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Emerging Anabolic and Regenerative Peptides in Athletic Body Re-composition and Bodybuilding: Mechanisms of Action, Dosing Strategies, and Evidence Review

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Abstract

This review examines prominent peptides used in athletic and bodybuilding contexts—such as BPC-157, HGH Fragment 176–191, CJC-1295, Ipamorelin, Apelin, Mitochondrial peptides, Folli statin (myostatin inhibitor), and GLP-1, detailing their mechanisms, typical dosing, purported benefits, and evidence base. It highlights the limitations of current data, safety concerns, and regulatory considerations.

Keywords

Body recomposition; Peptides; Mitochondrial peptides; Growth hormone; Myostatin inhibitor; Anabolic; Regenerative medicine; Bodybuilding.

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Introduction

Peptides - short chains of amino acids are increasingly being leveraged by athletes, bodybuilders, and biohackers aiming to enhance recovery, promote fat loss, stimulate anabolic pathways, and improve overall athletic performance. Their rising popularity is anchored in the belief that they mimic endogenous signaling more closely than traditional anabolic steroids or growth hormone therapy, offering a seemingly "natural" approach to performance enhancement. Enthusiasts tout peptides like BPC-157, CJC-1295, Ipamorelin, and Tesamorelin as tools to reduce inflammation, accelerate healing, and optimize body composition [1-3].

Despite the buzz, the supporting scientific evidence remains limited. Most claims stem from small-scale human studies, in vitro research, or animal models, rather than robust, randomized clinical trials in well-trained athletes [1,4]. For instance, collagen peptides have shown potential to support lean mass gains and strength improvements when paired with resistance training in some cohorts, yet other studies report mixed or non-significant outcomes [5,6]. A systematic review, meta-analysis and meta-regression of the effect of protein supplementation on resistance training-induced gains in muscle mass and strength in healthy adults [7,8]. Similarly, whey hydrolysate supplementation may accelerate force recovery and reduce biomarkers of muscle damage post-exercise, though the broader applicability remains uncertain [1].

Concurrently, interest in peptides extends beyond athletic performance. Peptides are central to therapeutic avenues ranging from insulin and GLP-1 analogs to skin and joint health supplements, leveraging their efficiency in cellular signaling and tissue repair [9,10].

Yet, concerns about safety, regulation, and quality control are mounting. Unlike approved pharmaceuticals, many performance-oriented peptides are unregulated "research chemicals" easily obtained online or at the gym or medical spa, thus raising risks of contamination, mislabeling, dosing inconsistencies, or harmful impurities [11]. The Food and Drug Administration (FDA) has responded by ramping up restrictions; for example, compounds previously permissible under compounding now face scrutiny, and the list of peptides considered unsafe has expanded dramatically [12,13].

The legal landscape adds further complexity. Many peptides commonly used for performance enhancement, such as growth hormone secretagogues including GHRP-6, GHRP-2, CJC-1295, and Ipamorelin, which are prohibited by the World Anti-Doping Agency (WADA), exposing athletes to the risk of doping violations and sanctions [11,14].

In summary, while peptides offer enticing potential for athletic body recomposition via recovery support, fat metabolism, and anabolic stimulation, current scientific validation is preliminary and fragmentary. Their appeal is heightened by narratives of safer, "natural" alternatives to traditional agents, yet these are often fueled by anecdotal reports and marketing rather than evidence. Growing regulatory enforcement, quality control challenges, and doping regulations underscore the pressing need for rigorous research and ethical considerations. Bodybuilders, athletes, and practitioners should approach

peptide use with caution, relying on clinical oversight, validated sourcing, and evolving scientific data but not the hype.

Objectives

In this review, we aim to guide readers through a comprehensive narrative that unfolds the science, practices, and governance surrounding emerging peptides in athletic body recomposition.

First, we will explore the biological mechanisms by which key peptides, ranging from growth hormone secretagogues (GHSs) like CJC-1295, Ipamorelin, and GHRP-6, to regenerative agents like BPC-157, support hypertrophy, enhance recovery, and modulate body composition. GHSs act through the GH–IGF-1 axis, often via GHRH analogs or ghrelin receptor agonism, to raise systemic anabolic hormone levels and trigger intracellular pathways such as JAK-STAT and MAPK/ERK. Meanwhile, peptides like BPC-157 facilitate tissue repair and inflammation control by promoting angiogenesis and cellular migration, the mechanisms that underscore their growing use among injury-prone athletes.

Next, we will detail typical dosing protocols as reported in lay and semi-clinical communities. We will then evaluate the supporting evidence, balancing the promises of these peptides against their risks and limitations. While anecdotal reports and small studies suggest benefits, such as enhanced lean mass and recovery, the scientific backing remains sparse, with few rigorous, randomized trials in athletic populations. Safety concerns include glucose metabolism disruption, fluid retention, potential desensitization, and uncharted long-term effects. Moreover, the preclinical and early-phase data do not yet justify widespread performance applications.

Taking into consideration recent interest to Glucagon-like peptide-1 receptor agonists (GLP-1 RAs), we will look into possible applications of this group of peptides in athletic body recomposition and bodybuilding.

Finally, we will discuss regulatory and safety considerations facing athletes and practitioners. Though peptides like Mitochondrial peptides, CJC-1295, Myostatin inhibitors and BPC-157 are frequently marketed for performance, they are not FDA-approved for such use, and enforcement actions are increasing—particularly against products labeled “for research only”. Furthermore, prominent anti-doping organizations, including WADA, prohibit many GH secretagogues and related compounds, exposing athletes to the risk of sanctions—even as detection methods continue to evolve. Given the variability in product quality and potential health hazards, caution and medical oversight remain essential.

Peptides Overview

Body protection compound 157 (BPC-157)

Mechanisms of action

BPC-157, a stable gastric-derived Penta decapeptide, has demonstrated potent regenerative effects by promoting angiogenesis and tissue repair in preclinical models. In rat injury models involving crushed muscle, transected tendon, and Achilles tendon repair, systemic administration of BPC-157 enhanced healing via upregulation of VEGF, CD34, and VEGFR-2, and activation of the VEGFR-2/Akt/eNOS signaling

pathway, resulting in improved endothelial protection, granulation tissue formation, and collagen organization [15,16].

Additional studies confirmed that BPC-157 promotes fibroblast proliferation, tendon outgrowth, and improved survival via the FAK–paxillin pathway, without triggering ossicle formation, suggesting its healing action is tissue-specific and avoids ectopic calcification [17].

BPC-157 possesses notable anti-Inflammatory and cytoprotective effects. Thus, in rodent models of gastrointestinal injury and adjuvant arthritis, BPC-157 administered intraperitoneally at doses as low as 10 µg/kg markedly reduced NSAID-induced mucosal lesions and mitigated arthritic progression, demonstrating both anti-inflammatory and mucosal protective properties [18].

Within the central nervous system, BPC-157 administered after ischemia–reperfusion in rat hippocampal tissue preserved neuromotor function, improved cognitive performance, and modulated gene expression by upregulating VEGFR2, Egr1, Nos3 while downregulating Nos2 and NF-κB, which is consistent with neuroprotective and antioxidant mechanisms [19].

