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Mitochondrial Peptides and Cell Extracts in Regenerative Medicine and Anti-Aging Therapies: Therapeutic Potential of Mito Organelles

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Abstract

Mitochondria-derived peptides (MDPs), including Humanin, MOTS-c, and small humanin-like peptides (SHLPs), constitute a novel class of micropeptides with potent cytoprotective, metabolic, and anti-aging properties. These molecules encoded in mitochondrial rRNA exert anti-apoptotic, anti-inflammatory, metabolic regulatory, and stress response functions. These therapeutic agents offer routes to modulate mitochondrial bioenergetics and apoptosis within specific pathological contexts. Additionally, cell extracts designated “Mito Organelles” fractionated mitochondrial proteins and peptides represent emergent therapeutic candidates for regenerative and anti-aging medicine. Here, we critically review the mechanisms, preclinical evidence, and translational applications of these MDPs, targeted peptides, and extracts, emphasizing their regenerative potential, safety profiles, and challenges in clinical translation. We also highlight gaps in current knowledge and propose future research directions to consolidate therapeutic applications.

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Keywords
Mitochondrial function; Regenerative medicine; Mitochondria; Peptides; Mito organelles; Anti-aging.

Introduction

Mitochondria are central regulators of cellular energy, apoptosis, and metabolic signaling. Over recent decades, mitochondria-derived peptides (MDPs), such as Humanin, MOTS-c, and several small humanin-like peptides (SHLPs), have revealed previously unrecognized bioactive functions from neuroprotection and metabolic regulation to anti-aging effects. These mitochondria-targeted peptides have emerged as promising therapeutic molecules in mitochondrial dysfunction and cancer. Concurrently, complex extracts enriched with mitochondrial proteins and peptides, referred to as "Mito Organelles" (MOs) are being explored for their regenerative potential. This review consolidates mechanistic insights, preclinical evidence, and safety data, framing their potential in regenerative medicine and anti-aging therapy.

Mitochondrial-Derived Peptides (MDPs)

Humanin

Humanin is a micropeptide encoded within the mitochondrial 16S rRNA gene (MT-RNR2), yielding either a 21-amino-acid mitochondrial-origin peptide or a 24-amino-acid cytosolic form, both biologically active [1]. It exerts potent anti-apoptotic effects by binding pro-apoptotic BAX and Bid proteins, blocking mitochondrial cytochrome c release and caspase activation [2]. Additionally, Humanin interacts with cell-surface receptor complexes including gp130/WSX-1/CNTFR and formyl peptide receptor-like 1 (FPRL1), activating STAT3 signaling [2].

Humanin protects neuronal cells against amyloid-β toxicity and oxidative stress in Alzheimer's disease models, and rescues retinal pigment epithelium (RPE) from oxidative injury by enhancing mitochondrial function and activating STAT3-mediated pathways [2,3]. It also preserves endothelial integrity in atherosclerosis and ameliorates vascular inflammation and remodeling in ApoE-knockout models, reducing oxidative stress and LDL-induced cytotoxicity [2].

Humanin levels decline with age across species including mice, primates, and humans, but remain relatively preserved in long-lived species like the naked mole rat [3]. Mid-life administration of Humanin in mice improved healthspan markers [4,5]. These findings support its geroprotective and cytoprotective therapeutic promise.

MOTS-c

MOTS-c is encoded by a small open reading frame within mitochondrial 12S rRNA. It functions as an exercise mimetic and is upregulated following physical activity [3]. MOTS-c enhances glucose regulation and insulin sensitivity, particularly in skeletal muscles, via AMPK activation and nuclear interactions with NRF2, modulating stress response genes and antioxidant pathways [7]. In mesenchymal stem cells (MSCs),

MOTS-c reduced mitochondrial ROS, normalized mitochondrial morphology and membrane potential, activated AMPK, and improved regenerative capacity [7].

MOTS-c levels decline with age in mice and human plasma; high levels correlate with metabolic health and decreased endothelial dysfunction. It mitigates insulin resistance in ovariectomy-induced metabolic dysfunction and protects bone in aged models. Acute aerobic exercise increases skeletal muscle and circulating MOTS-c, aligning with its role as an exercise-responsive peptide [3,4].

