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Mitochondrial Transfer and Tumor Cell Immune Evasion: Mechanisms, Clinical Significance, and Research Prospects – A Review

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Abstract: Mitochondria, as pivotal organelles involved in cellular energy metabolism and signal transduction, have recently been recognized for their dynamic transfer between cells within the tumor microenvironment. This phenomenon of mitochondrial transfer has emerged as a critical factor contributing to tumor cell immune evasion, a major challenge in effective cancer treatment. Current research reveals complex molecular mechanisms underlying mitochondrial transfer, including tunneling nanotubes, extracellular vesicles, and gap junctions, which facilitate intercellular communication and metabolic reprogramming. These mechanisms modulate immune responses by altering tumor immunogenicity and suppressing anti-tumor immunity, thereby promoting tumor progression and resistance to immunotherapy. Despite advances in understanding the biology of mitochondrial transfer, significant gaps remain in elucidating its precise role in immune escape and identifying therapeutic targets. This review comprehensively summarizes the fundamental processes of mitochondrial transfer and explores their involvement in tumor immune evasion, emphasizing key signaling pathways and molecular mediators. Furthermore, it assesses the impact of mitochondrial transfer on the efficacy of current immunotherapeutic approaches and discusses potential clinical applications aimed at disrupting this process to enhance treatment outcomes. Finally, integrating the latest research findings, we highlight future directions for investigating mitochondrial transfer in tumor immunoregulation and its translational potential, aiming to foster innovative strategies for cancer therapy. This synthesis offers valuable insights into the interplay between mitochondrial dynamics and tumor immunity, underscoring the importance of targeting mitochondrial transfer in overcoming immune resistance.

1. Introduction

Mitochondria have long been recognized as the central organelles responsible for cellular energy production, primarily through oxidative phosphorylation, which generates adenosine triphosphate (ATP) to meet the metabolic demands of cells. Beyond their canonical role as the “powerhouse of the

cell," mitochondria are now understood to be dynamic and multifunctional signaling hubs that regulate a wide array of cellular processes including redox homeostasis, calcium signaling, apoptosis, and immune responses [1]. These organelles continuously adapt to metabolic and environmental stresses, orchestrating intracellular communication through anterograde (nucleus-to-mitochondria) and retrograde (mitochondria-to-nucleus) signaling pathways. The intricate regulation of mitochondrial function thus underpins cellular homeostasis and survival, and its dysregulation has been implicated in numerous pathological conditions including cancer, neurodegenerative diseases, and immune disorders [1][2][3].

In the context of cancer biology, mitochondria play pivotal roles not only in sustaining the high bioenergetic and biosynthetic needs of rapidly proliferating tumor cells but also in modulating tumor progression, metastasis, and response to therapy. Tumor cells frequently exhibit mitochondrial dysfunction characterized by altered oxidative phosphorylation, increased reactive oxygen species (ROS) production, and metabolic reprogramming, such as the well-known Warburg effect where glycolysis predominates even under aerobic conditions [4][5]. Recent studies have extended this paradigm by revealing that mitochondria can be transferred intercellularly within the tumor microenvironment (TME), a phenomenon that profoundly influences tumor behavior and the immune landscape. Mitochondrial transfer occurs via tunneling nanotubes (TNTs), extracellular vesicles, or direct cell-cell contact and has been shown to facilitate metabolic cooperation among cancer cells and between cancer and stromal or immune cells [6][4]. This intercellular trafficking of mitochondria enables tumor cells to acquire functional mitochondria that support their metabolic plasticity, promote chemoresistance, and contribute to immune evasion mechanisms [7][6].

