, testable predictions regarding selective transport conditions [1–6].

IMPLICATIONS, SELECTIVITY CONDITIONS, AND TESTABLE PREDICTIONS

The mathematical framework developed above allows the physical implications of the proposed transport mechanism to be examined in a systematic manner. The central result is the existence of a **frequency-dependent condition** linking electromagnetic irradiation to membrane thickness through dissipative transport constraints. This relationship implies that selective membrane traversal is governed not by chemical specificity alone, but by the **interplay between electromagnetic energy, resistive losses, and membrane geometry**.

A direct implication of the model is that selectivity arises **only when a nonzero difference exists** between the effective membrane thickness of cancer cells and that of healthy cells. In the limiting case where the two membrane thicknesses are identical, the mathematical formulation predicts that no finite photon energy can discriminate between them. This outcome follows directly from the fourth-power frequency dependence derived in Section 4 and reflects the physical principle that **identical barriers cannot be selectively penetrated by a purely physical mechanism**. Conversely, even modest systematic differences in membrane thickness give rise to finite frequency windows in which transport into cancer cells is energetically permitted while transport into healthy cells remains suppressed.

The selectivity condition may be expressed qualitatively in terms of the balance between **dissipated power** and **transport efficiency**. For cancer cells, reduced membrane thickness corresponds to lower resistance and higher quality factor, allowing electromagnetic energy to be transmitted across the membrane with relatively low dissipation. For healthy cells, increased membrane thickness leads to higher resistance, lower quality factor, and enhanced conversion of electromagnetic energy into heat rather than transport. The operating point of selective transport therefore corresponds to a regime in which $P - Q$is positive for cancer cells and negative for healthy cells, as illustrated schematically by the spectral-area analogy shown in Fig. 3 [1–3].

An important feature of the model is that **selectivity does not require perfect transmission** into cancer cells. Indeed, fine-tuning the irradiation frequency to suppress transport into healthy cells necessarily reduces the absolute transport efficiency into cancer cells as well. The framework therefore predicts a trade-off: selective treatment may require increased irradiation intensity or increased concentration of metal complexes to compensate for reduced uptake, while still maintaining negligible absorption by healthy tissue. This trade-off is an inherent consequence of dissipative transport and does not undermine the selectivity principle itself [1].

The **asymmetric nature of membrane traversal** plays a decisive role in determining intracellular accumulation. Successful treatment requires penetration of the entrance membrane without penetration of the exit membrane on the opposite side of the cell. This two-stage constraint introduces a trapping condition that is explicitly frequency-dependent. As depicted schematically in Fig. 6, cancer cells are assumed to exhibit a resonance-like response in which photon absorption and transport are favored within a narrow frequency range, whereas healthy cells are tuned to reject photons within the same range due to increased dissipation [1]. The intersection of these response curves defines the **critical operating frequency** predicted by the model and represents the optimal regime for selective intracellular accumulation.

From a physical standpoint, the intracellular region between the two membranes functions as a **low-dissipation domain**. Once the complex has crossed the entrance membrane, no significant energy loss occurs until it encounters the exit membrane. This effectively decouples entry from exit and allows membrane thickness differences to determine whether the complex becomes trapped within the cell. The analogy with a zero-resistivity segment in an electrical circuit is therefore central to the predicted selectivity mechanism and provides a clear physical interpretation of the trapping condition [2,6].

The model yields several **qualitative predictions** that can, in principle, be subjected to experimental investigation. First, transport efficiency should exhibit a **sharp dependence on irradiation frequency**, with narrow frequency bands corresponding to enhanced uptake in cancer cells.

Second, increasing membrane thickness either chemically, mechanically, or through lipid composition changes should shift the optimal frequency upward and reduce overall transport efficiency.

Third, suppression of transport in healthy cells should persist even at increased irradiation intensity, provided the frequency remains outside the acceptance window defined by their membrane properties. These predictions follow directly from the frequency–geometry relationship derived in Section 4 and do not rely on additional assumptions [1–3].

Equally important are the **conditions under which the model would be falsified**. If experiments were to show frequency-independent uptake, identical transport behavior in cells with demonstrably different membrane thicknesses, or dominant bulk heating effects that overwhelm membrane-localized dissipation, the physical basis of the proposed mechanism would be undermined. The framework therefore makes clear, testable claims rather than unfalsifiable assertions.

