CLINICAL LEARNING GUIDE

HEXARELIN

Growth Hormone Secretagogue | GHS-R1a Agonist

Mechanisms, Evidence, and Clinical Applications

Based on lecture and slide materials by William Seeds, MD — SSRP Institute | Cellular Medicine Education

For educational and research purposes only. Not medical advice. Hexarelin is not FDA-approved for any indication — it is a research compound with no completed human outcomes trials. All clinical use is off-label and investigational. Consult qualified healthcare providers.

SECTION 1 · PROFILE OF THE PEPTIDE

Overview

Hexarelin is a synthetic hexapeptide growth hormone secretagogue, built on the GHRP-6 scaffold. Like the other GHRPs it activates the GHS-R1a (ghrelin) receptor to release GH — in fact about twice as much GH as an equimolar dose of GHRH in humans — but its defining feature is the strongest cardiac/CD36 character of the class. Through the CD36 scavenger receptor it drives a distinct set of GH-independent effects: cardioprotection, antifibrosis, and a notably strong influence on reverse cholesterol transport. It is more chemically stable than ghrelin and is active by multiple routes (IV, subcutaneous, intranasal, and even oral), though subcutaneous is the practical clinical route.

Two cautions travel with that potency. First, hexarelin shows the highest tachyphylaxis (receptor desensitization) of the GHRPs — although the attenuation is partial and reversible after a washout. Second, it raises cortisol and prolactin at higher doses, making it less “clean” than ipamorelin. As with all the secretagogues, it amplifies the body’s own pulsatile GH rather than replacing it, preserving the pituitary feedback that exogenous GH bypasses.

Peptide Profile

Property	Detail
Generic Name	Hexarelin
Classification	Synthetic hexapeptide (6 amino acids) GH secretagogue; ghrelin-receptor agonist; CD36 ligand
Sequence	His-D-2MeTrp-Ala-Trp-D-Phe-Lys-NH_2 (MW ~887 Da); derived from the GHRP-6 scaffold
Receptors	GHS-R1a (ghrelin receptor; EC50 ~1.7 nM) and CD36 scavenger receptor (cardiac)
GH Potency	~2× the GH release of GHRH at equimolar doses (humans)
Routes	IV, subcutaneous, intranasal, oral (SC is the practical clinical route)
Half-Life / GH Peak	GH peak ~15–30 min; effect duration ~60 min
Stability	More chemically stable than ghrelin; resistant to enzymatic degradation
FDA Status	NOT approved for any indication — research compound; no completed human outcomes RCTs

Property	Detail
Anti-Doping	WADA-prohibited at all times (Class S2, GH secretagogues)

Where Hexarelin Sits Among the GH Secretagogues

The GH secretagogues divide into GHRH analogs (sermorelin, Mod GRF 1-29, CJC-1295, tesamorelin), which act on the GHRH receptor, and the GHRPs (GHRP-6, GHRP-2, hexarelin, ipamorelin) plus the non-peptide MK-677, which act on the ghrelin receptor (GHS-R1a). All synthetic GHRPs are modeled on ghrelin, the body's own gut-derived GH-releasing peptide. For raw GH release Dr. Seeds's ordering is GHRP-2 strongest, hexarelin between GHRP-2 and GHRP-6, GHRP-6 strong, and ipamorelin moderate. For CD36/cardiac effects, hexarelin and GHRP-6 are the strong binders — with hexarelin the most pronounced — GHRP-2 milder, and ipamorelin minimal. Because GHRPs and GHRH analogs use separate receptors on the same somatotroph, combining the two is synergistic.