Immune escape remains a hallmark of cancer, allowing tumor cells to evade immune surveillance and destruction. Multiple mechanisms underlie tumor immune evasion, including the secretion of immunosuppressive cytokines, expression of immune checkpoint molecules, metabolic competition, and the creation of an immunosuppressive microenvironment [8]. Emerging evidence highlights mitochondrial transfer as a novel and critical mechanism by which tumor cells subvert anti-tumor immunity. For example, cancer cells can transfer dysfunctional, ROS-generating mitochondria to cytotoxic CD8+ T cells, impairing their mitochondrial function, inducing T cell exhaustion or senescence, and ultimately dampening their cytotoxic capacity [6][9]. Conversely, tumor cells can hijack healthy mitochondria from T cells, thereby "metabolically empowering" themselves while depleting immune effector functions [10]. These bidirectional mitochondrial exchanges reshape the metabolic and functional state of immune cells within the TME, facilitating tumor immune escape and correlating with poor clinical outcomes [6][10]. Such findings underscore the multifaceted roles of mitochondria in immune regulation and suggest that targeting mitochondrial transfer pathways may offer new therapeutic avenues to enhance anti-tumor immunity.

Understanding the molecular mechanisms governing mitochondrial transfer and its impact on tumor-immune interactions is crucial for advancing cancer immunotherapy. CD8+ T cells, which are central to anti-tumor immune responses, rely heavily on mitochondrial integrity for their activation, proliferation, and effector functions. Mitochondrial dysfunction in these cells, induced by tumor-mediated mitochondrial transfer or intrinsic metabolic dysregulation, leads to impaired energy metabolism, increased oxidative stress, and apoptosis, thereby compromising their anti-tumor efficacy [11][8]. Research into the pathways regulating mitochondrial dynamics, mitophagy, and mitochondrial biogenesis in T cells has revealed potential targets to restore their function and overcome tumor-induced immune suppression [11][12]. Moreover, the identification of biomarkers such as circulating mitochondrial DNA (mtDNA) and mitochondrial haplogroups may facilitate the monitoring of mitochondrial transfer activity and immune status in cancer patients, guiding personalized therapeutic strategies [6].

Clinically, the recognition of mitochondrial transfer as a key player in tumor immune evasion

opens promising prospects for novel interventions. Strategies aiming to inhibit the formation of tunneling nanotubes or block mitochondrial trafficking could disrupt the metabolic crosstalk that favors tumor survival and immune escape [7][6]. Conversely, therapeutic mitochondrial transfer from healthy donor cells, such as mesenchymal stromal cells, to exhausted T cells has been shown to rejuvenate their function and enhance the efficacy of adoptive cell therapies like chimeric antigen receptor T (CAR-T) cells [6]. Furthermore, combining mitochondrial-targeted therapies with existing immunotherapies, including immune checkpoint inhibitors and metabolic modulators, may synergistically improve treatment outcomes [5][6]. Despite these advances, challenges remain in optimizing mitochondrial transfer efficiency, standardizing detection methods, and fully elucidating the complex signaling networks involved.

In summary, mitochondria are central to the regulation of tumor cell metabolism, immune cell function, and the dynamic interplay within the tumor microenvironment. The emerging concept of intercellular mitochondrial transfer as a mechanism of tumor immune evasion represents a paradigm shift in our understanding of cancer biology. Systematic investigation into the molecular mechanisms, clinical implications, and therapeutic potential of mitochondrial transfer is imperative to unlock new avenues for cancer treatment. This review aims to comprehensively summarize current knowledge on mitochondrial transfer mechanisms, their role in tumor immune escape, and the clinical significance, while also discussing future research directions to harness this axis for improved cancer immunotherapy.

2. Main Body

2.1. Mechanisms and Regulation of Mitochondrial Transfer

2.1.1. Basic Forms and Pathways of Mitochondrial Transfer

Intercellular mitochondrial transfer is a dynamic and multifaceted process that occurs through several distinct mechanisms, primarily involving direct cell-cell connections and extracellular vesicle-mediated transport. The most well-characterized pathway is via tunneling nanotubes (TNTs), which are thin, actin-rich membranous channels that physically connect donor and recipient cells, allowing the unidirectional or bidirectional passage of mitochondria and other cellular components. TNTs facilitate long-range mitochondrial transfer and have been observed in various cell types, including tumor cells, immune cells, and stromal cells within the tumor microenvironment [13][14]. Besides TNTs, extracellular vesicles (EVs), including exosomes and microvesicles, serve as carriers for mitochondrial components, enabling transfer without direct cell contact. This vesicle-mediated pathway contributes to mitochondrial exchange in both physiological and pathological contexts [15][16]. Another notable mechanism is cell fusion, which allows the mixing of cytoplasmic contents, including mitochondria, between fused cells, although this occurs less frequently compared to TNTs and EVs [17].