Small humanin-like peptides (SHLPs 1–6)

SHLP1–6, encoded in the mitochondrial 16S rRNA gene, vary in length (20–38 amino acids) and tissue expression: SHLP1 (heart, kidney, spleen), SHLP2 (liver, kidney, muscle), SHLP3 (brain, spleen), SHLP4 (liver, prostate), SHLP6 (liver, kidney).

SHLP2 and SHLP3 enhance cell viability, inhibit apoptosis, boost cellular ATP levels, and increase oxygen consumption indicative of mitochondrial modulation. SHLP2 enhances insulin signaling (via Akt pathway), augments mitochondrial bioenergy, provides chaperone-like effects, and decreases caspase-mediated apoptosis in aging RPE models; levels decline with age [1].

SHLP3 promotes adipocyte differentiation, mediates ERK signaling, and enhances insulin sensitivity. In contrast, SHLP6 promotes apoptosis [4,7].

Mito Organelles Cellular Extracts

In the evolving landscape of regenerative medicine, a novel category of biologically derived preparations, termed “Mito Organelles” (MOs) cellular extracts, has gained emerging attention. These extracts comprise ultrafiltered cellular fractions (~300 kDa) enriched with mitochondrial proteins, peptides, and associated biomolecules, isolated from diverse tissues including heart, brain, kidney, placenta, thymus, cartilage, lungs, connective tissue, and composite sources labeled “LPPSIMKE” (liver, pancreas, placenta, kidney, intestine, retina). Though not yet mainstream in the peer-reviewed literature, these extracts are finding interest on specialized wellness platforms, such as European Wellness, which situates “Mito Organelles” within its Regenerative and Cell Therapy programs, purportedly leveraging mitochondrial biology for tissue rejuvenation [8,9].

The MOs organ-specific peptides are biologically extracted mixtures of cellular peptides that have predominantly mitochondria-specific functions [8]. Although cells of different organ systems have similar functions, variations in cellular functions between organs creates the differential expression of peptides, which can be utilized for various therapeutic purposes. MO peptides are organ-specific extracts that are aimed at revitalizing and rejuvenating mitochondrial activity, thereby regenerating cells and organisms as a whole [8,9]. One of the previous studies has investigated the peptide concentrations in serious of batches of MOs. The quantified analysis was done using the ThermoFisher™ Scientific BCA Protein Assay. Different batches exhibited an average protein concentration of 252.0 ± 12.83 µg/mL, and 260.6 ± 32.23 µg/mL. Statistical analysis revealed no significant difference between the two batches (p = not significant) [30].

The tested samples were separated based on molecular weight using SDS-PAGE, followed by enzymatic digestion with the protease trypsin. Subsequently, each sample was analyzed in triplicate using MALDI-TOF mass spectrometry, and chromatograms were generated (Figure 1).

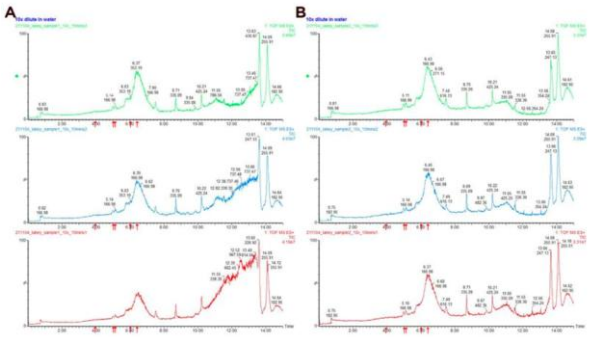

Figure 1: Chromatograms obtained from tested batches of MOs (from Jonathan RT Lakey et al., 2022).

Deconvoluted mass spectra from different batches of MOs revealed comparable profiles in their primary protein constituents (Figure 2). There were five predominant protein masses at 14,969 Da, 15,300 Da, 8,449 Da, 8,294 Da, and 4,618 Da identified.