It is important to emphasize that the present framework does not claim quantitative predictive accuracy in the absence of experimental calibration. Parameters such as effective resistance, inductance, and membrane thickness are treated phenomenologically rather than measured explicitly. Nonetheless, the existence of a frequency-selective transport regime is a **robust qualitative outcome** of the model, independent of the precise numerical values of these parameters.

The implications of this work are therefore primarily conceptual. By demonstrating that selective membrane transport can, in principle, arise from **purely physical constraints** rather than biochemical targeting alone, the model provides an alternative viewpoint on cancer treatment strategies. Whether this viewpoint can be translated into practical therapeutic protocols remains an open question that can only be resolved through experimental validation. However, the framework presented here establishes clear physical conditions under which such validation can be meaningfully pursued [1–6].

LIMITATIONS, SCOPE, AND OUTLOOK

The theoretical framework presented in this work is exploratory in nature and is subject to several important limitations that must be acknowledged explicitly. The model is intentionally idealized, focusing on physical transport mechanisms and electromagnetic constraints while neglecting much of the biochemical complexity inherent in real biological systems. Cellular membranes are treated as effective electromagnetic barriers characterized primarily by thickness and dissipation, whereas in reality they are heterogeneous, dynamic structures incorporating proteins, ion channels, membrane remodeling, and active transport mechanisms that are not included in the present formulation [1].

A further limitation arises from the phenomenological **treatment** of electromagnetic parameters such as resistance, inductance, and quality factor. These quantities are introduced to capture the dissipative and inductive behavior of localized transport pathways but are not derived from first-principles biophysical measurements. As a result, the mathematical relationships established in this work should be interpreted qualitatively rather than quantitatively. In particular, the fourth-power frequency dependence derived in Section 4 represents a structural outcome of the model rather than a numerically calibrated law [1–3].

The framework also relies on a set of physical assumptions concerning localized electromagnetic transport, flux-tube–like pathways, and coupled surface and axial modes.

These assumptions are motivated by earlier theoretical investigations in electrodynamics and flux-tube transport phenomena [2–6], but they have not been experimentally validated in the context of biological membranes. Consequently, the model does not claim to describe an established biological mechanism, but rather proposes a **physically self-consistent scenario** that could, in principle, operate under appropriate conditions.

From a biological perspective, the model abstracts away important processes such as endocytosis, membrane repair, thermal regulation, and cellular response to electromagnetic stress. These processes may significantly influence transport outcomes in practice and could either enhance or suppress the effects predicted by the present framework.

In particular, excessive local heating or radiation-induced damage could impose constraints on the range of permissible irradiation frequencies and intensities, independent of membrane selectivity considerations [1].

Scope and Experimental Outlook

The scope of the present work is therefore limited to establishing the **existence of a physically plausible selectivity mechanism** based on membrane thickness and

electromagnetic dissipation. It does not attempt to assess clinical feasibility, therapeutic safety, or biological efficacy. Such assessments would require extensive experimental investigation, including controlled studies of membrane properties, frequency-dependent irradiation experiments, and detailed analysis of cellular viability and DNA interaction following transport.

Despite these limitations, the framework provides a clear conceptual outlook for future research. In particular, it predicts the existence of **narrow frequency windows** in which selective transport may occur. These predictions suggest concrete experimental directions, such as systematic studies of frequency-dependent uptake of metal complexes in cell lines with well-characterized membrane properties.

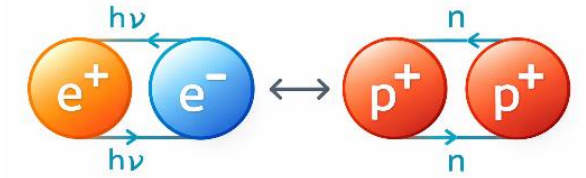

Figure 4. *Frequency-dependent transport efficiency for cancer and healthy cells.*

Schematic illustration of distinct acceptance windows arising from differences in membrane thickness and dissipation. Transport into cancer cells is permitted within a narrow frequency band, while transport into healthy cells is suppressed over the same range.

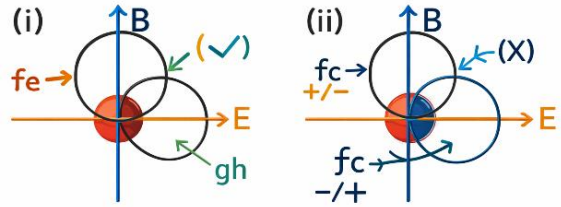

Figure 5. *Power–quality-factor competition as a function of membrane thickness.*

Illustration of the balance between dissipated power and quality factor for thin (cancer-like) and thick (healthy-like) membranes. The sign of $P - Q$ determines whether transport is energetically permitted or suppressed. These schematic representations emphasize that selectivity is governed by **physical constraints** rather than biochemical recognition and provide experimentally testable signatures of the proposed mechanism [1–3].