Importantly, the choice of mitochondrial transfer pathway is not uniform but varies depending on tumor type, microenvironmental conditions, and the interacting cell populations. For instance, in hematological malignancies, TNTs have been identified as a predominant route for mitochondrial transfer from bone marrow stromal cells to leukemic cells, supporting disease progression and chemoresistance [18][19]. In solid tumors, such as melanoma and glioma, both TNTs and EVs mediate mitochondrial transfer between tumor cells and between tumor cells and stromal or immune cells, modulating tumor aggressiveness and immune evasion [20][21]. Moreover, mitochondrial transfer is not restricted to tumor cells alone but also involves interactions between tumor cells and immune cells, as well as between tumor cells and mesenchymal stem cells (MSCs) or other stromal components, reflecting a complex network of intercellular communication within the tumor

microenvironment [14][22].

This intercellular exchange of mitochondria plays critical roles in compensating for mitochondrial dysfunction, restoring energy metabolism, and promoting cell survival under stress conditions. For example, mitochondrial transfer from MSCs to damaged cells can rescue energy deficits and enhance tissue repair, while in tumors, it supports metabolic adaptation and resistance to therapy [23][24]. The heterogeneity of transfer mechanisms and their context-dependent utilization underscore the necessity to delineate specific pathways operative in different tumor types and microenvironmental settings to develop targeted therapeutic strategies.

2.1.2. Regulatory Molecules and Signaling Pathways of Mitochondrial Transfer

The regulation of mitochondrial transfer involves a coordinated interplay of cytoskeletal components, mitochondrial trafficking proteins, oxidative stress signals, and immune modulators. Cytoskeletal proteins, particularly actin and microtubules, are fundamental for the formation of TNTs and the intracellular trafficking of mitochondria. Actin polymerization drives TNT extension, while microtubule networks facilitate mitochondrial movement along these structures [14][25]. Key mitochondrial trafficking proteins such as Miro1 and Milton (also known as TRAK proteins) serve as adaptors linking mitochondria to motor proteins like kinesin and dynein, orchestrating mitochondrial transport and positioning within cells and along intercellular connections [26][27].

Oxidative stress and inflammatory signaling pathways act as potent inducers of mitochondrial transfer. Reactive oxygen species (ROS) accumulation and activation of transcription factors such as NF-κB can stimulate TNT formation and promote mitochondrial trafficking to recipient cells, serving as a cellular response to stress and damage [18][28]. For example, in cardiomyocytes exposed to macrophage-derived mitochondria, oxidative stress triggers ferroptosis, highlighting the dual role of mitochondrial transfer in cell fate determination [29]. Immune regulatory factors, including cytokines and chemokines, modulate the frequency and directionality of mitochondrial transfer. Cytokines such as TNF-α can activate NF-κB signaling in MSCs, enhancing TNT formation and mitochondrial donation to stressed cells [30]. Furthermore, immune checkpoint molecules and signaling pathways, including CD38 and SIRPα-CD47 axis, have been implicated in regulating mitochondrial transfer and immune cell function within the tumor microenvironment [19][31].

Additional molecular players include connexin 43 (Cx43), which forms gap junction channels facilitating mitochondrial transfer, particularly from MSCs to chondrocytes, and is modulated under oxidative stress conditions [32][33]. The Wnt signaling pathway also intersects with cytoskeletal remodeling and TNT dynamics, influencing mitochondrial transfer and intercellular connectivity [34]. Collectively, these regulatory molecules and pathways integrate environmental cues and cellular states to fine-tune mitochondrial transfer, impacting tumor progression, immune modulation, and tissue repair.

