American Journal of
Biomedical Science & Research

www.biomedgrid.com
ISSN: 2642-1747

Mini Review

Next Gen in Neurorepair: Peptide Therapeutics for the Injured and Aging Brain

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To Cite This Article: *Mike K S Chan, Michelle B F Wong, Krista Casazza, Ian Jenkins and Jonathan R T Lakey. Next Gen in Neurorepair: Peptide Therapeutics for the Injured and Aging Brain. Am J Biomed Sci & Res. 2025 26(6) AJBSR.MS.ID.003509,* **DOI:** 10.34297/AJBSR.2025.26.003509

Received: May 06, 2025; **Published:** May 13, 2025

Abstract

CNS-specific NOPs and MO peptides show promise in enhancing mitochondrial function, reducing neuroinflammation, and supporting CNS-specific repair, offering a compelling alternative to conventional neuropharmacologic therapies. Advances in proteomic profiling, including MALDI-TOF mass spectrometry, have enabled the detailed characterization of neuroactive peptide formulations, revealing compositional diversity that informs optimization and standardization. Despite this progress, significant challenges persist, including limited blood-brain barrier permeability, peptide instability, potential immunogenicity, and high manufacturing costs. Emerging strategies such as nanoparticle-based CNS delivery, peptide-exosome hybrids, and precision neurotherapeutics tailored to genetic and inflammatory profiles aim to overcome these hurdles. As the field of neuroregenerative medicine matures, peptide therapies are positioned to become central to next-generation approaches for neurological repair, offering biologically precise, multifaceted tools to address the unmet clinical needs in CNS injury and neurodegeneration. This review outlines the current landscape, emerging innovations, and future directions of peptide-based therapeutics in neuroregeneration.

Keywords: Therapeutic Peptide, CNS Injury, Neuroinflammation, Axonal Regeneration

Introduction

The central nervous system (CNS) functions as the master regulator of bodily processes, integrating sensory input, coordinating motor activity, and supporting cognition and homeostasis. Due to its complexity and limited regenerative capacity, the CNS is particularly vulnerable to lasting damage following injury. Traumatic brain injury (TBI), spinal cord injury (SCI), stroke, neuroinflammatory conditions such as multiple sclerosis, and neurodegenerative diseases like amyotrophic lateral sclerosis (ALS) can all lead to profound and often irreversible neurological deficits. These insults disrupt neuronal circuitry, compromise the blood-brain barrier, and trigger cascades of inflammation, cell death, and tissue remodeling that can extend far beyond the initial injury site.

Unlike peripheral tissues, the CNS exhibits only modest intrinsic regenerative ability. While some neurogenesis and axonal sprouting can occur, particularly in neurogenic niches, repair is often thwarted by inhibitory environmental cues, glial scarring, and a lack of robust endogenous mechanisms for cell replacement. CNS healing involves a complex orchestration of cellular and molecular responses, including microglial and astrocyte activation, angiogenesis, axonal guidance, and synaptic reorganization. However, these responses are often maladaptive, leading to chronic inflammation, glial scar formation, and limited functional recovery.

In this challenging landscape, peptide-based therapies are emerging as promising tools to modulate the CNS microenvironment and promote repair. Peptides can mimic neurotrophic factors, support neuronal survival, attenuate neuroinflammation, and enhance neurogenesis and axonal regeneration. Several therapeutic peptides are in various stages of development, offering novel avenues to improve outcomes after CNS injury. As insights into neural repair mechanisms grow, peptide strategies are poised to become integral to regenerative approaches aiming to restore structure and function in the injured CNS.

CNS repair follows a temporally coordinated sequence of events, acute injury response, subacute remodeling, and chronic adaptation, with each phase presenting opportunities and challenges for intervention. The acute phase is marked by neuronal and glial cell death, blood-brain barrier disruption, and a rapid influx of immune cells. Microglia and infiltrating macrophages release cytokines and reactive oxygen species that clear debris but can also exacerbate tissue damage if unchecked. In the subacute phase, astrocytes proliferate and form a glial scar that walls off the injury but inhibits axonal regrowth. Endogenous progenitor cells may proliferate, and neurovascular remodeling begins, supported by angiogenic signaling and extracellular matrix reorganization. The chronic phase involves stabilization of the lesion site, with persistent inflammation, demyelination, and limited neuroregeneration. Successful long-term repair requires modulation of glial responses, re-establishment of synaptic connectivity, and integration of new or spared neural elements into functional networks.