> ⚠ **Hexarelin is not FDA-approved (a research compound) with no completed human outcomes trials, and is WADA-prohibited at all times. All clinical use is off-label and investigational, and competitive (drug-tested) athletes must avoid it entirely.**

SECTION 2 · MODES OF ACTION AND MECHANISMS

Hexarelin works through two receptors with two different jobs. GHS-R1a drives GH release (and somatostatin suppression); CD36 drives a set of GH-independent cardiovascular and metabolic effects. The CD36 arm is more developed for hexarelin than for any other GHRP, and Dr. Seeds treats it — especially its reverse-cholesterol-transport action — as the peptide's most distinctive property.

Receptor Mechanism: GHS-R1a Plus a Strong CD36 Arm

- **GHS-R1a (ghrelin receptor):** a GPCR in the pituitary, hypothalamus, and heart. Through it, hexarelin stimulates GH release from the pituitary, promotes hypothalamic GHRH release, and reduces somatostatin's inhibitory tone — a dual push (release + production) with the brake lifted. In the heart it mediates positive inotropic (stronger contraction) and anti-apoptotic effects.
- **CD36 (scavenger receptor B2):** a scavenger receptor in heart, macrophages, and adipocytes, confirmed as hexarelin's cardiac receptor by photoaffinity labeling. Through CD36, hexarelin drives cardioprotection, antifibrosis, and cholesterol efflux — effects that are entirely GH-independent and absent in CD36-null models.

Signaling Cascades

Pathway	Role
GHS-R1a → Gq/11 → PLC → IP3/DAG → Ca^{2+} + PKC	GH release (exocytosis); cAMP via Gs adds to pituitary GH release
CD36 → Src kinases (Fyn/Lyn) → MAPK/ERK1/2	Cardiac/cytoprotective signaling
CD36 → PPAR-γ → LXR-α → ABCA1/G1	Cholesterol efflux (reverse cholesterol transport) — see below
PI3K/Akt → ↑ Bcl-2 / ↓ Bax	Anti-apoptotic; shifts the survival ratio
↑ MMP-2/9, ↓ TIMP-1 (via GHS-R)	Antifibrotic — breaks down excess collagen/ECM

Pathway	Role
↓ IL-1β, ↑ IL-1Ra; SOD up / MDA down	Anti-inflammatory and antioxidant (I/R and HF models)

The Reverse-Cholesterol-Transport Mechanism (a Distinctive CD36 Effect)

Dr. Seeds gives special weight to one CD36 pathway. CD36 activation of PPAR-γ turns on the liver-X-receptor (LXR-α) — the cell's cholesterol sensor — which up-regulates the ABCA1 and ABCG1 transporters that move cholesterol out of the cell back to the liver as HDL ("reverse cholesterol transport," or cholesterol efflux). Crucially, hexarelin does this through a "differential" PPAR-γ effect: it enhances efflux without up-regulating CD36's own expression — so it does not increase the scavenging of oxidized LDL into macrophages (the step that forms atherosclerotic foam cells). In other words, it pushes cholesterol out without pulling more bad cholesterol in. This is why Dr. Seeds frames hexarelin's CD36 activity in the heart as cardioprotective rather than pro-atherosclerotic.

GH Axis: Pulsatile, With Feedback Preserved

Hexarelin produces pulsatile GH that raises IGF-1 through the liver while preserving pituitary feedback — physiologic pulses rather than the continuous square wave of injected GH, a lower receptor-desensitization risk than exogenous GH, and concurrent cytoprotective signaling. Dr. Seeds notes that even when GH output partially attenuates with chronic use, downstream biological effects can persist.

> **Key mechanistic point: Hexarelin is the most CD36-active GHRP. The GHS-R1a arm amplifies physiologic GH (and lifts the somatostatin brake); the CD36 arm delivers cardioprotection, antifibrosis, and — distinctively — reverse cholesterol transport that boosts efflux without increasing oxidized-LDL uptake.**

SECTION 3 · POINTS OF CLINICAL RELEVANCE

1. **Cardiac action is the signature.** Its dual GHS-R1a + CD36 mechanism gives it the strongest cardiac profile of the GHRPs.