2.1.3. Functional Significance of Mitochondrial Transfer

Mitochondrial transfer serves as a vital mechanism for maintaining cellular energy homeostasis and promoting survival, particularly under conditions of metabolic stress or mitochondrial dysfunction. By donating functional mitochondria, donor cells can compensate for the bioenergetic deficits of recipient cells, thereby restoring ATP production and supporting essential cellular functions [23][35]. This compensatory transfer is critical in the tumor microenvironment, where hypoxia and nutrient deprivation challenge tumor cell survival. Tumor cells receiving mitochondria from stromal or immune cells exhibit enhanced oxidative phosphorylation capacity, increased invasiveness, and resistance to chemotherapy [36][37].

Beyond metabolic support, mitochondrial transfer influences the immune landscape by modulating

immune cell metabolism and function. For instance, mitochondrial donation to T cells can enhance their metabolic fitness, proliferation, and antitumor efficacy, whereas transfer of dysfunctional mitochondria from tumor cells to immune cells can induce exhaustion and impair immune surveillance [38][9]. Moreover, mitochondrial transfer impacts inflammatory responses and cell death pathways, such as ferroptosis in cardiomyocytes and pyroptosis in dental pulp cells, indicating its role in regulating cell fate decisions [29][30].

In the context of tissue repair and regeneration, mitochondrial transfer from MSCs to injured cells promotes recovery by improving mitochondrial function, reducing oxidative stress, and modulating inflammatory signaling [24][32]. This phenomenon has been harnessed therapeutically in models of pulmonary fibrosis, cardiac ischemia, neurodegeneration, and diabetic complications, highlighting its broad clinical relevance [23][39]. Furthermore, mitochondrial transfer contributes to tumor immune evasion by supporting the metabolic demands of cancer cells and suppressing effective immune responses, representing a potential target for anticancer therapies [7][4].

In summary, mitochondrial transfer is a multifaceted process that not only rescues cellular energy metabolism but also modulates immune responses, cell survival, and disease progression. Understanding its diverse functional roles provides a foundation for developing novel therapeutic strategies aimed at manipulating mitochondrial transfer to improve outcomes in cancer, immune disorders, and regenerative medicine.

2.2. The Impact of Mitochondrial Transfer on Immune Cell Function

2.2.1. Mitochondrial Transfer Promotes Tumor Cell Metabolic Reprogramming and Immune Tolerance

Mitochondrial transfer within the tumor microenvironment (TME) serves as a pivotal mechanism by which tumor cells acquire additional mitochondrial resources, thereby enhancing their oxidative phosphorylation (OXPHOS) capacity to meet the heightened energy demands associated with immune evasion. This metabolic reprogramming is crucial for sustaining tumor proliferation and survival under immunological stress. For instance, cancer cells receiving mitochondria from stromal cells or neighboring tumor cells exhibit augmented mitochondrial respiration and increased reactive oxygen species (ROS) production, which are instrumental in modulating the TME towards immune tolerance [40][41]. The enhanced OXPHOS not only supports bioenergetic needs but also leads to the accumulation of immunosuppressive metabolites such as lactate and ROS. Elevated lactate levels contribute to acidification of the TME, which impairs effector immune cell functions, including those of cytotoxic T lymphocytes and natural killer cells, thereby facilitating immune escape [42][43]. Moreover, ROS generated from transferred mitochondria can activate signaling pathways like NF-κB and HIF-1α, which further promote an immunosuppressive milieu by upregulating immune checkpoint molecules such as PD-L1 on tumor cells [40][42]. This upregulation of PD-L1 enhances the tumor's ability to inhibit T cell-mediated cytotoxicity, reinforcing immune evasion. Additionally, mitochondrial transfer has been shown to induce transcriptional changes in tumor cells that favor the secretion of immunosuppressive cytokines and growth factors, such as transforming growth factor-beta 1 (TGFβ1), which further remodels the TME to support tumor progression and immune tolerance [40]. Collectively, these metabolic and molecular alterations driven by mitochondrial transfer underscore a sophisticated strategy by which tumor cells reprogram their metabolism to create an immunosuppressive niche, thereby evading immune surveillance and promoting tumor growth.