Given its promotion of angiogenesis, BPC-157 raises theoretical concerns for tumor progression. VEGF signaling stimulated by BPC-157 may potentially facilitate neovascularization within tumors, and elevated Egr-1 expression could further influence malignancy. Although one murine study did not show increased tumor size following BPC-157 administration, the long-term oncologic safety remains undetermined [20].

Dosing protocols

Reported dosing regimens are largely based on informal and non-clinical sources of usage of injectables and oral routes. Commonly observed protocols include:

- **Injectable:** 250–500 µg/day, divided into two administrations (e.g., before bed and post training); typical cycles last 4–12 weeks, followed by an equal-length rest period [21].
- **Oral:** Higher total doses ranging from 500 to 1,000 µg/day are often recommended for gastrointestinal indications, administered in two divided doses on an empty stomach.
- **Cycling Protocols:** A common community-derived pattern is 4 to 8 weeks on followed by 2 to 4 weeks off to mitigate potential tolerance effects [21].

Expected benefits

A growing body of preclinical research supports the regenerative properties of BPC-157, particularly in musculoskeletal and gastrointestinal tissue healing.

BPC-157 notably accelerated recovery from Achilles tendon transection in animal model, promoting granulation tissue formation, endothelial protection, and improved collagen organization. Its action seems mediated by enhanced NO synthesis, granulation, and upregulation of focal adhesion kinase (FAK) and paxillin pathways, facilitating tendon fibroblast migration and survival under oxidative stress [16].

Additional studies demonstrated improved outcomes in muscle, ligament, and bone healing, including restoration of tendon-to-bone and muscle-to-tendon continuity within six weeks, without ectopic bone formation that represents a key advantage over BMP-mediated regeneration [22].

A systematic review covering literature through mid-2025 identified 35 preclinical studies and only one human clinical case series—a small retrospective report in which 7 out of 12 patients experienced relief from chronic knee pain following intra-articular BPC-157 injections [21].

Safety and regulatory considerations

BPC-157 is not approved by the U.S. Food and Drug Administration for therapeutic or compounding use; it is classified as an unapproved new drug and is prohibited from inclusion in dietary supplements.

Under WADA's current Prohibited List (S0 category - Non-Approved Substances), BPC-157 is banned in athletes at all times (in and out of competition), with no exemptions permitted [24].

No controlled human trials have established the safety, tolerability, or effective dosing of BPC-157. Animal studies have not reported adverse events, but human data are lacking. Theoretical and practical risks include unregulated sourcing, when products labeled "for research only" may be contaminated, mislabeled, or inconsistent in purity and potency's; and angiogenesis-related tumor risk, especially in individuals with existing cancers. Systemic toxicity and long-term effects also remain unknown (chart 1).

Domain	Key Insights
Mechanisms	Enhances angiogenesis, fibroblast migration, anti-inflammatory and neuroprotective pathways; tissue-specific regeneration.
Dosing Practices	Informal protocols: injectable (250–500 µg/day), oral (500–1,000 µg/day), with 4–12 week cycles and break periods.
Evidence & Benefits	Preclinical evidence only; anecdotal claims of rapid recovery lacking robust clinical validation.
Safety & Regulatory	Not FDA-approved; banned by WADA; safety unproven in humans; potential risks include tumor growth and contamination concerns.

Chart 1: Summary for BPC-157.

Human Growth Hormone (HGH) Fragment 176–191

Mechanisms of action

HGH Fragment 176–191, also known as AOD9604 in its modified form, is a peptide derived from the C-terminal region of human growth hormone. Distinct from the full-length hormone, this fragment selectively promotes lipolysis, the breakdown of stored fat, while inhibiting lipogenesis, without triggering systemic growth effects or elevating IGF-1 levels [25].

Mechanistically, the fragment predominantly targets adipose tissue via activation of beta-3 adrenergic receptors (β3-AR), thereby increasing fatty acid mobilization and thermogenesis especially in visceral fat stores. Notably, rodent studies found that obese mice experienced approximately a 50% reduction in

weight gain over three weeks of treatment, compared to controls. Lean mice, however, maintained baseline body weight, suggesting a context-dependent lipolytic action [26]. Importantly, Fragment 176–191 does not elevate IGF-1, disturb glucose metabolism, or induce adverse metabolic effects typically associated with full-length HGH, reaffirming its targeted lipolytic profile [26].

Dosing protocols

Dosages commonly cited in user reports and research protocols range from 250 to 500 µg per day, typically administered via subcutaneous injection. Dosing is often split into one or two injections, with timing optimized for lipolysis—commonly before exercise, in a fasted state, or at bedtime [27].

Cycle durations vary from 4 to 12 weeks, particularly in fat loss contexts, followed by rest periods. Some protocols advocate 8–12-week cycles when focusing on body recomposition [27].

Expected benefits

In obese rodent models, Fragment 176–191 enhances thermogenesis, increases fat oxidation via β3-AR signaling, and suppresses weight gain without affecting lean body mass [28]. It suppresses lipoprotein lipase activity in adipose tissue, contributing to reduced fat deposition [26].

When combined with hyaluronic acid in osteoarthritic rabbit models, it promoted cartilage regeneration, therefore suggesting potential applications beyond fat loss [29].

Safety and metabolic profile

Across six clinical trials (IV and oral dosing), Fragment 176–191 did not alter vital signs, ECG findings, glucose levels, or serum IGF-1, and was devoid of insulin resistance or impaired glucose tolerance [30]. Adverse events reported were generally mild, such as headache, fatigue, or injection-site reactions, and were comparable to placebo. AOD9604's safety profile is favorable, lacking HGH-associated side effects such as edema, insulin resistance, or IGF-1 elevation, supporting a more favorable safety expectation for Fragment 176–191.

HGH Fragment 176–191 through its lipolytic-focused action and favorable preliminary safety profile represents an intriguing candidate for interventions aimed at fat reduction without the drawbacks of full-length HGH. Yet, the absence of direct human studies and formal regulatory approval necessitates cautious interpretation. Controlled clinical trials are essential to substantiate its efficacy and safety in humans before any therapeutic recommendation is justified.

CJC-1295 (with and without DAC)

Mechanism of action

CJC-1295 is a synthetic analog of growth hormone-releasing hormone (GHRH), designed to stimulate the pituitary gland to release growth hormone (GH), thereby increasing insulin-like growth factor-1 (IGF-1) levels. The DAC (Drug Affinity Complex) variant features an albumin-binding extension that prolongs its half-life to approximately 5.8–8 days, enabling sustained GH and IGF-1 stimulation following a single injection [31]. In healthy adult subjects, single subcutaneous doses of CJC-1295 led to a 2- to 10-fold rise

in GH for six days or more and a 1.5- to 3-fold increase in IGF-1 that persisted for 9–11 days; repeated administration maintained IGF-1 elevation for up to 28 days [31].

In contrast, the non-DAC variant (also called Mod GRF 1-29) mirrors GHRH's natural pulsatile release but has a short half-life of approximately 30 minutes, necessitating frequent dosing to mimic physiological GH dynamics [32,33].