In humans, a single dose produced a positive inotropic effect — LVEF rose from 64.0% to 70.7% in healthy volunteers (onset ~15 min, peak ~30 min), and increased LVEF, cardiac output, and cardiac index in coronary-bypass patients — without raising heart rate. Preclinically it reduced cardiac fibrosis, protected against ischemia/reperfusion injury, and was more effective than ghrelin in cardiac models. The cardiac effects are largely GH-independent (CD36-mediated).

2. **A cholesterol-efflux effect the other GHRPs lack.** It uniquely supports reverse cholesterol transport.

Through CD36 → PPAR-γ → LXR-α → ABCA1/G1, hexarelin enhances cholesterol efflux from macrophages — without up-regulating CD36 oxidized-LDL scavenging — reducing lipotoxicity. Dr. Seeds regards this anti-atherosclerotic mechanism as one of hexarelin's most clinically interesting and under-appreciated properties (preclinical/in vitro to date).

3. **Antifibrosis via MMP up / TIMP down.** It has robust antifibrotic effects — through a distinct mechanism.

Rather than only down-regulating TGF-β1 (the GHRP-6 route), hexarelin up-regulates matrix metalloproteinases (MMP-2/9) and down-regulates TIMP-1, actively breaking down excess

collagen and extracellular matrix. Preclinical models showed reduced cardiac collagen I/III and interstitial fibrosis with improved LVEF.

4. **Desensitization is the central management issue.** Tachyphylaxis is the highest of the GHRPs — but partial and reversible.

Over 16 weeks of SC dosing in elderly subjects, the GH response fell ~45% (significant by weeks 4 and 16) — but recovered fully to baseline after a 4-week washout, and IGF-1/IGFBP-3 were unchanged across 20 weeks. In children on long-term intranasal dosing, GH peaks dropped ~50% yet growth velocity still increased, suggesting biological effects can persist despite GH attenuation. The receptor can resensitize, which is why dosing discipline and cycling matter most for this peptide.

5. **Endocrine selectivity is dose-dependent.** Cortisol and prolactin rise with dose — less clean than ipamorelin.

Hexarelin's cortisol and prolactin effects are dose-dependent and can match or exceed GHRP-6 at higher doses. A revealing pattern: a low 0.125 mcg/kg dose combined with GHRH gives a massive GH release with minimal prolactin and no cortisol, whereas 1.0 mcg/kg alone drives significant GH plus prolactin and cortisol. This is the core argument for keeping the dose low.

6. **Low-dose GHRP + GHRH is the efficient strategy.** It synergizes powerfully with GHRH at low doses.

Because the GHRP (GHS-R1a → Ca^{2+}) and GHRH (GHRH-R → cAMP/PKA) pathways converge on GH release, a small hexarelin dose plus GHRH produces a synergistic, massive GH pulse — with minimal cortisol/prolactin. This allows lower doses, a cleaner endocrine profile, and less desensitization than pushing hexarelin alone.

7. **Understand the glucose/insulin interaction.** The transient glucose rise is an expected efficiency effect — not a warning sign.

Like all GH secretagogues, hexarelin transiently antagonizes insulin and increases fat oxidation, which can cause a small, temporary glucose rise on the way to greater mitochondrial efficiency over months. It is expected rather than alarming — but uncontrolled diabetes must be addressed first, ideally with a GLP-1 agent to set the metabolic foundation before a secretagogue is added.