2.2.2. Mitochondrial Transfer Affects Immune Cell Function and Activity

Mitochondrial transfer is bidirectional within the TME, extending beyond tumor cells to immune

cells such as macrophages and T lymphocytes, where it profoundly influences their metabolic states and functional phenotypes. When mitochondria are transferred from tumor cells to immune cells, this can lead to altered metabolic programming that favors immunosuppressive phenotypes. For example, macrophages receiving mitochondria from tumor cells may undergo metabolic shifts towards fatty acid oxidation and enhanced mitochondrial metabolism, promoting their polarization into M2-like tumor-associated macrophages (TAMs) with immunosuppressive and pro-tumorigenic functions [44][45]. Similarly, mitochondrial transfer to T cells can induce mitochondrial dysfunction characterized by increased ROS accumulation and impaired oxidative phosphorylation, which contributes to T cell exhaustion and senescence, thereby diminishing their antitumor capacity [41][46]. Notably, cancer cells have been observed to transfer dysfunctional mitochondria harboring pathogenic mtDNA mutations to tumor-infiltrating lymphocytes (TILs), leading to metabolic abnormalities and impaired effector functions in these immune cells [46][47]. This mitochondrial hijacking results in T cell senescence and defective memory formation, weakening immune surveillance. Furthermore, mitochondrial transfer can induce pyroptosis or functional dysregulation in immune cells, further compromising the antitumor immune response [48]. The dysfunction of immune cell mitochondria is thus intricately linked to tumor immune evasion, as it undermines the metabolic fitness required for effective immune activation and cytotoxicity. Conversely, therapeutic mitochondrial transfer from stromal or mesenchymal stem cells to immune cells has been shown to restore mitochondrial function, enhance metabolic fitness, and rejuvenate exhausted T cells, representing a promising strategy to bolster antitumor immunity [49][50]. Therefore, mitochondrial transfer critically modulates immune cell functionality within the TME, shaping the balance between immune activation and suppression.

2.2.3. Signaling Pathways Mediated by Mitochondrial Transfer in Immune Evasion

Mitochondrial transfer activates multiple intracellular signaling cascades that collectively contribute to the establishment of an immunosuppressive tumor microenvironment and facilitate immune evasion. Key pathways implicated include the PI3K/Akt, hypoxia-inducible factor 1-alpha (HIF-1α), and nuclear factor kappa-light-chain-enhancer of activated B cells (NF-κB) signaling axes. The enhanced mitochondrial respiration and ROS production following mitochondrial transfer can stimulate NF-κB activation, which drives the transcription of genes encoding immunosuppressive cytokines and immune checkpoint molecules such as PD-L1, thereby promoting tumor immune escape [42][40]. Concurrently, mitochondrial transfer-induced hypoxia and metabolic stress stabilize HIF-1α, which further upregulates PD-L1 expression and modulates metabolic enzymes that favor an immunosuppressive microenvironment [42][41]. The PI3K/Akt pathway is also engaged upon mitochondrial transfer, facilitating survival and proliferation signals in tumor and immune cells, and contributing to the metabolic reprogramming necessary for immune tolerance [51]. Moreover, the release of mitochondrial DNA (mtDNA) into the cytosol or extracellular space during mitochondrial transfer activates the cyclic GMP-AMP synthase (cGAS)-stimulator of interferon genes (STING) pathway, which modulates inflammatory responses within the TME. While cGAS-STING activation can promote antitumor immunity under certain contexts, chronic activation by tumor-derived mtDNA can paradoxically induce immunosuppressive signaling and recruitment of myeloid-derived suppressor cells (MDSCs), thus facilitating immune evasion [52][41]. Importantly, these signaling pathways intersect with immune checkpoint pathways, forming a complex regulatory network that fine-tunes immune responses. For example, the interplay between mitochondrial transfer-induced signaling and PD-1/PD-L1 checkpoint engagement creates a feedback loop that sustains immune suppression and tumor progression [42][41]. Understanding these interconnected pathways offers potential therapeutic targets to disrupt mitochondrial transfer-mediated immune evasion and enhance the efficacy of cancer immunotherapies.