Dosing protocols

- **With DAC:** Clinical investigations in humans have administered doses in the range of 30–60 µg/kg, translating to 2.5–5 mg weekly for an 80 kg individual [31]. Common practice suggests 1–2 mg per week, typically via one to two injections, across 8–12-week cycles, followed by 4–6-week rest [31,34].
- **Without DAC:** Dosing is user-dependent, ranging approximately 100–200 µg per injection, administered 2–3 times daily to maintain pulsatile GH release [35]. Unrecorded anecdotal protocols among bodybuilders also include daily administration 400-600 µg post resistance training.

Expected benefits

CJC-1295 consistently elevates GH and IGF-1 levels for extended durations, presenting potential benefits in muscle growth, fat loss, recovery, and anti-aging interventions. These effects are supported by its pharmacodynamics and early clinical biomarkers but remain to be validated in large-scale, outcome-based [36].

Safety and regulation

Clinical trials report that CJC-1295 is generally well tolerated, with mild side effects such as fluid retention and injection-site reactions; however, elevated GH levels may predispose to insulin resistance or carpal tunnel-like symptoms with prolonged exposure [31]. A trial involving CJC-1295 for lipodystrophy was halted after a participant death, though investigators suspected unrelated cardiac disease [37].

Like Ipamorelin and other peptide secretagogues, CJC-1295 is not FDA-approved for performance enhancement and is banned under WADA regulations in competitive sports [38].

Ipamorelin

Mechanism of action

Ipamorelin is a selective ghrelin receptor (GHSR-1a) agonist that stimulates GH release with minimal impact on cortisol, prolactin, or other pituitary hormones. Its selectivity distinguishes it from earlier GH secretagogues (e.g., GHRP-2, GHRP-6) known for broader endocrine effects [39].

Dosing protocols

Common usage involves 200–300 µg total per day, divided into 2–4 subcutaneous injections, frequently timed around training or sleep to align with natural GH pulses [40]. Coupling Ipamorelin with CJC-1295

(especially the non-DAC variant) is a popular stacking strategy aimed at producing synergistic GH release [39] (chart 2).

Benefits and regulation

Ipamorelin-supported treatment may offer improved fat metabolism, anabolic effects, and enhanced recovery, deriving largely from animal studies and small human trials. However, robust clinical evidence remains limited.

Ipamorelin shares the regulatory status of CJC-1295—it is unapproved for clinical use and prohibited in competitive athletics [38]. Long-term safety in humans is not well-established.

Peptide	Mechanism	Dosing (Typical)	Key Benefits	Safety / Regulation
CJC-1295 with DAC	Long-acting GHRH analog	~30–60 µg/kg weekly (~2–5 mg)	Sustained GH & IGF-1 elevation	Mild ADRs; unapproved, banned in sport
CJC-1295 no DAC	Short-acting GHRH analog	100–200 µg, 2–3× daily	Pulsatile GH mimicry	Similar to DAC version in regulation
Ipamorelin	Selective GHSR agonist	200–300 µg total, split daily	GH release with minimal side hormones	Unapproved, banned; safety not established

Chart 2: Summary for CJC-1295.

CJC-1295 (DAC and non-DAC forms) and Ipamorelin are potent GH secretagogues with compelling mechanistic and preliminary data supporting their roles in body composition modulation. However, despite their pharmacologic promise, clinical validation remains scarce, and safety, regulatory, and ethical constraints cannot be overlooked. High-quality controlled trials are necessary before these peptides can be considered for therapeutic or performance-related applications.

Apelin

Mechanisms of action

Apelin expression diminishes in skeletal muscle with aging across both rodents and humans. Experimental supplementation of aged mice with apelin restored muscle mass and strength, indicating its crucial role in muscle homeostasis and functions in age-related decline of the muscle function [41]. Apelin enhances mitochondrial biogenesis via activation of AMPK, which upregulates PGC-1α, NRF1, and TFAM, thereby improving oxidative capacity in insulin-resistant skeletal muscle [42,43].

In animal model (aged mice), apelin stimulates autophagy in myofibers, reduces inflammation, and supports muscle repair. It also promotes satellite (muscle stem) cell activation and differentiation, facilitating regeneration [41,43].

Apelin overexpression in aged mesenchymal stem cells (MSCs) upregulated autophagy via AMPK activation, reversed cellular senescence, and enhanced survival and angiogenesis post-myocardial infarction in mice [44].

Expected benefits

In aged murine models, exogenous apelin administration improved muscle strength, autophagic clearance, mitochondrial quality, and stem cell-mediated regeneration, thereby reversing features of age-related sarcopenia [41]. Apelin treatment in insulin-resistant mice increased fatty acid oxidation, mitochondrial function, and insulin sensitivity in skeletal muscle, thus highlighting its metabolic regulatory role [42].

Despite promising preclinical data, serum apelin levels in older adults did not correlate with muscle mass, strength, or performance, limiting its utility as a clinical biomarker for sarcopenia [45].

Human studies remain scarce and observational; no randomized controlled trials have yet assessed apelin's therapeutic potential in muscle-related aging or other pathologies.

Safety and regulations

The safety profile of Apelin is still unknown in humans. Apelin's safety, tolerability, effective dosing, and pharmacokinetics remain uncharacterized in human subjects. As an experimental molecule, apelin is not approved for human use and lacks presence in clinical or performance use contexts. Its therapeutic translation faces regulatory and ethical hurdles.

Apelin emerges as a compelling exercise-regulated myokine with potential roles in ameliorating age-related muscle decline, improving metabolic health, and enhancing tissue repair. Still, evidence remains restricted to preclinical contexts. Establishing its therapeutic validity demands well-designed human studies, detailed safety profiling, and translational efforts to determine clinical applicability.

MK-677 (Ibutamoren)

Mechanism of action

MK-677 (Ibutamoren) is a potent, orally active non-peptide agonist of the ghrelin receptor (GHSR-1a), acting as a GH secretagogue. It mimics the hunger hormone ghrelin, resulting in sustained enhancement of endogenous GH and IGF-1 secretion over extended periods following oral administration. Importantly, it does so without the need for injections, offering a convenient route for stimulating the GH–IGF-1 axis in both physiological and potentially therapeutic contexts [39].

Expected benefits

In a randomized, placebo-controlled trial involving healthy elderly adults aged 64–81, daily oral MK-677 (up to 25 mg/day) increased 24-hour GH levels by approximately 97% over baseline values, significantly heightening pulsatile GH release without affecting pulse frequency [46]. Correspondingly, IGF-1 levels increased into the range typical of younger adults, with a sustained effect over 4 weeks [46].

In a two-year randomized controlled trial, MK-677 (25 mg/day) preserved or increased fat-free mass (FFM) in older adults. Over twelve months, FFM rose by approximately 1.1 kg (vs. –0.5 kg in the placebo group), along with enhanced intracellular water and body cell mass [47,48]. Bone metabolism markers and bone mineral density also improved, suggesting anabolic bone effects.

In obese male subjects, MK-677 raised IGF-1 by ~40%, promoted FFM gains, and transiently increased basal metabolic rate, although total and visceral fat did not significantly decrease [39,49].