SECTION 4 · GENERAL DOSING INSTRUCTIONS AND DELIVERY OPTIONS

> **Hexarelin is not FDA-approved and has no validated human dosing regimen; use is off-label/investigational. The protocols below derive from research studies and Dr. Seeds's practice frameworks. Subcutaneous is the practical route.**

Published Research Protocols

Context	Dose / Route	Duration
GH release (humans)	1–2 mcg/kg IV; 1.5–3 mcg/kg SC	Single dose
GH release (humans)	20 mcg/kg intranasal; 20–40 mg oral	Single dose
Chronic GH (humans)	1.5 mcg/kg SC twice daily	16 weeks (showed tachyphylaxis)
Cardiac / antifibrotic (rats)	100 mcg/kg/day SC	5 weeks

Context	Dose / Route	Duration
Anti-cachexia (rats)	200 mcg/kg/day SC	3 weeks

Higher doses are needed for the intranasal and oral routes (and oral bioavailability is variable), which is why subcutaneous is preferred in practice.

Dr. Seeds's Dosing Philosophy: Keep It Low to Protect the Receptor

Because hexarelin has the highest desensitization risk of the GHRPs — and because cortisol/prolactin rise with dose — Dr. Seeds's consistent guidance is to keep the per-injection dose low and stay there rather than chase a bigger GH peak:

- 50–100 mcg SC per injection (50 mcg is a reasonable starting and maintenance dose; 100 mcg is the upper end);
- given once, twice, or three times daily (like the other GHRPs), with at least a 3-hour window between doses so the GH response curve can recalibrate;
- for a maximized but clean GH release, 25–50 mcg hexarelin combined with a GHRH analog (Mod GRF 1-29, CJC-1295, tesamorelin, or sermorelin) — the low-dose synergy strategy.

This low-dose approach is Dr. Seeds's answer to the two things that distinguish hexarelin from the cleaner GHRPs: desensitization and the dose-dependent cortisol/prolactin rise.

Cycling, Combinations, Administration

- Cycling: the most reproducible framework is 12 weeks on, 4 weeks off — the 4-week washout matches the interval shown to fully reverse tachyphylaxis. Protecting receptor sensitivity is the central goal.
- Synergy with GHRH analogs: pairing low-dose hexarelin with a GHRH analog is the preferred way to maximize GH while minimizing cortisol/prolactin and desensitization. Dr. Seeds favors a GHRP + GHRH pairing over stacking GHRPs.
- Timing & food: dose fasted — no carbohydrate or fat within ~30 minutes after, and an empty stomach ~1.5–2 hours before (pure protein is fine). Morning and bedtime are the natural fasted windows; evening aligns with the nocturnal GH pulse.
- Reconstitution & storage: reconstitute with bacteriostatic water; refrigerate at 2–8°C; use within ~28 days; protect from light; rotate SC sites (abdomen, thigh, arm).

SECTION 5 · EVIDENCE PROFILE

Human Data — GH Release & Cardiac

- GH release: confirmed dose-dependent across IV, SC, intranasal, and oral routes; ~2× GHRH at equimolar IV doses; GH peak ~15–30 min.
- Inotropic (7 healthy males): LVEF 64.0% → 70.7% ($p<0.03$), onset ~15 min, peak ~30 min.
- Coronary artery disease (24 bypass patients): increased LVEF, cardiac output, and cardiac index; reduced wedge pressure. LVEF also improved in GH-deficient and ischemic cardiomyopathy patients (no benefit in dilated cardiomyopathy).
- Tachyphylaxis (12 elderly, 16 weeks SC): GH AUC fell ~45% (significant at weeks 4 and 16), fully reversible after a 4-week washout; IGF-1/IGFBP-3 unchanged over 20 weeks.

- Children (intranasal, 6–10 months): GH peak dropped ~50% yet growth velocity rose from 5.3 to 7.4 cm/yr ($p<0.005$) — biological effects preserved despite GH attenuation.