A myriad of cellular players are integral to the three phases of CNS regeneration. Neurons and neural progenitor cells are central to restoring lost circuitry, with some endogenous neurogenesis occurring in specialized niches such as the subventricular zone and hippocampal dentate gyrus. Astrocytes, while often implicated in forming the glial scar, also contribute to repair by secreting neurotrophic factors, regulating extracellular ion balance, and supporting synaptogenesis. Oligodendrocyte precursor cells (OPCs) proliferate and differentiate to remyelinate axons, a critical step in restoring signal conduction. Endothelial cells facilitate angiogenesis, re-establishing vascular integrity and supplying nutrients and oxygen to regenerating tissue.

Immune modulation is equally essential in CNS repair. Microglia and infiltrating macrophages orchestrate the inflammatory response, clearing debris and secreting cytokines that can either support or inhibit regeneration depending on their activation state. The gut-brain axis, via circulating microbial metabolites, cytokines, and vagal signaling, has emerged as a key modulator of CNS immunity and repair, influencing microglial activation, neuroinflammation, and even neurogenesis.

Successful CNS regeneration also hinges on the activation of intrinsic signaling pathways that regulate progenitor cell fate, axon growth, and glial behavior. Wnt signaling plays a pivotal role in neural progenitor proliferation and neurogenesis, while Notch signaling is crucial for maintaining stem cell quiescence and directing lineage differentiation. Growth factors such as transforming growth factor-β (TGF-β) and brain-derived neurotrophic factor (BDNF) are vital for balancing inflammation, promoting neuronal survival, and guiding axonal sprouting. Epidermal growth factor (EGF) and fibroblast growth factor-2 (FGF-2) have been shown to enhance neural stem cell proliferation and glial responses during repair.

Collectively, these orchestrated cellular and molecular processes attempt to restore CNS function after injury. However, dysregulation-such as chronic inflammation, excessive gliosis, or insufficient neurogenesis-can severely impair repair, leading to permanent deficits. These complexities highlight the need for targeted therapies that can modulate the CNS microenvironment to support and enhance endogenous regenerative mechanisms.

The Emerging Role of Peptide-based Therapies

Peptide-based therapies have emerged as promising tools in regenerative medicine, particularly for promoting gut healing following injury [1-13]. Short chains of amino acids with diverse biological activities, therapeutic peptides can precisely target key pathways involved in tissue repair, offering advantages in specificity, bioavailability, and safety compared to larger biologic agents or small-molecule drugs [14]. A principal mechanism by which peptides facilitate gut regeneration is by stimulating epithelial proliferation and migration. Maintaining tight junction integrity is also essential for preserving the gut's selective permeability and defense

against luminal pathogens. Certain peptides enhance the expression and assembly of tight junction proteins, thereby reinforcing barrier function and preventing secondary infections or chronic inflammation. The modulation of inflammation is another critical action of regenerative peptides. Anti-inflammatory peptides can suppress excessive cytokine production while promoting the release of pro-repair factors, creating a controlled inflammatory environment that supports rather than impedes healing [13]. Peptides also contribute to angiogenesis, essential for restoring oxygen and nutrient supply to regenerating tissues. Pro-angiogenic peptides upregulate vascular endothelial growth factor (VEGF) signaling, stimulating the formation of new capillaries within damaged gut tissue. The anti-apoptotic effect on enterocytes, by which peptides can inhibit pro-apoptotic pathways and enhance cell survival under stress conditions such as ischemia, inflammation, or oxidative damage, preserving the epithelial cell pool needed are also requisite for effective regeneration [15]. Finally, emerging evidence suggests that peptides can influence the gut microbiome, fostering a microbial environment conducive to healing. Certain antimicrobial peptides selectively modulate bacterial populations, enhancing the growth of beneficial commensals while suppressing pathogens that exacerbate injury and inflammation [13].