A crossover study in healthy subjects undergoing calorie restriction demonstrated MK-677 reversed diet-induced nitrogen wasting, improving nitrogen balance compared to placebo [50].

Dosing protocols

Clinical trials predominantly employed 25 mg orally once daily over durations ranging from 2 weeks to 2 years [46,49]. In practice, particularly within fitness communities, MK-677 is commonly used at 20–25 mg per day, often in 8–12-week cycles, based on anecdotal discussions [47].

Safety and regulations

One of the most prominent side effects of MK-677 is appetite stimulation. The ghrelin-mimetic effect reliably increases hunger, which may challenge dietary control [47]; while community reports from the bodybuilders user frequently cite extreme hunger and rapid weight gain as side effects.

Secondly, edema and possible water retention. The mild lower-extremity edema and transient fluid retention are common, typically subsiding over time [47]. Users describe substantial bloating and restricted mobility in anecdotal accounts.

Among other side effects it worth mentioning possible insulin resistance and hyperglycemia. Clinical studies report elevated fasting glucose (by ~5 mg/dL) and decreased insulin sensitivity over longer use, raising concerns for metabolic disruption [46,49,51,52]. Users often report pre-diabetic readings after short-term use.

Also, among other effects some studies mention possible modest increase of prolactin, while cortisol remains largely unchanged [46]. Severe adverse events in elderly patients, including heart failure, have been reported, although data are limited [53].

The long-term safety is unclear. Comprehensive toxicology and long-term safety data remain unavailable [54].

MK-677 remains an investigational drug, not approved by the FDA for clinical or athletic use Wikipedia. It is banned by anti-doping authorities due to its GH/IGF-1 elevating potential [38] (Chart 3).

Domain	Key Insights
Mechanism	Oral ghrelin receptor agonist; elevates pulsatile GH and IGF-1 through sustained secretagogue action
Efficacy	Demonstrated increases in GH/IGF-1, preservation of lean mass, improved bone metabolism, and reversal of catabolism in human studies
Dosing	Typically 25 mg/day orally for weeks to years; commonly used in 8–12-week cycles in practice
Adverse Effects	Increased appetite, water retention, insulin resistance, possible joint discomfort and edema; limited long-term safety data
Regulatory Status	Experimental drug; not FDA-approved; prohibited in sports

Chart 3: Summary for MK-677.

MK-677 (Ibutamoren) is a compelling oral GH secretagogue with robust evidence for increasing GH/IGF-1, improving lean mass, and supporting bone metabolism in older adults. However, metabolic side effects, hunger surge, and lack of long-term safety data underscore caution. Its status as an investigational, non-approved compound further limits clinical uptake. Rigorous, long-term controlled trials are essential to establish both efficacy and safety before any therapeutic adoption.

IGF-1 LR3 (Long R3–Insulin-Like Growth Factor 1)

Mechanism of action

IGF-1 LR3 is a genetically engineered analogue of native IGF-1, featuring an arginine substitution at position 3 and an additional 13-amino-acid extension at the N-terminus. These modifications significantly reduce its binding affinity for IGF-binding proteins (IGFBPs) and markedly extend its half-life to approximately 56–72 hours, which is substantially longer than native IGF-1's ~12–15 hours, making it roughly threefold more potent [55].

Upon engaging the IGF-1 receptor, IGF-1 LR3 activates the canonical PI3K–AKT–mTOR signaling cascade, thereby promoting protein synthesis, satellite cell proliferation, glucose uptake, lipolysis, and inhibition of proteolysis [56].

Although direct peer-reviewed human trials are lacking, mechanistic evidence supports the anabolic and regenerative potential of IGF-1 signaling. In satellite muscle stem cells, IGF-1 promotes differentiation and myoblast activity via PI3K–AKT pathway activation, supporting muscle hypertrophy [56].

Expected benefits

Mainly derived from mechanistic insights and user reports, IGF-1 LR3 has been associated with the following effects. Muscle hypertrophy and hyperplasia, by recruiting satellite cells and enhancing protein synthesis [56]. Improved recovery post-exercise and potential benefits in tissue repair [57,58].

Dosing protocols

Although no standardized clinical dosing exists, community-based protocols in bodybuilding and athletic body recomposition are as follows. Typical use: 20–100 µg/day; beginners start with 20–50 µg/day, while advanced users may approach 50–100 µg/day [58]. IGF-1 LR3 is generally administered via subcutaneous injection for 4–6 weeks, followed by a 20–40 day rest period to mitigate receptor desensitization [59]. Commonly administered post-workout or before bedtime to align with anabolic windows.

The reports from bodybuilders user include the number of beneficial outcomes. “I ran 30 → 40 → 50 mcg over six weeks… incredible endurance, stronger, leaner, better pumps”. “100 mcg pre-workout—significant pump and size gains, enhanced quad growth”. “Started users at 40 mcg daily, zero side effects, good for body recomp and fat loss”.

Safety and regulations

The potential adverse effects, though largely anecdotal, may include the following findings. Hypoglycemia and insulin resistance, due to potent insulin-like effects [60]. Water retention, joint/muscle pain, and injection site discomfort. Long-term concerns include organomegaly, carpal tunnel, and theoretical tumor promotion.

No FDA approval exists for human use of IGF-1 LR3. Prohibited by WADA, making it banned in sports. Human usage is confined to unregulated, often illicit, and clinical risks and quality control pose significant concerns [38] (Chart 4).

Domain	Key Insights
Mechanism	Elevated receptor potency, extended half-life; activates PI3K–AKT–mTOR spectrum.
Benefits	Promotes muscle growth, anabolic signaling, glucose uptake, recovery.
Dosing	Community-based: 20–100 µg/day, 4–6 week cycles, with rest periods.
Safety & Risks	Hypoglycemia, water retention, joint pain, long-term unknowns.
Regulation	Unapproved, banned in sports, no clinical framework for human use.

Chart 4. Summary for IGF-1 LR3.

IGF-1 LR3 exemplifies a powerful experimental anabolic agent with a well-defined molecular mechanism and strong preclinical rationale for promoting muscle growth and recovery. However, the absence of human clinical data, coupled with regulatory restrictions, potential metabolic and proliferative risks, and reliance on anecdotal reports, underscores the urgent need for controlled studies to assess safety, efficacy, and therapeutic viability in humans.

Follistatin-344 (FS-344) Myostatin Inhibitor

Mechanisms of action

Follistatin-344 (FS-344) is a potent endogenous inhibitor of both myostatin and activin, key members of the TGF-β superfamily that suppress muscle growth. By neutralizing these negative regulators, FS-344

effectively removes natural constraints on muscle hypertrophy and hyperplasia. Crucially, its action enhances satellite-cell proliferation and muscle protein synthesis [61].

Satellite cells, speaking of which, we would like to emphasize on the crucial role that satellite cells contribute to muscle hypertrophy by fusing with mature muscle fibers to provide new myonuclei and cellular components, which are necessary for the increased protein synthesis and growth of the fiber. Satellite cells are muscle stem cells that, in response to resistance training, proliferate and then fuse with existing muscle fibers. This fusion process adds new myonuclei to the fiber. While hypertrophy can occur without satellite cells to a degree, their activation and fusion are crucial for achieving full and sustained long-term muscle growth in response to resistance exercise. More myonuclei mean a greater capacity for protein synthesis, which supports the increased size and mass of the muscle fiber. The fusion also brings cellular components from the satellite cells into the muscle fiber, further supporting its growth.