Preclinical — Cardiac, Metabolic, Neuro, Muscle

Domain	Key Preclinical Findings (animal / in vitro)
Cardiac fibrosis (rats, SHR)	Reduced collagen I/III and hydroxyproline; MMP-2/9 up, TIMP-1 down; reduced LV hypertrophy and BP
I/R protection (rats)	Improved systolic function, reduced MDA, more surviving cardiomyocytes (IL-1 pathway)
Lipid metabolism (mice)	Reduced visceral fat, improved insulin sensitivity (IGF-1 unchanged), ↓ hepatic triglyceride/lipogenesis
Cholesterol (in vitro/rats)	CD36 → PPAR-γ/LXR-α → ABCA1/G1 efflux; ↓ plasma cholesterol in obese Zucker rats
Neuroprotection (mice/in vitro)	↑ hippocampal neurogenesis after irradiation; protected neurons from oxidative damage (↑ Bcl-2, p-Akt)
Skeletal muscle (rats)	Protected mitochondrial homeostasis in cisplatin cachexia; restored PGC-1α, NRF-1, TFAM, mtDNA

On Preclinical Evidence

Most of hexarelin's cardiac, metabolic, neuro, and muscle evidence is preclinical (rats, mice, in vitro). Dr. Seeds's framing: the molecular pathways tend to cross species, so well-characterized mechanisms are informative — and, unusually, hexarelin has some human inotropic data that corroborates the preclinical cardiac signal. Still, these remain mechanistic rationale, not outcome proof, and controlled human trials are needed.

Hexarelin vs Ghrelin

Parameter	Hexarelin	Ghrelin
Type	Synthetic hexapeptide	Endogenous 28-aa peptide
GHS-R1a EC50	~1.7 nM	~1.0 nM
CD36 binding	Yes — identified cardiac receptor	Minimal
Chemical stability	High	Low (rapid degradation)
Oral activity	Yes (20–40 mg)	No
Cardiac potency	Superior (preclinical)	Moderate
Appetite	Orexigenic (less than ghrelin)	Strong orexigenic

Critical Evidence Gaps

- No completed human cardiac outcomes RCT — the single most critical gap.
- No human antifibrotic, anti-atherosclerotic, body-composition, or neuroprotection outcome data — all preclinical.
- No long-term (>16 week) human safety data; cancer-surveillance data absent (theoretical GH/IGF-1 concern).

- Tachyphylaxis-mitigation strategies and optimal cardiac-vs-endocrine dosing not formally established.

SECTION 6 · CLINICAL CONSIDERATIONS

Contraindications

- Active or prior malignancy — theoretical GH/IGF-1 concern.
- Uncontrolled diabetes mellitus — stabilize metabolism first (GLP-1 agents are the preferred bridge) before adding a secretagogue.
- Active coronary artery disease — CD36-mediated coronary perfusion-pressure increase warrants caution.
- Hypersensitivity to GHRP-class peptides.
- Pregnancy and lactation (no safety data).
- WADA-tested athletes — prohibited at all times.

Endocrine / Adverse Profile (Dose-Dependent)

Dose (mcg/kg)	GH	Prolactin	Cortisol
0.125 (+ GHRH)	Massive	Minimal	None
0.25	Significant	Moderate	Minimal
0.5	Large	Significant	Moderate
1.0	Very large	Significant	Significant

Other effects: partial, reversible GH tachyphylaxis with chronic use (the dominant concern); occasional water retention (may present as nighttime wrist pain / carpal tunnel); transient glucose elevation; mild injection-site reactions. Long-term human safety data is absent.

Drug Interactions & Cautions

- Glucocorticoids may blunt the GH response.
- Insulin enhances the GH response (and GH transiently opposes insulin).
- Carbohydrate and fat near dosing blunt GH release — dose fasted.
- Synergy with GHRH analogs is intended; Dr. Seeds advises against stacking two GHRPs.

Patient Selection

Potential candidates: age-related GH decline (somatopause); sarcopenia/muscle-wasting; post-surgical recovery; cachexia; and (research) cardioprotective protocols, where hexarelin's cardiac/CD36 profile is most distinctive. Given the desensitization and cortisol/prolactin considerations, it is best reserved for situations where its cardiac and reverse-cholesterol-transport properties are the goal, used at low doses.