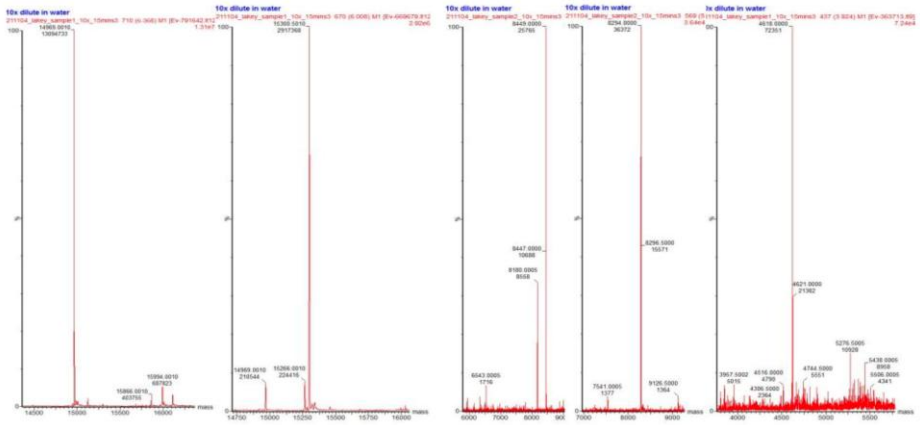

Figure 2: Deconvoluted mass spectrometry data obtained from 1st batch of MOs (from Jonathan RT Lakey et al., 2022).

In a parallel batch study, the tested products exhibited similar six principal protein components with molecular weights of 14,969 Da, 15,301 Da, 8,294 Da, 8,449 Da, 5,436 Da, and 6,214 Da (Figure 3).

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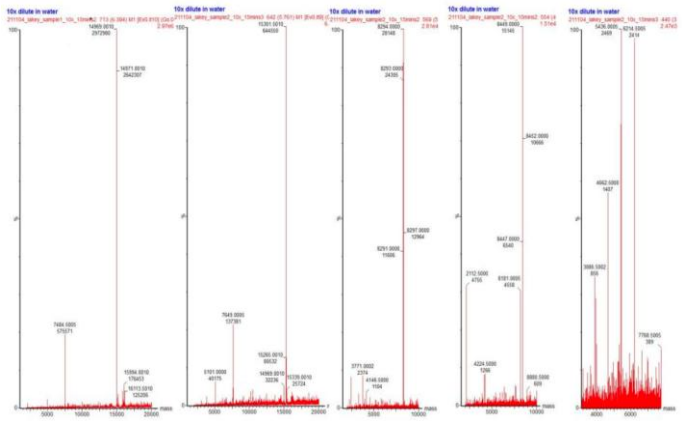

Figure 3: Deconvoluted mass spectrometry data obtained from 2nd batch of MOs (from Jonathan RT Lakey et al., 2022).

The foundational requirement for any therapeutic extract is safety. Preliminary in vitro studies, specifically MTT assays measuring mitochondrial metabolic activity and BALB/c-3T3 two-stage cell transformation assays, report that extraction of Mito Organelles exerts no cytotoxic effects on human-derived MCF-7 breast cancer cells, even under oxidative stress, and does not induce carcinogenic transformation or morphological abnormalities in murine fibroblasts [10]. These data are reassuring in demonstrating that Mito Organelles extracts, across a range of tissue sources, appear benign when applied to diverse cell types in laboratory settings.

While detailed mechanistic study remains absent, a compelling hypothesis guides interest: Mito Organelles extracts may deliver a concentrated complement of mitochondrial proteins and peptides akin to mitochondrial-derived peptides (MDPs) like Humanin or MOTS-c that function as endogenous regulators of cellular resilience, metabolism, and stress adaptation. Given that aging is intimately tied to mitochondrial dysfunction, oxidative stress, and impaired proteostasis, supplementation of mitochondrial components could theoretically reinvigorate cellular bioenergetics, enhance mitophagy, and support stem cell vigor [10,11].