Hence the morphological pathways of muscular hypertrophy represent the sequence of events followed by resistance exercise that triggers the activation and proliferation of satellite cells. The growth factors and HGH regulate satellite cell activation, proliferation, and their fusion. The satellite cells are essential for achieving the full adaptive potential of long-term muscle growth. Without satellite cell participation, muscle growth, strength, and other adaptations to exercise are blunted, indicating their requirement for sustained hypertrophic responses (figure. below).

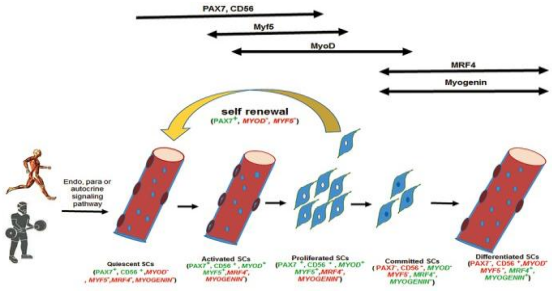

Figure 1: The morphological and functional changes of satellite cells (SCs) in response to exercise. From Bazgir B, Fathi R, Rezazadeh Valojerdi M, Mozdziak P, Asgari A. Satellite Cells Contribution to Exercise Mediated Muscle Hypertrophy and Repair. Cell J. 2017 Winter;18(4):473-484.

HGH regulates the size of the differentiated myotubes and auto renewal of satellite cells in autonomous manner while having no effect on size, proliferation, and differentiation of the myoblast precursor cells. The GH hypertrophic action leads to an increased myonuclear number, indicating that GH facilitates fusion of myoblasts with myotubes [62].

Follistatin (FS), being a myostatin inhibitor, promotes skeletal muscle hypertrophy through multiple interconnected mechanisms. Notably, satellite cell proliferation plays a pivotal role: in irradiated muscle (with impaired proliferative capacity), FS overexpression produced only ~20% muscle weight gain compared to ~37% in non-irradiated controls, indicating that cell proliferation is essential for the full hypertrophic effect [61]. Additionally, FS-induced hypertrophy is mediated via inhibition of both myostatin (Mstn) and activin (Act). Electroporation of an FS mutant with reduced activin affinity (FSI-I)

resulted in only ~14% increase in muscle weight versus ~32% by wild-type FS; notably, FSI-I failed to induce hypertrophy in Mstn-knockout mice, underscoring the contributions of both pathways [61].

In cynomolgus macaques, intramuscular delivery of the human FS344 isoform via AAV1 vector (AAV1-FS344) induced substantial and durable increases in quadriceps muscle size and strength with no detectable histopathological or functional abnormalities in key organs [63].

In rodent models of Muscular Dystrophy (a model of Duchenne muscular dystrophy), a single AAV1-FS344 administration led to long-term increases in muscle size and strength (observed over >2 years) and reduced features of dystrophic pathology such as fibrosis, necrosis, inflammation, and fatty infiltration [64].

A phase I study in patients with sporadic inclusion body myositis (sIBM) evaluated intramuscular injection of rAAV1.CMV.huFS344 at 6×10^{11} vg/kg into the quadriceps. Treated patients demonstrated a mean improvement in 6-minute walk distance of +56 m per year, compared to a –25.8 m decline in untreated controls ($P = 0.01$). Improvements were accompanied by reduced muscle fibrosis and enhanced regenerative markers [65].

The FS-344 enhances muscle regeneration through several mechanisms:

- Satellite-cell activation, which amplifies the hypertrophic response;
- Inhibition of myostatin and activin, thus releasing muscle growth constraints;
- Anti-fibrotic effects, especially in dystrophic or injured muscle contexts [61,64,65].

Dosing protocols

As for the specific dosing in Bodybuilding and Biohacking, the clinical dosing regimens for myostatin inhibitors in healthy individuals have not been established. The following reflects anecdotal community-reported practices only. The existing protocol reported mention daily subcutaneous injections of 100–200 µg for 10–30 days, with claims of rapid lean mass gains up to 4 kg within 10 days. These are unverified and subject to bias and reporting error. Because these practices are anecdotal and derive from unregulated sources, they carry substantial risks related to dosing accuracy, purity, and data reliability.

Safety and regulations

In controlled animal studies, including non-human primates, long-term expression of AAV1-FS344 did not disrupt endocrine parameters such as FSH, LH, testosterone, or estradiol [63,66].

Anecdotal bodybuilding reports include cases of central serous chorioretinopathy (CSCR) following high-dose follistatin use. While such data come from informal sources and lack peer review, they raise concern over potential ocular toxicity.

Follistatin is not FDA-approved for any indication and is prohibited in competitive sports under WADA guidelines. Use from unregulated sources presents significant quality control, purity, and ethical concerns [38] (Chart 5).

Domain	Key Findings
Mechanism	Satellite cell activation; inhibition of myostatin & activin
Nonhuman Primates	Durable ~10–15 % muscle growth, no organ pathology
Rodent Dystrophy	Long-term functional improvement, reduced fibrosis in mdx models
Clinical sIBM Trial	+56 m/year gain vs –25.8 m decline in controls; reduced fibrosis; regeneration
Anecdotal Use	100–200 µg/day SC for 10–30 d (unverified)
Safety Profile	No endocrine disruption; anecdotal CSCR in high-dose users
Regulatory Status	Not FDA-approved; banned in sports; unregulated human use

Chart 5: Summary for Folli statin.

Mitochondria-Derived Peptides: Humanin & SHLPs

Mechanisms & functions

Humanin is the first identified mitochondrial-derived peptide (MDP), encoded within the 16S rRNA region of mitochondrial DNA (MT-RNR2) [67]. It was originally discovered due to its neuroprotective ability in Alzheimer's-related neuronal models [68].

Humanin exerts cytoprotection by direct binding to multiple apoptosis-regulating proteins. It binds Bax, preventing its conformational activation and mitochondrial translocation, thereby inhibiting cytochrome c release and mitochondrial outer membrane permeabilization (MOMP) [69,70]. Additionally, Humanin engages BimEL, a BH3-only Bcl-2 family member, inhibiting its pro-apoptotic activity and downstream mitochondrial apoptosis signaling [71]. It also neutralizes Bid and its truncated form tBid, blocking their induction of Bax/Bak oligomerization and apoptogenic efflux [72].

Humanin has been shown to bind IGFBP-3 in vitro and in vivo, interfering with IGFBP-3–induced apoptosis. It inhibits the interaction between IGFBP-3 and importin-β1, thereby diminishing nuclear translocation of IGFBP-3 and reducing apoptosis in specific cell types [68].

Recent studies demonstrate that Humanin induces autophagy across multiple cell types and organisms. It enhances expression of autophagy-related genes, maintains autophagy flux in aged skeletal muscle, and extends lifespan in C. elegans [73].