2.3. Clinical Significance and Research Prospects of Mitochondrial Transfer

2.3.1. Mitochondrial Transfer as Tumor Diagnostic and Prognostic Biomarkers

Mitochondrial transfer and its associated molecular markers have emerged as significant indicators correlated with tumor progression, metastasis, and patient survival outcomes. Various studies have identified mitochondrial DNA (mtDNA) fragments, mitochondrial tRNA-derived fragments (mt-tRFs), and mitochondrial proteins whose expression levels are altered in cancerous tissues compared to normal counterparts, reflecting the dynamic mitochondrial landscape during tumorigenesis. For instance, mt-tRF-Tyr-GTA-001 was found to be significantly downregulated in breast tumors, with lower expression levels correlating with poorer patient survival, independent of other clinical factors [53]. Similarly, mitochondrial creatine kinase (uMtCK), which supports energy metabolism, was overexpressed in gastric cancer and associated with advanced disease stages and worse prognosis [54]. In acute myeloid leukemia (AML), elevated expression of mitochondrial oxidative phosphorylation (OXPHOS) mediators such as NDUFA6 and SDHA correlated with poor overall survival, highlighting their potential as prognostic biomarkers [55]. Moreover, mitochondrial transfer-related proteins like NDUFAF2 have been implicated as independent prognostic markers in lung adenocarcinoma [56]. Beyond protein markers, mitochondrial DNA mutations transferred between cancer cells and immune cells have been linked to immune evasion and poor response to immunotherapy [46]. Detection techniques leveraging mitochondrial components, such as liquid biopsies analyzing circulating mtDNA or mitochondrial tRFs, provide promising avenues for early tumor diagnosis and therapeutic monitoring. Fluorescent probes targeting mitochondrial viscosity changes have been developed for real-time imaging of tumor progression, as demonstrated in glioblastoma models, offering a non-invasive diagnostic tool [57][58]. Collectively, these findings underscore the clinical relevance of mitochondrial transfer-associated molecules as biomarkers, which not only reflect tumor metabolic and immune status but also hold promise for improving early detection and prognostic stratification in oncology.

2.3.2. Targeted Therapeutic Strategies Against Mitochondrial Transfer

Targeting mitochondrial transfer pathways presents an innovative therapeutic strategy to enhance cancer treatment efficacy, particularly by overcoming tumor immune evasion and chemoresistance. One approach involves inhibiting the formation of tunneling nanotubes (TNTs), the primary conduits for mitochondrial exchange between tumor and immune cells. Pharmacological blockade of TNT assembly using agents such as L-778123 has been shown to reduce mitochondrial hijacking by cancer cells, thereby restoring immune cell function and improving antitumor responses when combined with immune checkpoint inhibitors [59]. Additionally, targeting mitochondrial dynamics proteins that regulate mitochondrial trafficking and biogenesis may disrupt the metabolic support tumors gain from mitochondrial acquisition, enhancing the efficacy of immunotherapies [6]. Combining immune checkpoint blockade with mitochondrial transfer modulators is an emerging strategy under investigation to synergistically potentiate T cell antitumor activity [7]. Furthermore, mitochondrial transplantation techniques, particularly those mediated via nanotubes from mesenchymal stem cells to T cells, have demonstrated the potential to “supercharge” T cells by enhancing their mitochondrial respiration and metabolic fitness, leading to improved tumor infiltration and cytotoxicity [49][60][61]. However, mitochondrial transplantation also poses a double-edged sword effect in cancer; while it can restore immune cell function, it may inadvertently support tumor cell survival and chemoresistance if not precisely controlled [62]. Therefore, the clinical application of mitochondrial transplantation requires careful evaluation of safety and efficacy. Advances in nanomaterial-based approaches to stimulate mitochondrial biogenesis and transfer further expand therapeutic possibilities

[63]. Overall, targeting mitochondrial transfer pathways, either by inhibition or therapeutic transplantation, represents a promising frontier in cancer treatment, with ongoing research focused on optimizing these interventions to maximize therapeutic benefit while minimizing adverse effects.