Clinical Research Landscape

The capacity of peptides as critical mediators of communication between cells and tissues has rapidly advanced therapeutic treatment, but their structural complexity and organ/tissue specificity have posed challenges for medical understanding. Some peptides exert localized or systemic anti-inflammatory and regenerative effects by binding to organ- or tissue-specific receptors, while others, as some of the aforementioned act broadly across multiple tissues. Indeed, peptides perform diverse functions, relaying information, modulating metabolism, regulating inflammation, and serving as biomarkers, highlighting their essential roles in tissue regeneration, immune response, and neurological processes. Proteomic studies have detected the expression of over 150 distinct mature peptides. Through years of research and extensive global practice, MF-Plus has manufactured two products, Nano Organo Peptides (NOPs) and Mito Organelles (MO) Peptides, which are intended for use in both animals and humans as a revitalization therapy. Mito Organo (MO) peptides are biologically extracted mixtures of cellular peptides that have predominantly mitochondria-specific functions23. Although cells of different organ systems have similar functions, variations in cellular functions between organs create 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. Recently, MF-Plus manufactured and tested heart-oriented NOP for cardiac patients. The NOPs are organ-specific, thus making it possible to choose the range of the NOP needed for a particular patient. It could be injected intramuscularly and via noninvasive routes (sublingual, intranasal).

The brain is an energetically demanding organ, utilizing roughly 20% of the body's total caloric intake despite comprising only about 2% of total body weight. This high energy consumption is primarily driven by the need for adenosine triphosphate (ATP) to sustain ionic gradients that are essential for effective neurotransmission and neural plasticity. Mitochondria are central to these processes, playing crucial roles in neural development and function, including the proliferation, differentiation, and maturation of neural stem cells. They also support the extension of dendrites and contribute significantly to both developmental and synaptic plasticity. Moreover, mitochondria are key regulators of cell survival and programmed cell death. Peptides are intricately linked to NDDs. Over the past decades, researchers have tirelessly pursued understanding the pathogenesis of NDDs through extensive basic studies and clinical trials targeting peptides for treatment. This endeavor has led to the development of several marketed peptide-based drugs. However, the complexity of neurodegenerative disease mechanisms remains beyond our current understanding. Further investigation is needed to elucidate their mechanisms of action, and scientists must explore novel formulations and delivery methods to optimize the functionality of peptides.

Analogously, just as non-neural organs secrete neurotropic factors, peripheral tissues-including skeletal muscle, liver, and immune cells-release peptides and signaling molecules that influence CNS repair. As demonstrated in other organ systems, combinations of mitochondria-derived and organ-specific peptides (NOPs) sourced from muscle, endothelial cells, and hematopoietic tissues may be harnessed to support neuroregeneration. Neurons and glial cells are particularly reliant on mitochondrial function due to their high metabolic demands and limited regenerative capacity. In the setting of acute injury (e.g., stroke or spinal cord trauma) or chronic neurodegenerative disease (e.g., Alzheimer's, Parkinson's), mitochondrial dysfunction is a central driver of pathology-amplifying oxidative stress, disrupting ATP production, and impairing cellular signaling pathways.

Mitochondria, along with the nucleus, are the only organelles that harbor their own DNA. Human mitochondrial DNA (mtDNA) encodes 13 protein-coding genes and 24 RNA molecules essential for mitochondrial protein translation. Although the peptide repertoire encoded by mtDNA is small, it includes mitochondria-derived peptides (MDPs) which have demonstrated neuroprotective effects. The first MDP to be discovered was humanin, a 24–amino acid peptide encoded from the 16S rRNA region of mtDNA cloned from the resilient occipital lobe of an Alzheimer's disease patient's brain and found that the peptide protected against amyloid-β toxicity in neuronal cells. Humanin was also shown to protect neurons from amyloid-beta and oxidative stress-induced apoptosis . Recently, Kim showed a novel role of humanin administered intranasally to improve in mitochondrial function and neuronal survival in Parkinson's disease. Similarly, MOTS-c, a peptide encoded by the short open reading frame of the mitochondrial 12S rRNA gene significantly expressed in response to stress or exercise and

translocated to the nucleus, was shown to modulate inflammatory responses and metabolic signaling in glial cells . Moreover, small humanin-like peptide 2 (SHLP2), an MDP implicated in several biological processes such as aging and oxidative stress was shown to support mitochondrial homeostasis and neuron survival under stress conditions.

Recent studies suggest that mtRNA expression patterns are altered in specific CNS cell types following injury, influencing the local production of protective peptides. Region-specific or cell-specific mitochondrial transcriptomes-such as those in astrocytes, microglia, or oligodendrocyte precursor cells-may dictate the type and abundance of MDPs, potentially shaping the regenerative response. Additionally, peripheral administration of synthetic MDPs has been shown to cross the blood-brain barrier, opening the door to systemic delivery strategies for CNS repair.