Supporting this, research into agents that modulate mitochondrial quality control, such as dietary polyphenols (e.g., EGCG) and dietary interventions (e.g., caloric restriction, exercise) has shown robust induction of mitophagy, improved mitochondrial turnover, and reduction in reactive oxygen species. For example, green tea polyphenols have been shown to induce mitophagy in human fibroblasts at micromolar concentrations (around 5 µg/mL), thereby pointing toward a model where rejuvenation of mitochondrial populations equates to anti-aging effects. Similarly, natural antioxidants such as Coenzyme Q10, MitoQ, and others bolster mitochondrial efficiency and mitigate age-related oxidative damage. Thus, Mito Organelles extracts may offer a broader, more complex ingredient of mitochondria-associated factors, potentially delivering synergistic benefit in maintaining cellular and tissue-level mitochondrial homeostasis [10,11,12].

Beyond laboratory extracts, early-stage reports, from non-peer-reviewed sources like research communities describe experimental therapies involving direct mitochondrial transplantation. Albeit anecdotal and far from peer-reviewed validation, these narratives underscore growing creative enthusiasm for mitochondrial augmentation as a longevity strategy. Mito Organelles extracts may represent a more tractable and scalable alternative, still harness mitochondrial potency, but avoid the technical, immunologic, and regulatory hurdles posed by live mitochondrial transplant.

Integrating Mito Organelles into Regenerative and Anti-Aging Frameworks

Mito Organelles extracts are integrated into a multimodal regenerative paradigms.

Skin and dermatologic aging

Topical or subcutaneous application might deliver mitochondrial peptides and proteins that optimize local mitophagy and reduce collagen-degrading processes, reminiscent of how smart formulations (like peptides or niacin derivatives) target skin aging [14,15].

Cognitive and neurovascular support

Neurodegenerative disorders ranging from Alzheimer's and Parkinson's to vascular cognitive impairment are increasingly understood as conditions rooted in mitochondrial dysfunction and compromised neuronal bioenergetics. The fragile metabolic equilibrium of brain cells is easily perturbed by oxidative stress, amyloid or α-synuclein accumulation, and age-related mitochondrial decline. Hence, delivering mitochondrial-supportive agents across the blood–brain barrier (BBB) to bolster neuronal resilience and sustain neurovascular integrity represents a frontier in regenerative neurology.

Delivery across the blood–brain barrier (perhaps via modified nanoparticle systems) could reinforce neuronal mitochondrial resilience, potentially protecting against cumulative metabolic stress or incipient neurodegeneration [16,17,18,19].

Delivering Mito Organelles extracts across the BBB via engineered nanoparticles and non-invasive routes like intranasal delivery holds transformative potential for preserving brain mitochondrial function and halting early neurodegenerative changes. With mounting evidence from mitochondrial therapeutics and peptide delivery platforms, this strategy is poised at the confluence of regenerative neurology and mitochondrial medicine ready for systematic exploration and hopeful translation.

Musculoskeletal health

Systemic administration could aid regeneration of aged muscle or stem cell niches, in synergy with exercise-induced mitochondrial biogenesis [20,21]. This narrative aligns with broader anti-aging science, which increasingly focuses on restoring mitochondrial quality control via agents such as fisetin (a plant-derived senolytic that enhances autophagy and mitochondrial polarization), or CoQ10 analogs that preserve OXPHOS and blunt senescence in aging stem cells [22].

Osteoarthritis and muscle repair

In osteoarthritis (OA), impaired mitochondrial function in chondrocytes fuels inflammation and cartilage degradation. A landmark preclinical study demonstrated that mitochondria derived from mesenchymal stromal cells (Mito-MSC), when injected intra-articularly in mice, could home to chondrocytes and synovial cells, modulate gene expression related to DNA repair and antiviral stress responses, and significantly reduce cartilage breakdown, all without triggering inflammation or immune reactions [23]. This suggests that mitochondria, or their component extracts, may act as therapeutic regenerators of joint tissue.

Beyond cartilage, aging and injury compromise skeletal muscle's regenerative capacity, especially in energy-demanding satellite cells (muscle stem cells). The data on Mito Organelles research underscores that mitochondrial health is foundational to muscle repair: mitochondria deliver essential metabolic energy and signal fidelity for stem cell activation and myogenesis [24,25]. One can imagine that applying mitochondria-enriched extracts could bolster muscle recovery, particularly when combined with rehabilitation or resistance training.