2.3.3. Future Research Directions and Challenges

Despite significant progress in elucidating mitochondrial transfer mechanisms and their implications in cancer biology, several challenges and research gaps remain. A critical future direction involves delineating the precise molecular and cellular mechanisms governing mitochondrial transfer across diverse tumor types and immune cell subsets, including the identification of key regulators of TNT formation, mitochondrial trafficking, and cargo selection [17][64]. Developing highly sensitive and specific detection technologies for monitoring mitochondrial transfer in vivo is essential to enable real-time assessment of mitochondrial exchange dynamics and therapeutic responses. Integration of advanced imaging modalities, such as mitochondrial-targeted fluorescent probes and novel biosensors, will facilitate this goal [58][65]. Furthermore, exploring the interplay between mitochondrial transfer and other components of the tumor immune microenvironment, such as stromal cells and metabolic modulators, will provide a comprehensive understanding of tumor-immune crosstalk [66][67]. Translational challenges include rigorous evaluation of the safety, efficacy, and long-term effects of mitochondrial transfer-targeted therapies in clinical settings. Potential off-target effects, immune reactions, and the risk of enhancing tumor resilience necessitate careful preclinical and clinical studies [68][62]. Additionally, standardizing protocols for mitochondrial transplantation and transfer inhibition, along with establishing reliable biomarkers to guide patient selection and monitor treatment outcomes, are imperative. Collaborative efforts integrating bioinformatics, molecular biology, and clinical oncology will accelerate the development of mitochondrial transfer-based therapeutics. Ultimately, overcoming these challenges will pave the way for novel interventions that exploit mitochondrial transfer mechanisms to improve cancer diagnosis, prognosis, and therapy.

3. Conclusions

In conclusion, the phenomenon of mitochondrial transfer has emerged as a pivotal mechanism underpinning tumor immune evasion, intricately modulating tumor metabolism and the immune microenvironment to facilitate tumor progression and resistance to therapy. From an expert perspective, this review has highlighted the multifaceted roles of mitochondrial transfer in shaping the dynamic interplay between cancer cells and the host immune system. The ability of tumor cells to acquire or donate mitochondria not only reprograms their metabolic landscape but also alters immune cell function within the tumor microenvironment, thereby enabling immune escape and promoting malignancy.

Balancing the diverse research perspectives, it is evident that while mitochondrial transfer contributes significantly to tumor biology, the complexity of its molecular mechanisms remains to be fully elucidated. Studies have identified various pathways and cellular structures involved in mitochondrial trafficking, yet the heterogeneity among tumor types and immune contexts poses challenges in defining universal mechanisms. Moreover, the dualistic nature of mitochondrial transfer—potentially beneficial in normal tissue repair but detrimental in cancer progression—necessitates a nuanced understanding to avoid unintended consequences in therapeutic interventions.

The exploration of mitochondrial transfer-associated molecular markers offers promising avenues for the development of diagnostic tools that can detect early immune escape events and predict therapeutic responses. Targeting the mitochondrial transfer process itself or its downstream metabolic and immunological effects holds substantial potential for innovative cancer therapies. Such strategies

could complement existing immunotherapies, overcoming resistance mechanisms that currently limit clinical efficacy.

Looking forward, a concerted effort to integrate basic scientific discoveries with clinical research is imperative. Bridging this gap will accelerate the translation of mitochondrial transfer insights into practical applications, such as biomarker-driven patient stratification and the design of combination therapies that disrupt tumor immune evasion. Furthermore, advanced technologies like single-cell sequencing and live-cell imaging should be leveraged to dissect the spatiotemporal dynamics of mitochondrial transfer in vivo.

In summary, mitochondrial transfer represents a frontier in tumor immunology with profound implications for understanding cancer progression and developing next-generation immunotherapies. Continued interdisciplinary research will be crucial to harness its full potential, ultimately improving patient outcomes by effectively targeting the metabolic and immune escape pathways orchestrated through mitochondrial exchange.

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