Additionally, in pancreatic β-cells, Humanin activates AMPK, leading to upregulation of the mitochondrial biogenesis regulators PGC-1α, NRF1, and TFAM. This results in increased mitochondrial mass, mtDNA content, ATP production, and respiratory function [74].

Remarkably, in one of our previous experimental studies we have investigated anti-diabetic effects of Mitochondrial peptides MitoOrganelles (SBI, Germany) in animal model of auto-immune Type I Diabetes. The study demonstrated the beneficial effects of intramuscular delivery of stem cell derived peptides in delaying the onset of autoimmune diabetes in NOD mice. There was a 33% greater non-diabetic population in the MitoOrganelles-peptide treated mice versus the saline treated mice by the end of week 17 [75].

SHLPs (Small Humanin-Like Peptides) represent a family of six peptides (SHLP1–6) also encoded within the mitochondrial 16S rRNA locus [67]. Although each SHLP displays unique tissue expression patterns, SHLP2 and SHLP3 share certain functional attributes with Humanin [76].

In vitro, SHLP2 and SHLP3 significantly enhance cell viability, reduce apoptosis, and, in the case of SHLP2, promote cell proliferation in β-cell and prostate cancer models [67]. Both peptides also elevate oxygen consumption rate (OCR) and cellular ATP levels, indicating bolstered mitochondrial metabolism and energetics [67]. SHLP3 sand "MitoOrganelles" additionally suppresses reactive oxygen species (ROS) and influences ERK signaling and adipogenesis, while SHLP2 demonstrates protective effects in models of age-related macular degeneration (AMD) through preservation of mitochondrial oxidative phosphorylation complexes, maintenance of mtDNA, and anti-amyloidogenic activity [76].

MitoOrganelles, SHLP2 and SHLP3 have been shown to function as insulin sensitizers. In in vivo hyperinsulinemic-euglycemic studies, administered Mitochondrial peptides enhanced peripheral glucose uptake and suppressed hepatic glucose production [67]. The levels of Mitochondrial peptides decline with age, consistent with their proposed role in metabolism and survival [67].

Expected benefits

Humanin levels decrease with age in humans, with evidence linking its maintenance to longevity. Elevated Humanin is found in centenarian offspring and in long-lived species like the naked mole-rat, while reductions are observed in age-related conditions such as Alzheimer's disease and MELAS [68,77]. Experimental overexpression or analog administration (e.g., HNG) in model organisms improves healthspan-related phenotypes, reduces inflammation, and enhances survival under stress.

SHLPs, particularly SHLP2 and SHLP3, as well as "MitoOrganelles" mirror Humanin's protective effects by reducing apoptosis, improving mitochondrial function, and enhancing insulin responsiveness, all of which are central to the aging process and metabolic homeostasis [67].

Mitochondrial peptides protect neurons from amyloid-β toxicity and familial Alzheimer's mutant genes through anti-apoptotic mechanisms, positioning it as a promising candidate for neurodegenerative disease research [68]. In β-cells, Humanin supports mitochondrial health and energy generation, suggesting roles in diabetes prevention or management [74]. SHLP2 and SHLP3 exert favorable metabolic effects, including enhancement of insulin sensitivity and mitochondrial performance, which could translate to therapeutic angles in metabolic disorders [67,76] (Chart 6).

Peptide	Key Mechanisms	Functional Benefits	Research Stage
Humanin	Inhibits Bax, BimEL, Bid; modulates IGFBP-3; induces autophagy; promotes mitochondrial biogenesis	Cytoprotection, neuroprotection, metabolic health, autophagy, lifespan extension	Preclinical (cell/animal)
SHLP2/3	Enhances OCR & ATP, reduces apoptosis; modulates metabolism; improves insulin sensitivity	Cellular viability, mitochondrial metabolism, systemic metabolic regulation	Preclinical (cell/animal)

Chart 6. Summary for MDPs.

The Mitochondrial peptides remain preclinical, with evidence limited to cell culture and animal models. Human data are scarce, and no standardized dosing protocols or clinical trial data exist. Distribution via bodybuilding or self-experimentation protocols has not been documented in the peer-reviewed literature and therefore remains speculative and unverified. The long-term effects, safety, pharmacokinetics, and dosing in humans are currently unknown. Rigorous research is necessary to explore translational and therapeutic potential.

Glucagon-like peptide-1 receptor agonists (GLP-1 RAs)

This review won't be complete without mentioning the Glucagon-like peptide-1 receptor agonists (GLP-1 RAs). This group of peptides includes liraglutide, semaglutide, tirzepatide, and newer peptides are the incretin-based pharmacotherapies initially designed for type 2 diabetes but now widely employed for obesity and weight loss. These therapies dramatically influence body composition, notably reducing fat mass. However, they often also result in lean mass reduction, including skeletal muscle, raising concerns regarding sarcopenia, especially in susceptible populations [78,79].

Mechanisms and functions

GLP-1 RAs are potent agents, achieving substantial weight reduction. Clinical trials involving liraglutide, semaglutide, tirzepatide, and retatrutide report weight loss in the range of 15–24 % of baseline body weigh [79]. A meta-analysis of 22 randomized controlled trials (2,258 subjects) showed mean reductions of 3.55 kg total body weight, 2.95 kg fat mass, and 0.86 kg lean mass, meaning about 25 % of weight lost was lean mass [78]. A systematic review of semaglutide trials (1,541 adults) noted that while lean mass remained stable in some cases, other larger studies reported lean mass reductions ranging from 0% to 40% of total weight loss. Nevertheless, the relative proportion of lean mass typically increased [80]. Narrative reviews and studies comparing GLP-1RA-induced weight loss to caloric restriction emphasize a lean mass loss of 25–40 %, similar to calorie-restricted dieting. Nevertheless, the proportion of lean mass relative to total body weight often improves, indicating favorable body composition shifts [81,82]. A review stressed that loss of lean body mass may reach 20–50 % of total weight lost, resembling losses seen with diet-induced weight loss or bariatric surgery [82].

Expected benefits

Exercise, especially resistance training, plus optimal nutrition is key to preserving muscle mass during GLP-1 therapy. Supervised resistance training (>10 weeks) has demonstrated lean mass gains of ~3 kg and strength increase of ~25 % across both sexes [79]. Dietary strategies that emphasize higher protein intake (e.g., whey, branched-chain amino acids, high-protein diet) help preserve muscle during weight loss [83]. Emerging adjuncts like BCAAs, omega-3 fatty acids, vitamin D, and creatine have shown promise in mitigating muscle loss when used alongside resistance training and GLP-1 therapy [83, 84]. Combining resistance training with protein supplementation has yielded notable lean body mass improvements in older adults [84]. Resistance training stimulates muscle protein synthesis and strength, counteracting lean mass loss linked with appetite suppression and caloric deficits. GLP-1 therapy may induce fatigue and reduce energy, potentially curbing an individual's ability to train effectively; tailored training plans are vital.