Pre-treatment workup: baseline GH, IGF-1, fasting glucose/insulin, HbA1c, AM cortisol, prolactin, lipid panel, liver function, and body composition (DEXA/InBody). For chronic cardiac use, baseline echocardiography. Document informed consent for off-label/research use.

Monitoring Framework

Timepoint	Assessment
Baseline	GH, IGF-1, fasting glucose/insulin, HbA1c, AM cortisol, prolactin, lipid panel, LFTs, body composition; echo if cardiac use
During treatment	IGF-1 (trend), fasting glucose; cortisol/prolactin periodically; watch for GH attenuation (tachyphylaxis)
Chronic / annual	Cardiac function (echo) if used for cardioprotective indication; lipid panel; reassess risk–benefit

All monitoring is mechanistically derived — no clinical guidelines exist. As with the other secretagogues, treat a single IGF-1 value as a trend, not a verdict.

SECTION 7 · A FINAL NOTE

Hexarelin is the most cardiac of the growth hormone-releasing peptides. Built on the GHRP-6 scaffold, it releases roughly twice the GH of GHRH — but its defining character is the strongest CD36 arm of the class, which underwrites genuinely distinctive effects: positive inotropy (confirmed in humans), antifibrosis through MMP/TIMP remodeling, and a reverse-cholesterol-transport mechanism that enhances efflux without increasing oxidized-LDL uptake. That last property, Dr. Seeds argues, makes its cardiac CD36 activity protective rather than pro-atherosclerotic, and is one of the most clinically intriguing — if still preclinical — reasons to study it.

The trade-offs are equally distinctive. Hexarelin has the highest tachyphylaxis of the GHRPs (though partial and fully reversible after a 4-week washout), and its cortisol and prolactin effects climb with dose, making it less clean than ipamorelin. The honest framing is that much of its non-GH promise is preclinical, that there is no completed human cardiac outcomes trial, and that long-term safety is unknown — even as the human inotropic data and well-characterized molecular pathways give real reason to take the cardiac signal seriously.

The practical disciplines follow directly: keep the dose low (50–100 mcg, or 25–50 mcg paired with a GHRH analog), use the low-dose GHRP + GHRH synergy to get a clean, strong pulse, respect the 3-hour inter-dose window, and cycle 12 weeks on / 4 weeks off to protect and restore receptor sensitivity. As with the rest of the class, treat the transient glucose rise as an expected efficiency effect — provided uncontrolled diabetes is addressed first, often with a GLP-1 agent.

> **Bottom line: Hexarelin is the most CD36-active, most cardiac GHRP — a dual GHS-R1a + CD36 hexapeptide that amplifies physiologic GH (~2× GHRH) while adding human-confirmed inotropy, antifibrosis, and a reverse-cholesterol-transport mechanism that boosts efflux without raising oxidized-LDL uptake. Best used at low doses (50–100 mcg SC, or 25–50 mcg with a GHRH analog), cycled 12 weeks on / 4 off, with a 3-hour inter-dose window. Its distinguishing cautions are the highest (but reversible) tachyphylaxis of the class and dose-dependent cortisol/prolactin. A research compound; WADA-prohibited; no human outcomes RCTs; investigational/off-label.**

Selected References

1. Imbimbo BP et al. GH-releasing activity of hexarelin in humans: a dose-response study. Eur J Clin Pharmacol. 1994;46(5):421–425. [Clinical Trial]