Restoration or augmentation of these MDPs represents a promising direction in bioregenerative neurology. By targeting mitochondrial resilience and leveraging the crosstalk between CNS and peripheral tissues, peptide-based therapies aim to re-establish cellular homeostasis, reduce neuroinflammation, and promote synaptic and axonal repair. As understanding of mitochondrial signaling and neuro-immuno-metabolism grows, these strategies may become key tools in the treatment of stroke, neurodegeneration, and traumatic CNS injuries.

While peptides offer significant promise as therapeutics for CNS disorders, delivering them effectively to the brain remains a key challenge. Contrary to common belief, the primary barriers to CNS delivery are not always the blood-brain barrier (BBB) itself. Many peptides are capable of crossing the BBB through both saturable and non-saturable pathways, often reaching brain concentrations sufficient to trigger physiological effects. Instead, peripheral limitations-such as rapid degradation and short blood half-life-are often more critical in reducing therapeutic efficacy. Developing peptides that are resistant to enzymatic breakdown and have prolonged circulation times can enhance CNS delivery, even if BBB penetration is relatively low. Notably, peptides can exert CNS effects at surprisingly low concentrations, meaning that effective brain doses are typically smaller than those required for peripheral action. Additionally, brain-to-blood efflux mechanisms can significantly hinder peptide accumulation in the CNS, presenting another obstacle to therapeutic success. However, the exact makeup of these formulations is essential for effective transport. Matrix-assisted laser desorption/ionization time of flight (MALDI-ToF) mass spectrometry has been able to identify and quantify analytes in complex solutions and allows for highly sensitive, fast and high-throughput analysis [16-25].

By comparing experimental mass spectrometry (MS) data with that of well-established open-source databases, a determination can be made of the proposed identity of the molecules, peptides, or proteins found within a solution. Due to the low-cost and rapid application of MS in identifying the components of unknown solutions, our study employed MS as our primary method of identification [26]. Although our preliminary data relied upon LC-MS/MS based peptide sequencing techniques to produce chromatograms and deconvolute our data MALDI-TOF utilizes a protein fingerprinting method in which the sample is digested by a proteolytic enzyme such as trypsin and used to generate an MS spectrum that can be searched against existing databases [27]. Matched hits are ranked according to a scoring method in which the candidate protein that contains more proteolytic peptides has a higher score and generally represents the most probable protein/peptide. The desirability of MALDI-TOF also includes the speed at which each run is performed, often less than one minute to obtain– and the speed at which analysis can be performed against a database. MALDI-TOF mass spectrometry offers a powerful tool for characterizing the molecular composition of NOPs and MO peptides, enabling detailed profiling of peptide heterogeneity, structural integrity, and post-translational modifications critical for CNS applications. By precisely identifying and quantifying bioactive peptide species within complex NOP and MO formulations, MALDI-TOF helps refine therapeutic candidates for optimal blood-brain barrier penetration, enzymatic resistance, and receptor specificity. This analytical precision supports the development of peptide therapies tailored to target mitochondrial dysfunction and neuroinflammation-key drivers of CNS degeneration. Furthermore, MALDI-TOF facilitates batch-to-batch consistency and regulatory compliance by ensuring quality control and reproducibility, which are essential for translating peptide-based neuroregenerative therapies into clinical use.

Neuro-Regeneration: Trends, Innovations, Challenges, and Limitations

Recent trends in peptide therapy for neuro-regeneration reflect growing momentum toward harnessing targeted, multimodal approaches to repair and restore function in the injured or degenerating central nervous system. Innovations in delivery technologies (e.g., including intranasal administration, nanoparticle conjugation, and exosome-mediated transport) are being explored to facilitate the crossing of the BBB and ensure localized delivery of therapeutic peptides to affected neural tissues. Engineered peptides are being developed with enhanced receptor affinity and metabolic stability, addressing long-standing issues of rapid degradation and poor bioavailability. Combination strategies are also advancing, particularly those that pair neuroprotective peptides with anti-inflammatory agents, stem cell-based therapies, or neuromodulation techniques (e.g., electrical stimulation or transcranial magnetic stimulation). These integrative approaches aim to amplify regenerative signaling cascades while modulating the neuroimmune environment to support sustained repair. In parallel, efforts in personalized neurotherapeutics are beginning to incorporate patient-specific genetic, transcriptomic, and even gut-brain axis profiles to tailor peptide interventions and optimize outcomes.