Although direct studies combining Testosterone Replacement Therapy (TRT) with GLP-1 RAs are limited, TRT's anabolic properties offer theoretical muscle-preserving benefits. Testosterone promotes protein synthesis, muscle growth, and increased strength and bone density—key anabolic effects. In hypogonadal or aging male populations, TRT is well recognized for supporting lean mass and functional capacity. Thus, when low testosterone levels are present, integrating TRT with GLP-1 therapy and resistance training may further support muscle retention or gain. GLP-1 RAs drive fat loss and overall weight reduction but may compromise lean body mass. Resistance training plus adequate protein mitigates muscle loss, with evidence of strength and lean mass gains. TRT (in appropriately screened and treated hypogonadal males) could amplify the muscle-preserving and anabolic response.

GLP-1 peptides are robust pharmacologic tools for fat reduction and weight loss. However, they commonly induce a proportion of lean mass loss, raising sarcopenia concerns. Substantial evidence supports that resistance training combined with adequate protein intake significantly counters muscle loss. In hypogonadal men, TRT adds a potential anabolic boost enhancing muscle preservation. Together, these strategies facilitate not only slimming but a healthier, more functional body composition.

Dosing protocols

In clinical and investigational settings, GLP-1 receptor agonists are administered via subcutaneous, once-weekly injections, with established titration protocols to balance efficacy and tolerability. For instance, semaglutide - a long-acting GLP-1 RA approved for weight management, starts at 0.25 mg weekly, increasing every 4 weeks until the target maintenance dose of 2.4 mg weekly is reached; the escalation may be delayed if gastrointestinal side effects occur [85]. Tirzepatide (a dual GLP-1 and glucose-dependent insulinotropic polypeptide (GIP) agonist) typically initiates at 2.5 mg weekly, advancing every 4 weeks in 2.5 mg increments up to a maximum of 15 mg weekly. Emerging triple-agonists such as retatrutide (targeting GLP-1, GIP, and glucagon receptors) are still under research; however, clinical trials have tested doses ranging from approximately 0.5 mg to 12 mg weekly, with higher doses (e.g., at the upper end) achieving ~24 % weight reduction over 24 weeks.

While standardized protocols for stacking multiple GLP-1-based peptides remain uncommon in regulated medical practice, anecdotal and "biohacker"-oriented regimens have surfaced. For example, one user reported alternating injections, tirzepatide 8 mg and retatrutide 3.6 mg, split across multiple days (e.g., twice weekly for each) to mitigate side effects and improve appetite suppression. These community-derived approaches, although intriguing, lack rigorous clinical validation and should be approached with caution.

When combined with TRT, GLP-1 agonists may yield synergistic body recomposition benefits, i.e. significantly enhanced fat loss and lean-mass gain, especially in hypogonadal males. Real-world biomarker estimates suggest GLP-1 monotherapy typically achieves 8–15 % weight loss over 6–12 months, while TRT alone may lead to 3–8 % increases in lean body mass. When combined, these modalities are estimated to deliver 10–20 % weight loss, with net lean body mass increases of 3–8 %, along with greater improvements in insulin sensitivity, energy levels, and cardiovascular metabolic markers [86].

All GLP-1 agonist dosing regimens (semaglutide, tirzepatide, retatrutide) use stepwise titration, typically increasing dose every 4 weeks to minimize gastrointestinal adverse events. Combination protocols involving multiple GLP-1 or multi-agonist peptides are off-label and largely anecdotal, emerging from user forums (e.g., alternating injections of tirzepatide and retatrutide), and are not formally studied. TRT and GLP-1 combined therapy show promising metabolic and body composition enhancements, as indicated by estimated weight and lean mass changes in hypothetical or observational datasets; however, rigorous clinical trials are needed to substantiate these effects.

Stacking Strategies & Protocols

CJC-1295 + Ipamorelin (GH Stack)

This peptide combination seeks to synergistically enhance HGH output by leveraging two complementary mechanisms: CJC-1295, a long-acting synthetic GHRH analog that sustains elevated GH and IGF-1 levels through its albumin-binding Drug Affinity Complex (DAC), extending its half-life to 6–8 days and sustaining IGF-1 elevations up to 9–11 days after injection. Ipamorelin, a selective ghrelin receptor agonist, stimulates pulsatile GH release without affecting cortisol or prolactin levels (Chart 6). When administered together, these peptides mimic physiological GH release, achieving both baseline and pulsatile GH stimulation, while potentially reducing feedback suppression.

A range of dosing protocols exists, mostly derived from clinical clinics or self-experimentation contexts:

Source	Protocol Highlights
LifewellMD	300 µg each (CJC-1295 + Ipamorelin), 1–2× daily (pre-sleep, post-workout)
All About Peptides	200–300 µg each daily; 1–3×, pre-sleep or pre-workout; 8–12 week cycle, 4–8 weeks off
Lindy Health	Weekly CJC-1295: 1,000–2,000 µg; Ipamorelin: 200–300 µg, 1–3× daily; typical cycle: 12–16 weeks on/4–8 weeks off or 5 days on/2 off each week
Innerbody.com	5 days/week; nighttime administration; cycles of 3 months on / 1 month off

Chart 6: Stacking options for peptides in athletic body re-composition.

Reported benefits include improved body composition, enhanced recovery, fat loss, sleep quality, skin health, and connective tissue support. User-reported experiences offer additional detail: “Best base dose combo is 100 mcg CJC and 200 mcg Ipamorelin … cycle 6 weeks injecting 2× a day … off-cycle 4 weeks.” “I started with 300 mcg CJC w/ 500 mcg IPA nightly… splitting dosage to 150 mcg CJC + 250 mcg IPA AM and PM improved sleep.”

Evening or pre-sleep dosing aligns with natural GH circadian rhythms and may maximize effects. Some users report sleep disruptions initially with night-time administration, which may improve with time or by shifting dosing to morning/afternoon. Strict fasting around injection, typically 30–90 minutes before and after, is commonly described to avoid blunting GH response.

Though clinical safety data remain limited, potential adverse effects include injection site reactions, water retention, headaches, tingling or edema, nausea, and changes in hunger. Immunogenicity and cardiovascular effects (e.g., flushing, hypotension) have been noted as potential risks; use may be contraindicated in active or prior cancer due to elevated GH/IGF-1's mitotic effects. Many peptide preparations are research-grade, not for human use, and are banned in competitive sports by WADA. CJC-1295 (DAC) clinical trials were discontinued due to safety concerns, although causality remains uncertain.

HGH Fragment 176–191 with GH Stack

HGH Fragment 176–191 (also known as AOD 9604) is a C-terminal segment of the human GH molecule, theorized to produce lipolytic effects without raising IGF-1 levels. Initial human trials exhibited fat-loss promising results, though later studies failed to confirm consistent efficacy, and development was discontinued. Some users layer HGH-176–191 with CJC-1295 and/or Ipamorelin aiming to potentiate fat loss while maintaining anabolic and recovery benefits. One report describes a protocol of:

- HGH Fragment: 500 µg/day
- CJC-1295: 100 µg, 2–3× daily
- Ipamorelin: 300 µg nightly

These regimens remain anecdotal and unsupported in peer-reviewed literature (Chart 7).