2. Bodart V et al. CD36 mediates the cardiovascular action of growth hormone-releasing peptides in the heart. Circ Res. 2002;90(8):844–849. [Animal Study]
3. Ghigo E et al. GH-releasing activity of hexarelin after IV, SC, intranasal, and oral administration in man. J Clin Endocrinol Metab. 1994;78(3):693–698. [Clinical Trial]
4. Mao Y et al. The cardiovascular action of hexarelin. J Geriatr Cardiol. 2014;11(3):253–258. [Review]
5. Rahim A et al. Growth hormone status during long-term hexarelin therapy. J Clin Endocrinol Metab. 1998;83(5):1644–1649. [Clinical Trial]
6. Massoud AF et al. Hexarelin-induced GH, cortisol, and prolactin release: a dose-response study. J Clin Endocrinol Metab. 1996;81(12):4338–4341. [Clinical Trial]
7. Li Y et al. The growth hormone secretagogue receptor: its intracellular signaling and regulation. Int J Mol Sci. 2014;15(3):4837–4855. [Review]
8. Avallone R et al. A GHRP that binds scavenger receptor CD36 and ghrelin receptor up-regulates sterol transporters and cholesterol efflux. Mol Endocrinol. 2006;20(12):3165–3178. [In Vitro]
9. Conte E et al. Hexarelin exerts neuroprotective and antioxidant effects in Neuro-2A cells. bioRxiv. 2020. [In Vitro — Preprint]
10. Xu X et al. Chronic hexarelin attenuates cardiac fibrosis in the spontaneously hypertensive rat. Am J Physiol Heart Circ Physiol. 2012;303(6):H703–H711. [Animal Study]
11. Huang J et al. Hexarelin protects rat cardiomyocytes from ischemia/reperfusion injury through IL-1 signaling. Int Heart J. 2017;58(2):257–263. [Animal Study]
12. Wang Y et al. Modulation of PTEN by hexarelin attenuates CAL-induced heart failure in rats. Turk J Med Sci. 2020;50(1):227–236. [Animal Study]
13. Loche S et al. Desensitization from long-term intranasal hexarelin does not interfere with biological effects in short children. Eur J Endocrinol. 1996;134(6):716–719. [Clinical Trial]
14. Bisi G et al. Acute cardiovascular and hormonal effects of GH and hexarelin in humans. J Endocrinol Invest. 1999;22(4):266–272. [Clinical Trial]
15. Broglio F et al. Effects of acute hexarelin in patients with coronary artery disease during bypass surgery. [Clinical Trial] (cited in Mao 2014).
16. Mao Y et al. One dose of oral hexarelin protects chronic cardiac function after myocardial infarction. Peptides. 2014;56:156–162. [Animal Study]
17. Huang Z et al. Stimulation of endogenous pulsatile GH secretion by GHSR activation reduces fat accumulation in obese mice. FASEB J. 2021;35(1):e21269. [Animal Study]
18. Mosa R et al. Hexarelin improves lipid metabolic aberrations in nonobese insulin-resistant male MKR mice. Endocrinology. 2017;158(10):3174–3187. [Animal Study]
19. Rodrigue-Way A et al. Scavenger receptor CD36 mediates inhibition of cholesterol synthesis via PPAR-γ/PGC-1α and Insig1/2. FASEB J. 2014;28(4):1910–1923. [In Vitro]
20. Barlind A et al. Hexarelin increases cell proliferation in neurogenic regions of mouse hippocampus. Growth Horm IGF Res. 2010;20(1):49–54. [Animal Study]
21. Conte E et al. Hexarelin neuroprotective effects via MAPK and PI3K/Akt pathways. bioRxiv. 2020. [In Vitro — Preprint]

22. Liantonio A et al. Growth hormone secretagogues modulate electrical and contractile properties of rat skeletal muscle. Br J Pharmacol. 2003;139(3):575–584. [Animal Study]

23. Fonzino I et al. GHS hexarelin and JMV2894 protect skeletal muscle from mitochondrial damage in cisplatin-induced cachexia. Sci Rep. 2017;7(1):13364. [Animal Study]

For educational and research purposes only. Not medical advice. Hexarelin is not FDA-approved (a research compound) and is WADA-prohibited, with no completed human outcomes trials. Based on lecture materials by William Seeds, MD — SSRP Institute | Cellular Medicine Education.