Cardiovascular and ischemic tissue repair

Mitochondrial medicine has already crossed into early clinical exploration. In pediatric patients undergoing surgery with ischemia-reperfusion risk, autologous mitochondria injected into the heart improved cardiac recovery and cell viability for nearly 28 days, without adverse immune reactions [26]. Additionally, mitochondrial transfer has shown promise in enhancing endothelial cell bioenergetics, bolstering vascular regeneration, and possibly supporting angiogenesis in damaged tissues [27].

These findings point to potential uses of Mito Organelles extracts for ischemic injury, such as myocardial infarction or peripheral ischemia, especially if delivery systems can ensure local mitochondrial activity [28,29].

MOs a small peptide derived from the mitochondrial genome, has recently garnered attention for its role in regulating metabolic homeostasis and cardiac health. Unlike humanin, which is primarily involved in cell survival, MOs are implicated in metabolic regulation, particularly in the context of stress adaptation and cellular bioenergetics [30].

MOs functions by activating AMP-activated protein kinase (AMPK), a key regulator of cellular energy homeostasis [30]. Through AMPK activation, MOs enhances mitochondrial biogenesis, improves glucose metabolism, and reduces the accumulation of metabolic waste products. Additionally, MOs has been shown to modulate the expression of genes involved in oxidative phosphorylation and mitochondrial biogenesis [30].

In models of heart failure and myocardial ischemia, MOs has been shown to improve myocardial energetics, enhance mitochondrial function, and promote tissue regeneration. By enhancing mitochondrial function and cellular metabolism, MOs may reduce myocardial remodeling and fibrosis, ultimately improving cardiac function [28,30].

MOs also act by stabilizing the inner mitochondrial membrane and reducing mitochondrial oxidative stress. It enhances mitochondrial respiration and ATP production, while also protecting mitochondria from injury caused by ischemia or oxidative stress [54]. MOs has been shown to reduce mitochondrial dysfunction and apoptosis in cardiomyocytes following ischemic injury [28,30].

MOs have been tested in preclinical and clinical studies as a potential therapeutic agent in various cardiovascular diseases. In models of myocardial infarction and heart failure, MOs improves left ventricular function, reduces infarct size, and enhances myocardial regeneration. It has also shown promise in improving the bioenergetic capacity of cardiomyocytes, thereby improving cardiac output and reducing symptoms of heart failure [28].

Wound healing, skin rejuvenation, and hair follicle regeneration

Emerging research exploring mitochondrial transplantation in aging and tissue repair, across wound healing, hair rejuvenation, and skin regeneration has shown robust outcomes in rodent models: accelerated wound closure, reduced scarring, enhanced follicle density, and increased collagen deposition upon mitochondrial injection [31,32]. These hallmarks mirror the goals of anti-aging therapies in dermatology and regenerative aesthetics. Therefore, Mito Organelles extracts could be adapted for dermal or transdermal application, potentially activating local mitochondrial renewal and promoting skin health [33,34].

Discussions

The concept of Mito Organelles cellular extracts enriched in mitochondrial proteins, peptides, and associated biomolecules represents a daring leap in regenerative and anti-aging medicine. Although direct peer-reviewed studies remain conspicuously absent, a rich tapestry of related research provides a compelling theoretical and mechanistic foundation.

First, mitochondrial transplantation studies across cardiovascular, neurological, metabolic, and musculoskeletal disease models underscore the feasibility and potency of introducing functional mitochondria or their components into compromised tissues. For example cardioprotection - autologous mitochondrial injection into ischemic myocardium has demonstrated rapid and sustained improvements in ATP production, proteomic adaptation, and functional recovery, including decreased infarct sizes and enhanced mitochondrial respiration, lasting at least 28 days post-treatment.

Clinical studies in pediatric and adult patients with ischemia-reperfusion injury (IRI) have shown tangible benefits, improved ejection fraction (from ~36% to ~48%), higher exercise tolerance, and expedited weaning from ECMO support with no serious adverse events reported. From enhancing bone healing via mitochondrial transfer in mesenchymal stem cells (BMMSCs), to fostering cutaneous wound repair through platelet-derived mitochondrial potentiation of ADSCs, and promoting neuronal regeneration in central and peripheral nervous systems studies consistently report improved bioenergetics, reduced apoptosis, and functional tissue repair.