Experimental peptide combinations

Emerging combinations reflect users 'attempts to amplify benefits but lack verified clinical validation (Chart 8):

- MK-677 + IGF-1 LR3: Combining oral GH secretagogue with direct IGF-1 analog for enhanced anabolic signaling.
- Myostatin Inhibitor (FS-344, "Myopep" LabRMS) + IGF-1 LR3: Mixing myostatin inhibition with IGF-1 driven hypertrophy (discussed earlier in context of FS-344 research).
- MK-677 + GH Stack (CJC-1295/Ipamorelin): Attempting to unify continuous and pulsatile GH elevation strategies.
- Humanin/SHLPs + Other Mitochondrial Peptides: Experimental combinations aimed at supporting cellular resilience and anti-aging in "aging athlete" models and include various subtypes of "MitoOrganelles" (i.e. MO "skeletal muscles" - myocyte cell extracts, MO "placenta", et cetera). These protocols require further analysis, research and optimization.

Stack	Dosages (approx.)	Focus
BPC-157 + TB-500	BPC-157 200–500 µg/day TB-500 2–5 mg/week	Injury recovery
MK-677 + CJC-1295	MK-677 20–30 mg/day CJC-1295 100–200 µg/day	Lean gain
Follistatin-344 + PEG-MGF	Follistatin 100 µg EOD PEG-MGF 200–400 µg post-workout	Hypertrophy
AOD-9604 + CJC-1295	AOD-9604 300 µg/day CJC-1295 100–200 µg/day	Cutting, fat loss

Chart 7: Experimental stacking peptide protocols.

Stack Combination	Mechanistic Aim	Evidence Base
CJC-1295 + Ipamorelin (GH Stack)	Sustained and pulsatile GH release	Anecdotal + limited clinic
GH Stack + HGH 176–191 Fragment	Enhanced fat loss with GH-mediated benefits	Anecdotal only
MK-677 + IGF-1 LR3 or FS-344 + IGF-1 LR3	Combined GH axis and direct anabolic stimulation	Theoretical / preclinical
Humanin/SHLPs + GH peptides	Cellular health and anti-aging support	Experimental

Chart 8: Summary of peptide stack protocols.

All described stacking protocols and dosages are based on anecdotal, clinic-derived, or grey-literature sources. No combination has undergone rigorous clinical trials to establish efficacy, safety, dosing standards, or long-term outcomes. As such, any discussion regarding their use must emphasize the investigational nature of the data, potential risks, regulatory issues, and the need for medical supervision and further research.

Peptide	Mechanism	Dose (Typical)	Benefits	Notes & Risks
BPC-157	Angiogenesis, tissue repair	250–500 µg/day	Recovery, tendon healing	Unapproved; potential tumor risk
HGH Frag 176–191	Selective fat oxidation	250–500 µg/day	Fat loss w/o anabolic effects	Limited human data; banned
CJC-1295 (DAC/No DAC)	GHRH agonist, GH ↑ IGF-1 ↑	DAC: 1–2 mg/week; No DAC: 100–600 µg/day	Muscle growth, recovery	Hormonal side effects possible
Ipamorelin	Selective GH secretagogue	200–300 µg/day	Lean mass, recovery	Little safety data; banned
Apelin	Mitochondrial, regenerative	Unknown	Muscle regeneration (aging)	Preclinical only
CJC + Ipamorelin Stack	GH release synergy	Varies by protocol	Muscle, fat loss, recovery	Safety/legal unknown
Frag + GH Stack	Fat loss + GH anabolic overlay	Varied	Enhanced recomposition	Complex regime; safety needs study

Chart 9: Summary for peptides used in bodybuilding and athletic body recomposition.

Conclusions

Peptide therapeutics represent a compelling frontier in the domains of bodybuilding, body recomposition, performance enhancement, and healthy aging, owing to their ability to modulate highly specific

physiological pathways. In the context of athletic populations, peptides such as growth hormone–releasing hormone (GHRH) analogs, ghrelin mimetics, IGF-1 analogs, myostatin inhibitors, and mitochondria-derived peptides offer targeted approaches to enhance muscle anabolism, lipolysis, recovery, and mitochondrial efficiency (see Chart 9).

Mechanistically, these peptides act through well-characterized molecular and endocrine systems, including:

- GH/IGF-1 axis stimulation (e.g., CJC-1295, Ipamorelin, MK-677) to promote hypertrophy and recovery;
- Lipolytic signaling (e.g., AOD9604, GH fragment 176–191) aimed at regional fat reduction;
- Myostatin inhibition (e.g., FS-344, "Myopep/Myoslim" by LabRMS™) to release physiological brakes on muscle growth;
- Mitochondrial support and cytoprotection (Mitochondrial peptides, Humanin, SHLP2/3, "MitoOrganelles" from SBI™) with potential relevance for recovery and aging;
- Angiogenesis and satellite cell activation, contributing to regenerative potential and muscle hypertrophy.

However, despite the scientific plausibility and growing public interest, especially among bodybuilders, biohackers, and longevity enthusiasts, the clinical and regulatory foundations of peptide-based strategies remain immature and fragmented. While numerous peptides have shown favorable effects in preclinical models, including enhanced muscle protein synthesis, reduced fat mass, and improved endurance capacity, the robust, peer-reviewed clinical trials in healthy or athletic human populations are lacking. Most published studies focus on disease contexts (e.g., cachexia, GH deficiency, muscular dystrophies), limiting the generalizability of findings to fitness settings.

The vast majority of peptides marketed for bodybuilding or anti-aging purposes are not approved by the U.S. Food and Drug Administration (FDA) or equivalent regulatory bodies for these indications. Many are classified as research chemicals and are legally prohibited for human consumption outside of investigational settings. Furthermore, quality control and batch consistency from non-regulated suppliers are major concerns, introducing potential risks of contamination, mislabeling, or incorrect dosing.

Peptides are biologically active and may exert hormonal, immunologic, or metabolic effects far beyond their intended targets. Reported adverse events include edema, arthralgia, insulin resistance, visual disturbances, and even central serous chorioretinopathy in users of certain high-dose peptides (e.g., FS-344). Moreover, the long-term oncogenic potential of prolonged GH/IGF-1 elevation remains a critical unknown.

Many peptides fall under prohibited substances as outlined by the World Anti-Doping Agency (WADA). Their use in competitive sports may result in disqualification, sanctions, or reputational harm. Given the ease of online access and difficulty in detection, peptide misuse poses a growing challenge to fair play and athlete safety.

Individuals interested in peptide therapy, whether for physique goals or anti-aging, should prioritize consultation with licensed healthcare providers familiar with endocrinology and sports medicine. Dosing regimens, monitoring protocols, and off-cycle strategies require clinical judgment rather than anecdotal guidance. Moreover, evidence-based alternatives such as resistance training, protein periodization, and lifestyle optimization continue to outperform experimental peptides in safety and efficacy profiles.

While peptides hold significant therapeutic promise, particularly in targeted tissue regeneration, hormonal modulation, and mitochondrial function, their current application in bodybuilding and aesthetic enhancement remains speculative, unregulated, and often driven by anecdotal enthusiasm rather than scientific rigor. Future advances in clinical research, delivery technologies, and regulatory frameworks may clarify their role in human performance optimization. Until then, caution, critical evaluation, and adherence to medical ethics should guide their consideration in both recreational and professional athletic contexts.

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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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