CONCLUSION

The present work has developed a theoretical framework aimed at addressing one of the central challenges in metal-based cancer therapy: the **selective transport** of cytotoxic metal complexes into malignant cells while avoiding comparable uptake by healthy tissue. Rather than relying on biochemical targeting mechanisms, the approach explored here is grounded in **physical principles**, proposing that differences in membrane thickness and dissipation between cancerous and healthy cells can be exploited through controlled electromagnetic excitation.

At the core of the framework is the treatment of membrane traversal as a **localized electromagnetic transport process** occurring along flux-tube–like pathways embedded within the lipid bilayer. The conceptual geometry of this transport, illustrated in Fig. 1, allows membrane crossing to be analyzed in terms of coupled surface and axial transport modes, energy dissipation, and geometric constraints. Within this picture, membrane penetration is no longer viewed as a purely stochastic or diffusive event, but as an energetically constrained process governed by electromagnetic balance conditions.

Photon absorption and angular momentum transfer play a central role in initiating transport, as illustrated in Fig. 2. In the proposed model, photon–complex interaction imparts angular momentum that enables helical surface propagation, while axial transport facilitates traversal through the membrane interior. The equivalence of power dissipation in these two modes allows the transport process to be expressed using standard electromagnetic relations, providing a bridge between abstract theoretical constructs and physically interpretable quantities such as resistance, inductance, and dissipated power.

The mathematical formulation demonstrates that membrane thickness enters the problem in a **highly nonlinear manner**. Through the introduction of a quality factor and a spectral-area analogy (Fig. 3), the analysis reveals a fourth-power dependence of the required angular frequency on membrane resistance. This result has significant conceptual implications: even relatively small differences in membrane thickness between cancerous and healthy cells can lead to pronounced differences in transport behavior when electromagnetic irradiation is tuned appropriately.

A particularly important outcome of the analysis is the recognition that selective intracellular accumulation depends on **asymmetric membrane traversal**. As depicted schematically in Fig. 6, successful treatment requires that the metal complex penetrate the entrance membrane of a cancer cell while failing to traverse the exit membrane on the opposite side. This two-stage constraint transforms membrane thickness differences into a **trapping mechanism** rather than a simple transmission problem. The intracellular region between the membranes effectively functions as a low-dissipation domain, analogous to a zero-resistivity segment in an electrical circuit, while dissipation is localized primarily at the membrane interfaces.

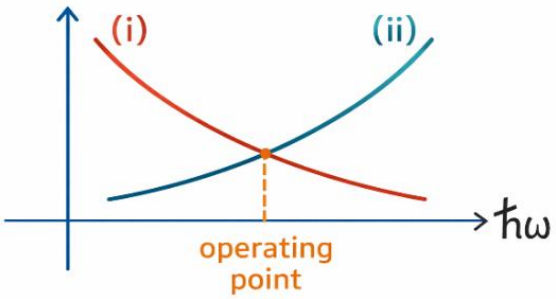

Figure 6. *Asymmetric membrane traversal and intracellular trapping.*

Schematic representation of frequency-dependent acceptance at the entrance membrane and rejection at the exit membrane. The intersection of these conditions defines the operating frequency for selective intracellular accumulation.

From a broader perspective, the framework developed here highlights the potential importance of **physical selectivity** in biological systems. While biochemical specificity has traditionally dominated approaches to targeted therapy, the present work suggests that electromagnetic and geometric constraints may provide an additional, orthogonal pathway to selectivity.

The model does not claim immediate clinical applicability, nor does it assert that such a mechanism operates naturally in biological systems. Instead, it establishes that, within a self-consistent physical description, selective membrane transport based on electromagnetic excitation is not forbidden by fundamental principles.

The limitations of the framework are therefore not weaknesses, but clarifications of scope. By deliberately abstracting away biological complexity, the present study isolates the physical conditions under which selective transport could occur. Whether or not the proposed mechanism can be realized experimentally, the analysis demonstrates that physical constraints alone are sufficient, in principle, to generate selective membrane transport. As such, the framework provides a foundation for further theoretical refinement and experimental testing, and contributes to a broader understanding of how electromagnetic dissipation and geometry may influence biological transport processes.

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