The translational logic is clear - Mito Organelles extracts, if they contain similar mitochondrial cargo, might confer comparable benefits, delivering a therapeutic payload of mitochondria-derived factors without the technical complexity, immunogenic risk, and regulatory burden of live organelle transplantation.

Osteoarthritis and age-related muscle degeneration are deeply linked with mitochondrial dysfunction. Injecting Mito Organelles, rich in mitochondrial proteins into affected joints or muscle tissue could energize chondrocytes and satellite cells, activate anabolic and regenerative programs, and inhibit inflammatory cascades. This is especially compelling when combined with physical therapy, where restored mitochondrial function could potentiate exercise response.

The established paradigm of mitochondrial therapy in ischemic heart models inspires a plausible route for Mito Organelles application via intracardiac or intracoronary delivery to support myocardial regeneration post-infarction or during cardioplegia, potentially reducing scarring and improving contractile function.

Skin aging is characterized by reduced collagen, compromised barrier function, and slower healing has been linked to mitochondrial decline. Topical or intradermal delivery of Mito Organelles might enhance keratinocyte and fibroblast bioenergetics, accelerate wound repair, and reduce visible signs of aging, paralleling or surpassing existing peptide-based cosmetic interventions.

Whether delivered locally via intraventricular routes or systemically with nanoparticle assistance, Mito Organelles may bolster neuronal resilience in contexts of stroke, neurodegeneration, or traumatic injury. The restoration of mitochondrial dynamics ATP production, mitophagy balance, and anti-apoptotic signaling—presents a direct intervention pathway in neurologic repair.

Obesity, type 2 diabetes, and nonalcoholic fatty liver disease (NAFLD) all feature mitochondrial dysfunction as a pathogenic hallmark. Although speculative, systemic or targeted delivery of mitochondrial extracts could enhance insulin sensitivity, reduce hepatic steatosis, and restore metabolic control part of a multi-pronged therapy that might include lifestyle modification, mitochondrial-targeting drugs, and personalized extract administration.

But no clinical translation is without significant hurdles, and there are some challenges involved. The molecular identity of Mito Organelles extracts would benefit from further characterization. By knowing which proteins, lipids, peptides, or nucleic acids they contain, reproducibility and mechanism of action can be further established.

Extraction protocols, stability of mitochondrial proteins, sterility, and batch consistency are paramount. Analogous mitochondrial therapies emphasize rigorous criteria for membrane integrity, respiration capacity, and mitochondrial quality markers none of which are publicly documented for Mito Organelles. Effective internalization by recipient cells is non-trivial. Mitochondria alone often require electroporation, conjugated cell-penetrating peptides, or encapsulation in extracellular vesicles (EVs) to achieve meaningful uptake.

Autologous sources minimize immune reaction, but allogeneic extracts (e.g., placenta, donor tissues) risk rejection or inflammatory response. Mitigating this requires thorough immunogenicity testing - a step historically bypassed in wellness-based offerings. Classified as neither drug nor simple supplement, these biologic extracts fall into a regulatory grey zone complicating pathways for clinical trials, marketing, and standardization.

Conclusions

The "Mito Organelles" represent a bold convergence of mitochondrial science and therapeutic innovation. On paper, they offer the promise of enhancing regenerative potential across multiple tissues, they distill the complexity of mitochondrial signaling into an administrable format. Yet, this promise must be tempered by the rigor of empirical validation, quality assurance, and ethical oversight.

If properly characterized, standardized, and tested, these mitochondrial extracts could become a cornerstone in regenerative and anti-aging medicine - a bridge between diminutive molecular regulators and whole-tissue restoration. Until then, scholarly curiosity must guide cautious advancement to avoid the pitfalls of overpromised yet unsubstantiated therapies.

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Institutional review board statement

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Data availability statement

The data presented in this study are available in the study outlined.

Conflicts of interest

The funders had no role in the design of the study; in the collection, analyses, or interpretation of data; in the writing of the manuscript, or in the decision to publish the results.

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