Despite these innovations, key challenges remain. Delivering peptides effectively to the CNS remains one of the most formidable

barriers, often requiring invasive techniques or complex carriers to bypass the BBB. Immunogenicity also poses a potential risk, particularly with synthetic or modified peptides. The therapeutic window for intervention, particularly in acute injuries like stroke or spinal cord trauma, can be narrow, making timing and dosing critically important. Moreover, the long and costly development pipelines for neuroactive peptides, combined with limited predictive biomarkers of neural repair, hinder the translation of promising candidates into clinical practice. There is also a need for more sophisticated models of CNS injury that capture the complexity of human neurodegeneration and regeneration, as traditional rodent models often fall short in replicating chronic or multifactorial conditions like Alzheimer's disease or traumatic encephalopathy. To fully unlock the potential of peptides in neuro-regeneration, continued innovation in peptide engineering, delivery systems, and precision medicine frameworks will be essential, alongside expanded clinical trials and regulatory pathways that recognize the nuanced challenges of CNS repair.

European Wellness

European Wellness Biomedical Group (EW) is a globally recognized, biological regenerative medicine, known for its groundbreaking advancements in precursor stem cell therapeutics, immunomodulation, and peptide-based innovations. EW has amassed over 40 international patents and contributed extensively to scientific literature surrounding peptide therapeutic option including neuro-regenerative conditions as well as others. Founded in the early 1990s, EW emerged from collaborative research across several countries, laying the foundation for a multinational enterprise with comprehensive capabilities in R&D, GMP bio-manufacturing, clinical application, and global distribution. Today, with operations spanning 80 countries, the EW is uniquely positioned to spearhead peptide-based therapies for neuroregeneration. EW is positioned to strategically leverage the growing understanding of brain cell diversity and intercellular interactions to advance its peptide therapy initiatives for neuroregeneration. By aligning R&D efforts with emerging insights into the distinct roles and communication pathways of neurons, glial cells, and other brain-resident cell types, EW is designing highly targeted peptide formulations that modulate specific cellular functions and repair mechanisms. This precision approach enables the development of therapies tailored to the unique regenerative needs of various neurological conditions, including neurodegenerative diseases, brain injuries, and psychiatric disorders. Coupled with their expertise in peptide bioengineering and analytical tools such as MALDI-TOF for quality control and peptide profiling, EW is well-positioned to translate cutting-edge brain science into clinically impactful neuroregenerative therapies that restore function and enhance brain resilience.

Future Directions

The future of peptide-based gut regeneration is rapidly evolving toward more targeted, personalized, and integrative approaches. One major direction involves the expansion of peptide libraries designed to mimic or enhance endogenous regenerative signals, such as Wnt, R-spondin, and Notch pathway modulators, offering the potential to fine-tune epithelial repair and stem cell niche function. Concurrently, the integration of peptides with stem cell and organoid-based therapies is opening new avenues for ex vivo modeling and in vivo tissue restoration, where peptides can promote engraftment, differentiation, or niche maintenance. Another emerging area is the study of peptide-microbiome interactions, which is revealing how microbial communities influence and respond to therapeutic peptides, shaping local immune responses and regenerative capacity. Looking forward, precision regenerative medicine strategies are gaining traction by leveraging genomic and proteomic profiling to match patients with optimal peptide therapies, enabling stratified interventions tailored to individual disease states or injury responses. Ongoing and upcoming clinical trials are increasingly testing these next-gen peptide constructs in conditions such as inflammatory bowel disease, short bowel syndrome, and radiation-induced enteropathy, suggesting a promising horizon for safe, modular, and biologically intelligent gut repair.

Conclusion

Peptide therapy has emerged as a promising modality for neuro-regeneration, with accumulating preclinical and early clinical evidence demonstrating its capacity to support neuronal survival, promote neurogenesis, enhance glial function, and modulate critical regenerative pathways such as Wnt, Notch, BDNF, and EGF signaling. Specific peptides, including MDPs (e.g., humanin, MOTS-c, engineered neurotrophic factors) have been shown to reduce neuroinflammation, protect against oxidative stress, and stimulate synaptic repair in models of stroke, traumatic brain injury, and neurodegenerative diseases such as Alzheimer's and Parkinson's. Despite these encouraging developments, comprehensive clinical research is needed to fully establish the safety, efficacy, and long-term viability of peptide therapies in diverse neurological conditions and patient populations. Rigorous, well-designed trials are essential to validate outcomes, refine delivery methods, determine optimal dosing regimens, and assess neurofunctional recovery over time. If these challenges can be addressed, peptide-based therapies hold strong potential to reshape current treatment paradigms, offering targeted, mechanism-driven alternatives to traditional neuropharmacologic or palliative interventions in CNS injury and neurodegeneration.

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